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Risk Factors for Stress Fractures

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Abstract

Preventing stress fractures requires knowledge of the risk factors that predispose to this injury. The aetiology of stress fractures is multifactorial, but methodological limitations and expediency often lead to research study designs that evaluate individual risk factors. Intrinsic risk factors include mechanical factors such as bone density, skeletal alignment and body size and composition, physiological factors such as bone turnover rate, flexibility, and muscular strength and

endurance, as well as hormonal and nutritional factors. Extrinsic risk factors include mechanical factors such as surface, footwear and external loading as well as physical training parameters. Psychological traits may also play a role in increasing stress fracture risk. Equally important to these types of analyses of individual risk factors is the integration of information to produce a composite picture of risk.

The purpose of this paper is to critically appraise the existing literature by evaluating study design and quality, in order to provide a current synopsis of the known scientific information related to stress fracture risk factors. The literature is not fully complete with well conducted studies on this topic, but a great deal of information has accumulated over the past 20 years. Although stress fractures result from repeated loading, the exact contribution of training factors (volume, intensity, surface) has not been clearly established. From what we do know, menstrual disturbances, caloric restriction, lower bone density, muscle weakness and leg length differences are risk factors for stress fracture. Other time-honoured risk factors such as lower extremity alignment have not been shown to be causative even though anecdotal evidence indicates they are likely to play an important role in stress fracture pathogenesis.

A stress fracture is a partial or complete fracture of a bone resulting from its inability to withstand stress applied in a rhythmic, repeated, subthreshold manner.[1] It is a common injury in physically active individuals, particularly track and field athletes, dancers and military recruits. Prevention of stress fractures is a major goal of sports medicine practitioners. In order to prevent injury there must be a clear understanding of the causative factors and the mechanisms by which they interact. With this knowledge, preventive measures can be evaluated (fig. 1). In this paper, we review the role of risk factors in the pathogenesis of stress fractures.

Risk factors for any injury may be classified as extrinsic (external) or intrinsic (internal). Extrinsic factors are characteristics of the environment in which the athlete participates, whereas intrinsic factors are characteristics of the athletes themselves. Injuries occur as a result of the summation of various extrinsic and intrinsic factors at a given point in time.[2] Potential extrinsic risk factors for stress fractures include training methods and equipment, while intrinsic factors can be muscular, mechanical, hormonal or nutritional (table I). In addition to the independent effects of a given risk factor and its mechanism of contribution to bone injury, interactions between risk factors are possible (table II).[3] Study of risk factors is also made difficult due to the fact that the relationship between various risk factors and stress fractures may not be linear but in fact curvilinear.

1. Pathogenesis of Stress Fractures

1.1 Factors Influencing Bone Loading

Bone is loaded during physical activity and this results in internal forces (stress) and deformation (strain). The complexity of the loading patterns has been demonstrated in studies measuring strains on the anteromedial surface of the human adult tibia during walking, running and vigorous activity.[4-7] The development of strain on bone during walking and running gait consists of a series of discrete events whereby bone is deformed from a particular direction, released, then loaded from another direction. Stress values calculated from their measurements showed that compressive stresses predominated at heel strike, followed by high tensile stresses at push-off.[8]

A number of factors influence bone loading, as well as the response of bone to loading, and hence its ability to withstand fracture. These include material density, geometry and quality, the activity of muscles and the loading parameters.

Experiments using bone specimens of standard

size have shown that bones with higher density are stronger.[9] Carter and Hayes[10] found that the compressive strength of all skeletal tissue was approximately proportional to the square of the apparent density. This suggests that small reductions in bone density may be associated with large reductions in bone strength. In addition to bone density, bone quality, referring to factors such as the number, spacing and connectivity of trabeculae, plays a role in the ability of bone to withstand fracture.

The geometry of a bone greatly influences its strength. For tension and compression loads, the strength of a bone is proportional to the bone cross-sectional area. Therefore, a larger bone is more resistant to fracture as it distributes the internal forces over a larger surface area, resulting in lower stresses.[11] With respect to bending loads, both the cross-sectional area and the distribution of bone tissue around a neutral axis are important geometrical features. The area moment of inertia is the index that takes into account these 2 factors in bending. A larger area moment of inertia means that the bone tissue is distributed further away from the neutral axis (the axis where the stresses and strains are 0) and is more efficient in resisting bending. The length of a bone also influences its strength in bending. The longer the bone, the greater the magnitude of the bending moment caused by the application of a force. For this reason, the long bones of the lower extremity are subjected to high bending moments and hence high tensile and compressive stresses.[12]

The strength of a bone also depends on the orientation of the bone microstructure with respect to the direction of loading, a characteristic known as anisotropy. In both transverse and longitudinal directions, human cortical bone withstands greater stress in compression than in tension, and greater stress in tension than in shear. With bending, bone is subjected to a combination of tensile loads on one side and compressive loads on the other. Failure begins on the tensile side, since adult bone is weaker in tension than in compression.[13] The rate at which bone is loaded will influence bone strength. Bone tissue which is loaded more rapidly absorbs considerably more energy than that which is loaded more slowly. This characteristic is known as viscoelasticity.

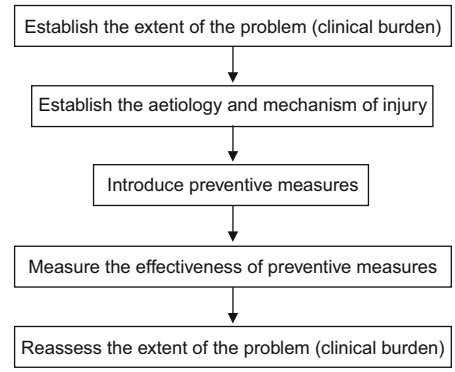

Fig. 1. Prevention of sports injuries. The sequence involved in the prevention of sports injuries includes first establishing the extent of the clinical burden (morbidity). This is followed by establishing the pathophysiology and aetiology of the injury and includes the identification of intrinsic and extrinsic risk factors and their relative importance. Next, preventive measures are evaluated for their effectiveness in reducing the risk of injury. Finally, the effect of the measures must be evaluated by again assessing the clinical burden (nature and extent of the problem).

When bone is loaded *in vivo*, the contraction of muscles attached to bone also influences the stress magnitude and distribution. Nordsletten and colleagues[14,15] found that intact soft tissues as well as muscle contraction substantially increased the structural capacity of the rat tibia. Muscles may serve to attenuate the loads applied to bone, as muscle fatigue has been shown to be associated with increased bone strain.[16]

1.2 Response of Bone to Loading

Repetitive strains are essential for the maintenance of normal bone strength, and physical activity can lead to increased bone mass as bone adapts to the additional loads placed upon it.[17,18] However, bone can also lose strength as a result of repetitive loads imposed during normal daily activity. This loss of strength is attributed to the formation and propagation of microscopic cracks within the bone. The existence of microdamage has been demonstrated following repetitive loading *in vivo*.[19-24] However, there appears to be a threshold

Table I. Potential risk factors for stress fracture

Intrinsic mechanical factors
Bone mineral density
Bone geometry
Skeletal alignment
Body size and composition
Physiological factors
Bone turnover
Muscle flexibility and joint range of motion
Muscular strength and endurance
Nutritional factors
Calcium intake
Caloric intake/eating disorders
Nutrient deficiencies
Hormonal factors
Sex hormones
Menarcheal age
Other hormones
Physical training
Physical fitness
Volume of training
Pace of training
Intensity of training
Recovery periods
Extrinsic mechanical factors
Surface
Footwear/insoles/orthotics
External loading
Others
Genetic predisposition
Psychological traits

level of strain needed for the accumulation of microdamage. Based on fatigue, clinical and pathological studies, Frost[25] suggested that this threshold is approximately 2000 microstrain, which represents the upper range of physiological values. If the load is continually applied, these 'microcracks' can spread and coalesce into 'macrocracks'. If repair does not occur, a stress fracture may eventually result.

Unlike structural material, bone has the capacity to remodel and to repair fatigue-damaged regions. Remodelling is a continuous process involving sequential breakdown and repair of microscopic cavities in bone. In this process lamellar bone is resorbed by osteoclasts, creating resorption cavities which are subsequently replaced with more dense bone by osteoblasts. Thus, remodelling allows bone to adapt to the mechanical loads imposed upon it and become stronger. However, since there is a lag between the increased osteoclastic activity and osteoblastic activity, bone is weakened during this time.[26,27] Microdamage may also occur at pre-existing sites of accelerated remodelling when the bone is in this weakened state.[28-31]

The processes of microdamage accumulation and bone remodelling, both resulting from bone strain, therefore play an important part in the development of a stress fracture. If microdamage accumulates, repetitive loading continues and remodelling cannot maintain the integrity of the bone, a stress fracture may result.[20,22,23] This may occur because the microdamage is too extensive to be repaired by normal remodelling, because depressed remodelling processes cannot adequately repair normally occurring microdamage, or because of a combination of these factors.[22] It is also possible that some stress fractures are secondary to high magnitude repetitive loads, which result in a loss of structural integrity independent of the remodelling process and prior to initiation of any attempts of repair by remodelling. The contribution of various risk factors to stress fracture pathogenesis is shown in figure 2.

2. Intrinsic Mechanical Risk Factors

2.1 Bone Density

Low bone density could contribute to the development of a stress fracture by reducing bone strength, hence increasing the accumulation of microdamage with repetitive loading.[32] Certainly, in the condition of osteoporosis, the association between fragility fractures and low bone density is well established and, clinically, bone density measurements are used to predict the likelihood of fracture.[33,34] However, unlike the elderly population, most active young people have bone density within the normal range, and in many cases it is well above that of their age-matched less active counterparts.[35] Nevertheless, it is feasible that the level of bone density required by physically active individuals to resist repetitive strains of high magnitude and rate without developing a fatigue fracture may be greater

than that of the less active population who subject their bones to much lower forces. It is usually only during special circumstances, such as endocrine disorders in the female athlete, that bone density is decreased and bone strength lowered.

Results of studies investigating the relationship between bone density and stress fracture risk have been contradictory (table III). This may reflect differences in populations (military *versus* athletic), type of sport (running, dancing, track and field), measurement techniques (single or dual photon absorptiometry, dual energy x-ray absorptiometry) and bone regions studied.

To date, 7 studies in athletes and 4 in military populations have investigated the relationship between bone density and stress fracture. Most of the athlete studies evaluated women (2 included both men and women, 4 included only women and one included men only), while most of the military studies evaluated men (3 out of 4 studies). Prospective cohort designs were employed in 3 studies with cohort numbers ranging from 95 to 610, while the others were cross-sectional designs with stress fracture group numbers ranging from 6 to 49. One problem with the cross-sectional studies was that the stress fracture and non-stress fracture groups were often inadequately matched and differed on other factors thought to influence bone density and stress fracture risk, such as menstrual status, body composition and training levels. Multivariate statistical analyses were not performed to take into account the influence of these confounding factors. It was also unclear how long after the injury the bone density measurements were taken. It is possible that enforced immobilisation or reduced activity levels following stress fracture may have led to a decrease in bone density.

For osteoporotic fractures, bone density measurements of the bone at risk of fracture are generally the best predictor of eventual fracture, although bone density at other sites is also predictive. To best provide evidence for a causal relationship between low bone density and stress fracture, measurements should ideally be taken at bone sites where stress fractures occur. While 7 studies included bone density measurements at lower limb sites, the others measured the lumbar spine, radius and/or proximal femur only. These latter sites may not necessarily reflect the bone status at stress fracture sites.

Table II. Risk factors for stress fractures: possible mechanisms and inter-relationships

Risk factor	Mechanisms and inter-relationships
Low bone density	Decreased bone strength
Small bone size	Decreased bone strength
Skeletal alignment	Elevated bone strain, unaccustomed bone strain, muscle fatigue
Body size and composition	Elevated bone strain, menstrual disturbances, muscle fatigue, low bone density
Bone turnover	Low bone density, elevated bone strain, inadequate repair of microdamage
Muscle flexibility and joint range	Elevated bone strain, unaccustomed bone strain, muscle fatigue
Muscle strength and endurance	Elevated bone strain, unaccustomed bone strain
Low calcium intake	Greater rate of bone turnover, low bone density, inadequate repair of microdamage
Nutritional factors	Altered body composition, low bone density, greater rate of bone turnover, reduced calcium absorption, menstrual disturbances, inadequate repair of microdamage
Menstrual disturbances	Low bone density, greater rate of bone remodelling, increased calcium excretion
Training	Elevated bone strain, unaccustomed bone strain, greater number of loading cycles, muscle fatigue, inadequate time for repair of microdamage, menstrual disturbances, altered body composition
Inappropriate surface	Elevated bone strain, unaccustomed bone strain, muscle fatigue
Inappropriate footwear	Elevated bone strain, unaccustomed bone strain, muscle fatigue
Higher external loading	Elevated bone strain, muscle fatigue
Genetic factors	Low bone density, greater rate of bone remodelling, psychological traits
Psychological traits	Excessive training, nutritional intake/eating disorders

Very little prospective evidence exists to support a clear causal relationship between bone density and risk of stress fractures in men. Giladi et al.[38] found no difference in tibial bone density in 91 recruits who developed stress fractures compared with 198 controls. This result held when the

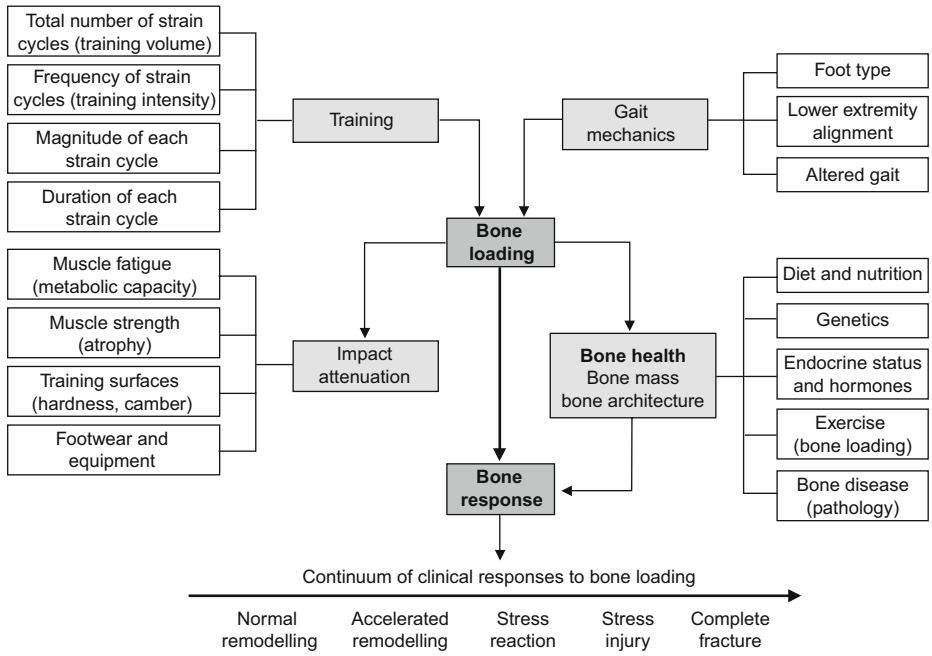

Fig. 2. The contribution of risk factors to stress fracture pathogenesis. Training influences bone loading and is itself affected by 4 factors. The volume of training is a function of the total number of strain cycles received by the bone and the intensity of training (load per unit time – pace, speed) is a function of the frequency of strain cycles applied to the bone. The magnitude of each strain and duration of each strain cycle are a function of bodyweight, muscular shock absorption capability, and lower extremity biomechanical alignment. Impact attenuation is both intrinsic (muscular factors) and extrinsic (equipment and training surfaces). Eccentric muscular strength is important but even more important is the muscle's ability to resist fatigue; to continue to contract effectively for a sustained period of time. This important factor is a function of metabolic adaptations that occur with training. Foot type and lower extremity biomechanical alignment may affect gait mechanics but altered gait may also occur from fatigue, disease, and injury. Finally, bone health is a major factor that determines the response of bone to loading and is affected by diet and nutrition, genetics, endocrine and hormonal status, the amount of regular exercise, and the presence of bone disease.

data were analyzed for all stress fractures combined as well as for femoral and tibial fractures only. Similar findings were reported in a cross-sectional study by Crossley et al.[48] who failed to find a difference in tibial bone density between male runners with and without a history of tibial stress fracture, and by Bennell et al.,[40] who used dual energy x-ray absorptiometry (DXA) to prospectively assess bone density at a number of regions in male track and field athletes. Although Beck et al.[39] found significantly lower tibial and femoral bone density in 23 male recruits who developed stress fractures compared with 587 control individuals, this result may be explained by differences in bodyweight as the stress fracture recruits were 11% lighter. Since bodyweight is a major predictor of bone density, it is important to ensure that the groups are matched on this factor, or that this factor is controlled statistically, otherwise the independent relationship between bone density and stress fractures cannot be determined.

A cross-sectional study[41] did show lower bone density at the femoral neck, Wards triangle and trochanter in 41 recruits with male stress fracture compared with 28 non-stress fracture recruits. None of the other studies in men included these proximal femoral sites, so direct comparisons cannot be made. When the group was subdivided by fracture site,

femoral bone density was still lower in recruits with femoral (n = 12) and calcaneal stress fractures (n = 10), but not in recruits with tibial, fibular or metatarsal fractures (n = 19). This site difference may be due to differences in the proportion of cortical to trabecular bone, and highlights the problem of the specificity of the measurement site.

Conversely, there is some evidence to suggest that lower bone density in women may play a role in stress fracture development. In the only prospective cohort study to date, female track and field athletes who sustained stress fractures had significantly lower total body bone mineral content and lower bone density at the lumbar spine and foot than those without a fracture.[40] The subgroup of women who developed tibial stress fractures had 8.1% lower bone density at the tibia/fibula. This deficit located at the site of fracture supports a possible cause and effect relationship, although the number in this subgroup was small. An important point to note is that although bone density was lower in the athletes with stress fractures, it nevertheless remained, as a group, higher than or similar to bone density of less active nonathletes. This implies that the level of bone density required by physically active individuals for short term bone health may be greater than that required by the general population. It also implies that the stress fracture individuals in this study would not have been identified as being at risk based on normative DXA values. At present there are no normative data bases specific to athletes of different sports to enable legitimate comparisons of an individual's bone density.

Findings of the cross-sectional studies in female athletes are contradictory. Again, this may reflect the small sample sizes as well as differences in the type of sport, the measurement techniques and the bone regions assessed. One study[45] of 14 female runners actually found significantly higher lumbar spine and femoral neck bone density in the stress fracture group. The authors speculated that greater external loading forces measured in the stress fracture athletes during running may have been responsible for their higher bone density. This may instead be a spurious result due to the fact that there were only 6 athletes in the stress fracture group and 8 in the control group. Other studies have reported either no difference or significantly lower bone density in the stress fracture group.[42-44,46,47]

Although the relationship between bone density and stress fracture development is still not clearly established, there is evidence that low bone density as a risk factor may be more common in women. Conflicting results from studies could indicate that the population of athletes with stress fractures is heterogeneous in terms of bone density and that other factors independent of bone mass contribute to the risk of fracture, particularly in men. In general, it would seem that bone densitometry does not have a place as a general screening tool to predict risk of stress fracture in otherwise healthy individuals. However, bone densitometry may be warranted in athletes with multiple stress fracture episodes or in females with menstrual disturbances.

2.2 Bone Geometry

Bone strength is related not only to bone mineral density but also to bone geometry. For bones loaded in tension or compression, the amount of load the bone can withstand prior to failure is proportional to the cross-sectional area of the bone. The larger the area, the stronger and stiffer the bone. For bending and torsional loads, both the cross-sectional area and the cross-sectional moments of inertia will determine bone strength. Bones with a larger cross-sectional area and with bone tissue distributed further away from the neutral axis will be stronger when subjected to load and hence less likely to fracture.[12]

The structural properties of long bones vary with age and gender and are largely dependent on body size.[49] However, even between individuals of similar age and build, there is great variation in bone geometry. In fact, there is much greater variation in structural geometry than in bone material properties, including bone mineral density.[50] Thus, differences in bone geometry might partly explain differences in stress fracture predisposition.

Table III. Summary of studies investigating the relationship between bone density and stress fractures. Studies were ordered according to the strength of their study design and then chronologically (adapted from Brukner and Bennell[36] and Bennell et al.,[37] with permission)

Reference	Study design	Participants	Gender	Sample size	Technique	Sites	Results[a] (% difference)
Giladi et al.[38]	PC	Military	M	91 SF, 198 NSF	SPA	Tibial shaft	–6.0%
Beck et al.[39]	PC	Military	M	23 SF, 587 NSF	DXA	Femur	–3.9%*
						Tibia	–5.6%*
						Fibula	–5.2%
Bennell et al.[40]	PC	Track and field athletes	F	10 SF, 36 NSF	DXA	Upper limb	–3.3%
						Thoracic spine	–6.7%
						Lumbar spine	–11.9%*
						Femur	–2.2%
						Tibia/fibula	–4.2%
						Foot	–6.6%*
			M	10 SF, 39 NSF	DXA	Upper limb	–4.9%
						Thoracic spine	–4.1%
						Lumbar spine	–0.8%
						Femur	–2.9%
						Tibia/fibula	–4.0%
						Foot	–0.3%
Pouilles et al.[41]	XS	Military	M	41 SF, 48 NSF	DPA	Femoral neck	–5.7%*
						Wards triangle	–7.1%*
						Trochanter	–7.4%*
Carbon et al.[42]	XS	Various athletes	F	9 SF, 9 NSF	DPA	Lumbar spine	–4.0%*
						Femoral neck	–7.0%
					SPA	Distal radius	–7.7%
						Ultradistal radius	0.0%
Frusztajer et al.[43]	XS	Ballet dancers	F	10 SF, 10 NSF	DPA	Lumbar spine	–4.1%
						1st metatarsal	0.0%
					SPA	Radial shaft	0.0%
Myburgh et al.[44]	XS	Various athletes	6 M, 19 F	25 SF, 25 NSF	DXA	Lumbar spine	–8.5%*
						Femoral neck	–6.7%*
						Wards triangle	–9.0%*
						Trochanter	–8.6%*
						Intertrochanter	–5.5%
						Proximal femur	–6.5%*
Grimston et al.[45]	XS	Runners	F	6 SF, 8 NSF	DPA	Lumbar spine	8.2%*
						Femoral neck	7.6%*
						Tibial shaft	9.7%
Bennell et al.[46]	XS	Track and field athletes	F	22 SF, 31 NSF	DXA	Lumbar spine	–3.5%
						Lower limb	–0.9%
						Tibia/fibula	–2.0%
Cline et al.[47]	XS	Military	F	49 SF, 78 NSF	DXA	Lumbar spine	–2.4%
						Femoral neck	0%
						Wards triangle	–2.2%
						Trochanter	0%
						Radial shaft	1.5%
Crossley et al.[48]	XS	Athletes	M	23 SF, 23 NSF	DXA	Tibial shaft	8.1%

a Results are given as the % difference comparing stress fracture subjects (SF) with non-stress fracture subjects (NSF).

DPA = dual photon absorptiometry; **DXA** = dual energy x-ray absorptiometry; **F** = females; **M** = males; **PC** = observational analytical prospective cohort; **SPA** = single photon absorptiometry; **XS** = observational descriptive cross-sectional; * statistically significant ($p < 0.05$).

A prospective observational cohort study of 295 male Israeli military recruits[51] found that those who developed stress fractures had narrower tibias in the mediolateral plane at 3 different levels (measured using radiographs) than those without stress fractures. This result was found for total stress fractures as well as for stress fractures in the tibia and in the femur alone. The authors suggested that the

reason the result was seen for femoral stress fractures as well was because the size of the tibia is an indication of the size of tubular bones in general.

Further work by the same research group used these widths to calculate the cross-sectional moment of inertia. Using multivariate statistical techniques, they found that the cross-sectional moment of inertia about the anteroposterior axis (CSMIAP), an estimate of the ability of bone to resist bending, was an even better indicator of the risk of stress fracture than tibial width.[52] Stress fractures developed in 31% of recruits with a low CSMIAP compared with only 14% of recruits with a high CSMIAP.[53] However, it must be pointed out that tibial geometry is complex and changes continuously along the length of the tibia. The researchers derived tibial widths from standard radiographs and based their calculations on the assumption that the tibia is an elliptical ring with an eccentric hole. This assumption may not necessarily be valid. From our experience, tibial cross-sectional shape varies widely between individuals and in many cases is more closely aligned to a triangle rather than an ellipse.

Nevertheless, the results showing differences in tibial geometry between stress fracture and non-stress fracture recruits were supported by a recent prospective study of more than 600 military recruits undergoing 12 weeks of basic training. Bone mineral data acquired from a DXA scanner were used to derive cross-sectional geometric properties of the tibia, fibula and femur.[39] This method is likely to be more accurate than radiographs as it does not entail assumptions of cross-sectional shape and manual measurements of cortical thickness. The results showed that even after adjusting for differences in bodyweight, the stress fracture recruits had smaller tibial width ($p = 0.03$), cross-sectional area ($p = 0.03$), moment of inertia ($p = 0.07$) and modulus ($p = 0.05$) than the non-stress fracture group. In fact, the average reduction in tibial bone geometry of the stress fracture subjects appears to be similar in the 2 studies.

Another interesting finding in the study by Beck and colleagues[39] was that the smaller dimensions in the fracture group were limited to long bone diaphyses and not joint size, suggesting a specificity in the structural deficit in the fracture group. There is evidence that diaphyseal cross-sectional dimensions are more environmentally influenced than joint size. This could indicate that the bones of the stress fracture group had not been loaded sufficiently prior to basic training to develop cortices strong enough to withstand the subsequent stresses.

The presence of smaller and weaker bones in military recruits subjected to intense, unaccustomed physical activity may lead to a higher rate of bone microdamage. Without adequate time for adaptive cortical remodelling to occur, a stress fracture could then result. There is also evidence to suggest that a smaller bone is a risk factor for stress fractures in athletes, even though their bones are loaded more gradually over a longer period of time. In a cross-sectional study of 46 male runners, CT scanning was used to evaluate tibial geometry at the level of the middle and distal third.[48] Those with a history of tibial stress fracture had a significantly smaller tibial cross sectional area (after adjusting for body mass and height) than the non-stress fracture group. Whether this is also a risk factor in female athletes has not yet been investigated.

Even if bone geometry plays a role in stress fracture development, the clinical relevance of this risk factor is limited. Large scale screening of tibial geometry using plain radiographs or DXA techniques is impractical and costly. With further research, it may be possible to develop surrogate indicators of tibial geometry via simple anthropometric measurements.

2.3 Skeletal Alignment

Lower limb and foot alignment may predispose to stress fractures by creating areas of stress concentration in bone or by promoting muscle fatigue. While an association between various factors influencing skeletal alignment and stress fracture has been sought in military populations, there are few data pertaining to athletes. Furthermore, the way in which the factors were defined and measured is

inconsistent, and reliability and validity of the measurements was often not addressed. Studies that have attempted to evaluate the link between skeletal alignment and stress fractures are summarised in table IV.

The structure of the foot will help determine how much force is absorbed by the foot and how much is transferred to bone during ground contact. The high arched (pes cavus) foot is more rigid and less able to absorb shock, resulting in more force passing to the tibia and femur. The low arched (pes planus) foot is more flexible, allowing stress to be absorbed by the musculoskeletal structures of the foot. But it is less stable during weight bearing, which may contribute to muscle fatigue as the muscles have to work harder to control the excessive motion, especially at toe-off. Pes planus is also very often associated with prolonged pronation or hyperpronation, which can induce a great amount of torsion on the tibia. Theoretically, either foot type could predispose to a stress fracture. Several studies have indicated that the risk of stress fracture is greater for male recruits with high foot arches than with low arches.[55,58,64]

In a prospective cohort study, the overall incidence of stress fracture in the low-arched group was 10% as opposed to 40% in the high-arched group.[55] A similar trend was noted when tibial and femoral stress fractures were analysed separately. However, assessment of foot type was based on observation in a non-functional position, and recruits with extreme pes planus were excluded. Furthermore, those trainees with 'average' arches had a stress fracture incidence (31%) similar to the high arched group. Nevertheless, these findings were supported in a cross-sectional study using a contact pressure display method to provide foot-ground pressure patterns and derived stress intensity parameters.[64] In this study, recruits with high arches were more likely to have sustained a stress fracture than those with lower arches. In contrast, not all military studies have reported an association between foot type and stress fracture risk. Montgomery and colleagues[57] used visual inspection of foot posture in standing and found that the incidence of stress fracture was similar in recruits with cavus, neutral or planus feet.

However, a relationship between foot type and stress fracture may vary depending on the site of stress fracture. Using radiographs to assess foot type, femoral and tibial stress fractures were more prevalent in the presence of higher arches whereas the incidence of metatarsal fractures was higher with lower arches.[58] Therefore, studies may fail to find an association between certain foot types and stress fractures because they have not grouped the data by stress fracture site.

Most of the studies in athletes are case series which do not allow comparison of injured and uninjured athletes. While pes planus may be the most common foot type in athletes presenting to sports clinics with stress fractures,[67,68] pes planus may be equally common in athletes who remain uninjured. In a large series of 320 stress fractures, pes planus was more frequent in athletes with tibial and tarsal bone stress fractures and least common in athletes with metatarsal stress fractures.[66] This tends to differ from military findings where foot fractures appear to be more prevalent in recruits with pes planus. In the only prospective cohort study in athletes, foot type assessed visually in weight bearing did not predict the likelihood of stress fracture.[40] However, there were insufficient numbers to evaluate the results according to stress fracture site. Even when assessing a single stress fracture site, foot type measured using contact pressure from a force platform during walking did not appear to play a role. In 29 consecutive patients presenting with unilateral tibial stress fractures, there was no difference in foot type between injured and uninjured legs, and between injured athletes and a reference group of 30 sedentary and athletic individuals.[65]

A leg length discrepancy has been postulated as a potential risk factor for stress fracture due to resulting skeletal realignment and asymmetries in loading, bone torsion and muscle contraction.[69] One study in male recruits failed to show a relationship between leg length differences (measured from computer digitisation of highlighted landmarks from photographs) and likelihood of stress frac-

Table IV. Studies investigating the association between skeletal alignment and stress fractures. Studies were ranked firstly according to the strength of their study design and then chronologically

Reference	Study design	Participants	Sample size	Factors analysed	Method of measurement	Results
Friberg[54]	XS, PC	Army (Finland)	371 M	Leg length difference	X-ray – WB	Increased incidence with increased difference
Giladi et al.[55]	PC	Army (Israel)	295 M	Foot type	Observation – NWB	SF risk greater in high arch than low arch
Giladi et al.[56]	PC	Army (Israel)	295 M	Genu valgum/varum	Observation	No relationship with SF
				Tibial torsion	NS	No relationship with SF
				Gait in toe/out toe	Observation	No relationship with SF
Montgomery et al.[57]	PC	SEAL (USA)	505 M	Genu recurvatum	Distance heels to bed – supine	No relationship with SF
				Genu valgum/varum	Distance between condyles – WB	No relationship with SF
				Q angle	Goniometer – supine	No relationship with SF
				Foot type	Observation – WB	No relationship with SF
Simkin et al.[58]	PC	Army (Israel)	295 M	Foot type	X-ray – WB	High arch – higher risk of femoral and tibial SF; low arch – higher risk of metatarsal SF
Milgrom et al.[59]	PC	Army (Israel)	783 M	Genu valgum/varum	Distance between condyles	No relationship with SF
Bennell et al.[40]	PC	Athletes	53 F, 58 M	Leg length difference	Tape measure – NWB	Leg length difference – higher incidence of SF
				Genu valgum/varum	Observation – NWB	No relationship with SF
				Foot type	Observation – WB	No relationship with SF
Cowan et al.[60]	PC	Army (USA)	294 M	Genu valgum/varum	Computer digitisation of photographs showing highlighted anatomic landmarks – WB	Increased SF risk with increased valgus
				Genu recurvatum		No relationship with SF
				Q angle		Q angle >15° = increased risk for SF
				Leg length difference		No relationship with SF
Winfield et al.[61]	PC	Marines (USA)	101 F	Q angle	NS	No relationship with SF
Hughes[62]	XS	Army (USA)	47 M	Forefoot varus	Goniometer – NWB	Greater FFV 8.3 times at risk of metatarsal SF
				Rearfoot valgus	Goniometer – WB	No relationship with SF
Brunet et al.[63]	XS	Athletes	375 F, 1130 M	Leg length difference	Self-report questionnaire	
				Foot type	Self-report questionnaire	
Brosh and Arcan[64]	XS	NS	42 M	Foot type	Contact pressure display	
Ekenman et al.[65]	XS	Athletes	29 SF M and F; 30 NSF M and F	Foot type	Contact pressure during gait	
				Rearfoot valgus	NS	
				Forefoot varus	NS	
Matheson et al.[66]	CS	Athletes	175 F, 145 M	Subtalar varus	>3°	Not related to site of SF
				Foot type	NS	Pron – tibia and tarsal SF; cavus – metatarsal and femoral SF
				Forefoot varus	>2°	Not related to site of SF
				Genu valgum/varum	Distance between condyles	Not related to site of SF
				Tibial varum	>10°	Not related to site of SF

CS = case series; **F** = female; **FFV** = forefoot varus; **M** = male; **NS** = not stated; **NSF** = non stress fracture; **NWB** = non weight bearing; **PC** = prospective cohort; **SF** = stress fracture; **WB** = weight bearing; **XS** = cross-sectional.

tures.[60] However, the results of all other studies in this area do suggest an association. Using a radiological method to assess leg length in standing, Friberg[54] found that in 130 cases of stress fracture in military recruits, the longer leg was associated with 73% of tibial, metatarsal and femoral fractures while 60% of fibular fractures were found in the shorter leg.

In a prospective analysis following a group of 102 parachutists over 330 days, he observed a positive correlation between the degree of leg length inequality and the incidence of stress fractures. The incidence of stress fractures in those with equal leg lengths was 15.4%. This increased to 24.3% in those with 5 to 9mm difference and to 66.7% in those with 15 to 20mm difference. However, no statistical analyses were performed to assess the significance of these results. Similar findings have been reported in a cross-sectional survey of male and female runners. Using a self-administered questionnaire, those who claimed to have a leg length difference were more likely to have sustained a stress fracture in the past.[63] In a cohort study, 70% of women who developed stress fractures displayed a leg length difference of more than 0.5 cm (measured using a tape measure in supine) compared with 36% of women without stress fractures.[40] Based on these reports it would seem appropriate to correct leg length discrepancies should they exist.

The other alignment features to have been assessed in relation to stress fractures include the presence of genu varum, valgum or recurvatum, an increased Q angle, and tibial torsion. Of these, only the Q angle has been found in association with stress fractures. Using computer digitization of highlighted landmarks from photographs, male recruits with a Q angle of greater than 15° had a relative risk of stress fracture that was 5.4 times that of recruits with an angle less than 15°.[60] Conversely, other studies have failed to show that Q angle is related to stress fracture occurrence.[57,61]

The literature suggests that foot type may play a role in stress fracture development, but the exact relationship may depend upon the anatomical location of the injured region and the activities undertaken by the individual. However, a leg length discrepancy does appear to be a risk factor in both military and civilian populations. The failure to find an association between other biomechanical features and stress fractures in cohort studies does not necessarily rule out their importance for individuals. A thorough biomechanical assessment is an essential part of both treatment and prevention of stress fractures. Until the contribution of biomechanical abnormalities to stress fracture risk is clarified through scientific research, correction of such abnormalities should be attempted, if possible. This is particularly so in individuals who present with recurrent stress fractures.

2.4 Body Size and Composition

Theoretically, body size and soft tissue composition could affect stress fracture risk directly by influencing the forces applied to bones. For example, as bodyweight is positively related to ground reaction force, heavier individuals would generate higher forces during physical activity.[70] This could increase the likelihood of stress fracture. Body size and composition could also have indirect effects on stress fracture risk by influencing bone density or menstrual function. Smaller, leaner athletes are more likely to have lower bone density as bodyweight, lean mass and fat mass are all significant determinants of bone density. Similarly, menstrual disturbances are more prevalent in female athletes who are light and have little body fat.

A number of potential risk factors related to body size and composition have been reported in the stress fracture literature including height, bodyweight, body mass index (BMI), skinfold thickness, total and regional lean mass and fat mass, limb and segment lengths, and body girths and widths. Measurements have generally been obtained by simple anthropometric techniques, although one study used DXA to measure fat and lean mass.[40]

Although several studies have evaluated the role of body habitus variables, none have reported differences in height, weight, body mass index or fat mass of athletes who have sustained a stress fracture compared with those without.[40,71-74] Failure to find an association between these factors and stress fracture incidence may be due to the fact that athletes of a particular sport tend to be relatively homogenous in terms of somatotype and body composition. Also, any relationship that exists may be nonlinear. For example, both lighter and heavier athletes may be at risk but the data has not been

analysed appropriately to detect such a relationship. Furthermore, these parameters are unlikely to be stable and their measurement in cross-sectional studies may not reflect their status prior to injury.

Body size may be a risk factor in military recruits where size variations are likely to be greater than in athletes. In a recent study, the incidence of stress fracture was greater in smaller individuals.[39] The authors surmised that this may be because of common training requirements where similar weight packs and other equipment are carried regardless of recruit bodyweight. It is also possible that the lower BMI of the fracture group was indicative of relatively lower muscle mass and/or poorer physical conditioning prior to training. On the other hand, overweight individuals may be at increased risk for stress fracture as these populations also tend to be less physically active. However, many military studies have failed to find an association between stress fractures and various parameters of body size and composition in both men and women.[38,61,75,47]

In female Marine trainees, a narrow pelvis (≤26 cm) was associated with a greater risk of stress fracture ($p < 0.09$).[61] The incidence of stress fracture in those with a narrow pelvis was 14% compared with 4% in those with a wider pelvis. Thus, the relative risk of stress fracture was 3.57 for recruits with a narrow pelvis compared with 'normals'. An explanation for this finding is not clear, as typically a wider pelvis has been attributed to increased biomechanical stresses by increasing the Q angle. It is possible that a narrow pelvis in this group of female Marines is a marker for some other risk factor for stress fractures.

In general, there are no consistent relationships observed between body size or composition and risk of stress fracture. However, monitoring body fat and lean mass may be useful to indicate health and nutritional status, particularly in female athletes where disordered patterns of eating are relatively common.

3. Physiological Risk Factors

3.1 Bone Turnover

It is apparent that bone remodelling plays a role in stress fracture pathogenesis and that perturbations in bone remodelling, either generalised or focal, may predispose to this injury. Stress fractures develop if microdamage cannot be successfully repaired by the remodelling process and thus accumulate to form symptomatic 'macrocracks' in bone. However, accelerated remodelling, resulting from excessive bone strain or from the influence of systemic factors, may also weaken bone because bone resorption occurs before new bone is formed. This could allow the accumulation of microdamage with repetitive mechanical loading at remodelling sites. Conversely, depressed bone remodelling, in particular bone formation, may not allow normal skeletal repair of naturally occurring microdamage. It is conceivable that either sequence could lead to the development of a stress fracture in individuals training intensely.

Because direct assessment of bone remodelling in humans is invasive and impractical, measurement of biochemical markers of bone turnover may prove useful in a clinical setting to aid identification of individuals most at risk for this injury. A prospective cohort study of 104 male military recruits[76] found that a single measurement of plasma hydroxyproline (a nonspecific indicator of bone resorption), taken in the first week of a training programme, was significantly higher in 5 recruits who subsequently sustained stress fractures than in those who remained uninjured. While this supports the concept that elevated bone turnover may be a stimulus for stress fracture development, hydroxyproline is not specific to bone and thus the elevated levels may well reflect nonskeletal sources.

A limited number of cross-sectional studies have measured biochemical markers of bone turnover in small samples of female athletes with and without a history of stress fracture.[42,44,72] These studies have found no difference in bone turnover levels between groups. However, single measure-

Table V. Studies investigating the association between muscle and joint flexibility and stress fractures. Studies were ranked according to the strength of their study design and then chronologically

Reference	Study design	Participants	Sample size	Factors analysed	Method of measurement	Results
Giladi et al.[56]	PC	Army (Israel)	295 M	Hip internal/external rotation	Goniometer – 90° hip flexion	Increased risk of SF with greater external rotation
				Ankle DF/PF	Goniometer – 90° knee flexion	No relationship with SF
				Rearfoot inversion/eversion	Goniometer	No relationship with SF
				Generalised ligament laxity	Thumb extension test	No relationship with SF
Montgomery et al.[57]	PC	SEAL (USA)	505 M	Hip extension	Thomas test – distance to table	No relationship with SF
				Hip internal/external rotation	Goniometer – prone	No relationship with SF
				Knee extension/flexion	Passive – distance	No relationship with SF
				Ankle dorsiflexion	Knee extension – distance	No relationship with SF
Milgrom et al.[59]	PC	Army (Israel)	783 M	Hip external rotation	Goniometer – 90° hip flexion	Increased risk of SF with greater external rotation
Bennell et al.[40]	PC	Athletes	53 F, 58 M	Hamstring/lumbar spine	Sit-and-reach test	No relationship with SF
				Hip internal/external rotation	Goniometer – 90° hip flexion and extension	No relationship with SF
				Ankle dorsiflexion	DF lunge test – distance	No relationship with SF
				Calf length	Goniometer – knee extension	No relationship with SF
Winfield et al.[61]	PC	Marines (USA)	101 F	Rearfoot inversion/eversion	Goniometer – passive	No relationship with SF
Hughes[62]	XS	Army (USA)	47 M	Ankle dorsiflexion	Goniometer – NWB passive	Reduced range 4.6 times at risk of metatarsal SF
Ekenman et al.[65]	XS	Athletes	29 SF M and F	Big toe flexion/extension	NS	No relationship with SF
			30 NSF M and F	Ankle DF/PF	NS	No relationship with SF
				Rearfoot inversion/eversion	NS	No relationship with SF
				Hamstrings	NS	No relationship with SF
				Quadriceps	NS	No relationship with SF
				Hip adductors	NS	No relationship with SF
				Hip flexors	NS	No relationship with SF

DF = dorsiflexion; F = female; M = male; NS = not stated; NSF = non-stress fracture; NWB = non-weight bearing; PC = prospective cohort; PF = plantarflexion; SF = stress fracture; XS = cross-sectional.

ments of less sensitive markers were taken at variable times after diagnosis.

A 12-month prospective study[77] evaluated bone turnover in 46 female and 49 male track and field athletes aged 17 to 26 years, of whom 20 developed a stress fracture. Baseline levels of bone turnover were evaluated in all athletes, while monthly bone turnover levels were evaluated in a subset consisting of the 20 athletes who sustained a stress fracture and a matched comparison group who did not sustain a stress fracture. Bone formation was assessed using serum osteocalcin and bone resorption by urinary excretion of pyridinium cross-links and *N*-telopeptides of type 1 collagen. Athletes who developed stress fractures had similar baseline levels of bone turnover compared with their non-stress fracture counterparts while serial measurements showed no differences in average levels of

bone turnover in those who developed stress fractures compared with the control group. There was also no difference in bone turnover levels prior to or following the onset of bony pain in the athletes with stress fractures.

These results show that single and multiple measurements of bone turnover are not clinically useful predictors of the likelihood of stress fractures in athletes. However, they do not negate the possible pathogenetic role of local changes in bone remodelling at stress fracture sites, given the high biological variability of bone turnover markers and the fact that levels of bone turnover reflect the integration of all bone remodelling throughout the skeleton. If trabecular bone, with its greater metabolic activity, contributes more to bone turnover levels than cortical bone, this may explain the relative insensitivity of bone turnover markers to stress fractures which are primarily cortical lesions.

3.2 Muscle Flexibility and Joint Range of Motion

The role of flexibility in stress fracture risk is difficult to evaluate as flexibility encompasses a number of characteristics including active joint mobility, ligamentous laxity and muscle length. It is likely that each of these characteristics, or even a combination of 2 or more (for example the paradoxical combination of joint laxity and muscle tightness), has its specific relation to stress injuries. Nevertheless, flexibility of muscles and joints may directly influence stress fracture risk by altering the forces applied to bone. Numerous variables have been assessed including range of rearfoot inversion/eversion, ankle dorsiflexion/plantarflexion, knee flexion/extension and hip rotation/extension together with length of calf, hamstring, quadriceps, hip adductors and hip flexor muscles (table V). Of these, only range of hip external rotation[38,56,59] and range of ankle dorsiflexion[62] have been associated with stress fracture development, and even these findings have been inconsistent.

Large prospective cohort studies in the Israeli military included an orthopaedic examination in addition to assessment of other risk factors for stress fractures.[38,59] Soldiers in whom hip external rotation was greater than 65° were at a higher risk for tibial and total stress fractures than those with a range less than 65°. The risk for tibial stress fracture increased 2% for every 1° increase in hip external rotation range.[59] However, 2 prospective studies, one in male American recruits[57] and the other in male and female athletes[40] failed to confirm these findings. It is possible that the Israeli recruits represent a separate population as their average hip external rotation range was much higher than that reported for other populations.

In a cross-sectional study of 47 male recruits, restricted ankle joint dorsiflexion was related to an increased risk of metatarsal stress fracture.[62] Those with a reduced range were 4.6 times more likely to develop a metatarsal stress fracture. Conversely, others have measured ankle dorsiflexion and failed to find a relationship. This may be because the data were analysed for all stress fracture sites combined, which may have masked a true relationship.

The difficulty in assessing the role of muscle and joint flexibility in stress fractures may relate to a number of factors including the relatively imprecise methods of measurement, the heterogeneity of these variables and the fact that both increased and decreased flexibility may be contributory. From a clinical perspective, assessment of muscle and joint flexibility should be included in the prevention and management of stress fractures until scientific evidence clarifies the role of these factors. If individuals are found to have restricted flexibility in comparison with individuals of similar age and sport, then measures can be introduced to stretch the tight structures. If increased flexibility is noted, it is important to ensure that there is adequate muscular stabilisation to control the greater range of motion.

3.3 Muscular Strength and Endurance

Skeletal muscle may play a dual role in stress fracture development. Some investigators consider that muscles act dynamically to cause stress fractures by increasing bone strain at sites of muscle

attachment.[78,79] If this is the mechanism of injury for stress fractures, then larger and stronger muscles with greater ability to generate force should be associated with an increased risk for stress fracture. Although there are no experimental studies showing that stress fractures develop from muscular pull at bony attachments, this would appear to be true in upper extremity stress fractures such as those of the ulna or radius in swimmers.

Skeletal muscle attenuates and dissipates forces applied to bone.[80] During running, each foot strikes the ground approximately 500 times per kilometre. Each heel strike generates vertical ground reaction forces (GRF) varying from 2 to 5 times bodyweight.[81,82] These can be considerably higher, up to 12 times bodyweight, during jumping and landing activities.[83] A shock wave travelling in bone results in vibrations from 25 to 100Hz.[84] These shock waves travel up the axial skeleton and are attenuated by muscles, bones and joint structures along the way. For example, 54% of the shock measured at the medial femoral condyle has been absorbed by the time it reaches the forehead.[80] It is therefore apparent that bone actually 'sees' only a small fraction of the total force, largely because of attenuation by muscles.

Therefore, muscle weakness or fatigue could predispose to stress fracture by causing an increase or redistribution of stress to bone.[85,86] In military recruits, accelerometry was used to assess the amount of shock transmitted to bone.[87] Vertical accelerations in the tibiae of recruits during walking were measured before and after a fast cadence march of 24km. A control group of soldiers who did not participate in the march were also measured. After the march, there were increases in acceleration amplitudes of between 20 and 30%, implying that reduced shock absorption capabilities resulted from skeletal muscle fatigue.

Using a biomechanical model, Scott and Winter[88] calculated that during running the tibia is subjected to a large forward bending moment as a result of ground reaction force. The calf muscles oppose this large bending moment by applying a backward moment as they contract to control the rotation of the tibia and the lowering of the foot to the ground. The total effect is a smaller bending moment. Extrapolating from this, a stress fracture could result if the calf muscles were unable to produce adequate eccentric force to counteract the loading at ground contact and decrease excessive bone strain.

Although most studies directly evaluating muscle strength and muscle fatigue have not found these to be related to stress fracture occurrence,[38,59,65] this may be due to the study design or the methods used to assess muscle function. Furthermore, it may not only be absolute strength that is important, but strength ratios between agonists and antagonists or muscle coordination, neither of which have been assessed adequately in relation to stress fractures.

In male recruits, relatively crude tests were used, including isometric quadriceps strength measured at 1 knee angle and the number of leg thrusts performed in 30 seconds. Despite the relative insensitivity of these tests, one recent study[89] found that recruits who were 1 standard deviation below the population mean for both absolute and relative maximal leg press strength had a 5 times greater risk for stress fracture than stronger recruits. In athletes, an isokinetic dynamometer was used to measure maximal concentric strength and endurance (during 100 repetitive maximal concentric contractions) of the ankle plantar flexor muscles. Although no difference was reported between stress fracture and non-stress fracture athletes, this study was cross-sectional in design so it is impossible to know whether the measurements reflect muscle function prior to the stress fracture. Furthermore, since the calf muscles act eccentrically to reduce bone load at ground contact, it may have been better to evaluate eccentric rather than concentric activity.

Some indirect evidence for muscle fatigue as a risk factor in stress fracture development comes from a study by Grimston and colleagues.[90] They found that during the latter stages of a 45-minute run, women with a past history of stress fracture recorded increased GRF whereas in the control group GRF did not vary during the run. The authors surmised that this may indicate differences in fa-

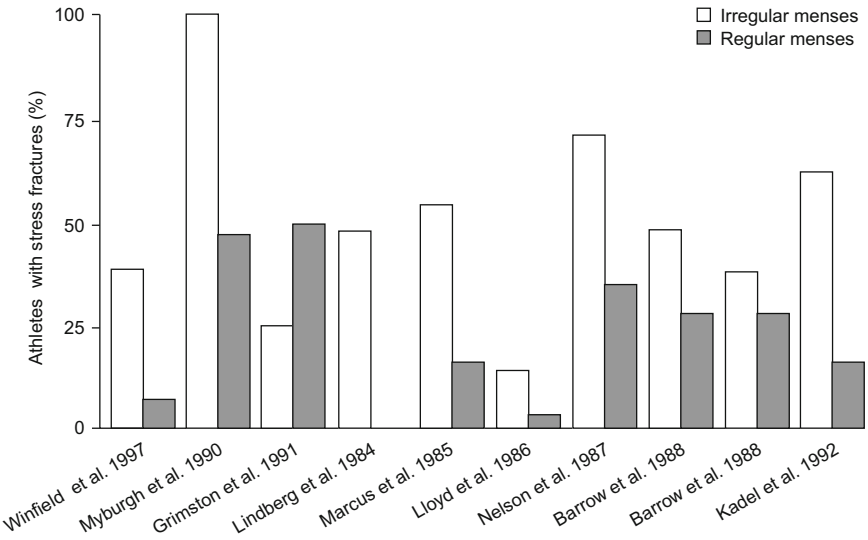

Fig. 3. Studies where the percentage of athletes/recruits with stress fractures were compared in groups with and without menstrual irregularity.

tigue adaptation and muscle activity. It is probable that muscular fatigue and reduced shock absorption are risk factors for athletes engaged in long distance running and recruits involved in extreme physical activity, but further research needs to confirm this contention.

Measurements of muscle size can be indicative of the ability of that muscle to generate force. Male recruits with a larger calf circumference developed significantly fewer femoral and tibial stress fractures.[87] This finding was also evident in female athletes (but not male athletes) where every 1cm decrease in calf girth was associated with a 4-fold greater risk of stress fracture.[40] In both studies, the calf girth was corrected for skinfold thickness to ensure that the measurement was a better indicator of calf muscle size. In the military study, the variable of calf girth circumference was also found to be independent of tibial bone width. The finding of a smaller calf girth in those who go on to develop stress fractures tends to support the hypothesis that muscles act to protect against rather than cause stress fractures. In order to establish a causal relationship, the effectiveness of a calf strengthening programme in reducing the incidence of stress fractures should be evaluated in a randomised, controlled trial.

4. Hormonal Risk Factors

4.1 Sex Hormones

Compared with the general female population, athletes have a higher prevalence of menstrual disturbances, including delayed menarche, anovulation, abnormal luteal phase, oligomenorrhoea and amenorrhoea.[91-93] Younger, nulliparous women of excessive leanness who train intensely appear particularly at risk of developing menstrual disturbances. In a questionnaire survey of 226 elite athletes, the prevalence of menstrual disturbances was higher in ballet (52%), gymnastics (100%), lightweight rowing (67%) and distance running (65%) than in swimming (31%) or team sports (17%).[94] Thus, it is apparent that menstrual disturbances are relatively common in the female athletic population.

Stress fractures may in fact be more frequent in female athletes with menstrual disturbances. The cause may be lowered estrogen levels resulting in lower bone density, accelerated bone remodelling

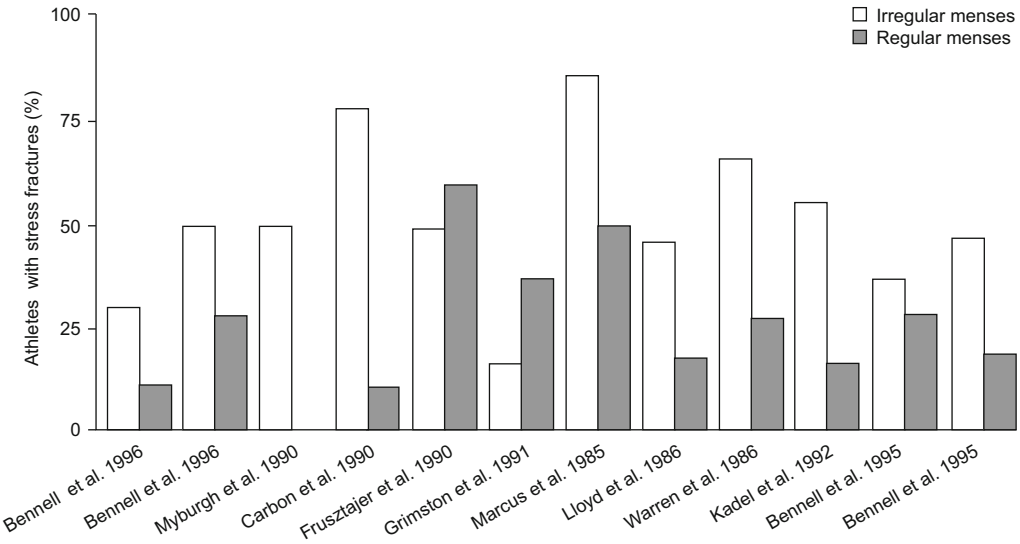

Fig. 4. Studies where the percentage of athletes/recruits with menstrual irregularity were compared in groups with and without stress fractures.

or negative calcium balance in these athletes, or the interaction of these variables. Studies have shown lower axial bone density in athletes with amenorrhoea or oligomenorrhoea compared with their eumenorrhoeic counterparts and/or sedentary controls.[95-98] Appendicular bone density may also be lower in female athletes with menstrual disturbances,[97,98] although this has been a less consistent finding. Estrogen deficiency leads to accelerated bone remodelling. Since bone resorption occurs before bone formation in this process, the bone is in a weakened state and hence more likely to accumulate microdamage if subjected to repeated loading. Estrogen loss also causes increased calcium excretion, which can result in negative calcium balance if dietary calcium is inadequate. Although this may seem analogous to the situation of increased risk of osteoporotic fracture in hypoestrogenic postmenopausal women, athletes, in contrast to postmenopausal women, typically sustain their bony injuries in cortical rather than trabecular bone. Furthermore, there may be decreased rather than increased remodelling in athletes with athletic amenorrhoea.

4.1.1 Relationship Between Shortened Luteal Phase and Stress Fractures

Although amenorrhoea is the most obvious sign of reproductive hormone disturbance, exercise may cause subtle changes in reproductive hormone levels that are too small to produce amenorrhoea. A decrease in progesterone production associated with short luteal phases and anovulation can be present in women despite normal menstrual cycle duration and flow characteristics.[99,100] Lowered progesterone levels may be detrimental to bone health, as progesterone might promote bone formation particularly in cortical bone.[101-103] In a prospective study involving eumenorrhoeic women, two-thirds of whom were runners, Prior et al.[104] found that recurrent short luteal phase cycles and anovulation were associated with spinal bone loss of approximately 2 to 4% per year. Serum progesterone levels and the proportion of the total menstrual cycle spent in luteal phase have also been found to be significant predictors of lumbar spine bone density,[105] as well as rate of change of bone mass at this site.[104] However, a cross-sectional study failed to find a significant difference in spinal bone density between groups with short and long luteal phase

lengths.[106] Despite the possible detrimental effects of luteal phase deficiency on bone, a link between this and stress fracture risk has not yet been sought.

4.1.2 Relationship Between Amenorrhoea, Oligomenorrhoea and Stress Fractures

The relationship between amenorrhoea or oligomenorrhoea and stress fracture risk has been the subject of a number of studies, mainly retrospective cross-sectional surveys of runners and ballet dancers.[71-74,107,108] Many of these studies are characterised by small samples and low questionnaire response rates. In other studies, volunteers were specifically recruited according to certain criteria, either stress fracture history or menstrual status.[42-45,96,109-112] Categorisation of menstrual status is based on number of menses per year, rather than on analysis of hormonal levels, and definitions of menstrual status vary between studies. Where hormonal assessment is included, most are single measurements, often non-standardised with respect to menstrual cycle phase. The length of exposure to amenorrhoea also differs within and between studies, and this may influence the risk of stress fracture.

Despite the methodology limitations, the findings generally suggest that stress fractures are more common in athletes with menstrual disturbances (figs 3 and 4).[40,42-46,61,71-74,107,109,110] Athletes with menstrual disturbances have a relative risk for stress fracture that is 2 to 4 times greater than that of their eumenorrhoeic counterparts. However, logistic regression analysis in ballet dancers showed that amenorrhoea for longer than 6 months' duration was an independent contributor to the risk of stress fracture and that the estimated risk was 93 times that of a dancer with regular menses.[74] While this risk seems extraordinarily high, there were only 6 dancers with regular menses in this sample of 54 dancers and this may have affected the statistical analyses.

The risk of multiple stress fractures also seems to be increased in those with menstrual disturbances.[73,108] Clark et al.[108] found that while amenorrhoeic and eumenorrhoeic groups reported a similar prevalence of single stress fractures, 50% of the amenorrhoeic runners reported multiple stress fractures compared with only 9% of those regularly menstruating. In female distance runners, the amenorrhoeic group was the only one to have a runner who had sustained 6 stress fractures, whereas in the 120 eumenorrhoeic runners, none had more than 3 stress fractures.[73]

Grimston and colleagues[113] developed a menstrual index that summarised previous and present menstrual status. The index quantified the average number of menses per year since menarche. They found no relationship between this menstrual index and the incidence of stress fractures in 16 female runners. Conversely, track and field athletes with a lower menstrual index, indicating less menses per year since menarche, were at greater risk of stress fracture than those with a higher index.[40] Barrow and Saha[73] also found that lifetime menstrual history affected the risk of stress fracture. They showed the incidence of stress fracture to be 29% in the regular group and 49% in the very irregular group.

Although methodological differences make direct comparison of results difficult, it would appear that there is a higher incidence of menstrual disturbances in female athletes with stress fracture than in those without. These findings have led some authors to assume that this is a direct result of lowered estrogen levels and decreased bone mineral density in athletes with menstrual disturbances. However, athletes with menstrual disturbances also exhibit other risk factors such as lower calcium intake,[92] greater training load[114] and differences in soft tissue composition.[94] Since these were not always controlled for in the studies discussed, it is difficult to ascertain which are the contributory factors.

Menstrual disturbances may also predispose to stress fractures in female recruits. In a recent prospective cohort study of 101 female Marines,[61] the incidence of stress fractures in those with fewer than 10 periods per year was 37.5% compared with 6.7% in those with 10 to 13 periods per year. Conversely, in a study of 49 female soldiers with stress fractures and 78 soldiers with no orthopaedic injuries, menstrual patterns did not differ between

groups.[47] However, the number of soldiers with menstrual disturbances was relatively low.

Given the association between menstrual irregularity and risk of stress fracture, it is important to question physically active females about their current and past menstrual status and then seek appropriate medical opinion if necessary. Since menstrual disturbances are often found together with eating disorders and osteopenia, commonly referred to as the 'female athlete triad', the presence of one of these factors should alert the practitioner to the possibility of the others.

4.1.3 Relationship Between Oral Contraceptive Pill Use and Stress Fractures

Some authors have claimed that the oral contraceptive pill (OCP) may protect against stress fracture development by providing an exogenous source of estrogen to reduce the remodelling rate and improve bone quality and/or density. There have been no randomised intervention trials to show that use of the OCP reduces the stress fracture rate in athletes, particularly in those with prior or current menstrual disturbances.

Two prospective cohort studies, one in athletes[40] and one in female Marines,[61] have failed to support a protective effect of OCP use on stress fracture development, although numbers in the stress fracture groups were relatively small.

The results of cross-sectional studies are contradictory. Barrow and Saha[73] found that runners using the OCP for at least 1 year had significantly fewer stress fractures (12%) than nonusers (29%). This was supported by the findings of Myburgh et al.[44] Conversely, no difference in OCP use was reported in ballet dancers with and without stress fractures.[74] However, few dancers were taking the OCP. Since these studies are retrospective in nature, it is not known whether the athletes were taking the OCP prior to or following the stress fracture episode. In addition, athletes may or may not take the OCP for reasons which in themselves could influence stress fracture risk. It is not known whether the risk of stress fracture is decreased in athletes with menstrual disturbances who subsequently take the OCP.

4.1.4 Relationship Between Testosterone Levels and Stress Fractures in Male Athletes

The relationship of testosterone levels to bone density and stress fracture risk in young male athletes has not been well investigated. However, the results of a limited number of studies[115-117] have failed to establish a relationship between lowered circulating testosterone levels and osteopenia in male athletes. A case report[118] described the clinical features of a 29-year-old male distance runner who presented with a pelvic stress fracture, markedly decreased bone density and symptomatic hypogonadotropic hypogonadism. Using this case as an index, the authors hypothesised that exercise-induced hypogonadotropic hypogonadism could be identified in male athletes by the presence of one or more specific risk factors, which included the presence of sexual dysfunction, a history of fracture and the initiation of endurance exercise before age 18 years.[119] They compared levels of free testosterone and luteinising hormone in 15 male runners with one or more of the above risk factors and 13 runners with none of the risk factors. Only one of the runners in the first group was identified as having primary hypogonadism and there was no significant difference between groups for hormone levels. However, bone density was not measured in these runners and correlated with testosterone levels.

From a clinical perspective, it is important to clarify that although some male athletes do present with reduced testosterone levels, these concentrations are generally still within the normal range for adult men. Therefore, stress fracture risk may not be increased and detrimental effects on bone density may not be as dramatic as those described for females with athletic amenorrhoea where estradiol levels are well below normal.

4.2 Menarcheal Age

Menarche is attained later in athletes compared with nonathletes, particularly in certain sports such as ballet, gymnastics and running.[120,121] The relationship between age of menarche and risk of stress fracture is unclear. Some authors have found that athletes with stress fractures have a later age of

menarche,[40,42,72,112] while others have found no difference.[74,43,44] In a prospective cohort study,[40] age of menarche was an independent risk factor for stress fracture in female track and field athletes, with the risk increasing by a factor of 4.1 for every additional year of age at menarche.

An association between delayed menarche and stress fractures may be caused by a lower rate of bone mineral accretion during adolescence and therefore decreased peak bone mass.[122,123] However, the relationship between age of menarche and bone density in female athletes is unclear, with some investigators finding significant, but moderate to weak, negative correlations at a number of bone sites[112,124,125] and others not.[96,126,127] Although these results suggest that delayed sexual maturation is not strongly associated with lower bone density in female athletes, many of the samples have been small and the influence of confounding variables has not been taken into account. In larger cohorts of healthy adolescents and pre- and post-menopausal women, the most common finding is that a later age of menarche is related to lower bone density.[122,128-131] However, this does not imply a causal relationship since other factors such as genetic background may be major determinants of both variables.

A later age of menarche has also been found in association with menstrual disturbance, lowered energy intake, decreased body fat or bodyweight and excessive premenarcheal training.[132,133] All of these could feasibly influence stress fracture risk. Whatever the reason for an association, athletes should be questioned about when they commenced their periods. A later age of menarche could then be used as a marker to identify a possible increased risk of fracture.

4.3 Other Hormones

Although alterations in calcium metabolism could affect bone remodelling and bone density and thus predispose to stress fracture, there is no evidence to support such a relationship. Single measurements of serum calcium, parathyroid hormone, 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D did not differ between stress fracture and non-stress fracture groups in military recruits[134] or athletes.[42,44,72] These findings may reflect the sampling procedures, as single measurements were taken at some time point following stress fracture. Conversely, since many of these biochemical parameters are tightly regulated, alterations in calcium metabolism may not be a factor in stress fracture development in healthy individuals. Other endocrine factors which have the potential to influence bone health and hence stress fracture risk include glucocorticoids, growth hormone and thyroxin.

5. Nutritional Risk Factors

Dietary surveys of various sporting groups often reveal inadequate intakes of macro- and micronutrients. This is particularly true in females and in sports where low weight is desirable, such as gymnastics, ballet or distance running. The presence of abnormal eating behaviours including disorders such as anorexia nervosa and bulimia nervosa will also contribute to inadequate dietary intake and appear to be more common in certain athletes.[93]

Dietary deficiencies, in particular dietary calcium, may contribute to the development of stress fractures by influencing bone density and bone remodelling. In animal studies, a calcium-deficient diet decreases the ability of bone to adapt to mechanical strain,[135] while high dietary calcium intake has a favourable effect on bone biomechanical properties.[136] In humans, some studies have found a positive relationship between dietary calcium intake and bone mass,[137,138] while others have noted small gains in bone mass resulting from calcium supplementation.[139,140]

Several factors make it difficult to clarify the role of diet in bone health and stress fracture development:

- The accurate assessment of habitual dietary intake is problematic.
- Nutrients may exert their effects on bone over a number of years and hence measurement of current intake may not represent lifetime status.

- Calcium balance is negatively influenced by other dietary factors including high intakes of salt, phosphorus, fibre, protein, caffeine and alcohol.
- Calcium operates as a threshold nutrient whereby intakes above a certain level produce no additional effects on bone.[141]
- The recommended daily allowance for calcium differs across the life span and may not adequately address the specific needs of physically active individuals or female athletes with menstrual disturbances.

The only randomised, intervention study to assess the relationship of calcium intake to stress fracture development was conducted in military recruits. Schwellnus and Jordaan[142] found a similar incidence of stress fractures during a 9-week training programme in 247 male recruits taking supplementation of calcium 500mg daily, and in 1151 controls not taking additional calcium. This result does not appear to support a role for calcium in stress fracture prevention. However, 9 weeks is probably not long enough for any effects of calcium to become apparent, particularly at cortical lower limb sites where the bone turnover rate is slower. Furthermore, both groups had a baseline dietary calcium intake greater than 800 mg/day which is the recommended daily allowance for adults. This intake may have been sufficient to protect against stress fracture with additional calcium offering no added benefit. No studies have evaluated the effect of calcium supplementation on stress fracture incidence in individuals whose usual dietary calcium intake is below the recommended daily allowance.

A recent cross-sectional study in female military recruits did not find a difference in dietary calcium intake between the stress fracture and non-stress fracture groups,[47] confirming the results of the intervention study in male recruits discussed previously.[142]

There is scant evidence to show that lower calcium intake is associated with an increased risk for stress fracture in athletes. In a cross-sectional study, Myburgh et al.[143] found a significantly lower intake of calcium in athletes with shin soreness compared with a matched control group. However, since exact diagnoses were not made, stress fracture may not have been the only pathology included in the shin soreness group. A follow-up study in athletes with scintigraphically confirmed stress fractures did show similar results.[44] Current calcium intake was significantly lower in the stress fracture group, being 87% of the recommended daily intake.

Other studies in athletes have failed to confirm a relationship between stress fractures and current dietary calcium intake.[40,42,43,45,74,112] Ballet dancers were found to consume less than the recommended daily allowance for calcium regardless of their stress fracture status,[43,74] implying that other factors may be more important as risk factors in dancers. Calcium intake in male and female track and field athletes, assessed using 4-day food records as well as food frequency questionnaires, was similar in those with and without stress fractures.[40] This latter result does not necessarily exclude calcium deficiency as a risk factor for stress fracture, as the majority of athletes in this study were consuming more than the recommended daily allowance of 800 mg/day and hence would not be regarded as calcium deficient. Only one study has evaluated the relationship of historical calcium intake and stress fracture occurrence.[45] A calcium index, based on the variability in calcium intake between the ages of 12 to 23 years, was found to be similar in runners with and without stress fractures.

Negative influences on calcium balance can include high intakes of salt, protein, phosphorus, caffeine and alcohol. At present, there are no reports of any associations between these and the incidence of stress fractures in athletes.[40,42-44,112]

Dietary behaviours and eating patterns may differ in those with stress fractures. Ballet dancers with stress fractures were more likely to restrict caloric intake, avoid high fat dairy foods, consume low calorie products, have a self-reported history of an eating disorder and have lower percentages of ideal bodyweight than those without stress fractures.[43] Similarly, in a cross-sectional study of young adult female track and field athletes,[46] those with a his-

tory of stress fracture scored higher on the Eating Attitudes Test (EAT)-40 (a validated test relating to dieting, bulimia and food preoccupation, and oral control) and were more likely to engage in restrictive eating patterns and dieting than those without stress fractures. In this same group followed prospectively, 4-day food records revealed a lower fat diet in the females who went on to develop stress fractures during the year of the study.[40]

More recently, a large multicentre survey of 2298 US collegiate athletes[93] revealed that White females with a history of pathogenic weight control behaviours had a relative odds ratio of 1.96 of having a stress fracture compared with those who did not have this history. These studies suggest that disordered patterns of eating are associated with a higher risk for stress fracture. Whether this association is causal or due to some other factor is not clear.

In summary, there is currently little evidence to support dietary deficiencies, in particular low calcium intake, as a risk factor in otherwise healthy recreational and elite athletes or military recruits. Conversely, abnormal and restrictive eating behaviours do seem to be related to a greater likelihood of fracture. Healthy eating habits should be promoted in all individuals. If one is concerned about dietary intake in athletes, food records as well as biochemical and anthropometric indices can be used to assess dietary adequacy and nutritional status.

6. Physical Training Risk Factors

Repetitive mechanical loading arising from athletic training contributes to stress fracture development. However, the contribution of each training component (type, volume, intensity, frequency and rate of change) to the risk of stress fracture has not been elucidated. Training may also influence bone indirectly through changes in levels of circulating hormones, effects on soft tissue composition and associations with menstrual disturbances.

6.1 Physical Fitness

Whether lack of prior physical activity and poor physical conditioning predisposes to stress fracture is unclear, as many studies have relied on self-reporting rather than on standardised fitness tests before commencement of the exercise programme. Most of the literature focuses on military recruits who are subjected to a short burst of intense, unaccustomed activity and are often unfit.

Some military studies have reported a correlation between self-reported previous physical activity levels and rate of stress fracture during basic training, while others have failed to corroborate a relationship. Montgomery et al.[57] found that male trainees with a running history averaging at least 25 miles per week in the previous year had a lower incidence of stress fracture (3%) than trainees averaging less than 4 miles per week (11.5%). Similarly, Gardner et al.[144] found the stress fracture rate to be 24 times greater in the previously inactive group than in the very active group.

In a prospective cohort study in female US Marines, those who reported running less than 2.8 miles per session had a 16.3% incidence of stress fractures during basic training compared with 3.8% who ran more than 2.8 miles per session.[61] However, there was no relationship between stress fractures and the frequency of running sessions nor a battery of fitness tests including running, sit-ups and push-ups. In a study of female US military recruits, the authors reported that higher leisure activity energy expenditure tended to be associated with a lower stress fracture risk ($p = 0.06$).[47] In a recent study, an algorithm of 5 physical activity questions and a 2.4km run time revealed that 21.6% of 'high risk' individuals experienced more than 3 times as many stress fractures as 'low risk' individuals, suggesting that risk of stress fracture is increased by poor physical fitness and low levels of physical activity prior to entry into recruit training.[145]

Conversely, in a large study,[146] neither aerobic fitness, measured by calculating the predicted $\dot{V}O_{2max}$, nor self-reported pretraining participation in sport activities was related to stress fracture in-

cidence in 295 male recruits aged 18 to 20 years. This lack of association is confirmed in other large studies of male recruits.[38,89]

Although the data are conflicting, the majority of studies tend to suggest that physical fitness or prior physical activity may be a predictor of stress fracture risk in individuals undergoing basic military training. Poor physical conditioning does not seem to apply to athletes, as stress fractures often occur in well conditioned individuals who have been training for years.

6.2 Training Regimen

Aspects of the training regimen can influence stress fracture development. Military studies have shown that various training modifications can decrease the incidence of stress fractures in recruits. These interventions include rest periods,[147,148] elimination of running and marching on concrete,[149,150] use of running shoes rather than combat boots[150,151] and reduction of high impact activity.[75,148,152] These may reduce stress fracture risk by allowing time for bone microdamage to be repaired and by decreasing the load applied to bone.

In contrast, there is little controlled research in athletes. Most are anecdotal observations or case-series where training parameters are examined only in those athletes with stress fractures. For example, surveys reporting that up to 86% of athletes can identify some change in their training prior to the onset of the stress fracture[68,153] do not provide a similar comparison in uninjured athletes. Other researchers have blamed training 'errors' in a varying proportion of cases but do not adequately define these 'errors'.[1,67,154,155] Brunet et al.[63] surveyed 1505 runners and found that increasing mileage correlated with an increase in stress fractures in women but not men. An explanation for the apparent gender difference is unclear. In a study of ballet dancers, a dancer who trained for more than 5 hours per day had an estimated risk for stress fracture that was 16 times greater than a dancer who trained for less than 5 hours per day.[74] These studies support a role for training volume as a risk factor for stress fracture.

It is apparent that bone remodelling is able to repair microdamage if given adequate time, but that a stress fracture results with the repeated application of a load. Hence, one would surmise that cyclic training would be preferable to progressive training in order to allow both bone and soft tissue to rest from repetitive loading. Athletes and military personnel should perform alternative aerobic exercise with low impact loading for a week after 2 to 3 weeks of training. Furthermore, alternative exercise such as cycling or swimming should be added into weekly training in place of running.

In the clinical setting, it is imperative to obtain a detailed training history to try and identify any training parameters that may have contributed to that individual's stress fracture. In particular, questions should be directed at establishing volume, intensity, degree of rest periods, type of training and any recent changes in these parameters. Furthermore, athletes should be encouraged to keep an accurate training log book. This will allow the athlete to monitor his or her training and gauge the appropriateness of training changes. Athletes should be counselled about the nature of overuse injuries and should be advised to seek medical assistance at the first onset of any pain. If overuse is identified early in the continuum, then a period of reduced activity may break the cycle and allow a stress reaction to heal rather than developing into an actual stress fracture.

7. Extrinsic Mechanical Risk Factors

7.1 Surface

Training surface has long been considered a contributor to stress fracture development.[156] Anatomical and biomechanical problems can be accentuated by cambered or uneven surfaces, while GRF are increased by less compliant surfaces.[157,158] Alternatively, running on softer surfaces may hasten muscle fatigue. Large epidemiological studies of overall running injuries fail to show an association between training surface or terrain after controlling for effects of weekly running distance.[159,160] However, this may be related to the difficulty in

accurately quantifying running surface parameters and to sampling bias. While there are no data assessing the relationship of training surface and stress fractures specifically, it still may be prudent to advise athletes to minimise the time spent training on hard, uneven surfaces.

7.2 Footwear/Insoles/Orthotics

Athletic footwear and insoles/orthotics aim to attenuate shock with ground contact and to control motion of the foot and ankle. The material characteristics and construction of the midsole of an athletic shoe mainly determine its shock absorbing and attenuating properties.[161] Using price as a surrogate measure of the quality of running shoes, a large prospective study failed to find a significant difference in stress fracture rates in recruits wearing high quality *versus* low quality runners.[144] However, the assumption that price equates to quality is not necessarily valid. The age of a shoe is thought to provide an indication of the condition of the midsole. In the same study, a significantly higher stress fracture rate was found in recruits wearing older or worn running shoes.[144] While this could be because of decreased shock absorption in older shoes, age also has a detrimental effect on the mechanical support provided by the shoe.[162] Similar results were reported in a study of 25 patients with shin soreness and 25 matched control patients, where 80% of injured patients were wearing worn shoes compared with 44% of control patients.[143]

Changing from military boots to athletic shoes may reduce the incidence of stress fractures in the foot.[150,163] An experimental study[163] was conducted in 390 infantry recruits to evaluate whether the incidence of overuse injuries was affected by type of footwear. Basketball shoes were provided to 187 randomly selected recruits while the remainder wore standard military boots. After 14 weeks of basic training, there was no significant difference between overall stress fractures rates in the 2 footwear groups. However, those training in basketball shoes had a significantly lower incidence of overuse injuries of the foot, suggesting that the effect may be limited to injuries resulting from vertical impact loads. Injuries such as tibial stress fractures which are secondary to bending forces were not affected by the use of a more cushioned shoe.[161] However, the results of a pilot study[164] where rosette strain gauges were mounted onto the tibial mid shaft in a human volunteer showed that different shoes can influence tibial strains during walking and running. Surprisingly, in this study a new form of infantry boot produced the lowest compressive strains compared with various sport shoes, despite the relatively higher weight and sole durometry of the boot.

Shock absorbing insoles are often used in an attempt to reduce the incidence of overuse injuries. There are many different types of insoles on the market which vary in their ability to absorb shock and change foot biomechanics. For example, Cinats et al.[165] found that the use of Sorbothane® inserts reduced the final transmitted stress over the duration of heel strike by less than 10%, while Voloshin and Wosk[166] found that viscoelastic inserts reduced the amplitudes of shock waves during gait by an average of 42%. There are conflicting reports about whether insoles can prevent stress fracture.

An animal model was used to assess the effect of viscoelastic orthotics on the incidence of tibial stress fracture.[167] The hindlimbs of skeletally mature rabbits were passively loaded 5 days per week over a 6- or 9-week period. Tibial stress fractures were sustained by 90% of rabbits who trained without orthotics and 92% who trained with orthotics.

In a randomised experimental study in the Israeli army, use of a semi-rigid orthotic device made no significant difference to the overall incidence of stress fractures during basic training, but did reduce the incidence of femoral stress fractures.[168] Why an insole effect was limited to femoral stress fractures only is unclear. When the data were further analysed, there appeared to be an interaction between foot type, the orthotic and the site of stress fracture.[58] For recruits wearing the orthotic, the incidence of femoral stress fractures was reduced in those with high arched feet, while the incidence of metatarsal fractures was reduced in those with

low arched feet. The incidence of tibial stress fractures was not affected by the use of this orthotic device. Since the device had a hindfoot post at 3° varus altering the biomechanics of the foot, it is difficult to know whether the results of the study can be attributed to this feature or to the shock absorption capability.

In another large intervention study of 3025 US Marines,[144] there was no difference between stress fracture rates in the experimental group wearing the viscoelastic polymer insole and the control group. This contrasts with the results of a similar study in South African recruits where 250 recruits wore neoprene insoles in their shoes during 9 weeks of basic training while 1151 recruits acted as a control group.[169] Compliance was good, with 85% of recruits reporting that they wore the insoles daily. Results showed that the insoles significantly reduced the overall incidence of overuse injuries with a noticeable trend for stress fractures (0% in the experimental group and 1.4% in the control group). The difference in results may be due to differences in the insoles. Viscoelastic insoles have not been shown to significantly reduce vertical impact forces when compared with conventional running shoe insoles.[170] Furthermore, neoprene insoles have been found to be less rigid, more resistant to shear compression forces and able to reduce transmitted force better than viscoelastic insoles.[171]

In track and field, clinical observation suggests that the use of running spikes may influence the likelihood of stress fracture. To date, there is insufficient research on the relationship of this form of footwear and the risk of stress fracture development.

From a practical perspective, it is important that individuals train in shoes that are appropriate for their foot type. When selecting an athletic shoe, the important features to consider are midsole hardness, midsole width, heel flare, heel height, stability devices and torsional flexibility. Those with high arches should consider shock absorbing features a priority, while those with low arches need support for excessive motion.

7.3 External Loading

In humans, direct measurement of bone strain through the surgical attachment of a bone strain gauge has both ethical and methodological constraints. GRF, measured by a force platform set into the floor, can provide an indirect measure of both the magnitude and rate of external load on the lower extremity during physical activity.[172]

There is conflicting evidence about the role of external loading as measured by GRF in predicting stress fracture risk. In 2 cross-sectional studies, Grimston and colleagues[45,90] found significant differences in GRF during running between the stress fracture and non-stress fracture groups. However, in their initial study the forces were higher in the stress fracture group, while they were lower in the subsequent study. Sample characteristics and testing procedures differed between their two studies and in some aspects were not well controlled, which may have contributed to the inconsistent findings. In particular, the samples were small and heterogeneous, there was an insufficient number of running trials and the running speed was not standardised. A larger, more recent, cross-sectional study in 46 male runners[48] failed to support a role for external loading kinetics in stress fracture development.

8. Other Risk Factors

8.1 Age

Age may be a risk factor for stress fractures, but whether the rates increase or decrease with age is controversial. Certainly bone density decreases with age and this could reduce the ability of bone to withstand repetitive loading in older individuals. Furthermore, matrix microdamage accumulation in human compact bone has been shown to increase with increasing age, particularly in women, and that the increasing microdamage accumulates more rapidly than intrinsic processes can repair it.[173] This has implications for the postmenopausal athlete as it results in a reduced resistance to fracture. On the other hand, children and adolescents with immature bones may also be at risk, as peak bone mass and strength are not reached until around the

late teens and early 20s.[122,174] Other factors, including training habits and hormonal status, may also explain a possible relationship between age and stress fracture risk.

Military studies allow stress fracture rates to be compared in individuals of different ages engaged in identical or similar training programmes. However, the age range is relatively narrow and is confined to early adulthood in most cases. In a retrospective cohort study of 20 422 military recruits,[175] a review of clinical records found that the incidence of stress fracture increased from 1.3% for army recruits aged 17 to 22 years, to 2.3% for those aged 23 to 28 years, and to 5.0% for those aged 29 to 34 years. The relative risk of stress fracture for this latter age group compared with the youngest age group was 3.9. In the recruits aged over 35 years the incidence was 2.4%, but the numbers in this group were small and thus considered unreliable. This higher risk of stress fracture with age was also seen when men and women were evaluated separately. Similar results have been reported in prospective cohort studies. Even after adjusting for pre-training physical activity levels, recruits over the age of 21 years (of the 934 trainees in this group, only 37 were older than 25 years) had a relative risk of stress fracture of 1.7 (95% CI; 0.92, 3.21) compared with recruits aged 18 to 20 years.[144]

Conversely, two other studies[61,59] have found that younger recruits are more likely to develop stress fractures than are older recruits. In female Marine trainees the stress fracture incidence rate was 17% in those under the age of 23 years but only 2% in those over 23 years.[61]

A prospective study by Milgrom et al.[59] among male Israeli army recruits also reported that those with stress fractures were significantly younger than those without, but the mean difference was small, approximately 1 month (18.58 years versus 18.70 years). Multivariate statistical analysis revealed that for each year of increase in age from 17 to 26 years, the risk of stress fracture at all sites decreased by 28%. However, of the 783 recruits, only 26 were over the age of 19 years. Thus, the statistical results would seem questionable, particularly for the post-teenage years. The other studies[144,175] finding an increasing stress fracture risk with increasing age had a greater spread of ages and analysed their results differently by grouping ages together into categories. It is possible that between 17 and 19 years of age there is a decreasing risk for fracture, but this risk may still be less than those in the early to mid-20s.

Whether or not age is a risk factor for stress fractures in athletes is not known. The difficulty in athletes is quantifying the amount and intensity of activity in different aged populations to allow an accurate comparison of stress fracture rates.[176] If differences in rates are found, these may be simply due to differences in exposure to physical activity and not to age-related factors.

One case series of 1407 patients presenting to a sports medicine centre found that stress fractures/periostitis comprised a greater percentage of injuries in the 'younger' group (mean age of 30 years) compared with that in the 'older' group (mean age of 57 years).[177] However, due to the study design, it is not known whether this reflects selection of stress fracture resistant individuals in the older group, modification of training regimens to lower musculoskeletal stress or an independent age effect on stress fracture development.

There is discrepancy in the results from military studies,[59,61,144,175] with some finding an increase in stress fracture risk with increasing age from the late teens to the mid-30s and others finding a decreasing risk. At this stage, the athletic literature does not provide enough evidence to evaluate an age effect.

8.2 Psychological Traits

There is little information about psychological traits and stress fractures, particularly in athletes. Of the three prospective studies conducted in the military, two failed to find an association between psychological factors and incidence of stress fractures. Pre-training motivation, assessed by the Quality Index, was not related to the 31% incidence of stress fractures in male Israeli military recruits.[38] Similarly, Taimela et al.[75] found that,

in multivariate analysis, psychomotor reaction time parameters, mental ability parameters and specific personality traits were not predictive of stress fractures. Conversely, low achievement and high obedience personality traits were related to an increased incidence of stress fractures in 108 army conscripts.[178] However, these results may not be extrapolated directly to athletes involved in voluntary exercise. It could be speculated that, instead, high achievement and motivation may be related to greater training volume and intensity and perhaps disordered patterns of eating, all of which could contribute to stress fracture development.

9. Conclusions and Clinical Implications

Although most studies that have evaluated risk factors for stress fractures do not permit the assignment of causality, recommendations for clinical practice can be made. Anecdotally, it is apparent that training factors are important for stress fracture development. Therefore, individuals should ensure that they gradually increase intensity and total load and include periods of cross training and rest days to allow time for remodelling processes to repair microdamage. It may be necessary to include exercises into training to help specifically strengthen bone and increase its resistance to fracture. From studies utilising animal models, the best types of exercise appear to be those that primarily involve high strain rates.[179] These could be incorporated into a progressive training programme or commenced prior to the introduction of basic training in military recruits. Since muscle fatigue may be an important factor in the pathogenesis of stress fractures, exercises should be included to improve muscular resistance to fatigue. This occurs only through sport specific training, and muscular adaptations require a minimum of 8 weeks of training, but in fact improvements in fatiguability resistance increase for many months with training.

Attention should be paid to lower limb alignment and differences in leg length should be corrected with the use of a heel raise. Abnormal foot posture may need to be addressed with appropriate orthotics and footwear, particularly in those with recurrent stress fractures.

For exercising females, the absence of regular menstrual cycles appears to be associated with a greater risk of stress fracture. For this reason, it is important to screen for menstrual irregularities and investigate the cause of these. If there is prolonged amenorrhoea and/or recurrent stress fractures, it may be worth including a bone density scan to assess for the presence of osteopenia as this may influence the athlete's management. Although it is not clear whether or not taking the OCP assists with reducing the risk of stress fracture, it may be an option for women, particularly those whose endogenous oestrogen levels are low. Encouraging physically active individuals to adopt healthy eating patterns and to consume a well balanced diet with adequate calcium intake is also sensible.

Finally, since stress fractures occur along a continuum, monitoring for signs and symptoms of excessive bone strain with a subsequent reduction or cessation of training may circumvent the development of a full stress fracture.

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