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Tibial Stress Injuries: Aetiology, Classification, Biomechanics and the Failure of Bone

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1. Introduction

Stress fractures (SFs) were originally recognised in 1855 by Breithaupt, a Prussian military surgeon, who noticed that young military recruits suffered painful swelling of the forefoot after long marches (Carlson and Wertz, 1943). Initially called March Fractures, he believed the condition to be inflammatory, but in 1897 the bony nature of the affliction was identified when Stechow performed Roentgen studies on the metatarsals (Carlson and Wertz, 1943).

During World War II, the incidence of March fractures was prominent in new military recruits who were unaccustomed to long route marches with heavy packs (Carlson and Wertz, 1943; Bernstein and Stone, 1944). When the training programme became more intense, it was noticed that the frequency of the injury increased (Bernstein and Stone, 1944; Bernstein et al., 1946). By this time, 'March' fractures were also known as insufficiency, exhaustion, fatigue and creeping fractures (Hullinger, 1944). Although diagnosed in bones other than the metatarsals, they were still called March fractures (Carlson and Wertz, 1943; Hullinger, 1944; Bertram, 1944). The classic military type March fractures, however, are found in the neck and the mid-shaft of the second metatarsal.

Medial Tibial Stress Syndrome (MTSS) was probably first identified in 1913 when Hutchins discovered what he called 'spike soreness' in runners (as they were wearing running spikes). He described it as an area of tenderness in the posteromedial distal tibial region sustained in new athletes learning running techniques, or athletes altering their training regimens. Although there was no name for the injury at the time, Hutchins noticed the involvement of the periosteum and attributed the cause of pain to the flexor digitorum longus (FDL) tibial origin (Hutchins, 1913).

MTSS was not originally recognised as a separate entity to an overt stress fracture (SF) of the tibia, which is not surprising as at the time, patient examination and radiographs were the only clinical tools available for the diagnosis of these injuries. Usually MTSS patients demonstrated no abnormal signs on plane X-rays, but this common observation was due to lack of refinement of the imaging technology available at the time.

Devas (1958) was one of the first clinicians to extensively study tibial SFs and 'shin soreness' in athletes by using clinical observations in conjunction with plane radiographic interpretations. Assuming only one type of injury under study, Devas (1958) described a

'shin soreness type of SF involving a disruption of the periosteum over a varying distance' and described other symptoms i.e. tibial tenderness with soft tissue 'thickening' of the subcutaneous surface of the tibia and periosteal oedema. He also noted that plane radiological changes in this type of so-called SF were either of late onset, or not seen at all.

The term *shin splints* was originally used as a non-specific clinical term to describe distal tibial pain caused by repeated impact in the absence of any other injuries to the region. However, an improved understanding of the aetiology in conjunction with advances in nuclear medicine diagnostic techniques led researchers and clinicians to realise that shin splints, later defined more specifically as MTSS, was a separate condition which could be differentiated from other forms of distal leg pain such as SFs or compartment syndrome.

The distinction between tibial SFs and MTSS was becoming clearer and in 1966, the American Medical Association (AMA) defined the shin splint syndrome as "pain and discomfort in leg from repetitive running on hard surface or forcible excessive use of foot flexors; diagnosis should be limited to musculotendinous inflammations, excluding fatigue fracture or ischemic disorder" (AMA, 1966). It was not until the late 1960s and in the 1970s that a large range of Technitium-99 labelled radiopharmaceuticals were developed (Seibert, 1995), making Triple Phase Bone Scintigraphy (TPBS) available as a diagnostic tool. TPBS was initially believed by some clinicians to be of no advantage in the diagnosis of MTSS as it was claimed that the uptake of radionuclide was related to increased activity of the patient rather than being specific to a particular pathology (Rorabeck et al., 1983; Wallensten, 1983; Allen, 1996). However, abnormal scintigraph findings in patients with clinical signs of MTSS who did not subsequently develop a SF lead to the scintigraph definition of MTSS (Holder and Michael, 1984).

About this time, probably the first comprehensive and now classic clinical study of SFs was published (Matheson et al., 1987). Using plane film radiography coupled with nuclear medicine imaging, the pattern of SFs in athletes was found to differ from military recruits, with the most common SF type in athletes being the tibia (49.9%). This was followed by the tarsus (25.3%), metatarsus (8.8%), femur (7.2%), fibula (6.6%), pelvis (1.6%), sesamoids (0.9%) and lastly, the spine (0.6%). Bilateral stress fractures were observed in 16.6% of the cases. The femoral and tarsal SFs were more frequent in older athletes, whilst the fibula and tibial stress fractures were more common in the younger athletes. Matheson et al. (1987) radiographed 43.3% of these cases at presentation, and only 9.8% were abnormal. They recognised tibial periostitis ('tibial stress syndrome') as a separate entity to a tibial SF. This supported earlier studies such as Mubarak et al. (1982), who found that MTSS was due to periostitis rather than elevated compartment pressure or an overt tibial SF.

In the 1980's, a series of nuclear medicine studies on MTSS was published, leading to more specific diagnostic criteria for the injury. These criteria included recognising that MTSS had a characteristic scintigraphic appearance comprising of an elongated linear deposition of medium intensity radionucleotide along the posterior medial cortex of the tibia (Deutsch et al., 1997; Holder and Michael 1984; Matin, 1988). This differed from the more intense localised fusiform pattern typical of a SF (Matin, 1988), and highlighted that MTSS was a specific injury rather than just a precursor to a SF (Macleod, 1999). Despite these advances in nuclear medicine, MTSS is still used as a generic term for distal tibial pain. However, this perception is changing as more studies are published on the nature of this injury.

2. Diagnosis and classification

2.1 Definitions

From an aetiologic perspective, stress fractures can be divided into two types: insufficiency/ pathological fractures and fatigue fractures. An insufficiency fracture occurs when normal loads are applied to bone which has mineral or elastic resistance deficiencies, such as in the case of osteoporosis, where there is a loss of normal bone per unit volume of bone tissue. On the other hand, fatigue fractures develop in normal bone which is exposed to atypical and/or more frequent loading. It is believed that this load alteration causes muscular fatigue, which then results in an altered stress state in the bone, initiating a microfracture. The muscular fatigue assumption is supported by the fact that non-weight bearing bones such as the ribs, humerus, radius and ulna, e.g. in tennis players, can sustain SFs (Devas, 1975; Bruckner, 1998).

Less is known about the aetiology of MTSS. Initially believed to be only tibial periostitis, current evidence indicates it is, at least in many cases, also a bone injury (Johnell et al., 1982; Magnusson et al., 2001; Magnusson et al., 2003, Franklyn et al., 2008). Additionally, clinical data indicates that there is more than one type of MTSS, with specific aetiology and injury mechanisms for each type. This is discussed further later.

2.2 Diagnosis and classification of tibial SFs

All tibial SFs fall into several different categories depending on location and fracture type/injury mechanism (Table 1). This was first recognised by Devas (1975), who initially categorised tibial SFs into two different types, compression and distraction (i.e. tensile) SFs, based on X-ray findings and clinical studies. Tensile tibial SFs were then further subdivided into transverse, oblique and longitudinal fractures.

Tibial SF type	Incidence	Most common age	Most common location
Compression	50%	Children or elderly	Upper third in children or lower third in elderly
Transverse	2.5%	Young adults, particularly physically active ones	Shaft
Oblique	42.5%	Young adults, particularly athletes and military recruits	Lower third
Longitudinal	5%	Mature adults	Shaft

Table 1. Various classifications of tibial stress fractures as defined by their characteristics, incidence and location (reproduced from material contained in Devas, 1975).

Devas found that young active individuals are likely to sustain either transverse or oblique tibial SFs. The transverse SFs, which he observed in the tibial shaft, were prevalent in athletes who performed plyometric activities, e.g. ballet or jumping sports, where powerful plantar-flexion of the foot occurs. He attributed these SFs to the dynamic pull of the calf muscles (Soleus and Gastrocnemius) loading the tibia, causing the tibia to bend and become more convex anteriorly; subsequently producing high tensile stresses on the anterior tibial mid-shaft. On the other hand, Devas (1975) believed oblique SFs of the distal third of the tibia, found in athletes and military recruits, to be the result of bending forces which subject the injury site to excessive tension. He found that the propagation of the oblique crack generally begins at the posteromedial border of the tibia and occurs in conjunction with

mild inflammation around the bone and a small swelling on the medial tibia. He also noted that thickening of the cortex may occur around the SF site due to an associated attempted healing periostitis.

Much of Devas's original observations form the basis for the current knowledge regarding SFs. In the current clinical setting, focal tenderness (due to periosteal swelling and probable attempted bone repair with cortical thickening) is the key for diagnosis of a SF. In the tibia, the whole anteromedial surface of the bone is subcutaneous; hence, there may be overt anteromedial subcutaneous pitting oedema on firm digital palpation reflecting the underlying response of the periosteum to micro-fracture formation (Johnell et al., 1982; Matin, 1988). Additionally, there is sometimes linear tenderness along the whole anterior margin of the tibia to which the deep fascia of the leg has a strong attachment for the anterior compartment. The tenderness sometimes also extends to the posteromedial longitudinal tibial margin or border, where the deep fascia also attaches in addition to the origin of the FDL. Medial tenderness may also arise from the medial belly of the Soleus, which is attached to the proximal medial tibial and the deep fascia. Intense localised posteromedial margin tenderness in addition to significant oedema and early callus formation may be palpable in athletes who have a delayed presentation of several weeks.

In addition to the above observations, the clinical exam of a potential tibial SF patient should also include an examination of lower limb alignment and foot types. The presence of foot pronation, and in particular, weak inverter muscles (Hinterman et al., 1998; Oakes, 1993), has been shown to predispose the running athlete's tibia to excessive medial torque during weight bearing, thus potentially altering the stress distribution in the tibia (Figure 1).

2.3 Diagnosis and classification of MTSS

MTSS has been defined as a condition resulting in intermittent pain in the lower extremities, in particular, tibial periostitis associated with a specific scintigraphic appearance (Macleod, 1999; Macleod et al., 1999). Probably the most widely accepted definition of MTSS is a condition comprising of tibial anteromedial surface subcutaneous periostitis in the vicinity of the junction of the middle and distal thirds on the medial border (Holder and Michael, 1984; Macleod, 1999) due to an osteoblastic irritation and stimulation of the periosteum (Deutsch et al., 1997). Oakes proposed that this was also potentially associated with outer cortical bone microfractures (Oakes, 1988).

The research of Holder and Michael (1984), which was later verified by other studies (e.g. Matin, 1988) is used by most medical imaging specialists as the standard reference for the correct definition and diagnosis of MTSS, and was supported by the British Medical Journal (Macleod, 1999). Holder and Michael (1984) described shin splints to be exercise-induced pain and tenderness to palpation along the posterior medial border of the tibia. In a study of 10 patients (5 males and 5 females), they described the injury as:

1. Exercise-induced pain initially relieved by rest and exacerbated by exercise;
2. Usually subacute onset of pain, initially dull and aching;
3. Pain and palpable tenderness along the posterior medial border of the tibia in the distal region of the middle third; diffuse and less focal than with an acute SF;
4. Hindfoot abnormality with heel valgus and excess pronation of the forefoot.

Foot pronation has been consistently identified as a significant risk factor for MTSS (Matheson et al., 1987; Moen et al., 2009), although not in all affected patients. As discussed by Hinterman et al., (1998), individuals with overuse lower limb injuries typically have a 2-4 degrees greater pronation than those with no injuries, although 40-50% of runners with excessive pronation have no overuse injuries.

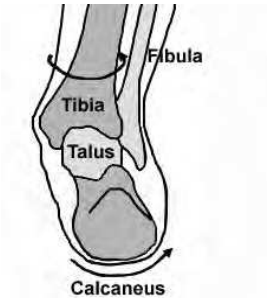

Fig. 1. Posterior view of the right foot. Notice the calcaneal eversion, which is associated with forefoot pronation and principally occurs at the subbtalar and midtarsal joints. This movement of the forefoot is associated with medial tibial rotation.

Using Cybex isometric leg muscle testing, Oakes demonstrated that MTSS patients had weaker inverter muscles than uninjured control subjects. He hypothesised that the weak inverters lead to excessive foot pronation due to eversion of the foot at the subtalar joint and subsequent medial rotation of the tibia (Oakes, 1993). Other authors have supported this injury mechanism (e.g. Hinterman et al., 1998), stating that the tibial rotation leads to injuries on the medial aspect of the tibia. This rotation would result in altered stress distribution in the bone, potentially increasing the tension on the medial border.
In-shoe orthotics can be beneficial for MTSS patients as they attempt to statically raise the medial arch of the foot, thereby preventing excess medial tibial torque or rotation by attempting to minimise forefoot pronation. However, orthotics have not consistently shown to be effective (Craig, 2008), which is not surprising, as not all MTSS patients show excessive pronation. This highlights the importance for the treating physician to identify the type of MTSS and therefore ensure correct management. It also emphasises that medial arch maintenance is mainly under the dynamic muscle control of the tibialis posterior and cannot be corrected optimally in the athlete by the use of simple static medial arch orthotics.

2.4 Nuclear medicine and imaging tibial SFs and MTSS

SFs and MTSS have been classified by both nuclear medicine and by clinical findings. Matin (1988) used a five-stage classification, where the initial two stages are defined as MTSS and the final three stages are a SF (Table 2). It is important to note that although there is bone involvement in Stages I and II, MTSS is not considered to be a precursor to a tibial SF in this system. Accuracies of 75% or greater have been found for scintigraphy (Lieberman and Hemingway, 1980; Allen, 1996; Gaeta et al., 2005), although false positives do occur, thus highlighting the need for a clinical diagnosis in conjunction with nuclear imaging.

Stage of injury	Percentage of bone cross-section involved	Description
I	0-20%	Minimal periosteal reaction
II	20-40%	Moderate periosteal reaction
III	40-60%	Early stress fracture
IV	60-80%	True stress fracture
V	80-100%	Full thickness stress fracture

Table 2. Classification of a stress fracture and MTSS using nuclear medicine techniques (Matin, 1988).

Figure 2 demonstrates a TPBS image from a patient showing three features of interest: a SF of the fourth metatarsal, MTSS, and bone bruising (i.e. increased bone oedema due to greater water content as a result of bone micro-damage or micro-fracture) of the calcaneus, where the patient had pronounced clinical symptoms of the SF and MTSS. The focal uptake at the location of the SF and the diffuse uptake at the MTSS site are readily apparent. Although the uptake at the calcaneus is considerable, the patient only experienced periodic mild tenderness at this site.

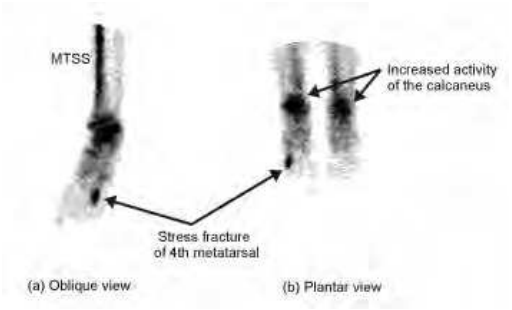

Fig. 2. Patient scintigraph illustrating three conditions (1) MTSS, (2) stress fracture of the fourth metatarsal and (3) mildly symptomatic bone bruising of the calcaneus.

Based on extensive clinical observations, Oakes (1988) proposed there were two distinct types of MTSS, where they can occur in isolation, or in conjunction to form a third type:

1. Tenderness on the distal tibia which when overt, can result in subcutaneous oedema or periostitis on the anteromedial surface of the distal third of the tibia. He proposed this was a result of tibial flexion from contraction of the two heads of the Gastrocnemius and the Soleus muscle causing tibial bending moments during the push-off stage of running. This type of MTSS subsequently results in microtrauma (microcracks between Haversian systems or osteons) to the underlying cortical superficial cortical bone and potentially a tibial SF at the thinnest tibial profile (i.e. the lowest tibial total cortical cross-sectional area).
2. Posteromedial linear pain and tenderness, predominantly due to the strong deep fascia of the posterior calf muscle compartment attaching to the linear posteromedial border of the tibia, but also from the tibial origin of the FDL. The cause of this longitudinal pain is tension in the tibial attachment of the deep fascia in conjunction with the origins of the powerful action of the Gastrocnemius and Soleus muscles proximally.
3. A combination of the above seen in serious long and middle distance runners.

As discussed further later, the authors have observed that some cases of MTSS may lead to a tibial SF, while most cases will not.

In two complementary studies, the first involving ten athletic patients diagnosed with MTSS by a TPBS and the second where anatomical dissection was performed on the lower limb of fourteen cadavers; Holder and Michael (Holder and Michael, 1984; Michael and Holder, 1985) found both pain and abnormal tracer accumulation were present at the origin of the Soleus. This study supports the Soleus involvement in MTSS, although Holder and Michael believed this to be the only cause of the injury. They further postulated the periosteal irritation stimulated afferent pain nerve fibres and activated an osteoblastic response. This

can be observed clinically from distal tibial tenderness, and possibly oedema, over the subcutaneous surface of the tibia.

Matin (1988) attributed the bony changes in MTSS to the insertion of Sharpey's fibres. He suggested that abnormal stress on the Sharpey's fibres not only led to the periosteal reaction with an elongated radionucleotide pattern, but also resulted in increased bone remodelling. He further postulated that Sharpey's fibres, which originate from muscles and other fasciae, increase stress on the superficial region of the tibial cortex as the fibres extend through the periosteum into the mineralised bone matrix of the outer circumferential cortical lamellae.

Radiographic findings are likely to be absent in the case of MTSS (Matin, 1988; Deutsch et al., 1997) with some clinicians stating they will always be radiographically occult (Matin, 1988). As opposed to a TPBS where bone turnover/remodelling is positively imaged due to increased bone vascularity, radiography relies on bone density changes for visualisation of pathology. Therefore, a tibial SF will be several millimetres in length and thus provides sufficient density contrast for radiographic visualisation, but only with optimal imaging conditions required for 'early' SF identification. On the other hand, even when MTSS is associated with microfractures, it is likely that they will never be large enough to be visible on plane radiography.

More recently, CT and MRI have been used to classify both SFs (Bergman and Fredericson, 1999; Deutsch et al., 1997; Feydy et al., 1998; Matin, 1988; Reeder et. al., 1996) and MTSS (Matin, 1988; Deutsch et al., 1997; Beck, 1998; Bergman and Fredericson, 1999). The main advantage of CT is that it provides a good delineation of fine bone detail (Feydy et al., 1998) so both SFs and partial SFs can be more easily observed. Unlike X-ray scans, cross-sectional images are possible using CT as this imaging modality uses a large number of X-ray beams to reconstruct a slice of bone. However, like radiography, the fracture line cannot always be observed readily as visualisation of the fracture line depends critically on the views imaged.

Fredericson et al. (1995) first reported MRI to be significantly better than other techniques for the early diagnosis of tibial and other SFs. They performed a study on 14 runners with 18 symptomatic legs within 10 days of referral for radiology. A TPBS and an MRI exam were also performed and the three imaging modes were compared. From this small number of patients, they concluded that MRI was more sensitive and more accurate in its correlation with the clinical symptoms and signs than a TPBS.

The main limitation with MRI is the lack of sensitivity in the assessment of cortical bone. Although Fredericson et al. (1995) found MRI to be superior to other imaging techniques in their 1995 study, SFs are often present without the fissure being observable, and the absence of a fracture line is considered a negative diagnosis (Bergman and Fredericson, 1999). The main advantages with MRI are that the patient is not subjected to ionising radiation and soft tissue changes can be easily depicted (Deutsch et al., 1997) as well as 'bone-bruising'. However, like TPBS, positive MRI images are possible in the absence of clinical symptoms (Kiuru et al., 2005). Additionally, oedema visible on MRI can be due to other conditions e.g. osteomyelitis.

In the original MRI study by Fredericson et al. (1995), the images were graded into four groups (Table 3). This was further modified by Pomeranz (2011) by dividing Group 4 into two separate categories: Group 4a (partial cortical fracture) and Group 4b (complete cortical fracture.

Grade	Clinical Exam	MRI
1	Periosteal tenderness at the distal 1/3-1/2 of the anteromedial tibial surface. Requires **firm** palpation with thumb.	Periosteal oedema: mild to moderate on T2 weighted images. Marrow normal on T1 and T2-weighted images.
2	Tenderness as above. Requires **less firm** palpation with thumb & may have linear tenderness along the posteromedial tibial border.	Periosteal oedema: moderate to severe on T2 weighted images. Marrow oedema on STIR or T2-weighted images. T1 normal.
3	Tenderness as above. Requires **less firm** palpation and may have linear tenderness as above. May have subcutaneous anteromedial tibial oedema.	Periosteal oedema: moderate to severe on T2 weighted images. Marrow oedema on T1 & STIR-T2-weighted images.
4	Tenderness as above. Requires **less firm** palpation and may have linear tenderness as above. A discrete region of maximal tenderness/thickening (early callus formation) over the fracture site will be palpable. Obvious tibial subcutaneous oedema is usually present.	Periosteal oedema: moderate to severe on T2 weighted images. Marrow oedema on T1-STIR or T2-weighted FS images. Fracture line clearly visible as low fuzzy incomplete (4a) or complete (4b) line. May see oedema in proximal tibial origins of Tibialis Posterior, FDL and Soleus.

Table 3. Modified by Oakes from Fredericson et al. (1995) and Pomeranz (2011).

As shown by the categories in Table 3, MRI can be used for the early detection of periosteal oedema, which is a reflection of microcracks between and through the Haversian systems in cortical bone in the region subjected to excessive repetitive loading. Fredericson et al. (1995) noted that the average marrow oedema extended over 5.2 cm and penetrated the outer one third of the marrow diameter. This indicated endosteal oedema as well as periosteal oedema.

For the managing physician, MRI can also be used to determine rehabilitation protocols and return to activity time without fear of reoccurance of a further episode of disabling bone pain due to inappropriate premature activity by often over-enthusiastic athletes, especially high-earning athletes. The soft-tissue sensitivity in detecting oedema due to microfractures can be used to determine the level of injury, as the extent of the bone oedema is a direct reflection of the strain damage to the tibial cortical bone. As a guide, Grade 1 injuries may be able to return to activity within 4-6 weeks if the aetiology of their stress reaction or SF is rectified. The most severe injuries, Grade 4a and 4b, can take as long or even longer to heal than the more conventional tibial fractures depending on the cause. For example, if the patient has poor biomechanical lower limb alignment coupled with excessive pronation due to unilateral weak inverter muscle groups (i.e. Tibialis Anterior and Posterior), 12 weeks or more might be needed for full recovery as the muscles will need strengthening prior to full safe return to activity or competition. Shoe orthotics can be beneficial in partial prevention of excess forefoot pronation and therefore excess medial tibial torque or rotation; however, the weak inverter musculature must also be addressed as outlined above.

2.5 Differential diagnosis

As a positive scintigraph is a non-specific finding (i.e. fracture lines cannot be observed) and MRI has a number of limitations, a differential diagnosis should be considered:

1. Insufficiency fracture: described earlier, in this case the bone has normal loading or low-level repetitive loading but is weak due to loss of bone mass, mineral or elastic resistance. The prime aetiological cause is osteoporosis (i.e. a loss of normal bone volume), and it should be considered as a potential diagnosis, particularly in older female patients.
2. Anterior tibial compartment syndrome: this condition is not common; however, it should be suspected when the pain is localised to the proximal anterior muscle compartment following intense exercise. This injury is aggravated by impact exercise and relieved when the exercise ceases. A measurement of compartment pressure during leg exercise can be used to confirm this diagnosis.
3. Popliteal artery entrapment syndrome (rare): muscle ischaemia from stenosis of the popliteal artery, which may pass through the medial head of Gastrocnemius. As with anterior tibial compartment syndrome, this condition also worsens during exercise. Dopler flow studies of the popliteal artery may help confirm the diagnosis prior to the use of femoral artery angiography.
4. Tibial tumours: Both benign and malignant tumours are possible. These tumours can usually be identified by X-ray; therefore, in order to exclude bone tumour, X-ray should be performed prior to scintigraphy.
5. Bone infection/osteomyelitis: This should be suspected in young athletes, especially if it involves the proximal tibial epiphysis. MRI can be used to establish the diagnosis and its exact location and extent.

Clinical symptoms and patient history should be used in conjunction with medical imaging for correct diagnosis of these injuries.

3. Mechanics of the tibia and bone failure

Numerous risk factors have been associated with tibial SFs (e.g. Burr, 1997; Bennell et al., 1999; Brukner et al., 2000), and to a lesser extent, MTSS (e.g. Moen et al., 2009). As these have been extensively published in the literature, they are not discussed here. The following sections focus on the biomechanical cortical bone parameters relating to tibial stress injuries, bone failure under various loading conditions, and *in-vivo* tests on humans and animals.

3.1 Tibial cortical bone parameters

The preponderance of the literature on cortical bone parameters originates from research on Israeli and US military recruits, with some studies having been performed on athletes. Most of this work has focused on male rather than female populations; hence the significant bone geometry related factors in females are less well understood. In male military cohorts, parameters which have been associated with tibial SFs include a narrow mediolateral (ML) width at the narrowest tibial cross-section (Giladi et al., 1991; Giladi et al., 1987) and small diaphyseal dimensions relative to body weight (Beck et al., 1996). In both male military cohorts and male athletes, a low cortical bone cross-sectional area (Beck et al., 1996; Crossley et al., 1999; Franklyn et al., 2008), small second moments of area (Milgrom et al., 1989;

Franklyn et al., 2008) and a small section modulus (Beck et al., 1996; Franklyn et al., 2008) have been associated with increased tibial SF risk.

A major limitation with many of these studies is that only basic cross-sectional dimensions have been measured from the images, and when they are then used to calculate parameters such as the cross-sectional area, introduce significant error for an irregularly shaped object such as a tibial section. Additionally, in some instances, the correct mechanical parameter has not been measured. This is most obvious in the case of the section modulus, which is often described as the cross-sectional area divided by the half-width of the cross-section. Lastly, using a formula to approximate tibial cross-sections to a shape such as en ellipse introduces a similar error; this error is compounded by the fact that tibial cross-sections can differ considerably from subject to subject (Figure 3).

Using a sample of 130 tibial CT cross-sections derived from a population of athletic and sedentary subjects, both male and female, Franklyn (2004) demonstrated that for irregularly-shaped sections such as tibial cross-sections, use of a formula involves a highly statistically significant degree of error. In this study, a series of cross-sectional parameters were calculated using various formulae from the literature and compared to the values of the parameter computed numerically by a validated code. For example, Milgrom et al. (1989) used a formula to calculate second moments of area about two planes; these moments are cross-sectional properties related to bending strength. Franklyn (2004) found that the formula for the second moment of area about the ML axis resulted in an overestimation by 1.09 (p = 3.15E-19), while the formula for the second moment of area about the anteroposterior (AP) axis resulted in an underestimation of 1.23 (p = 9.87E-32) when compared to the values of parameters computed numerically, with the error being larger for bigger sections. Other formulae in the literature tested resulted in similar levels of error. As these errors depend on both the parameter and the formula used, there was no one mathematical transformation which could be used to correct these values for all cross-sectional parameters.

(a) (b) (c)

Fig. 3. Normal variation observed in cross-sections of the human tibia at the same cross-sectional level (a) elliptical, (b) triangular and (c) a hybrid of both shapes.

If basic methods are used to calculate tibial parameters, the error introduced is generally not a problem if, for example, cross-sectional areas are compared between groups of subjects within a specific study. However, problems arise when specific values need to be compared (i.e. values compared between two different studies) or the magnitude of the parameter is needed to better understand the injury mechanisms. It introduces doubt as to whether, for instance, an accurate cross-sectional area or section modulus is actually being calculated. Conclusions drawn from a particular study should be considered in light of the error implicit in the method used.

A further concern is the type of imaging modality used to scan the bone. Some imaging types, e.g. DEXA (Dual Energy X-ray Absorptiometry), have poor cortical bone resolution, but the technique has previously been used in a number of military or marine studies to calculate bone dimensions (e.g. Beck et al., 1996). DEXA was designed for bone mineral density (BMD) computation; hence dimensions measured from these images are likely to involve considerable error. DEXA can be used to directly compute geometric properties, but this technique also has error: a series of scan lines is performed through the bone at a specific cross-section, producing a profile of bone content in that section. For each line, the bone content is summed over the region; hence the distribution of bone cannot be determined (see Figure 4).

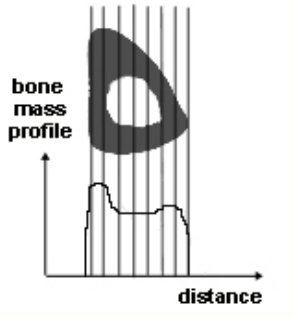

Fig. 4. DEXA uses a series of scan lines to produce a profile of bone content.

In order to overcome some of the abovementioned limitations, the current authors examined tibial geometry in athletes using CT imaging in conjunction with numerical methods to compute the true mechanical parameters (Franklyn et al., 2008). Bone geometry of MTSS patients was also analysed in order to make comparisons with the tibial SF patients. These results are discussed in detail later.

4. Bone alterations due to injury

It is now evident that MTSS involves alterations to at least the cortical bone of the tibia (Johnell et al., 1982; Magnusson et al., 2001; Magnusson et al., 2003; Franklyn et al., 2008; Murrihy, 2009), although whether the trabecular bone is also involved is unknown. This was first evident in the study by Johnell et al. (1982), where cortical bone and soft tissue biopsies were obtained from control (non-injured) patients and patients with chronic MTSS. The MTSS patients, who had one month's rest prior to the biopsies, were diagnosed by patient history and clinical examination in conjunction with radiography to exclude those with a stress fracture (scintigraphy was not widely available at the time).

Johnell et al. (1982) found there were no bone or inflammatory changes in any of the control subjects, while patients with MTSS had alterations such as increased osteoblastic activity at the medial surface of the tibia (where the biopsies were performed) and vascular ingrowth in conjunction with the soft tissue inflammatory changes. These findings were consistent with nuclear medicine studies at the time which demonstrated that MTSS patients had positive scintigraphy, indicating changes in bone metabolism (Matin, 1988). However, not all bone biopsies demonstrated changes: from a total of 35 cortical bone biopsies, 22 specimens had at least one sign of bony changes, but 13 did not. Consequently, they concluded that microfractures were a cause of MTSS, but not necessarily in all cases.

In other research performed since this time, BMD and cortical bone geometry in MTSS and tibial SF patients have been examined. These studies provide corroboration that MTSS involves alterations to the cortical bone, at least in many cases of MTSS, but not necessarily identical changes to those seen in tibial SF patients.

4.1 Bone mineral density in tibial SF and MTSS patients

In previous studies, BMD has been found to not differ between tibial SF patients and exercising controls subjects in most cases. This has been shown in male military recruits (Giladi et al., 1991; Milgrom et al., 1989), male marine recruits (Beck et al., 2000), male athletes (Crossley et al., 1999) and female athletes (Bennell et al, 1999). However, BMD differences have been found between female marine recruits with and without a tibial SF, (Beck et al., 2000). Nevertheless there is strong evidence to suggest the differences found in female subjects are due to hormonal effects such as menstrual irregularities or use of oral contraceptives (Myburg et al., 1990).

There are only a few studies where BMD has been analysed in MTSS patients. Magnusson et al. (2001) measured BMD in 18 male athletes sustaining clinically and scintigraphy-diagnosed MTSS, 18 competitive athletic controls (exercising 3-15 hours/week) and 16 control subjects who exercised at the non-professional level (0 to 5 hours per week). The authors demonstrated that at the injury site, male athletes with chronic MTSS had localised decreased BMD, and this reduction was bilateral even when the injury was unilateral. Additionally, they found that BMD normalises after recovery from the injury (Magnusson et al., 2003). At other sites of the tibia, the MTSS patients had higher BMD than the control group but lower BMD than the athletic control group.

The Magnusson study was limited by several factors. Firstly, subjects in the control group performed some exercise and were comprised of both manual and non-manual workers; hence they were not a true sedentary control group. A second limitation was the large range in number of hours of exercise per week, and both control groups contained a combination of subjects with manual and sedentary occupations; hence the groups were not uniform with regards to exposure. It is known that BMD increases due to impact exercise (e.g. Etherington et al., 1996), but these results show that BMD is reduced at the injury site in MTSS patients. It is likely that the reduced BMD is not inherent but develops in conjunction with the symptoms.

Differences in BMD at the injury site have not only been found between (male) MTSS and non-injured control subjects, but also between (female) MTSS and SF patients at the injury site. The authors of this chapter measured BMD from DEXA scans on 5 SF patients (n = 10 scans) and 10 MTSS patients (n = 20 scans), all of whom performed impact exercise a minimum of 3 to 4 times per week and had a minimum training history of 2 years (study criteria was described in Franklyn et al., 2008). All scans were performed at the same medical clinic with a Norland XR-36 scanner (Norland Medical Systems Inc.), and each subject was scanned in three regions 2.1 cm in length. Although only a small number of subjects, it was found that MTSS patients had significantly lower localised BMD (1.46 g/cm^2) than tibial SF patients (1.63 g/cm^2) at the injury site, but not at sites in the proximal and distal tibia (Table 4 and Figure 5).

Hence, from these studies, it can be concluded that male MTSS patients have localised low BMD at the injury site compared to non-injured exercising controls, and the BMD returns to normal after the symptoms have resolved. Also, at the injury site, female MTSS patients

have lower BMD than female tibial SF patients. As subjects with a tibial SF have been shown to have normal BMD, MTSS patients clearly have reduced BMD at the injury site.

BMD (g/cm²)	SF (n = 10)	MTSS (n = 20)	Significance
Proximal	1.2757	1.2139	0.136
33% level (injury site)	1.6354	1.4598	0.013[a]
Distal	0.9439	0.9023	0.403

[a] Statistically significant $p < 0.05$

Table 4. Statistical analysis of BMD in female tibial SF and MTSS patients (Oakes and Franklyn, 1998).

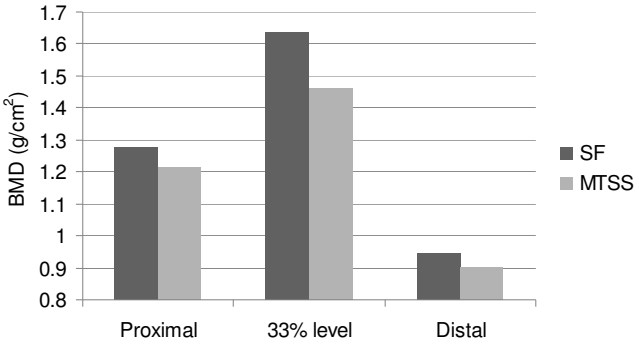

Fig. 5. BMD in female tibial SF and MTSS patients at three tibial sites (Murrihy, 2009).

4.2 Bone geometry in tibial SFs and MTSS patients

It has been shown that tibial SF and MTSS athletes have lower values of some cortical bone geometrical properties when compared to uninjured aerobic control subjects (Franklyn et al., 2008). These findings may imply that MTSS and tibial SFs are a continuum of injury, with MTSS being the precursory state of a tibial SF, and some researchers and clinicians believe this is the case. However, it is the belief of the authors that tibial SFs and MTSS are two separate injuries with some common aetiology and mechanisms. This is probably most strongly evidenced by the fact that not all cases of MTSS lead to a tibial SF. If they were one injury on a continuum, all MTSS patients would eventually sustain a tibial SF with continued exposure to the same impact forces, yet this does not occur. Additionally, tibial SFs are a localised injury whereas MTSS is diffuse. Lastly, it has never been demonstrated that MTSS and tibial SFs fall on a continuum of injury.

The results of the study by Franklyn et al. (2008) showed that the tibiae of male athletes with a tibial SF or MTSS have less cortical bone cross-sectional area (A) than uninjured athletes, resulting in lower values of some other mechanical parameters such as the polar moment of area (J), the maximum and minimum second moments of area (I_{max} and I_{min} respectively) and the section modulus (Z). These mechanical parameters determine the strength of a beam, such as bone, under different types of loading (see Table 5).

Parameter	Symbol	Type of loading it represents
Cross-sectional area	A	Axial loading
Polar moment of area	J	Torsion
Maximum second moment of area	I_{max}	Maximum bending rigidity
Minimum second moment of area	I_{min}	Minimum bending rigidity
Section modulus	Z	Pure bending

Table 5. Geometric parameters with engineering denotations and meanings.

Thus, injured males are less adapted to axial loading, torsion, maximum and minimum bending rigidity and pure bending (a state where there are no axial, shear or torsional forces). The lower values of these parameters in the injured males were due to less cortical bone in the medullary region (primarily in the AP medullary region) rather than from differences in external tibial widths. These results suggest that in males, cortical bone loss occurs from the medullary region prior to, or as a result of, these injuries.
In this study discussed above (Franklyn et al., 2008), it was found that females with a tibial SF or MTSS had smaller section moduli than uninjured females, but as other cross-sectional parameters did not differ, it was not due to less cortical bone area. Instead, injured females are less adapted to pure bending, but the results show that this occurs by a redistribution of the cortical bone about the centroid (centre of mass) so that bending forces are less tolerated by the tibia. Figure 6 shows typical tibial cross-sections from injured male and female subjects compared to uninjured control subjects.

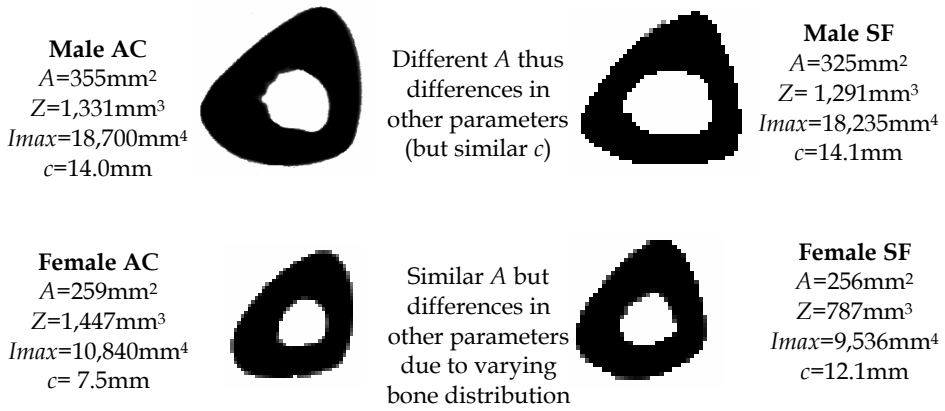

Fig. 6. Examples of typical male and female cross-sections from the mid-distal junction of the tibia showing the characteristic differences in geometry AC = aerobic control.

In mechanics, Z is a measure of a specific type of bending (pure bending). It depends on both the amount of material (cortical bone area) as well as its distribution, and is defined as:

$$Z = I_{max} / c$$

where c is the distance from the centre of mass to the outmost fibre of the cross-section (on the anterior border or tensile side). This outermost point is important as it is where the stress is highest under bending and therefore where failure is predicted to occur. If Z is larger, the structure can support a greater load under bending. This can occur due to a higher value of $I_{\max}$ (due to more bone) or lower value of c (the bone is closer to the centre of mass). In the study by Franklyn et al. (2008), the lower values of Z in the injured males were predominately due to lower values of $I_{\max}$, whereas in the females, it was from higher values of c, consistent with the fact that injured males had less cortical bone area, but injured females had a different bone distribution less favourable for bending forces.

Alterations in bone shape can occur as osteoblasts in the periosteum create compact bone around the external bone surface while osteoclasts in the endosteum remove bone on the internal medullary cavity. Two mechanisms in which bone can adapt to mechanical loading have been proposed in the literature: (1) periosteal expansion (reshaping) and (2) redistribution of bone mineral from trabecular to cortical components (Adami et al., 1999), although the validity of the former has been disputed (Jarvinen et al., 1999). Although not a longitudinal study, the results from Franklyn et al. (2008) suggest in injured males, cortical bone could be lost to trabecular bone either before or during the injury, whereas in females, cortical reshaping may occur in conjunction with the injury. It is difficult to hypothesise further on these mechanisms; however, it is apparent that longitudinal studies examining cortical bone alterations prior to and during injury progression are needed.

4.3 Conclusions on bone characteristics and tibial stress injuries

These more recent studies on cortical bone and tibial stress injuries clearly demonstrate MTSS is, in many cases, an injury involving microfractures in the cortical bone in addition to low BMD, and cortical bone geometry which is less adapted to some mechanical modes of failure such as bending. Matin (1988) suggested that in MTSS patients, the deposition of radionuclide around the injured region was due to the response of the periostium to the developing abnormality in the cortical bone. However, he also proposed that abnormal stress on the Sharpey's fibres from the tissues increases stress on the outer circumferential lamellae of cortical bone, implying the tissue response may occur first. It seems unclear as to whether the cortical bone alterations occur before the inflammatory response of the tissue. In cases of MTSS which do not involve microfractures (Oakes Type II), the periosteal response would have to be due to, or a result of, a factor other than bone microfractures.

Most previous studies have shown BMD does not differ between uninjured control subjects and tibial SF patients. However, patients sustaining MTSS have reduced BMD at the site of the injury, and lower BMD than tibial SF patients at the injury site (consistent with tibial SF patients having normal BMD). This provides further evidence that MTSS and tibial SFs are two distinct injuries. Both MTSS and tibial SF patients have cortical bone geometry which is less adapted to dynamic mechanical loads imparted by the musculature. In males, there is less cortical bone area, which results in a decreased ability to tolerate different loading conditions such as axial load, torsion and various bending loads. In injured females, cortical bone area is not affected but there is decreased ability to tolerate pure bending. More work is needed in this area as there is a lack of longitudinal studies to provide more information on cortical bone changes and development of both MTSS and tibial SFs.

5. Cortical bone failure and fatigue

5.1 Bone as an engineering material

Bone is composed of two types of osseous tissue: cortical bone and trabecular bone, where the main distinction between the two is the density and the degree of porosity (Carter and Hayes, 1977). Compared to trabecular bone, cortical bone is quite stiff. Hence it is able to endure greater stress (force per unit area) but less strain (deformation) before failure. On the other hand, trabecular bone can withstand greater deformation before failure, and as a result, has a large capacity for energy storage (Keaveny and Hayes, 1993). In a long bone, a SF occurs in cortical bone as this tissue type is subjected to higher stresses, particularly around the external or superficial surface. Under the right conditions, this eventually leads to cracks (failure).

In engineering, materials can be classified as ductile i.e. have the ability to deform, such as in a soft metal, or brittle i.e. breaks with little deformation, for example, glass. As shown in Figure 7, each type of material has a typical fracture type: a ductile material has a characteristic 'cup and cone' fracture shape, while a brittle material has little yield and then fractures at an oblique angle.

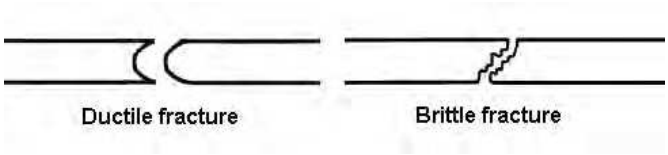

Fig. 7. Typical fractures of a ductile material and a brittle material.

Cortical bone does not act like a typical engineering material; it fractures in an oblique plane like a brittle material, but it also displays ductile behaviour. In addition, cortical bone demonstrates anisotropic properties i.e. the properties vary in different directions. For example, when subjected to tension transversely, cortical bone displays brittle behaviour, while if it is subjected to tension longitudinally, it appears to be ductile. Therefore, the type of behaviour depends on the loading conditions and the bone microstructure.

Although mechanical failure theories can be used to understand bone behaviour, like most biological materials, cortical bone exhibits unique characteristics that are different to standard engineering materials, and as such there are no mechanical theories of failure. Additionally, as the skeleton is subjected to complex loading conditions, it can be difficult to predict when and where failure will occur.

5.2 The biological basis for bone failure

According to the clinical evidence presented by Burr (1997), the most likely biological explanation for the initiation and/or propagation of a stress fracture is adaptive bone remodelling. This strain-mediated process is outlined below:

1. The stress is applied to the bone;
2. Osteoclastic resorption, which occurs as a part of the normal bone remodelling process, creates a reabsorption space that increases the bone porosity, reduces bone mass and exponentially decreases bone strength and stiffness. Osteoclasts reabsorb areas of bone, thereby forming hollow channels;
3. There is less bone, hence the strains on remaining bone increase;
4. The increased stress on the bone causes a new remodelling cycle to commence.

At Stage IV, there are two possibilities (Matin, 1988). First, the bone is allowed to rest so that osteoblastic bone regeneration can occur, with more dense bone replacing the lost bone so that the stress site is strengthened. Alternatively, at Stage IV, if there is no rest, the bone becomes weaker after the period of strain and resorption. This leads to the individual bone trabeculae eventually collapsing, subsequently causing microfractures in the bone which then may eventually lead to an overt SF.

In the initial osteoblastic stage, immature bone is laid down and eventually matures over time. Johnson (1963) found that it takes 90 days to fill a reabsorption space with mature bone. According to Reeder et al. (1996), the cross-sectional area decreases during this time period, which consequently subjects the bone to a potentially higher local stress. As a result, it is probable that the weakened state of the bone during the 90-day reparative period is when the bone is most susceptible to an injury such as a SF. Robling et al. (2001) demonstrated the importance of recovery time in restoring mechanosensitivity (i.e. the capability of sensing and responding to mechanical forces) to bone cells. Loading rat bones *in situ* using a four-point bending apparatus, tissue histology was examined when the rats were killed at various days after the loading commenced. They found that approximately 8 hours of recovery was required in the rat tibia to restore full mechanosensitivity to the cells after the cells had been desensitised from the application of repetitive mechanical loads for an extended period.

The theory outlined above is supported by other research. For example, Li et al. (1985) conducted an experiment where 20 rabbits were induced to run and jump by subjecting them to an electrical impulse at various intervals for a period of 60 days. Using radiographic and histological analyses on this group and a control (non-exercising) group, the authors found that osteoclastic reabsorption occurred before the presence of any cracks in the cortical bone. Furthermore, only some rabbits developed cracks in the bone after the period of exercise, suggesting that in the majority of cases, the rabbit tibiae adapted to changes in the applied stress.

Martin et al. (1997) performed a study *ex vivo* on deceased racehorses using the common SF site of the third metacarpal. Using the contralateral bone as a control, they found that if three-point cyclic bending loads were applied to the right bone for as many cycles as a racehorse would experience during its training and racing lifetime, then the elastic modulus and yield strength were not affected. This suggested that equine bone was not weakened by this loading *ex vivo*, and that SFs are not simply fatigue failure, but a result of the inability of the repair mechanism (remodelling) to sustain a level of equilibrium with the damage produced by fatigue. This implies that another mechanism, such as adaptive bone remodelling, is involved in SF development *in vivo*.

5.3 Fatigue failure in cortical bone specimens

In mechanics, ductile materials generally fail from a tensile load rather than a compressive load. Similarly, bone can withstand greater loads under compression than under tension; therefore, bone generally fails due to tensile stress. Hence, under *static* bending of a symmetrical specimen, bone will yield from tensile stresses rather than compressive stresses as bone is weaker in tension (Evans, 1957).

Currey and Brear (1974) tested cortical bone specimens under (non-fatigue) loading at different strain rates; some of the samples were subjected to compressive loads, while others were subjected to different types of bending loads. They demonstrated that cortical bone can fail under both tension and compression, but the modes of failure differed. When cortical bone

is compressed longitudinally, shear lines develop at an angle of approximately 30 degrees with respect to the load line (rather than 45 degrees, as in a normal isotropic material) due to the anisotropy of bone). These shear lines are believed to be due to buckling of the bone lamellae. However, cortical bone under tensile stress does not develop shear lines (Currey and Brear, 1974), but instead tensile lines that show yield (Caler and Carter, 1989).

In mechanics, failure of a structure is often from a time-varying load rather than from a constant load; this type of failure is known as *fatigue failure*. In this case, failure of the structure will occur at a *lower* stress level than would otherwise be the case for a standard static load. Most materials under cyclic loading fail as a result of a crack which develops from *tensile* stress. This crack then leads to stress concentrations, which subsequently initiate unstable crack propagation in the material. Alternatively, cracks will tend to form at any pre-exiting stress concentrator (imperfection) in the material, leading to crack propagation.

This mechanism differs in cortical bone, which fails under both tension and compression, i.e. there are two separate fracture regions, although the tensile failure occurs first. This was demonstrated by Carter and Hayes (1977) and Carter et al. (1981) who found that under cyclic loading, *tensile* loads result in *tensile stresses* which cause failure at osteon cement lines so that the osteons debond from the surrounding interstitial bone. On the other hand, *compressive* loads result in the formation of oblique microcracks along the planes of high *shear stress* before the crack from the tensile load had extended throughout the entire specimen. The shear stress tends to initiate near blood channels (Currey and Brear, 1974), which can act as stress concentrators in bone and therefore initiate crack propagation.

The studies described above do not take into account the remodelling process, which is a critical difference between bone and standard engineering materials. This was examined by Pattin et al. (1996), who studied energy dissipation under fatigue failure. Using human femoral cortical bone specimens, they performed fatigue to fracture testing under different types of cyclic loading. They found that above specific strain thresholds, tensile-loaded fatigue specimens dissipate 6-7 times more energy than compressive loaded fatigue specimens when subjected to the same loading magnitude. These results suggest that bone remodelling may be favoured under tensile load, since more energy is available to activate a remodelling response. This is consistent with other studies showing that SFs occur due to tensile failure.

Failure of cortical bone specimens are also affected by other factors such as the frequency of loading (Caler and Carter, 1989) and the strain range (amplitude). However, the mean strain and maximum strain do not affect the fatigue life (Caler and Carter, 1989; Carter et al., 1981). Compared to most engineering materials, cortical bone has a poor fatigue resistance, but a longer fatigue life than trabecular bone (Carter and Hayes, 1977).

In summary, the fact that cortical bone fails in tension under cyclic loading is not surprising, as according to mechanical engineering theory, tensile loads cause fatigue crack propagation in ductile materials (although bone is neither ductile or brittle). However, it is apparent that bone differs from most mechanical structures in that it demonstrates failure from both tensile and compressive components of a cyclic load, although the tension load will cause failure before the compressive component. Under each of these load types, the mode of failure is different. The bone specimen tests described above can describe the behaviour of cortical bone under load; however, they do not factor bone remodelling, which will influence the number of cycles to failure. Additionally, the applied loading to bone is likely to reduce when a crack initiates as continued loading becomes painful for the individual, consequently leading to a reduction in physical activity.

5.4 Fatigue failure in cortical bone *in-vivo*

Using patient X-rays and clinical examinations, Devas (1975) was probably the first to hypothesise that the tibial SFs which occur in athletes and military recruits, i.e. oblique SFs at the junction of the mid and distal thirds of the tibia, are the result of bending forces subjecting the site to excessive tension. This is a similar mechanism to the Oakes Type I MTSS (Oakes, 1988) mentioned earlier, where he proposed that the gastrocnemius and soleus muscles caused bending moments in the tibia, subsequently resulting in injury at the smallest tibial cross-sectional profile.

Lanyon et al. (1975) bonded a strain gauge rosette to the anteromedial aspect of the tibial midshaft of a 35-year-old human male, measuring the principal strains in the bone (i.e. the maximum and minimum strains, which are the most tensile and the most compressive strains respectively). When the subject was running with shoes, the maximum tensile strain, which occurred during the push-off phase, was greater than the maximal compressive strain, and the tensile strain was in-line with the long axis of the bone. This finding suggests that tibial SFs which occur at the midshaft are due to tensile forces causing tensile stress, and is consistent with the cortical bone specimen experiments mentioned earlier by Carter and Hayes (1977) and Carter et al. (1981), who found that tensile loads result in tensile stresses, which then cause failure at osteon cement lines.

The principal strains from the Lanyon et al. (1975) study were converted into principal stresses by Carter (1978). Carter found that the longitudinal stress on the anteromedial aspect of the tibial midshaft during running was primarily compressive at the heel-strike stage, while during the push-off stage, the longitudinal stress was highly tensile. On the other hand, the transverse and shear stresses were found to be small throughout the entire running gait. This suggests that if bone does fail under tensile stress when subjected to cyclic loading, then loads from the push-off stage are a significant contributor to the development of microcracks which lead to a tibial SF.

Milgrom et al. (1999) attached strain gauges directly to the mid-diaphysis of the medial cortex of the tibia in five male and three female subjects and measured strain magnitude and strain rates. They found that, in general, both strain magnitude and strain rate increased due to muscular fatigue, but values were not presented for different stages of the gait cycle. Similarly, Burr et al. (1996) conducted a study where strain gauges were attached to the medial tibial cortex at both the tibial midshaft and 2 cm distal to the first gauge in two male subjects, although data was only presented for the midshaft. Strains and strain rates where shown to be higher when running than walking, but the phase of the gait cycle producing these strains was again not presented.

5.5 Fatigue failure in cortical bone in animals

A number of experimental studies on rabbit tibiae have been performed to determine the aetiology of stress fractures. In humans, it is ethically difficult to instrument bone then load it until fatigue or injury; however, this is possible in animals.

As mentioned in Section 5.2, Li et al. (1985) conducted an experiment where rabbits were induced to run and jump for approximately 2 hours per day by being subjected to a pulse via an electric cage, where the frequency and period of the pulse was controlled. Radiographic and histological changes in the bone were examined over a 60-day period after sacrificing the exercising rabbits at various stages during the test. Two rabbits were also sacrificed from a control group, the first at the beginning and the second at the

conclusion of the experiment. From the radiographs, Li et al. (1985) found that there was a progressive periosteal reaction in 18 of the 20 rabbits, whereas the remaining tibiae showed soft tissue swelling with no radiographical changes (changes were found in 16 tibial mid-shaft, 3 distal and 1 upper third). Osteoclastic reabsorption was evident as early as the seventh day after exercise commenced, but cracks were not visible until the tenth day after loading. The histological analysis demonstrated that cracks developed on the cement lines of the Haversian systems, particularly on the anterior and medial aspects of the tibia, and that fracture lines were subsequently formed by convergence of adjacent cracks from the Haversian systems.

The experiment by Li and colleagues provided *in-vivo* verification of the early cortical bone specimen tests under cyclic loading performed by Carter and Hayes, which were discussed earlier under Section 5.3. Carter and Hayes found that tensile failure occurs first under cyclic loading, and that the tensile stresses caused failure through osteon debonding at the cement lines. Li et al. (1985) did not specify which types of cracks (longitudinal, transverse or oblique) occurred in the different locations of the tibiae. However, they did observe that most cracks occurred in the midshaft, which is consistent with other research to date on tensile stresses and tensile failure at this site.

Burr et al. (1990) applied cyclic loads to the hind limbs of 31 rabbits; one limb was subjected to compressive loads while the other limb acted as a control. The loads were applied using a specifically designed apparatus designed to apply cyclic loading of 1.5 times the body weight of the rabbit to simulate running. SFs were successfully produced in 68% of the rabbits within 6 weeks of loading and were verified by scintigraphy. Burr and colleagues stated that 89% of the SF were in the midshaft (implying that 11% were distal) and 74% were anteromedial; however, it was not clear how many of the midshaft SFs were anteromedial. As rabbit bones are quite small, it would have been difficult to visualise exact locations using scintigraphy. In addition, the rabbits were not under anaesthetic; hence their muscles could involuntary contract. This means that the loading applied to the tibia was not purely compressive as the involuntary contractions apply other loads to the bone such as bending.

Burr's group followed-up the above work with another rabbit experimental study analysing strain rate versus strain magnitude. Strain gauges were bonded to the midshaft and mid-distal third of the medial, lateral and posterior aspects of the tibia, but not the anterior border. The authors concluded that SFs were a result of increased strain rate at the mid-distal third of the tibia; however, the data presented showed that both strain rate and strain magnitude were higher in this location than in the midshaft. Hence, it could not be deduced from the results whether strain magnitude or strain rate is most likely to be associated with tibial SFs, or if both parameters in combination are significant.

6. Computer models of the tibia

More recently, computer models such as Finite Element (FE) models have been developed to examine the stresses in the tibial bone. FE models are advantageous in that the stresses can be analysed in any region of the bone modelled, loading and other boundary conditions can be readily controlled, and unlike human and animal experiments, a large number of loading conditions can be analysed.

In Section 5.5, a rabbit experimental model developed by Burr et al. (1990) was discussed. Burr and colleagues subsequently developed an FE computer model of the rabbit tibia (Burr, 1997; Burr 2001) where compressive loading only was applied. However, there were a

number of discrepancies with this model. For example, the model did not have any other loads from the musculature applied other than compression, yet it is probable that the tibia was subjected to other loads such as bending in the rabbit experiments. Additionally, the results of the FE model showed that high compressive stresses occurred on the anterior border of the tibia, yet from clinical research and knowledge of fracture types at this site, SFs on the anterior border are a result of tensile failure due to tensile or bending forces. Lastly, to produce compression on the anterior border, the applied compressive load would need to be significantly anterior to the centroidal axis of the tibia, particularly as the tibia is bent anteriorly and the rabbit leg is partially flexed. However, this is not consistent with the load position being applied to the heel in the experiment.

Using an MTSS patient, Franklyn (2004) developed a human tibial FE model to examine the relationship between ground reaction forces, bone geometry and maximum principal stress (Figure 8). The model was analysed similar to a 'free-body' analysis in engineering, where a section of the tibia was modelled, and the forces acting on the free body (tibial model) were applied. The forces were derived from gait analysis data of ground reaction forces which were then mathematically transposed to the equivalent forces acting on the free body. The major muscle forces were included. The model was then validated using *in-vivo* strain gauge data available in the literature such as the data from Lanyon et al. (1975), although this validation was not extensive due to the lack of *in-vivo* cyclic loading data available in the literature for all regions of the tibia.

The model was analysed using different time steps in the running gait cycle. It was found that the highest magnitude of principal stress was tensile, disperse, located on the external surface of the cortical bone on the medial tibial midshaft and occurs during the push-off stage of the gait cycle. This high stress region was due to a specific combination of high transverse and compressive loads during the latter part of the gait cycle. These findings are all consistent with previous work where bone fails in tension, and the push-off stage of the gait cycle has been shown to result in maximal tensile strain at the midshaft during running (Lanyon et al., 1975). Additionally, cracks have been shown to initially develop on the exterior cortical surface, which is also consistent with mechanical theory, which predicts stresses are greatest on the external surfaces.

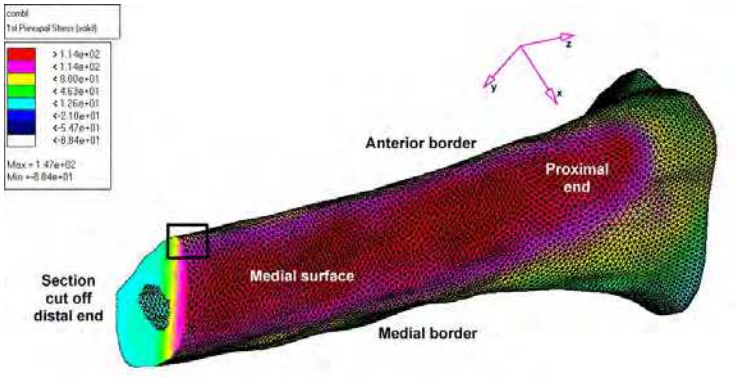

Fig. 8. Tibial FE model of an MTSS patient sectioned at the midshaft. Maximum principal stresses on the medial surface are diffuse and originate from the exterior surface (Franklyn, 2004).

Franklyn (2004) also conducted a preliminary analysis on load versus bone geometry in the FE model to replicate the stress pattern typical of a tibial SF. It was found that a localised SF pattern could not be produced by altering the loads alone, but only by changing the geometry. These results suggest that bone geometry is more influential than loading conditions in the development of tibial SFs and indicate that graded training programmes may be the most effective countermeasure for SF prevention.

7. Conclusion

Current knowledge of the aetiology and mechanics of tibial SF and MTSS development has come from a combination of clinical research, cohort studies, *in-vitro* cortical bone specimen experiments and *in-vivo* tests on both humans and animals. More recently, FE computer models have been used to better understand the relationship between tibial bone geometry, applied loads and stress distribution in the cortical bone.

Although there has been considerable research on the mechanisms behind these injuries, they are still not fully understood. However, a number of conclusions are evident. SFs of the tibial midshaft, which are longitudinal or transverse, arise from tensile and bending loads respectively. These loads produce tensile stresses which cause osteons to debond from the surrounding tissue, resulting in cracks between and through Haversian systems. On the other hand, SFs of the mid-distal junction are oblique; hence they could be due to shear stress and subsequent lamellae buckling from compressive loads, or from tensile stresses in an oblique plane due to torsional load. Clinical findings suggest tensile failure occurs at this site; hence torsional load appears to be a more likely mechanism.

Cortical bone geometry is significantly different between injured patients and non-injured control subjects. It is probable that the bone geometry alters due to impact loading rather than being inherent, but longitudinal studies are needed to determine if bone geometry alters prior to the injury or as a result of the injury. These types of studies may lead to the development of reliable prediction tools for tibial stress injuries.

Despite some common aetiology and mechanisms between tibial SFs and MTSS, it is unlikely that they are one injury on a continuum. However, it is evident that more research is needed in this area, as prevention of these debilitating injuries remains a problem which can affect successful sporting and military careers as well as the large recreational athletic population.

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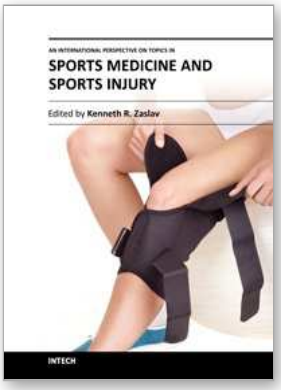

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For the past two decades, Sports Medicine has been a burgeoning science in the USA and Western Europe. Great strides have been made in understanding the basic physiology of exercise, energy consumption and the mechanisms of sports injury. Additionally, through advances in minimally invasive surgical treatment and physical rehabilitation, athletes have been returning to sports quicker and at higher levels after injury. This book contains new information from basic scientists on the physiology of exercise and sports performance, updates on medical diseases treated in athletes and excellent summaries of treatment options for common sports-related injuries to the skeletal system.

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