Sarah Morrison

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Tibial Shaft Stress Fractures in Endurance Runners

**Intro**

Long-distance running has become a popular form of exercise and competition for many individuals today. However, the high magnitude of repetitive loading can place runners at increased risk for overuse bone stress injury. Lower extremity stress fractures occur relatively commonly in endurance athletes, reportedly making up for between 50% and 90% of all stress fractures in sports medicine1,2. In endurance runners specifically, lower extremity stress fractures may account for approximately 16% of all injuries3. Fracture of the tibial shaft has been shown to be the most common bone stress injury in runners, comprising between a quarter to a half of all stress fractures in this population3,4­.

Such injury can significantly limit an individual’s ability to exercise, compete, and perform daily functions for extended periods of time. Additionally, recurrence rates for repetitive stress fractures or bone stress injury are relatively high, further increasing the likelihood of long-term and chronic deficits after an initial incident1,4. The aim of this paper is to identify the contributing bone composition and anatomy, fracture pathophysiology, risk factors, and evidence-based prevention and treatment strategies related to tibial shaft stress fractures in order to help minimize the impact these injuries have on the at-risk endurance runner population.

**Properties of Bone**

Human bone’s properties and composition lend it significant strength and stiffness to provide structure, support, and protection to the body and resist against the repetitive stresses imposed on bone during activities such as endurance running. Like other connective tissues, bone is composed of a fiber component, ground substance, and cellular component. About 90% of the organic fiber matrix is collagen, and about 45% of the inorganic ground component is composed of calcium salts and other mineral constituents, which afford bone its relatively great ultimate strength or ability to withstand a large amount of stress before failure and injury occurs. Osteoblasts and osteoclasts reside in the endosteum, a thin membrane surrounding the inner cavity of long bones such as the tibia, and are the unique cellular components of bone. They promote bone formation and resorption, respectively, and thus play an important role in bone’s ability to remodel according to the mechanical demands it must meet.5

This ability of bone to remodel in response to stress is described by Wolff’s Law, which states that the type of change and extent to which bone structure and fiber orientation change are directly influenced by the type and magnitude of stress placed upon them6. Tibial bone may undergo all types of stress in many directions, including compressive, tensile, shear, torsional, and combined stresses. However, since the tibia is a weight-bearing structure, it mainly experiences compressive stress during which equal and opposite loads are applied to the proximal and distal ends of the tibia in the vertical direction5. Consequently, collagen fibers in the diaphysis, or central shaft, of the tibia are oriented mainly in a vertical direction best suited to resist this daily compressive stress and prevent failure5. However, this also means that the diaphysis of the tibia is more apt to fail when other types of stress are involved in loading4.

While all of these factors work in favor of bone maintaining its healthy structure, it may still fail if overloaded. Important factors affecting bone’s mechanical behavior and integrity include not only the loading mode and direction of loading, but also bone’s inherent mechanical properties. The load-deformation curve for bone (Figure 1) demonstrates how bone biomechanically responds to increasing loads. The initial elastic region represents bone’s capacity to return to its original shape after a load is applied. Once the yield point is reached, outer fibers may begin to deform permanently. If loading is continued, bone will reach the plastic region in which its fibers will not return to their original state after the load has been removed and eventually bone will reach ultimate failure in the form of a fracture5. Similarly, the strain-stress curve (Figure 2) demonstrates that with progressively increased levels of stress, or load across a unit of area, bone will experience a certain amount of deformation relative to its original state, known as strain, before reaching failure and fracture. The steep slopes of this graph along with the large ultimate strength emphasize that bone may undergo only a small amount of strain but a relatively large amount of stress before more permanent changes or failure takes place5.

This risk for failure is further influenced by too high a frequency of loading or too high a rate of loading5. Bone’s strength and stiffness increases with increased loading rates, which is beneficial for protection during more vigorous activities such as running. However at very high loading rates, bone becomes more brittle and must release more energy, increasing risk for fracture2,5, Additionally, because stress and strain occur relative to a specific cross-sectional area of bone, the thinner geometry of the tibial shaft may place it at higher risk of fracture since force is exerted over a smaller area4,5.

**Pathophysiology of Overuse Stress Fracture**

A stress fracture is defined as a partial or complete fracture of bone as a result of repetitive sub-maximal loading2. As noted previously, bone will fracture if overloaded past its elastic range to its ultimate failure point. While compressive microstrain in the tibia during running (417 to 2456 microstrain) is usually measured much below that needed to cause ultimate failure of cortical bone, it causes varying amounts of microdamage depending on the number of bone strain cycles, strain magnitude, and strain rate7. This microdamage stimulates beneficial bone remodeling in the affected areas in order to strengthen the bone according to Wolff’s Law as described above. It is only when loading rate, frequency or magnitude results in such increased strain rate, frequency or magnitude that bone remodeling processes cannot keep up and bone stress injury occurs1,4,5,8.

This imbalance is more likely to occur in the diaphysis of the tibia due to the predominance of poorly vascularized cortical bone8. This type of bone remodels and heals mainly via endochondral ossification, an indirect mechanism that takes increased overall time and involves the formation of an un-mineralized cartilaginous callus, which is structurally weaker than normal bone5. Microdamage to osteocytes signals the activation of remodeling units that target the specific area of damage. Osteoclasts arrive first since they must tunnel through the tissue to reach the targeted area and remove damaged particles, and osteoblasts follow replacing the resorbed bone with un-mineralized osteoid that is mineralized over time4,8. This process usually maintains a homeostasis between microdamage and remodeling, resulting in a beneficial ability of bone to adapt to increased loads by lessening the strain bone experiences at a certain level of loading. However, one cycle of remodeling takes about three to four months to complete in cortical bone9. Thus, if any combination of too much frequency, magnitude or rate of loading, surpasses this time threshold, as may happen with long-distance running, more damage may occur due to the weakened state of the un-mineralized bone during the healing period4,5,8. Consequently, a feedforward loop of damage ensues as increased remodeling units are recruited and the initial osteoclastic activity leads to locally reduced bone mass and energy-absorbing capability. If this feedforward mechanism is allowed to continue, a negative stress reaction associated with excess bone resorption will lead to the physical fracture line associated with stress fracture4,8.

**Risk Factors**

As noted before, factors influencing the likelihood of a tibial stress fracture in runners are all those elements that affect the load applied to the tibia, including the frequency, magnitude, rate, direction, and duration of both internal and external forces. In addition to these force factors, characteristics of the bone itself and things that influence healthy bone’s ability to effectively resist the strain that occurs in response to loading also influence risk for tibial stress fractures.

*Biomechanical*

The ground reaction force (GRF) is the force exerted by the ground on a body that comes in contact with it. During gait, there is an initial peak GRF at heel strike, followed by a maximum GRF during late stance phase (Figure 3). It has been suggested that runners with increased GRF may be at higher risk of tibial stress fracture due to higher loading with each repetition shifting them closer to the injury threshold. However, research has not supported this idea. A meta-analysis including 10 studies of tibial stress fractures found no overall significant difference in peak GRF values between tibial stress fracture groups and controls. However, the vertical loading rate (VLR), or change in GRF over a specified time period, was shown to be significantly higher in those with a history of tibial stress fracture2. Greater peak braking forces at impact have also been found in runners with a history of tibial stress fracture10.

Those with a history of tibial stress fracture may demonstrate higher peak hip adduction and greater rearfoot eversion angles during stance phase of running, possibly increasing the torsional forces on the tibia during stance phase11. As the trabeculae of the tibial bone are more predominantly aligned to resist vertical stress, this increased torsional stress could increase risk for bone injury. Greater pronation and velocity of pronation may also be associated with increased risk of tibial stress fracture due to the increased tensile stress on the posterior tibia at the insertion of the tibialis posterior muscle that is being stretched12, as well as the consequent increased loading velocity incurred with faster pronation4. Runners with increased pes cavus or planus, leg length discrepancy, or increased hip external rotation range of motion may have also been shown to be more likely to have tibial stress fractures due to the abnormal stiffness or direction of loading caused by these biomechanics4.

*Training*

Increased duration or distance during training increases the total number of loading cycles, which can cause bone to reach its microdamage threshold causing fracture if enough time is not allowed for healing. Increased running speed leads to increased GRF and VLR13, which can surpass the ultimate strength of bone if not progressed gradually to allow remodeling units to adapt to the increased loads. These effects may be especially augmented for individuals with a history of lesser physical activity who progress training too quickly since their bone has not had the same load-bearing stimulation14. Thus, a similar change in training may upset the homeostasis of bone microdamage and remodeling to a greater degree in these individuals compared to those with a history of higher magnitude, frequency or durations in their training programs.

Changes in the running surface for training can also affect risk for tibial stress fracture. A systematic review by Warden et al. found that moving training to more uneven surfaces, less compliant surfaces, or more downhill slopes may all be associated with increased tibial stress fracture risk4. These factors work to increase risk by increasing bone strain magnitude and rate, as well as altering normal kinematics so that loads are placed on different bone areas which may be less adapted to the higher loads associated with running4.

*Running Style*

Adoption of various running styles that shift impact from the heel to the mid- or forefoot have been proposed to decrease overuse injury rates by decreasing the GRF and VLR. One study comparing Chi running to traditional heel-strike running found significantly lower VLR’s and peak braking forces in Chi runners15. These decreases in force and rate may be due to the shorter stride length, increased cadence, and decreased vertical displacement of the runner’s center of mass that are characteristic of such an alternative running style16,17. Landing on the forefoot rather than directly on the heel may increase the time to reach a velocity of zero at impact, and lessening the vertical displacement may decrease the total change in velocity, which leads to an overall decreased GRF according to the impulse-momentum equation Force = mass x (velocity change)/(time change)16.

While the initial GRF may be less with these alternative forefoot strike patterns than traditional heel-strike styles, the mid-stance propulsive GRF may be greater18 having possible implications for bone injury. At this point, there is not enough research or a consensus to support or negate the hypothesis that these alternative running styles decrease bone injury risk, and further studies are necessary to come to any strong conclusion on the topic16.

*Muscle*

Under normal conditions, muscle functions to help protect bone and take on some of the loads experienced during running. However, muscle fatigue may lead to dysfunction in this relationship, causing higher GRF peak and VLR19 as well as higher bone strain magnitude and rate20, increasing risk for bone injury. Surrounding muscle size and strength have also been repeatedly associated with increased risk for stress fracture, specifically for the gastroc-soleus, tibialis anterior and tibialis posterior for tibial fractures4. Additionally, weak knee extensors have been associated with increased risk for tibial stress fracture21.

*Footwear*

Shoes and foot orthotics may be able to absorb shock to lessen the GRF and VLR. By altering foot positioning, shoes and orthotics may also be able to influence the previously described influential biomechanics of the kinetic chain moving proximally from the foot. However, research shows mixed results as to how influential footwear is on tibial stress fracture risk. A review of stress fracture in the military population found significantly decreased stress fracture risk with the use of orthoses, but it is unclear whether or not these results are generalizable to runners22. One recent review found lesser VLR’s when runners wore traditional running shoes versus barefoot runners. Overall, the authors concluded that no well-designed studies have demonstrated significant injury reduction by matching shoe type to foot morphology16. In another study, researchers found that individuals running in minimalist shoes with a rear-foot strike pattern experienced significantly higher VLR’s than runners wearing traditional shoes with a rear-foot strike or runners wearing minimalist shoes with an anterior-foot strike23. As the VLR is a strong risk factor for tibial stress fractures, it may be that assessing the match between running style or ground-striking technique with footwear is more important than the match between foot morphology and specific shoe type.

*Nutrition & Energy*

Any factor that decreases the mass or structure of bone itself will allow more strain and faster strain rates, putting bone at higher risk of stress fracture. This is partially due to a given load being distributed over a smaller area, causing more stress. Additionally, these effects decrease bone’s area moment of inertia and polar moment of inertia, lessening its capacity to resist bending and torsional loads4,5. Both decreased mass and cross-sectional area have been directly linked to bone stress injuries and tibial stress fractures4.

Calcium and vitamin D intake both affect the rigidity of bone since calcium binds with phosphate to provide the mineral strength of bone, and vitamin D drives calcium resorption in the kidneys. Deficits in both have been directly associated with increased incidence of tibial stress fractures in athletes and military who practice long-distance running24,25. Research has come to a general consensus that reduced overall energy availability has also been shown to decrease bone mineral density and strength, as well as lessen its ability to resist loads and repair itself4. This risk factor may be especially important for female endurance runners as energy availability directly influences not only adequate bone formation, but also regulation of normal menses and hormonal balance, which further affect healthy bone mass and cross-sectional area3,21. Due to the increased energy usage inherent to endurance sports, these athletes are at especially high risk of energy and nutritional insufficiency. Other factors that can affect bone mass and cross-sectional area include high caffeine intake, contraceptive use, very high alcohol intake, prednisone, and nicotine use26,27,28,29.

**Management**

Stress fractures to the anterior cortex of the tibia, while relatively uncommon, are more severe and will usually require a non-weight-bearing cast and/or surgical intervention with rod fixation or anterior tension plate banding. However, most commonly tibial stress fractures occur in the posteromedial tibia and heal with conservative treatment in 8-12 weeks1, in line with the general healing period necessary for bone remodeling as discussed previously. Initially, these injuries are managed with modified activity and assessment of the risk factors noted above1,4. Pain at the injury site during or after activity may indicate over-loading of the weakened bone and necessitate further reduction in activity or weight-bearing. Suitable activities may include stationary bicycling, pool running or swimming, antigravity treadmill running1. Deep water running and anti-gravity treadmill running may be the most beneficial for runners since they mimic the neuromuscular recruitment patterns involved in running4. A pneumatic leg brace may be utilized to promote normal pain-free gait and decrease abnormal compensatory mechanisms that may lead to bad habits4. Therapeutic low-intensity pulsed ultrasound may also help speed bone remodeling and union, but might be most effective in more severe fractures and cases of delayed healing30.

The second phase of rehabilitation is generally started 2 weeks after the runner can ambulate and perform non-impact cross-training pain-free and is no longer point tender over the injury location. This phase emphasizes muscular endurance training, core stabilization, balance and proprioceptive training, flexibility, gait training, and a gradual return to running1. The gradual running progression should gradually increase frequency and duration over the course of 3 to 6 weeks until the runner has reached their original training levels, and then speed is increased1,4. Warden provides a useful guideline for running progression (Figure 4) in which the runner can gradually increasing running duration, frequency and speed as long as pain is not provoked during or after completing each level.

Gait training throughout the rehabilitation process should focus on changing any biomechanical risk factors, as well as minimizing compensatory mechanisms adopted following the injury in order to decrease risk for re-injury. Also, since VLR and possibly GRF have been implicated in stress fracture occurrence, strategies to minimize these variables are important to address1,4. According to the impulse-momentum equation F = m x (change in v)/(change in t), effective strategies should be aimed at decreasing the change in velocity and increasing the change in time to keep GRF and VLR low. It may be helpful to provide runners with feedback instructing them to “run softer” as well as biofeedback aimed at decreasing tibial acceleration4. Adoption of some of the proposed mechanisms for decreasing VLR and GRF during running, such as decreased stride length and increased cadence while maintaining the same running speed may effectively decrease VLR, GRF and other biomechanical risk factors associated with stress fracture1,4,16,17. A metronome can be a helpful tool for encouraging these changes4. As noted before, no clinical or research consensus has been reached concerning other gait alterations, such as transition to a forefoot-strike pattern4,16. Use of more cushioned shoes to increase loading time via shock absorption may be helpful, but this also may encourage a more rearfoot heel-strike, possibly increasing GRF and VLR and off-setting these beneficial effects4,16. All in all, gait mechanics and individual risk factors of each runner should be taken into account when re-training running after stress fracture1,4,16. Other risk factors, such as inadequate nutrition or energy consumption, must also be addressed and any necessary referrals made to ensure comprehensive management.

**Conclusion**

An understanding of tibial stress fracture injury mechanisms, risk factors, and management is important for physical therapists and other healthcare professionals working with endurance runners due to the relatively high occurrence and morbidity in this population. Stress fracture formation results from an imbalance of microdamage and bone remodeling, which is influenced by training frequency, duration, intensity and magnitude. A progressive return to weight-bearing activities and running can effectively return individuals to running in 8-12 weeks. However, addressing risk factors that affect VLR and bone strength and cross-sectional area are essential to preventing repetitive injury. These factors may include running form and movement patterns, malalignments, muscle strength and endurance, and appropriate nutrition.

**Figures**

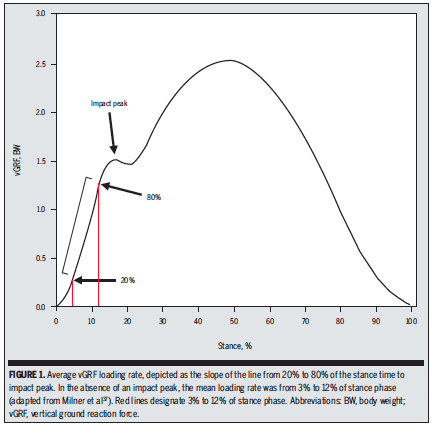
***Figure 1: Load-Deformation Curve5***



***Figure 2: Stress-Strain Curve5***



***Figure 3: Ground Reaction Force during Running15***



***Figure 4:* Graduated Running Program to Return a Runner to 30 Minutes of Pain-Free Running4**

|  |  |
| --- | --- |
| **Stage/Level** | **Description** |
| 0 (Pain during walking in normal activities of daily living) | Pre-entry to graduated running program |
| 1 | Initial loading and jogging (50% normal pace) with increasing duration |
| A | Walk 30 minutes |
| B | Rest |
| C | Walk 9 minutes and jog 1 minute (3 repetitions) |
| D | Rest |
| E | Walk 8 minutes and jog 2 minutes (3 repetitions) |
| F | Rest |
| G | Walk 7 minutes and jog 3 minutes (3 repetitions) |
| H | Rest |
| I | Walk 6 minutes and jog 4 minutes (3 repetitions) |
| J | Rest |
| K | Walk 4 minutes and jog 6 minutes (3 repetitions) |
| L | Rest |
| M | Walk 2 minutes and jog 8 minutes (3 repetitions) |
| N | Rest |
| 2 | Running with increasing intensity |
| A | Jog 30 minutes |
| B | Rest |
| C | Run 30 minutes at 60% normal pace |
| D | Rest |
| E | Run 30 minutes at 60% normal pace |
| F | Rest |
| G | Run 30 minutes at 70% normal pace |
| H | Rest |
| I | Run 30 minutes at 80% normal pace |
| J | Rest |
| K | Run 30 minutes at 90% normal pace |
| L | Rest |
| M | Run 30 minutes at full pace |
| N | Rest |
| 3 | Running on consecutive days |
| A | Run 30 minutes at full pace |
| B | Run 30 minutes at full pace |
| C | Rest |
| D | Run 30 minutes at full pace |
| E | Run 30 minutes at full pace |
| F | Rest |
| G | Run 30 minutes at full pace |
| 4 | Return to running |

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