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Preventing bone stress injuries in runners with optimal workload

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Abstract

Bone stress injuries (BSIs) occur at inopportune times to invariably interrupt training. All BSIs in runners occur due to an ‘error’ in workload wherein the interaction between the number and magnitude of bone tissue loading cycles exceeds the ability of the tissue to resist the repetitive loads. There is not a single optimal bone workload rather a range which is influenced by the prevailing scenario. In prepubertal athletes, optimal bone workload consists of low-repetitions of fast, high-magnitude, multidirectional loads introduced a few times per day to induce bone adaptation. Premature sports specialization should be avoided so as to develop a robust skeleton that is structurally optimized to withstand multidirectional loading. In the mature skeleton, optimal workload enables gains in running performance, but minimizes bone damage accumulation by sensibly progressing training, particularly training intensity. When indicated (e.g. following repeated BSIs) attempts to reduce bone loading magnitude should be considered, such as increasing running cadence. Determining the optimal bone workload for an individual athlete to

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prevent and manage BSIs requires consistent monitoring. In the future, it may be possible to clinically determine bone loads at the tissue level to facilitate workload progressions and prescriptions.

Keywords

exercise; relative energy deficiency in sport; running; stress fracture; stress reaction

Bone stress injuries (BSIs), including those presenting as a radiographically-confirmed cortical defect (i.e. stress fracture), are frustrating. Frequently occurring in the lead up to a major running event, these injuries invariably require interruption to training as the prodromal pain and risk of progression to complete fracture are real. It is well established cumulative loading, inherent to running, contributes to bone fatigue, which presents as microscopic damage (i.e. microdamage) in bone tissue [1, 2]. Microdamage is a normal and necessary phenomenon occurring in all skeletons independent from athletic ability. It triggers targeted remodeling where bone-resorbing osteoclasts remove damaged regions of bone before bone-forming osteoblasts fill the void with new, undamaged bone [3].

It can be argued all BSIs occur due to an ‘error’ in workload, whereby microdamage accumulation in response to cumulative loading outweighs the ability to repair or resist the damage. Assuming a suitable workload and healthy athlete, microdamage is removed at the same rate new microdamage forms; however, the process takes time. Osteoclast activation and resorption in cortical bone takes approximately 4 weeks, and replacement with new bone can take three months and up to a year for full mineralization [4]. The process is longer in trabecular bone, which may explain why BSIs at trabecular rich sites have more prolonged healing times [5].

Within the remodeling timeline, the transition period between resorption and formation is consequential to BSIs. Bone resorption creates porosity, which influences mechanical properties and reduces fatigue resistance (Fig. 1) [6]. This creates the potential for a feedforward loop whereby suboptimal workload (e.g. too rapid progression of training) can heighten microdamage formation, and its accumulation, coalescence, and progression to a BSI. BSIs begin appearing approximately 3-4 weeks following a major workload ‘error’, as observed in military recruits experiencing a large change in workload as they transitioned from a sedentary lifestyle to the rigors of basic training [7].

What is bone workload?

There is no uniform definition of workload. From the perspective of athletic performance, there is consensus workload comprises a combination of internal and external factors which combine to determine training and/or competition stress [8]. Because microdamage occurs at the tissue level, bone workload leading to microdamage formation and its progression to a BSI is best described at this level.

Applied loads produce stresses and strains within bone tissue which in turn can produce microdamage. Here, stress is a localized measure of load intensity defined as the force per

unit area of tissue and strain is a normalized measure of tissue deformation created by stress. Microdamage formation is threshold-driven and depends on the interaction between the number of times a bone is loaded, and the magnitude and rate that stresses and strains are generated. Of these factors, load magnitude appears most important in terms of bone fatigue and BSI risk [9].

There is a strong correlation between the magnitude of bone tissue stress/strain and the number of cycles before bone fatigue failure. The relationship is often described by an inverse power law which indicates small increases in stress/strain dramatically reduce the number of cycles until fatigue failure, and vice versa. In vitro, the rate of microdamage accumulation increases with strain magnitude by an exponent of 17 [10]. For the loads relevant to running, it has been estimated a 10% increase in tissue stress/strain results in halving the number of loading cycles before failure [11].

The stresses and strains experienced within a bone depend on the magnitude of the applied load and the ability of the tissue to resist this load. Greater applied loads generate greater stresses and strains whereas weaker bones experience greater stresses and strains for a given applied load. Thus, workload approaches to minimize microdamage and reduce BSI risk aim to: 1) improve the ability of the skeleton to resist load by inducing mechanoadaptation and 2) manage the loads being introduced to the skeleton to reduce damage accumulation. These two approaches are somewhat paradoxical, suggesting loading both protects against and causes BSI development. Ultimately, optimal bone workload can be defined as: *the interaction between the number and magnitude of bone loading cycles that induces adaptation to best enhance function and reduce the risk of re/injury.*

Optimal workload to induce skeletal mechanoadaptation

The ability of the skeleton to resist load is determined by its mass, structure and material quality. There is great potential for the skeleton to adapt to mechanical loads to improve its strength. For instance, baseball players have nearly double the strength of the humeral diaphysis in their throwing arm compared to their contralateral non-throwing arm [12]. The adaptation reduces tissue stresses and strains such that they remain safely below fracture thresholds during throwing. If the same throwing-related forces were introduced to the contralateral non-adapted humerus it would catastrophically fail in a single pitch [12]. In terms of BSI risk, an animal study showed a moderate (<10%) gain in bone mass induced by a loading program can generate a large and exponential (>100-fold) gain in bone fatigue resistance as a result of less tissue level strain being experienced during each fatigue loading cycle [13].

Not all athletes have good skeletons

There is a presumption all athletes have good bone health as they are regularly exposed to elevated loads; however, this is not always true. The mechanosensitive machinery in bone responds best to high magnitude loads introduced at high rates. Weight bearing activities incorporating impulsive loading, particularly those involving some degree of intermittent, explosive jumping and/or sprinting with rapid changes in direction, have the greatest osteogenic potential (Fig. 2a).

Gymnastic activities generate some of the greatest osteogenic stimuli and accordingly gymnasts have high bone mass [14]. Interestingly, gymnasts also have a high incidence of BSIs [15] suggesting a finite ability by their well-adapted skeletons to tolerate applied loads (Fig. 2b). Similarly, basketball players experience a relatively high number of BSIs despite typically good skeletal health. Conversely, swimmers and cyclists experience limited osteogenic stimuli resulting in low bone mass compared to other athletes [16], yet experience few BSIs due to low bone workloads (Fig. 2b) [15]. These findings underscore the importance of understanding the balance between applied bone loads and underlying bone health across a spectrum of athletes.

Distance runners have some of the highest incidences of BSIs (Fig. 2b). A contributing factor in many is poor bone health. For instance, up to 40% of female adolescent cross-country runners have a dual-energy x-ray absorptiometry (DXA) z-score of below −1 for spine areal bone mineral density (aBMD) [17]. There are numerous possible reasons for the inferior bone health in cross-country athletes. The most obvious is the high occurrence of Relative Energy Deficiency in Sport (RED-S) and Female Athlete Triad in this population, with up to 50% meeting criteria for disordered eating and/or reporting menstrual dysfunction [17-19]. However, another important contributing factor is that distance running simply is not a good bone building activity. The latter is supported by the observation that gymnasts exhibit higher bone mass than runners despite both populations having a similar prevalence of menstrual dysfunction [20].

Distance running does not build good bones

Bone cells desensitize or become ‘deaf’ to repetitive loading. They lose 95% of their mechanosensitivity after only 20 back-to-back loading cycles and introducing additional cycles does not yield proportional adaptation [21]. The implication is that after a few minutes of running, bone cells find the monotonous, unidirectional loading to be boring and they stop responding.

A period of relative rest enables the system to regain mechanosensitivity to support further adaptation. Over 90% of mechanosensitivity is restored with 4-8 hours of rest between repeat loading bouts [22]. Thus, a few minutes of a bone-centric exercise (e.g. plyometrics) later in the day after a running session may generate further bone adaptation over and above that generated solely by running. The addition of short bouts of bone-centric exercises requires consideration of cumulative bone workload, but the approach has been used in other sports to improve bone health while limiting exposure to excessive loading cycles [23].

Use periodization to build more bone

Bone cells also lose sensitivity over a block of training (e.g. across a sport season). The adaptive response of bone cells to loading is proportional to the difference between the applied and routine loads [24, 25]. When routine bone loads are high, a greater stimulus is required to create an adaptive response as the threshold to respond is raised [26]. Progressive overload does not address this issue as each load increment leads to accommodation creating a situation where the difference between the new and routine load is small [27].

Periodization can be used to improve bone cell mechanosensitivity, particularly in individuals participating in year-round sports such as distance running. In an animal model, bone adaptation was compared between groups that received either continuous loading for 15 weeks versus a periodized approach of two 5-week blocks separated by a 5-week 'rest' period [28]. Despite receiving one-third less cumulative load, bone adaptation in the periodized group was greater since the rest period restored the mechanosensitivity prior to the recommencement of loading. Clinically, "rest" would involve other conditioning activities (such as cycling, swimming, water running) that load alternate skeletal sites, rather than absolute rest.

Early sport specialization likely contributes to BSIs

Early sport specialization (i.e. intensive prepubertal participation in a single sport for more than 8-months of year at the expense of other sports) has been associated with an increased risk of overuse injury [29], which may include BSIs. The years from birth to the pubertal growth period provide a window of opportunity to accrue bone mass. This was eloquently shown in racquet sport players. Girls who began playing before puberty had more than twice as much adaptation between their racquet and non-racquet arms compared to girls who began playing after puberty [30]. Athletes who specialize early in sports with low osteogenic potential (e.g. distance running, swimming, cycling) may enter adolescence and young adulthood with low bone mass and elevated BSI risk [31, 32].

Structural optimization is critical and only develops before puberty

More importantly than facilitating bone mass accrual, loading during growth provides a *once-in-a-lifetime* opportunity to optimize bone size. Bone size develops principally due to modeling (not remodeling) which involves new bone forming on existing surfaces without prior resorption. Growth is associated with rapid modeling on the periosteal/outer surface which increases bone girth. Loading when young encourages additional bone to be added periosteally to further increase bone size [33] and disproportionately increase bone strength and fatigue resistance for the amount of material added (Fig. 3) [13, 34]. Also, the bone size, but not mass, benefits of loading when young persist long term [12, 35]. In contrast, there is no consistent evidence mechanical loading impacts bone size once skeletal maturity is reached, with the less consequential gains in bone mass occurring at the inner endocortical surface post-puberty [33].

Load in multiple directions to build a robust skeleton

Bone adaptation to loading has directionality, with bone mass being added and size developing in accordance with the direction of loading [36]. The implication is that athletes who specialize in unidirectional sports from a young age (e.g. long-distance road running) may not have the ability to resist loading in alternate directions (e.g. such as during trail running, with running on natural terrain changing the principal loading axes [37]).

Activities requiring jumping and landing in different directions or running with rapid changes in direction should be encouraged during growth, such as occurs during basketball, volleyball, soccer, and gymnastics, to name a few sports. Participants in these sports exhibit more structurally robust lower extremity bones which may be more resistant to BSI [38, 39].

Indeed, military recruits with a prior history of playing ball sports had nearly 1/3rd the odds of developing a BSI in basic training than those without a history of playing these sports [40]. Multidirectional activities should be encouraged from a very young age when the skeleton is most permissive, and single sport specialization should be delayed at least until high school so to develop a robust skeleton that can withstand multidirectional loading.

Managing the load being introduced to the skeleton

If skeletal mechanoadaptation requires proactive attention when very young and continuing through puberty, what do we do for skeletally mature athletes? In this case, attempts at improving skeletal health are still important, particularly in athletes with poor bone health (e.g. distance runners suffering from RED-S with associated low bone mass and a history of repeat BSIs). Nevertheless, bone gains are more difficult to achieve in mature athletes, particularly in terms of optimizing bone size. For those who have reached skeletal maturity, attention shifts more towards managing applied loads.

The load applied to bone represents the summation of external and internal forces, which are influenced by a range of variables including biomechanical factors, muscle performance, and environmental characteristics (e.g. training surface/s, shoes and inserts, etc.). However, training factors are by far the leading contributor with which all other factors interact.

All BSIs occur due to training errors

All running injuries are training load injuries [41], and BSIs are no exception. Evidence suggests rapid increases in training loads increase the risk of running injuries [42, 43]. Historically, the ‘10% rule’ has been used to guide increases in running volume on a weekly basis [44]. More recently, an athlete’s acute:chronic workload ratio (ACWR) [45] has been proposed to guide training load prescriptions. The ACWR is most commonly defined by the ratio of workload for the previous week to the average workload for the previous three to six weeks, with the training goal being to avoid large increases or ‘spikes’ in this measure. However, there is much individual variability with respect to tolerance to changes in training loads and it is unlikely a single “rule” can be uniformly applied to eliminate running injuries [46]. Also, different skeletal sites may respond differently to changes in workload [47]. Training workloads should be individualized since two runners may have identical training loads but have different injury patterns. Individualized risk of BSI likely relates to the complex interaction between rapid changes in workload and a runner’s biomechanics, psychology, physiology, musculoskeletal qualities, and energy availability [48, 49].

Changes in workload occur at specific times depending on sport

Identifying scenarios when large changes in workload are likely enables implementation of preventative strategies. The most hazardous times for BSIs in seasonal sports (e.g. basketball, soccer, outdoor track) are during preseason and with the intensity increase from preseason to competition [15, 50]. These times present particular risk for those returning from off-season surgery and those transitioning between competitive levels (e.g. moving from high-school to collegiate to professional level).

Athletes competing in seasonal sports should be afforded a workload reduction at the end of the competitive season to take advantage of periodization. However, a progressive bone loading and general conditioning program should be considered leading up to preseason to dampen the spike in workload with the return from the off-season and to help reduce seasonal variations in bone health. A lead-in program is particularly important in those with history of BSis as they are at high risk of another BSI [51].

In more year-round sports where training is more constant and progressive (e.g. gymnastics and cross-country), BSI risk progressively increases across the competitive season [15]. However, the risk patterns vary by sex. Risk in male cross-country runners is fairly consistent across the season, whereas it progressively increases in females [15]—possibly due to increasing effects of RED-S as training progresses, with RED-S being more common in females [52]. In athletes competing in more year-round sports, BSI mitigating strategies include substituting training sessions with activities requiring reduced load (discussed later) and incorporating rest periods (e.g. at least 1 rest day per week and 1-to-2 weeks rest every 3 months [53]).

Monitoring bone workload

The often weeks delay between workload change and BSI development necessitates clinicians, coaches, and athletes closely track workload. While the majority of runners tend to overestimate their training volume [54], individuals who develop a BSI tend to *under-report* their training volume and intensities when compared with wearable monitors [49]. Thus, using objective means to monitor training load should be the cornerstone of managing risk of BSI.

Unfortunately, there is no prospectively established workload metric that accurately predicts BSIs. The proposed microdamage origin of BSIs suggests monitoring of tissue level loading is the ultimate goal, but determining bone tissue loading is neither trivial or currently available clinically. Surrogate metrics, such as GRFs and segmental accelerations, and the development of wearable sensors accompanied with software algorithms and user interfaces (collectively known as ‘wearables’) are an attempt to fill this void [55].

GRFs and segmental accelerations are used to quantify and monitor the intensity of foot-ground impacts, with the presumption that their magnitudes and rates are related to BSI risk. However, limited evidence supports a causal link between foot-ground impact characteristics and BSIs, with currently available data derived mostly from retrospective cohort studies [56]. The lack of data supporting causality has raised conjecture as to the relative contributions of the initial foot-ground impact versus muscle contraction in BSI genesis [57].

Internal tissue loads are much greater than those predicted from external measures because of the pull of muscle [58]; this was illustrated decades ago for running [59]. Differentiating impact-related versus muscle-generated bone loading is challenging given most impulsive (i.e. high impact) activities are also those with greatest muscular loading. However, muscle (and, consequently, bone) loads peak well after initial foot-ground impact and nearer midstance of the running gait cycle—near the second or ‘active’ peak of the ground reaction

force (Fig. 4) [57, 60, 61]. Foot-ground impact may generate greater loading rates, but the majority of cadaveric research suggests bone is able to better withstand repetitive loads when applied over shorter durations (i.e. higher loading rate) [11], tilting the balance towards greater importance of high-magnitude muscle-derived bone loading. Unfortunately, at the moment, clinicians cannot capture muscle-derived loading.

Progress training duration before intensity

The non-linear relationship between loading cycles and their magnitude before bone fatigue failure can be used to guide training progression. Assuming all other risk factors remain constant (e.g. energy availability) there is a linear one-to-one increase in BSI risk for an increase in running volume (i.e. number of loading cycles). In contrast, the disproportionate reduction in bone fatigue life with increasing loading magnitude means BSI risk increases more rapidly with increases in running velocity. These observations suggest it is safer to initially increase training volume than intensity, and have led to the concept of high volume, low velocity training (e.g. ‘train slow to race fast’). In a probabilistic model, running the same distance but with decreased speed from 3.5 to 2.5 m/sec reduced tibial BSI likelihood by half [62]. Ultimately, bouts of high-speed running should be performed judiciously and progressions in high speed running should be coupled with temporary reductions in running volume.

Training with reduced bone workload

Beyond careful management of high-speed running volume, there are other practical methods of reducing bone loads without compromising training benefits. For example, treadmill running may engender lower tibial bone strain than overground training [63], despite minimal differences in running mechanics and physiological metrics between the two running modes [64]. Thus, treadmill running may be substituted for one or more overground sessions per week to reduce cumulative bone workload.

Treadmill running can also be coupled with body weight support (e.g. with lower body positive pressure or a mounted upper body support system) to further reduce bone workload, and be performed on steady incline. The role of incline running (and, similarly stairclimbing) on BSI risk remains unknown. On one hand, incline running may reduce BSI risk as it reduces impact loads and accelerations [65]. On the other hand, it may increase risk by increasing muscle-induced bone loading or by shifting risk to an alternate site (e.g. metatarsals).

Can we alter running mechanics to alter bone workload?

It may be possible to alter bone workload through gait retraining. Gait retraining is usually reserved for runners with repeat BSIs and currently involves implementing techniques to reduce GRFs and/or bone accelerations, despite evidence lacking that these metrics are valid surrogates for tissue-level loading. Techniques currently being applied include increasing stride rate (i.e. cadence) [66], cueing a softer landing by providing feedback on peak positive tibial acceleration [67], or transitioning an athlete to a forefoot (FFS) strike pattern [68]. Ultimately, retraining interventions should aim to reduce bone tissue loads, not surrogates

for bone loads such as vertical GRFs [57]. For instance, cueing a softer landing may reduce GRFs but may require higher muscle forces ultimately resulting in greater bone loads [69].

Of the techniques listed, cueing an increase in cadence appears to have considerable clinical promise. Increasing running cadence above a preferred rate results in a proportional decrease in stride length at a given speed. The net result is an increase in the number of loading cycles for a given running distance, but a concomitant reduction in the vertical excursion and velocity of the center of mass, reduced peak hip adduction angle and moment, reduced GRFs and tibial accelerations, and reduced demands on lower extremity joints [70, 71]. The combination of these changes may improve running economy [72] and has been modeled to reduce internal loading, which was predicted to more than offset any increase in tibial BSI risk associated with the increased loading cycles [73]. In high school cross-country runners, those in the lowest quartile for step rate were 6.7 times more likely to experience a shin injury compared to runners in the highest step rate quartile [74], and prescriptive decreases in step length (e.g. via increased cadence) contributed to a reduction in the incidence of BSIs in female military recruits [75, 76]. Runners can be retrained to increase their running cadence easily in the clinic, and most commercially available running watches enable runners to retrain and monitor cadence during routine runs [66].

Does muscle strengthening increase or decrease BSI risk?

There is no doubt muscle loads bone, but debate remains as to whether muscle-induced loading is causative or protective of BSIs. Biomechanical data suggests most bone loading is muscle-induced [59, 57]; however, clinical data points to a potential protective role of muscle on BSI risk. In particular, prospective clinical studies demonstrated BSI susceptibility was directly related to muscle size (girth and cross-sectional area) [7, 77, 78] and strength [79].

Enhanced muscle properties may aid in diffusing forces across the bone cortex during running or reduce bending moments induced by external loads. Alternatively, they may protect against the skeletal consequences of fatigue. Runners exhibit greater tibial stress and strain during running after an exertion protocol [80, 81], suggesting poor endurance may elevate the risk of BSI. In addition, intense running can lead to altered kinematics, which may modify the direction of bone loading resulting in increased strain at less accustomed sites [82].

Overall, these data suggest improving muscular endurance and strength may benefit runners at risk for BSI. Unfortunately, much of the evidence supporting resistance training to reduce BSI risk is retrospective. For example, female military cadets with <7 months of resistance training prior to basic combat training had a 4-fold greater risk of sustaining a BSI than cadets who habitually strength trained [83], and adolescent runners who did not strength train were more likely to sustain a BSI during a cross country season [84]. Lastly, greater bone density is observed in runners and athletes who regularly participate in heavy resistance training compared with those who solely did their sport [85].

Summary

Optimal bone workload promotes beneficial adaptation to best enhance function and reduce the risk of re/injury. There is not a single optimal workload, rather a range which are influenced by the current scenario. In athletes before their adolescent pubertal growth period, optimal bone workload consists of low-repetitions of fast, high-magnitude, multidirectional, novel loads introduced a few times per day. Care needs to be taken to avoid premature sports specialization to develop a robust skeleton that is structurally optimized and can withstand loading in multiple directions. In the mature skeleton, tracking of workload is indicated to avoid acute spikes. Rest periods should be incorporated into each program, at least 1 d/wk and 1 wk every 3 months. When indicated (e.g. following repeated BSIs), attempts to reduce bone loading magnitude such as increasing cadence should be considered.

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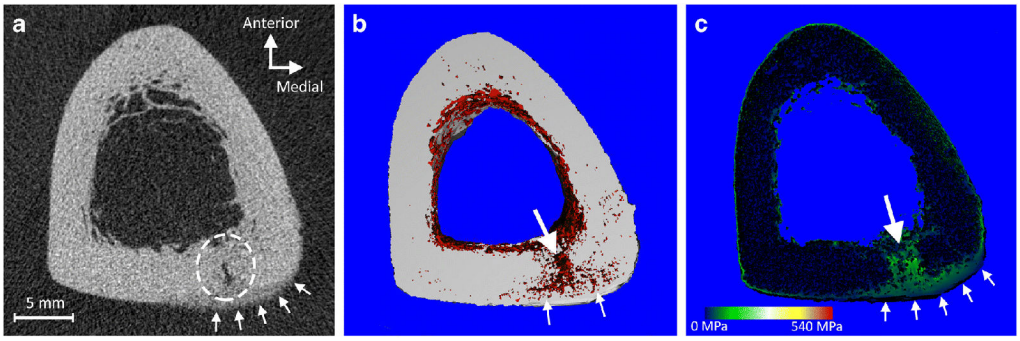


Figure 1.

Bone stress injuries (BSI) cause porosity and reduced localized mechanical properties. **A)** Tomographic image of a posteromedial tibial cortex BSI (broken circle) in a 22-year-old female distance runner, acquired using high-resolution peripheral quantitative computed tomography (voxel resolution = 61 μm). Note the presence of undermineralized callus bridging the periosteal surface at the injury site (arrows). **B)** 3D map showing regions of porosity in red. The majority of the tibial cortex has limited porosity, including the newly formed undermineralized callus. However, there is prevalent porosity at the BSI site (large arrow) and branching medially and laterally along the original periosteal layer of bone (small arrows). **C)** Finite element model of the stress distribution in response to axial compressive loading. Stresses are concentrated on the regions of the BSI (large arrow) and the immature undermineralized callus (small arrows).

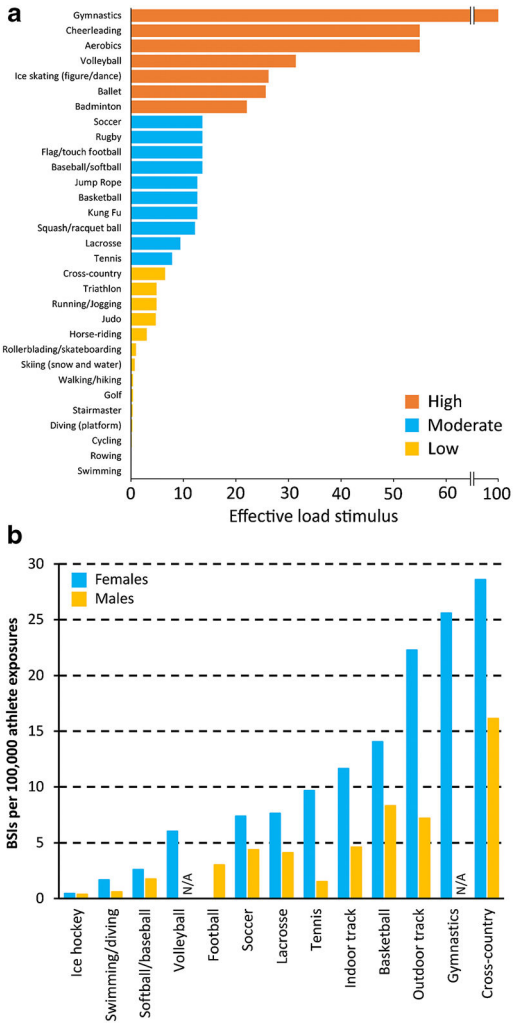
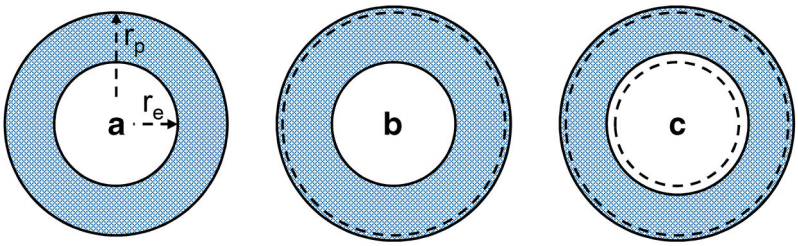


Figure 2.

A) Lower extremity effective load ratings for common physical activities, with higher load ratings being representative of a greater bone osteogenic stimulus. Effective load ratings were estimated from the magnitude and rate of ground reaction force generation during representative actions (or similar actions when reaction forces could not be directly measured). Data from Weeks and Beck.[86] **B)** Incidence of bone stress injuries (BSIs) in females and males across collegiate sports in the United States over a 10-year period. N/A = data not available. Data from Rizzone et al.[15]



Bone property	a	b	c
Periosteal radius (r_p)	1.00	1.05	1.05
Endosteal radius (r_e)	1.00	1.00	1.15
Bone mineral content	1.00	1.15	1.00
Polar moment of inertia	1.00	1.24	1.16

Figure 3.

Loading-induced addition of bone on the outer periosteal surface is functionally important, helping the skeleton meet its dual needs of being strong to resist injury, but lightweight for energy efficient motion. A) The polar moment of inertia (i.e. strength) of a bone is proportional to the radii of its outer periosteal (r_p) and inner endocortical (r_e) surfaces according to the relationship $\pi(r_p^4-r_e^4)/2$. This relationship illustrates that periosteal surface changes have a greater influence on strength than changes on the endocortical surface. B) For example, a 5% increase in r_p (equating to a 15% increase in bone mineral content [i.e. mass]) results in a disproportionate 24% increase in strength, assuming constant bone material properties (i.e. volumetric bone mineral density) and an initial r_p-to-r_e ratio of 1.8. C) If the same mass of bone added to the periosteal surface was simultaneously removed from the endocortical surface, r_e would increase by 15%, but the bone would still be 16% stronger than the bone with same mass in A) because of its greater size (i.e. 5% greater r_p). Broken lines in B) and C) indicate the original bone surfaces in A).

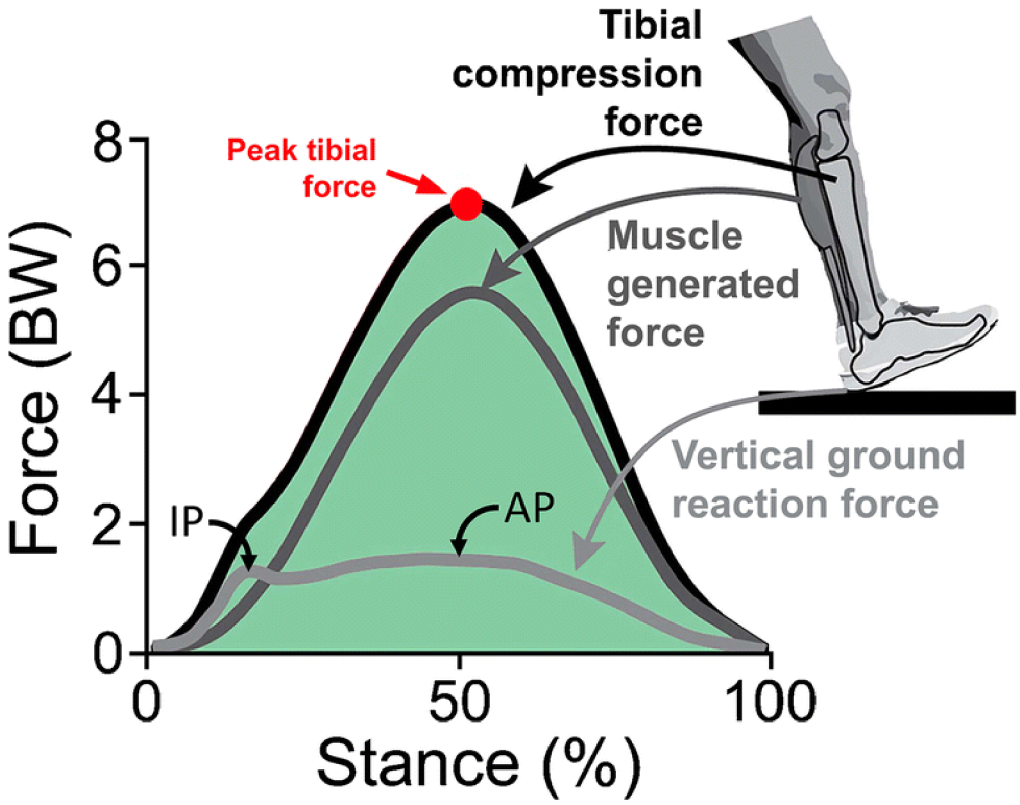


Figure 4.

Vertical ground reaction force, and computed muscle generated and tibial compression forces during running with a typical rear-foot strike pattern. The external ground reaction force has two peaks—an initial rapidly reached impact peak (IP) and a second slower, but higher magnitude active peak (AP). Internal muscle generated and tibial compressive forces, computed via subject-specific musculoskeletal modeling, far exceed ground reaction forces and peak near the active peak of the ground reaction force. The later peak of tibial forces has raised the question of the relative contribution of initial foot-ground impact versus later muscle generated forces in BSI genesis. Image adapted from: Matijevich E, Scott L, Volgyesi P, Derry K, Zelik K. Combining wearable sensor signals, machine learning and biomechanics to estimate tibial bone force and damage during running. *Human Movement Science* 2020;74:102690, with permission from Elsevier.