ARTICLE

Modeling Overuse Injuries in Sport as a Mechanical Fatigue Phenomenon

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EDWARDS, W.B. Modeling overuse injuries in sport as a mechanical fatigue phenomenon. *Exerc. Sport Sci. Rev.*, Vol. 46, No. 4, pp. 224–231, 2018. *This paper postulates that overuse injury in sport is a biomechanical event resulting from the mechanical fatigue of biological tissue. A theoretical foundation and operational framework necessary to model overuse injury as a mechanical fatigue phenomenon is introduced. Adopting this framework may provide a more mechanistic understanding of overuse injury and inform training and preventive strategies to reduce their occurrence.* **Key Words:** loading magnitude, tissue damage, cumulative damage, Weibull analysis, loading rate

Key Points

- Overuse injuries result from repetitive loading and cumulative bouts of activity.
- Repetitive loading of biological tissues illustrates damage accumulation and failure consistent with a mechanical fatigue process.
- Information from mechanical fatigue tests suggest that the risk of overuse injury should increase much more rapidly with loading magnitude than loading cycles.
- Mechanical fatigue tests also do not support the general notion that higher loading rates are deleterious to the musculoskeletal system.
- Probabilistic modeling of the mechanical fatigue process may be used to examine the relative risk of injury associated with sudden changes in training activity.

INTRODUCTION

Overuse injuries in sport are thought to have a diverse and multifactorial etiology (1,2), but this is true only when overuse injury is defined at the whole-body, or systems, level. Overuse injuries manifest at the tissue level, and previous definitions of overuse injuries in sport (3,4), including the word "overuse" itself, suggests that these injuries result from a mechanical fatigue phenomenon. Here, mechanical fatigue refers to the accumulation of tissue damage and progressive loss of stiffness and strength associated with repetitive loading and cumulative bouts of activity. This damage serves as a stimulus for remodeling and adaptation (5,6), but without adequate rest and repair, continued loading will cause damage accumulation that may eventually lead to failure (*i.e.*, tear, rupture, fracture) (7,8).

Despite sharing a common philosophy that overuse injuries in sport result from repetitive loading and cumulative bouts of activity, few researchers in this field have incorporated the fundamental principles of mechanical fatigue into their applied and clinical investigations. This article hypothesizes that overuse injury is ultimately a biomechanical event resulting from the mechanical fatigue of biological tissue. Thus, any risk factor, whether intrinsic or extrinsic to the athlete, will contribute to overuse injury through this dynamic process. The fundamental principles of mechanical fatigue can be readily applied to overuse injury when one centers their focus of inquiry at the level of the tissue. In this regard, there are several lessons that can be learned from mechanical fatigue tests, which may provide a more mechanistic understanding of the etiology of overuse injury.

This article briefly reviews evidence from *ex vivo* and *in vivo* studies that repetitive loading of biological tissue results in mechanical fatigue. The predominant role that loading magnitude plays in the mechanical fatigue process and its interaction with loading cycles will subsequently be discussed followed by the important distinction between cumulative load and cumulative damage, and its implication for injury risk. The stochastic nature of mechanical fatigue is described, in addition to probabilistic analyses that incorporate mechanical fatigue uncertainty into injury risk estimations. Finally, the topical debate of loading rate and its potential role in overuse injury is discussed, along with the fact that repetitive loading may not only induce

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damage as a function of loading cycles, but also as a function of loading duration.

MECHANICAL FATIGUE IN BIOLOGICAL TISSUE

Mechanical fatigue is characterized by microstructural damage, or microdamage, in response to cyclic or repetitive loading. Over time, this microdamage may grow and reach a critical size (9,10), resulting in complete failure at loading magnitudes much lower than the material's monotonic strength (11–13). The phenomenon of mechanical fatigue has been well described in load-bearing biological tissues, either through *ex vivo* cadaveric testing (11–13) or *in vivo* animal studies (14,15). Microdamage can take many forms depending on the specific tissue or mode of loading. Cyclic loading of bone, for example, may illicit linear microcracks approximately 50–100 μm in length, or diffuse clusters of matrix cracks on the order of 1 μm or less (16). Tendon damage on the other hand presents as kinked fibers, or localized fiber dissociation and ruptures, depending on the degree of fatigue loading (10).

Mechanical fatigue–induced damage is often, but not necessarily, accompanied by measureable changes in material properties, such as reductions in modulus or increases in residual strain. Fung *et al.* (10), cyclically loaded rat flexor digitorum longus tendons to low, medium, and high levels of microdamage. Changes in stiffness and hysteresis were not observed until high levels of microdamage, while tendon strain progressively increased with the level of microdamage. Burr *et al.*, (17) tested canine femora in four-point cyclic bending and observed an almost opposite trend; reductions in stiffness preceded any observable microdamage, such that the presence of microcracks was always accompanied by material property degradation, but not the other way around. In uniaxial loading, the rate of material property degradation in bone was highly influenced by the specific mode of loading (*i.e.*, tension versus compression); however, the total energy dissipated before failure was similar between loading modes (18).

Whereas the general pathway from early stage mechanical fatigue to subsequent pain and overuse injury is understood, specific details of this pathway related to the importance of mechanobiology remain undefined. In bone, it is not known if the microdamage itself or an excessive remodeling response is ultimately responsible for stress fracture development. Indeed, strong theoretical arguments have been made that bone remodeling in response to microdamage accumulation may create localized porosity and elevated mechanical stress/strain, which in turn leads to more microdamage and bone remodeling activity with continued loading (19). Similarly, the extent to which tendinopathy is an exclusive degenerative disorder versus an inflammatory mediated response to microdamage accumulation remains a topic of debate (20). But, whether biologically mediated or solely due to mechanical degradation, the rate of mechanical fatigue is always a strong function of loading magnitude.

THE INFLUENCE OF LOADING MAGNITUDE

The fatigue life of a material is defined as the number of repetitive loading cycles it can endure before complete failure. This fatigue behavior often is described by a stress-life plot (Fig. 1), or S-N curve, which characterizes the damage nucleation, damage accumulation, and failure processes of a material

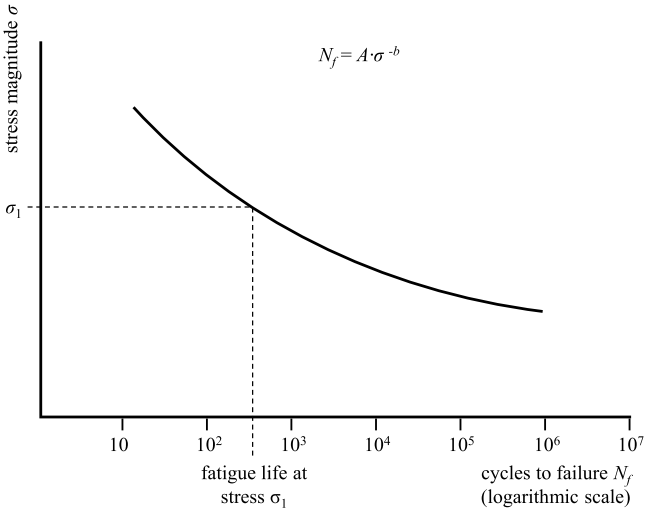

Figure 1. Theoretical stressed-life plot, or S-N curve, for a material subjected to cyclic loading. Fatigue life is defined as the number of cycles to failure N_f at a particular stress magnitude σ.

into a single, empirical relation. For engineering materials, the relation between peak stress magnitude σ, and the number of cycles to failure N_f is well described by an inverse power law:

$$N_f = A \cdot \sigma^{-b} \quad (1)$$

where A is a proportionality constant, and b is the slope of the S-N curve. When cyclically loaded *ex vivo*, biological materials illustrate this same exemplar behavior (13); alternatively, logarithmic or exponential decay curves are predictive of fatigue life (11,12). Although it may be more appropriate to write Eq. 1 in terms of stress range Δσ (21), *i.e.*, the difference between the maximum and minimum stress per cycle, biological tissues *in vivo* often range between some peak stress magnitude (*i.e.*, tension or compression) and zero, and so here, they may be used interchangeably. What is important to recognize is that small changes in σ often will result in large changes in N_f. For the loading magnitudes relevant to running, a 10% reduction in stress generally is associated with a corresponding 100% increase, or more, in the number of cycles to failure (11–13).

If overuse injuries in sport were to result from a mechanical fatigue phenomenon, then Eq. 1 would suggest that the risk of injury within a given sport would increase much more rapidly with loading magnitude than loading cycles (*i.e.*, similar to the theoretical relation illustrated in Figure 2a). Unfortunately, the field of exercise and sport science has yet to report the necessary data to test this hypothesis; however, convincing evidence of a mechanical fatigue-type risk profile has been reported for musculoskeletal disorders such as carpal tunnel syndrome, hand-wrist tendinopathy and pain, epicondylitis or tennis elbow, and lower back disorders. Gallagher and Heberger (22) performed a literature review of 12 epidemiological studies that examined musculoskeletal disorder risk as a function of loading magnitude and loading cycles. They reported evidence for the presence of a load × cycle interaction in 10 of these studies. The interactions were such that increased loading cycles resulted in moderate increases in musculoskeletal disorder risk for low-magnitude loading tasks, but rapid increases in risk for high-magnitude loading tasks (Fig. 2b). In other words, it

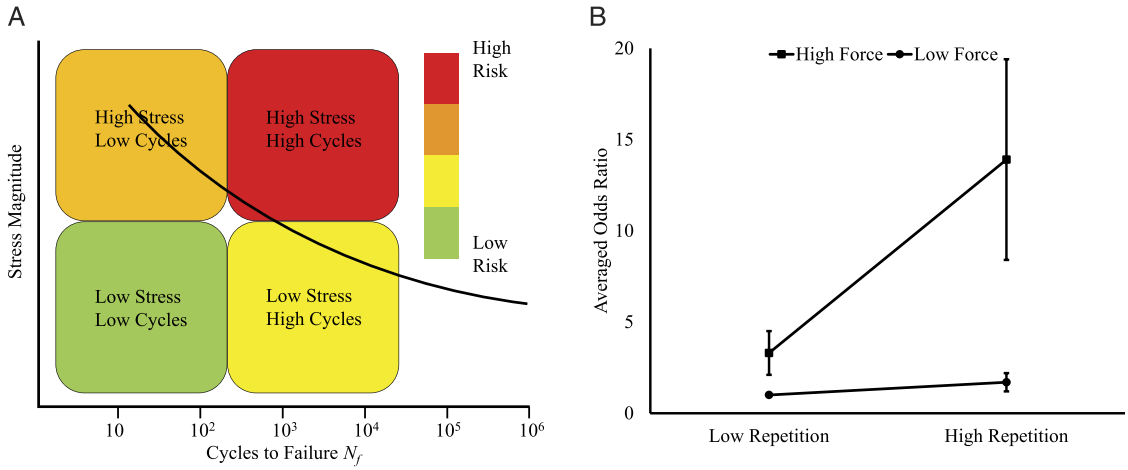

Figure 2. A. Theoretical stress-life plot and patterns of fatigue failure risk for different combinations of loading magnitude and loading cycles. B. Averaged odds ratios for epidemiological studies that examined musculoskeletal disorder risk as a function of loading magnitude and loading cycles (22). [Adapted from (23). Copyright © 2017 Taylor & Francis Group. Used with permission.]

may be insufficient to examine the main effects of loading magnitude and loading cycles independently when examining injury risk; rather, measurements that account for their potential interaction may be required.

DIFFERENCE BETWEEN CUMULATIVE LOAD AND CUMULATIVE DAMAGE

The complex interaction of loading magnitude and loading cycles is inherently captured by cumulative damage models of mechanical fatigue. The simplest of all cumulative damage models is the Palmgren-Miner rule, which states that a material will fail when a specific amount of damage D, as defined by the load-time history and S-N curve, has been accumulated. If a material experiences m different stress cases (*i.e.*, walking, running, jumping, etc.), then the fractional damage caused by the i^{th} case of peak stress magnitude σ_i is simply the number of cycles n_i at σ_i divided by the number of cycles to failure N_{fi} at σ_i. Thus, according to the Palmgren-Miner rule, failure will take place when:

$$D = \sum_{i=1}^{m} \frac{n_i}{N_{fi}} = 1. \quad (2)$$

Substituting Eq. 1 into Eq. 2 provides:

$$D \propto \sum_{i=1}^{m} \frac{n_i}{\sigma_i^{-b}} = \sum_{i=1}^{m} n_i \sigma_i^{\,b}. \quad (3)$$

Although beyond the topic of this review, there are a number of proposed methods that may be used to reduce a complex load-time history with variable stress amplitude into a series of simplified cycle/stress case scenarios that may be used with the Palmgren-Miner rule (24). For the case of a single peak stress magnitude, Eq. 3 may be written as:

$$D \propto n\sigma^{b}. \quad (4)$$

Some individuals will recognize Eq. 4 to be similar to the daily loading stimulus equation proposed by Carter's group to predict load-induced tissue adaptation (25). This is not a coincidence because Carter based his theory on the Palmgren-Miner rule, and there exists considerable evidence that load-induced tissue damage serves as a stimulus for biological remodeling (5,6).

It should be clearly stated that the measurement of cumulative damage, and therefore injury risk from a mechanical fatigue perspective, is not linearly proportional to the measurement of cumulative load, which often has been defined as the product of the number of loading cycles and peak load, or some impulse equivalent (26,27). Although two cycles at loading magnitude σ would provide the same cumulative load as one cycle at loading magnitude 2σ, according to the Palmgren-Miner rule, the damage incurred by these specific loading scenarios would differ by a factor of 2^{b-1}. Therefore, using cumulative load as a proxy for injury risk may lead to erroneous conclusions for loading scenarios that differ in terms of their magnitude and number of cycles, even when their cumulative loads are identical.

To illustrate this concept, data from a previously reported study (28) were used to calculate Achilles tendon force for ten participants running overground at three different speeds (*i.e.*, 2.5, 3.5, and 4.5 m/s). The Achilles tendon impulse per step was calculated as the time integral of the Achilles tendon force curve during stance, and a weighted impulse measure was quantified where Achilles tendon force was raised to the power of 9 before impulse calculation. This value for b was derived from *ex vivo* tendon testing data and is described in more detail in the following section. The cumulative impulse and cumulative weighted impulse were calculated as their respective impulse per step measures multiplied by the number of steps necessary to run 1 km. Whereas the cumulative impulse decreased with running speed (Fig. 3a), the cumulative weighted impulse increased with running speed (Fig. 3b). In other words, using a traditional measure of cumulative load suggested that running faster might actually decrease the risk of Achilles tendon injury, although this rather counterintuitive conclusion was not reached when using a weighted impulse measure that considered the stress-life behavior of tendon.

MECHANICAL FATIGUE IS A STOCHASTIC PROCESS

Cumulative damage models like the Palmgren-Miner rule rely on equations from S-N curves representing the arithmetic mean of sample fatigue behavior derived from one or multiple

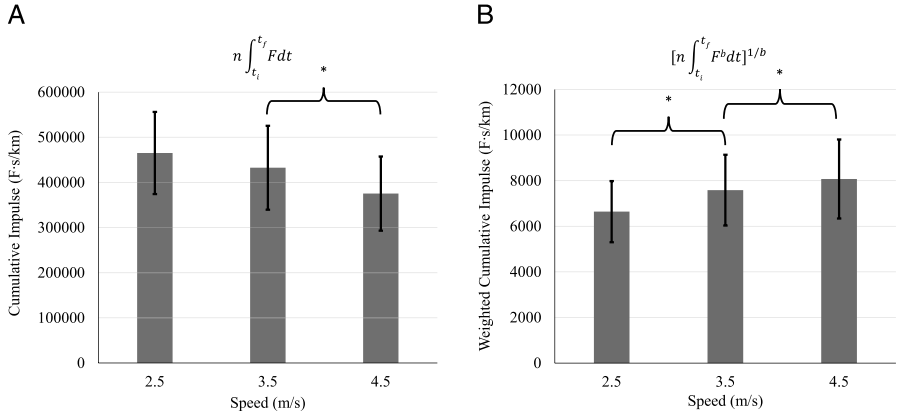

Figure 3. The mean (A) cumulative impulse and (B) weighted cumulative impulse as a function of speed calculated from the time integral of the Achilles tendon force curves during the stance phase of running (*$P \leq 0.05$). Note that the weighted cumulative impulse has been raised to the power of 1/b to provide identical units.

experiments. However, owing to the inherent variability in tissue microstructure, two otherwise seemingly identical samples will inevitably exhibit scatter in their fatigue behavior. In fact, there is a high probability that a tissue will fail either before or after what is predicted by the S-N curve, and because of the logarithmic scale, it may be extremely important to account for this variability.

The Weibull analysis is a well-known technique used for the treatment of scatter observed in the fatigue of materials, based on a distribution function, which mathematically describes the "weakest-link-in-the-chain" concept. Briefly, if a series of specimens with identical macrostructure are cyclically loaded to failure at stress magnitude σ, their measured fatigue strength will be expected to vary according to:

$$p_f = 1 - \exp[-(\sigma/\sigma^*)^m] \quad (5)$$

where P_f is the probability that the fatigue strength of a given specimen will be less than or equal to σ, and σ* and m are experimentally derived constants. The reference stress σ* is a measure of the material's fatigue strength, defined as the σ at which the probability of failure is 63% for a specific number of loading cycles. The Weibull modulus m defines the degree of scatter observed in fatigue-life measurements, where higher values indicate a lower range of variation. Despite its relative simplicity, the Weibull distribution performs remarkably well at describing the P_f for many types of biological materials.

Considerable work has used the Weibull distribution to examine the risk of stress fracture development (28–30). Here, a Weibull analysis will be used to estimate the probability of patellar tendinopathy, a common overuse injury associated with repeated bouts of jumping and landing activity. Raw data were first obtained from fatigue tests of human tendon across a range of strain magnitudes (12). These data were then fit to Eq. 1 (Fig. 4a), where peak strain magnitude ε explained 59% of the variance in N_f with a slope b equal to 9.3. When a Weibull distribution was optimized to the data and normalized to the mean fatigue strength ε_{mean} estimated from the S-N curve to allow for comparison of data at different absolute strain levels, m = 6.7 and $\varepsilon^* = 1.076 \cdot \varepsilon_{mean}$ (Fig. 4b). Using these constants, the equation explained 97% of the variance in experimentally measured fatigue strength. Therefore, if ε can be calculated for a particular activity, the P_f associated with a specific number of loading cycles can be determined.

To quantify ε, data were collected from six university basketball players performing maximum vertical jump landings in two footwear conditions while motion capture and force platform data were collected concurrently. The footwear conditions differed only in their outsole cushioning material, with a traditional shoe being 42% stiffer and providing 11% less energy return than an energy return shoe. An inverse dynamics approach was used to quantify intersegmental forces and moments of the lower extremity, and patellar tendon force F was quantified using a musculoskeletal model of the knee that accounted for antagonistic coactivation (31). After the calculation of F, ε for the patellar tendon during landing was calculated as:

$$\varepsilon = F/(A \cdot E) \quad (6)$$

where A and E are the cross-sectional area and elastic modulus of the patellar tendon, respectively. Tendon dimensions and material properties were obtained from the literature (32,33). Peak patellar tendon strain during landing was on average 14% lower in the energy return shoe compared with the traditional shoe (Fig. 4c).

The probability of patellar tendon failure P_f associated with ε from the maximum vertical jump landings in the two footwear conditions was calculated from Eq. 5. The P_f was examined as a function of time in days, assuming a typical basketball player experienced 45 jumps per day, based on the average number of jumps per elite-level game (34). Note that for these calculations, ε* from Eq. 5 also is a function of time, computed from the fatigue-life data. The P_f increased rapidly and in a nonlinear fashion as a function of time, demonstrating a greater risk of failure for landing in the traditional shoe (Fig. 4d). To compare these values with the prevalence of patellar tendinopathy observed in basketball players, we can look at the P_f associated with 1000 cycles of loading, which is just slightly less than 1 month of activity, or the approximate length of time it takes to induce deleterious structural changes in a rabbit tendon model (35). The P_f values for 1000 cycles were 14% and 34% for the energy return and traditional shoe, respectively. In this particular case, the numbers correspond very well with the prevalence of patellar tendinopathy in basketball

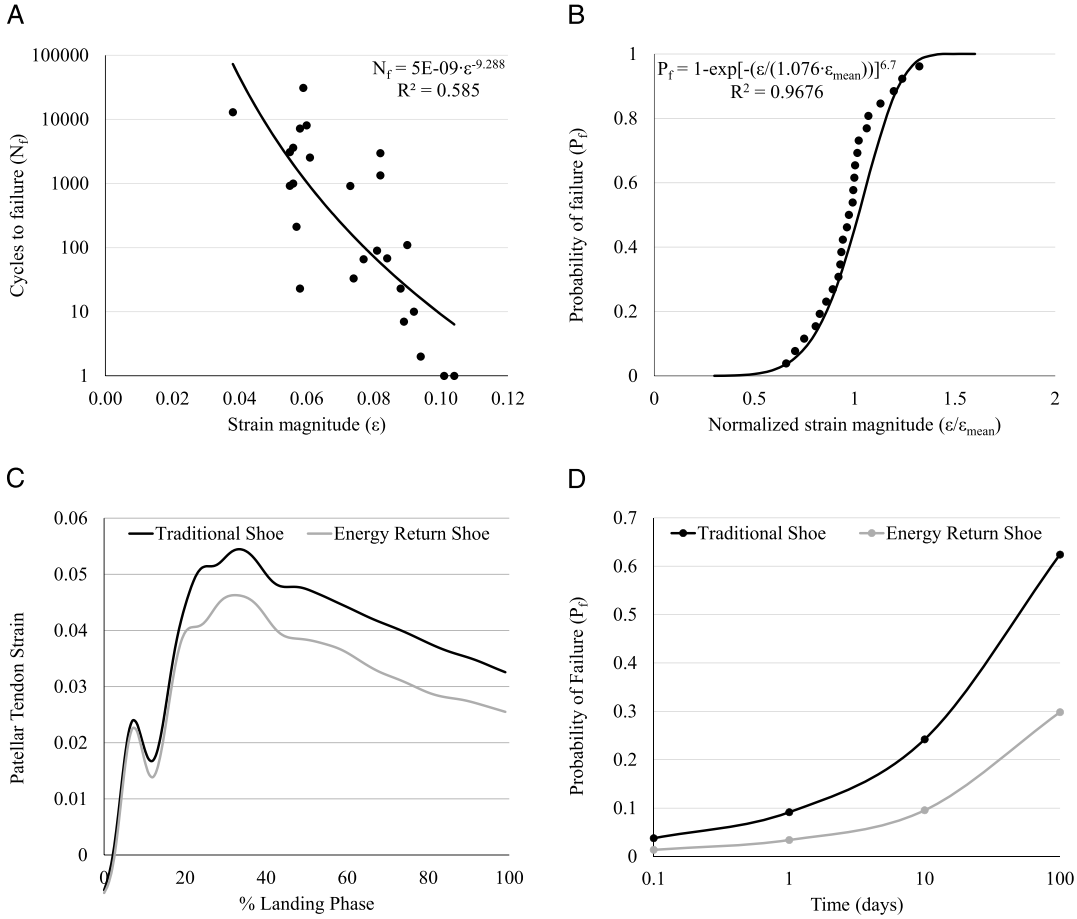

Figure 4. A. S-N curve for human Achilles tendon data from Wren *et al.* (12) (B) Weibull distribution optimized to the data. Strain axis has been normalized by the S-N curve trend line. C. Ensemble average curves of patellar tendon strain for basketball players landing in two different shoe conditions after maximal vertical jumps. D. Ensemble average probability of tendon failure curves associated with landing in two different footwear conditions as a function of time assuming 45 jumps per day.

players, which ranges from 12% to 32% (36,37). However, the model in its current form is too simplistic for long-term injury prediction, where remodeling and adaptation are likely to play a key role in the fatigue failure process. The strength of the model lies in its utility to examine the relative risk of injury associated with sudden changes in training activity, which for this shoe example ranged from 2.7 to 2.1, depending on the number of days.

LOADING CYCLES VERSUS LOADING DURATION

Until now, this article has focused solely on mechanical fatigue and damage accumulation in regards to stress or strain magnitude and their interdependence with loading cycles. Of course, increased loading rate has frequently been implicated in the etiology of overuse injuries, at least in runners (38). These studies relied on data from surrogate measures of tissue loading such as the vertical ground reaction force (vGRF) or peak tibial acceleration. But what effect might loading rate actually have on the mechanical fatigue of biological tissue? Cyclically loading bovine cortical bone samples using vGRF-like waveforms in running suggested that the loading rates associated with impact have little influence on the mechanical fatigue behavior of bone when compared to loading magnitude (39). The presence of an impact and higher initial loading rate in a vGRF profile characteristic of rearfoot running did not reduce fatigue life relative to a loading profile with no impact, even though samples from both groups were loaded to the same peak stress. Similarly, seminal work in this area, which manipulated loading rate through different sinusoidal frequencies, has consistently demonstrated that the fatigue life of bone and tendon actually increases with loading frequency (40,41). These findings cannot be explained by the viscoelastic response of biological tissue, because changes in loading rate must reach orders of magnitude in order to observe an appreciable change in material properties – changes well beyond what would be expected for different physiological movement patterns (41,42).

As previously mentioned, repetitive loads *in vivo* typically range between zero-tension or zero-compression, and this means that a nonzero mean stress exists. Consequently, damage may accumulate as a function of loading duration (*i.e.*, creep, or time-dependent damage), loading cycles (*i.e.*, fatigue, or cycle-dependent damage), or both. Figure 5 illustrates the number of cycles and time to failure as a function of peak stress for bone samples loaded in zero-compression at either 3 or 9 Hz. In general, samples loaded at 9 Hz survived more cycles of loading, but no difference between frequencies was observed as a function of time (39). Zioupos *et al.* (40) demonstrated this same phenomenon for human and bovine bone loaded in zero-tension. In other words, fatigue loading can be considered a "continually interrupted creep test," where the frequency of loading has no

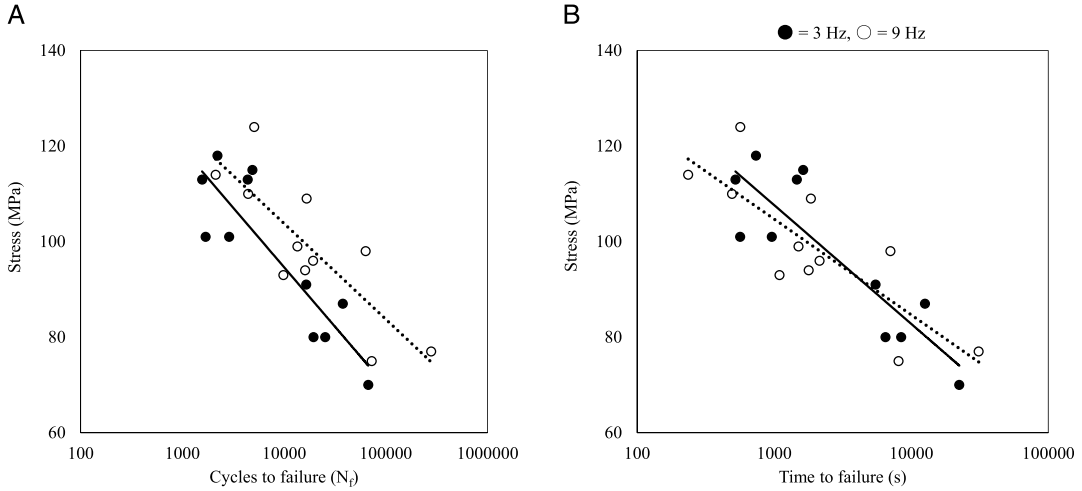

Figure 5. A. Cycles to failure and (B) time to failure as a function of stress magnitude for bovine cortical bone samples cyclically loaded at 3 and 9 Hz. Samples loaded at 9 Hz illustrated a longer fatigue life, although no differences in time to failure was observed. [Adapted from (39). Copyright © 2018 The American Society of Mechanical Engineers. Used with permission.]

other effect than to influence the impulse and thus the damage accumulated per cycle, but the total damage as a function of time remains the same. These results relate explicitly to bone-fatigue; damage in tendon will demonstrate both time- and cycle- dependency, though the spread in loading frequency is much greater as a function of cycles than time (41). In other words, it seems that musculoskeletal tissues are better able to tolerate loads when applied over shorter time durations, specifically because less damage is accumulated at a given stress level.

So why have so many studies in the running literature reported an association between individuals who experience high rates of loading and those with prevalent or incident overuse injury (38)? It is important to note that not all studies have reported this association (43,44); in fact, some studies suggest that increased loading rate has a protective effect (45,46). I suspect much of this discrepancy arises from the complex relation between external transducer measurement (*e.g.*, force platform and accelerometer) and tissue-level stress/strain (30,47). Stress and strain are not only dependent on applied load, but also on tissue size, geometry, material properties, and how the applied load is transferred to the tissue locally through forces and moments (48,49). I speculate that the positive relation between loading rate and injury observed in previous studies is real, but that increased loading rates are associated with lower-extremity mechanics that also increase stress and strain magnitudes. Increased cadence, for example, would be expected to lower both loading rates at impact (50) and loading magnitudes throughout stance (27,29).

LIMITATIONS AND FUTURE DIRECTIONS

This article has focused on the damaging effects that repetitive loading and cumulative bouts of activity may have on musculoskeletal tissues, with no regard to the repair and adaptation responses that play an important role in the fatigue failure process. These natural defense mechanisms may improve fatigue resistance at a particular stress level (*i.e.*, a shift in the S-N curve up and to the right) and reduce the stress magnitude associated with a particular activity. Previous work has incorporated repair and adaptation into predictive models of stress fracture, and these were necessary to provide risk estimations inline with prevalence rates in athletes (28,29). The primary limitation of these models was that repair and adaptation parameters were not subject-specific and as such they are unlikely to provide improved discriminatory power when assessing injury risk between individuals. Indeed, the greatest challenge moving forward with models of overuse injury is the appropriate representation of subject-specific remodeling and adaptation behavior.

An accurate prediction of cumulative damage requires both an accurate estimation of tissue stress/strain and the number of loading cycles. In regards to the latter, the emergence of wireless sensor technology to track physical activity has great potential; data from inertial measurement units (IMUs) can directly monitor loading exposure with a high degree of accuracy. However, barring the development of wireless sensors capable of direct tissue stress/strain measurement, the information provided by current IMUs is unlikely to have a substantial impact in the field of injury risk surveillance, unless the objective is to identify hazardous training patterns within a particular athlete. The optimal approach will likely require bringing athletes into the laboratory for a series of calibration trials, where both sophisticated subject-specific computational modeling approaches and data from IMUs can be used to generate a surrogate model (51), which may subsequently be used to estimate tissue stress/strain in a more ecologically valid environment outside of the laboratory.

CONCLUSIONS

This article argues that overuse injury is ultimately a biomechanical event resulting from the mechanical fatigue of biological tissue and Figure 6 provides a conceptive framework summarizing this hypothesis. Both *ex vivo* cadaveric and *in vivo* animal studies of biological tissues illustrate damage accumulation and stress-life behavior that is exponentially related to stress magnitude. There is no reason to believe that human tissues behave any differently when loaded *in vivo* as suggested by epidemiological data of musculoskeletal disorders such as low back pain and carpal tunnel syndrome. Attempts to characterize material property degradation in response to repetitive loading *in vivo* should recognize that the relation between damage accumulation and material property

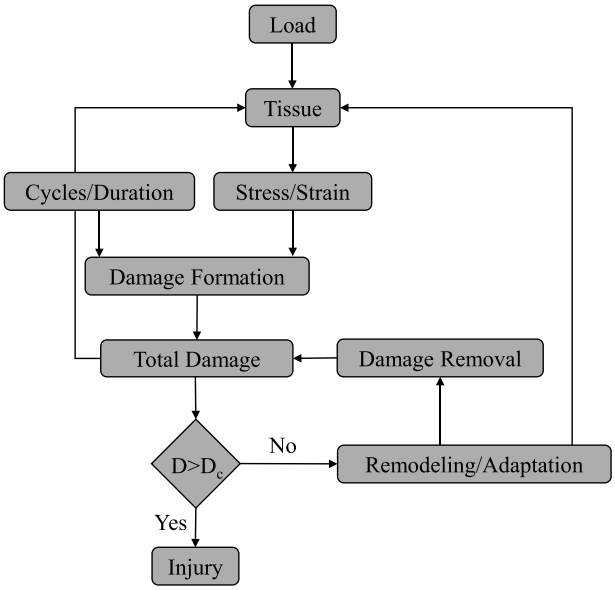

Figure 6. Conceptual framework for the potential role of mechanical fatigue in overuse injury. Load applied to tissue results in stress/strain, which in combination with loading cycles/duration causes damage formation that is highly dependent on loading magnitude. Damage formation contributes to the total damage in the tissue, resulting in material property degradation and increased stress/strain. When total damage (D) is less than some critical damage (D_c), a stochastic parameter, this damage induces tissue remodeling/adaptation. Damage induced tissue remodeling/adaptation may be positive, resulting in damage removal and improved tissue integrity (*e.g.*, material properties, geometry, size), or negative, resulting in diminished tissue integrity. This feedback loop continues until D becomes greater than D_c, leading to overuse injury.

degradation manifests differently in different tissues and under different modes of loading.

Much of the epidemiological research into overuse injury has focused on either the main effects of loading magnitude (*i.e.*, activity, intensity) or loading exposure (*i.e.*, duration, mileage), while ignoring the potential interaction these parameters may have in the mechanical fatigue process. Similar to the presence of a statistical interaction, it could be argued that the main effects of loading magnitude and loading cycles should not be examined independently, as doing so may lead to erroneous conclusions regarding injury risk. Although, traditional measurements of cumulative load consider both loading magnitude and loading cycles in the overuse injury process, the relative influence of these two components are not appropriately weighted according to S-N data. In this regard, the cumulative damage model based on the Palmgren-Miner rule is expected to provide a more sensitive framework to account for the potential interaction of loading magnitude and loading cycles. The most meaningful predictions of overuse injury based on S-N data will likely require probabilistic methods to account for the extreme variability observed in the mechanical fatigue of materials.

Assuming that damage accumulation resulting from repetitive loading and cumulative bouts of activity is the precursor to overuse injury in sport, results from mechanical fatigue tests do not support the general notion that higher loading rates are deleterious to the musculoskeletal system. On the contrary, it seems that biological materials are better able to withstand mechanical loads when applied over shorter durations. More work is needed to elucidate the mechanism by which increased loading rates at impact are associated with overuse injury. Mechanical fatigue principles would suggest that future areas of research should focus on a potential relation between external loading rate at impact and tissue stress/strain magnitude and loading duration throughout stance.

SUMMARY

Although current definitions of overuse injury in the field of exercise and sport science are consistent with a fatigue failure process, few studies have incorporated the fundamental principles of mechanical fatigue into their investigations of overuse injury. The information outlined in this article provides a theoretical foundation and operational framework necessary to model overuse injuries in sport as a mechanical fatigue phenomenon. In doing so, future work may provide a more mechanistic understanding of these injuries as well as training programs and preventive strategies to minimize their occurrence.

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