REVIEW ARTICLE

Training Load and Injury: Causal Pathways and Future Directions

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Abstract

Causal pathways between training loads and the mechanisms of tissue damage and athletic injury are poorly understood. Here, the relation between specifc training load measures and metrics, and causal pathways of gradual onset and traumatic injury are examined. Currently, a wide variety of internal and external training load measures and metrics exist, with many of these being commonly utilized to evaluate injury risk. These measures and metrics can conceptually be related to athletic injury through the mechanical load-response pathway, the psycho-physiological load-response pathway, or both. However, the contributions of these pathways to injury vary. Importantly, tissue fatigue damage and trauma through the mechanical loadresponse pathway is poorly understood. Furthermore, considerable challenges in quantifying this pathway exist within applied settings, evidenced by a notable absence of validation between current training load measures and tissue-level mechanical loads. Within this context, the accurate quantifcation of mechanical loads holds considerable importance for the estimation of tissue damage and the development of more thorough understandings of injury risk. Despite internal load measures of psycho-physiological load speculatively being conceptually linked to athletic injury through training intensity and the efects of psycho-physiological fatigue, these measures are likely too far removed from injury causation to provide meaningful, reliable relationships with injury. Finally, we used a common training load metric as a case study to show how the absence of a sound conceptual rationale and spurious links to causal mechanisms can disclose the weaknesses of candidate measures as tools for altering the likelihood of injuries, aiding the future development of more refned injury risk assessment methods.

1 Introduction

Training loads have been described as the input variable that is manipulated to elicit a desired training response in athletes [[1](#page-10-0)] and can be described as being internal or external depending on whether the measurable aspect in question is occurring internally or externally to the athlete [[1](#page-10-0)]. It follows that a range of internal and external training load measures and metrics exist, with many of these being commonly utilised across the sports science literature. Notably, the monitoring and management of training loads has been an area of substantial interest for practitioners and athletes in sport, with recent interests pertaining to its relationship

Key Points

A clear aetiology between athletic injuries and training load is yet to be established.

Training loads may be related to certain types of injuries through the mechanical load-response pathway, the psycho-physiological load-response pathway, or both. However, the capacity of currently available training load measures and metrics to reflect either of these pathways is notably limited.

Current training load measures and metrics provide unreliable assessments of injury risk. It appears that a more detailed approach centered on the specifc causal mechanisms of injury should be sought to provide more rigorous assessments of injury risk.

with injury. However, despite an abundance of literature, causal pathways between training load, tissue damage and injury remain poorly understood. Understandings of the mechanisms underpinning tissue damage and injury

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are important for evaluating the conceptual viability and limitations of current training load measures and metrics for injury risk assessment. This may, in turn, assist sporting practitioners and researchers to avoid the collection and utilization of redundant or unreliable data, correctly interpret scientifc research fndings, align expectations regarding specifc metrics more appropriately, and assist with the facilitation of future research and the development of training load metrics that assess athletic injury risks more efectively.

While their manifestation or clinical presentation may difer, injury occurs when either singular or repetitive forces are applied to a tissue that result in stresses and strains that exceed tissue strength and repairability [[2–](#page-10-1)[7](#page-10-2)]. It is clear from such mechanisms that mechanical loading (the forces experienced by specifc biological tissues) is a fundamental contributor to athletic injury. Accordingly, within this context, current training load measures and metrics should be considered based on their representation of the mechanical load-response pathway i.e., the mechanical loading experienced and the internal stress, strain, and subsequent mechanically induced tissue damage that ensues. Although athletic injuries share the common characteristic of mechanical loading, certain critical features along the causal pathway to injury may vary depending on the wider causal factors that can infuence injury mechanisms and particular injury events [[6\]](#page-10-3). For this reason, it seems prudent to also consider specifc training load measures and metrics relative to the psycho-physiological loadresponse pathway, which may also infuence injury risk.

A variety of internal and external training load measures and relative metrics are commonly reported across the research and sporting landscape. These metrics differ in value and applicability regarding injury risk quantifcation and their capacity to refect causal pathways to injury. Recently, metrics that claim to allow for the utilisation of a variety of training load measures, such as the acute:chronic workload ratio (ACWR) have also been proposed [\[8\]](#page-10-4). Accordingly, this paper seeks to clarify conceptual understandings relating training load to injury and investigates how currently available training load measures and metrics may relate to the causal pathways of tissue damage and injury incidence. This will be undertaken by evaluating the mechanical and psycho-physiological load-response pathways, whilst also considering the loading patterns experienced and the non-linear relationship between load magnitude and damage [[5,](#page-10-5) [9](#page-10-6), [10\]](#page-10-7). A further purpose of this article is to provide a conceptual foundation for the selection and evaluation of training load measures and metrics when forming etiological models and assumptions, contributing a stronger conceptual basis for future injury research studies. For detailed defnitions of relevant nomenclature in this article, please see Table [1](#page-2-0).

2 Training Load and the Mechanisms of Tissue Damage and Injury

To understand the causal pathways to injury, it is important to address the core mechanical principles surrounding mechanical fatigue damage and failure in biological tissues (e.g., bones, muscles, tendons etc.). Mechanical failure occurs when the ultimate strength of a material is surpassed by excessive stress and strain induced by the application of a singular high-magnitude force, or when repeated applications of sub-ultimate loads exceeds the material's fatigue strength [\[7](#page-10-2)]. Within this context, "stress" is defned as the force per unit of area and is descriptive of the internal forces neighbouring particles of a given material exert on one another, while "strain" is defned as the amount of deformation expressed as a normalized change in shape or size [[11,](#page-10-8) [12\]](#page-10-9). Whilst it is important to acknowledge that human tissue resides within a dynamic environment whereby physiological processes contribute to function, remodelling and recovery, tissues are also materials that exhibit many of the same fundamental principles as non-biological materials in response to applied forces $[5, 13]$ $[5, 13]$ $[5, 13]$ $[5, 13]$ $[5, 13]$. Accordingly, whilst tissue pathology may be an important factor that contributes to various injuries, without the application of force and the stresses and strain that ensue $[2, 3, 5-7, 14]$ $[2, 3, 5-7, 14]$ $[2, 3, 5-7, 14]$ $[2, 3, 5-7, 14]$ $[2, 3, 5-7, 14]$ $[2, 3, 5-7, 14]$ $[2, 3, 5-7, 14]$ $[2, 3, 5-7, 14]$, athletic injury does not occur. It follows that the vast majority of, if not all, contact and non-contact athletic injuries occur as a result of exposure to either singular, or repetitive, applied forces $[5, 15-17]$ $[5, 15-17]$ $[5, 15-17]$ $[5, 15-17]$.

Supporting mechanical loading as a fundamental contributor to athletic injury occurrence, a recent review highlighted that biological tissues demonstrate exponential relationships between the force applied to a specifc tissue and the number of load cycles to failure [[10](#page-10-7)]. Although the majority of these studies were conducted in vitro [[18–](#page-10-15)[27\]](#page-10-16), in vivo animal studies that utilized a variety of loading conditions have also been conducted. These studies investigated the infuence of tissue loading on tendons and cartilage in rats [[28](#page-10-17)] and mice [[29](#page-10-18)], respectively, demonstrating several interactions between the critical musculoskeletal risk factors of force and repetition in relation to tissue damage and inflammation across a variety of tissues. Furthermore, epidemiological studies that have examined a force–repetition interaction have shown a pattern of risk consistent with a mechanical fatigue failure process [[9](#page-10-6), [30\]](#page-10-19). Importantly, these fndings also support suggestions that various markers of mechanically induced tissue damage, such as muscle damage [\[31,](#page-10-20) [32\]](#page-10-21), kinked fbers in tendons [[33\]](#page-10-22), and microcracks in bone [[34](#page-10-23)], may act as precursors to more severe injury [[14,](#page-10-12) [35](#page-11-0)[–38](#page-11-1)]. Of notable recent signifcance, the accumulation of collagen molecular unfolding has been identifed as

Table 1 Relevant nomenclature

the "micro-damage" mechanism of cyclic fatigue damage and failure in collagenous tissues [[39](#page-11-2)]. Considering the prominent role of mechanical loading in tissue damage accumulation and injury occurrence, there have recently been calls to explore musculoskeletal injury, and more specifcally overuse injury [\[5\]](#page-10-5), as a mechanical fatigue phenomenon [[5,](#page-10-5) [9](#page-10-6), [10\]](#page-10-7). This is a most prudent suggestion considering the growing body of research demonstrating that several tissues follow a number of common engineering principles regarding mechanical fatigue [\[9](#page-10-6), [10](#page-10-7), [40\]](#page-11-3).

Although the contributions of mechanical loading to tissue damage formation are well established, it is important to note that further tissue damage may emanate through physiological mechanisms (Fig. [1](#page-3-0)). This additional tissue damage is facilitated by cellular mediated processes and apoptosis

that form part of the remodelling and tissue recovery process [[41,](#page-11-4) [42](#page-11-5)], initiated in response to mechanical loading and the mechanically induced tissue damage that ensues [\[41,](#page-11-4) [42](#page-11-5)]. Accordingly, for the purposes of this paper, these processes are considered supplementary to mechanical loading and the mechanically induced tissue damage that presents, and further exploration of these processes falls outside the scope of this article. Despite this, the contributions of these processes to tissue damage, pathology, overuse/gradual onset injury and recovery remain acknowledged. It is also worth noting that certain additional mechanical factors such as strain rate may also be of importance to damage and injury outcomes; however, deeper exploration of this aspect is similarly beyond the scope of this article.

Considering the evidence supporting mechanical loading and a mechanical fatigue failure process as being etiologically relevant to tissue damage, investigation of a mechanical fatigue phenomenon in athletic populations is needed. However, the accurate quantifcation of mechanical loading at the tissue-specifc level is essential to this process. It follows that within the context of injury, available training load measures and metrics should be evaluated based on their capacity to quantify or refect mechanical loading and the mechanical load-response pathway, tissue damage and subsequent injury.

2.1 Mechanical Load and the Mechanical Load‑Response Pathway

Considering the evident contributions of mechanical loading to tissue damage accumulation and injury, it is worthwhile exploring some key concepts that underpin the mechanical load-response pathway within a sports setting. Within this context and for the purposes of this paper the force applied to a tissue is referred to as the mechanical load, whilst the stress and strain that results in mechanically induced tissue damage is referred to as the mechanical load-response.

Although the mechanical load-response pathway is of heightened relevance to tissue damage outcomes, a series of challenges surrounding its quantifcation exist within athletic settings. Most notably, the tissue response (stress and strain) is not solely dependent upon the force applied to a tissue, but also to additional factors such as tissue morphology and material properties including tissue cross-sectional area, density and stifness [[2,](#page-10-1) [5,](#page-10-5) [6](#page-10-3), [14\]](#page-10-12). This makes the accurate quantifcation and assessment of the mechanical load-response pathway, depicted in Fig. [2](#page-4-0), extremely challenging. Despite these infuencing factors, attempts to quantify the internal forces experienced by specifc tissues have been made [[43](#page-11-6)[–46\]](#page-11-7), with such endeavours requiring the insertion of optic fibres $[43, 44]$ $[43, 44]$ $[43, 44]$ $[43, 44]$ or strain gauges $[45]$ $[45]$ into various tissues. However, these methods often require

laboratory-based settings, and their typically invasive nature makes their application problematic in applied sporting settings. Accordingly, the non-invasive, accurate quantifcation of the mechanical loads experienced by specifc tissues is a more feasible alternative that would provide value to injury risk assessments. Furthermore, the accurate quantifcation of mechanical loading may open up exciting possibilities regarding the formation and application of computational models for determining the mechanical load-responses of specific tissues [\[46](#page-11-7)[–50\]](#page-11-10).

The measurement and modelling of forces is common practice in laboratory-based settings. However, a number of challenges regarding the measurement of mechanical loads exist within applied settings [[49\]](#page-11-11). For this reason, the development of appropriate, more convenient, proxy measures of force may hold considerable value. A range of external training load measures are currently used in applied sport settings, with the use of certain spatio-temporal measures, such as those derived from global positioning systems (GPS) and accelerometers, being common practice. However, the capacity of current popular external load measures and metrics, such as those derived from GPS, to accurately quantify mechanical loading in a reliable and valid capacity is unviable, especially when considering the movement patterns and variable loadings typically experienced by athletes. This concern is further emphasised when considering the lack of precision with which this equipment can quantify certain spatio-temporal variables such as changes in velocity $[51, 52]$ $[51, 52]$ $[51, 52]$ $[51, 52]$ $[51, 52]$ or high-speed running $[51, 53]$ $[51, 53]$ $[51, 53]$ $[51, 53]$, as well as other potentially relevant activities such as collisions [[54](#page-11-15)]. Accordingly, GPS does not provide a feasible proxy measure of the mechanical loadings experienced by specifc tissues and these shortcomings inevitably contribute to many of the inconsistent results associating GPS data with injury $[55-57]$ $[55-57]$.

To improve upon estimates of tissue damage and athletic injury risks, external training load measures and metrics should be included or dismissed based on their capacity

Fig. 1 Conceptual figure highlighting contributions to tissue damage in athletes

Fig. 2 A proposed sequence of steps required to enable a more precise assessment of the mechanical load-response pathway

to represent force and repetition. Importantly, considering the consistent statistical interactions between force and repetition that have been reported in relation to musculoskeletal disorders [\[9](#page-10-6)], the efects of force and repetition must be specifcally explored in combination [\[5](#page-10-5), [10](#page-10-7)], as the isolated efects of these components would provide unreliable estimates of risk in the presence of an interaction [\[58](#page-11-18)]. Notably, when determining proxies of force and repetition, appropriate measures would be expected to difer between specifc sporting contexts. For example, in baseball, acceptable proxies for pitchers may be centred on the number of pitches thrown and the forces focused around the upper body, pitching arm and its various components. However, in running, the number of steps taken may act as an appropriate proxy for the number of load cycles, while ground reaction forces (GRF), lower limb accelerations or running speed may provide the best available measures, or proxy measures, of force acting on the lower body and its various components.

It has been suggested that acceleration-based metrics utilising accelerometers or other wearable technologies may assist with the accurate quantifcation of tissue-specifc [[55,](#page-11-16) [56](#page-11-19), [59\]](#page-11-20) and whole-body [\[60](#page-11-21)] mechanical loading. Although these technologies are widespread and have demonstrated potential for the accurate estimation of GRFs [[61,](#page-11-22) [62](#page-11-23)], and GRFs have been associated with various types of injury such as patellofemoral pain, plantar fasciitis and Achilles tendinopathy [\[63](#page-11-24)], considerable limitations to this approach should be noted. Specifcally, the accelerations of body segments and the correlates of GRF impact peaks [[64,](#page-11-25) [65\]](#page-11-26) or loading rates [[63\]](#page-11-24) that are commonly derived from current running wearable technologies are not equivalent to the forces experienced by specifc tissues inside the body (e.g., bones, muscles, tendons) [[66\]](#page-11-27). It follows that even a seemingly ecologically valid metric such as GRF may poorly refect the loads experienced at a tissue-specifc level [[47,](#page-11-28) [66](#page-11-27), [67](#page-11-29)]. This concern is emphasized when considering that the distribution of forces across specifc tissues is typically unknown, while peaks in GRF often do not coincide temporally with the peak forces experienced by specifc tissues [[66,](#page-11-27) [68](#page-11-30)]. This can, in part, be attributed to the majority of mechanical loading being internally sourced for certain tissues, e.g., bone loading is primarily due to muscle contractions [[14](#page-10-12)]. Importantly, the shortcomings of current wearable technologies are notably problematic as modest errors in the measurement of the exact forces experienced by specifc tissues result in notably large errors when attempting to estimate tissue damage [[69\]](#page-11-31). Accordingly, the validity and reliability of these potential proxies remain questionable and their limitations emphasized. Currently, these tools may provide the best available feld-based estimates of mechanical loading; however, their relationship to the internal forces experienced by biological tissues should be viewed with extreme caution. Recent research has shown promising approaches using novel measurement modalities [\[70\]](#page-11-32) or multiple wearable sensors in combination with biomechanics and machine learning [[69](#page-11-31)] to provide targeted estimates of the loading experienced by specifc biological tissues. Detailed, tissue-specifc approaches such as these are encouraged as the accurate estimation or quantifcation of the actual forces experienced by biological tissue is of utmost importance. This will allow for the non-linear relationship between loading magnitude and damage to be accounted for and permit the valid application of damage estimation methods, enabling more reliable assessments of injury risk.

2.2 Estimating Mechanically Induced Damage: The Non‑linear Relationship Between Load Magnitude and Damage

To assist with the determination of material damage accumulation, validated methods for predicting and estimating damage accumulation have been formed [\[71,](#page-11-33) [72\]](#page-11-34). Such methods specifcally assist with capturing the combined efects of stress magnitude and the number of load cycles on material fatigue damage, which when excessive, eventually results in failure. Considering the evidence demonstrating that biological tissue follows many of the same principles as non-biological material when exposed to repetitive mechanical loads, particularly regarding mechanical fatigue and microdamage formation [[13](#page-10-10), [33,](#page-10-22) [34](#page-10-23), [73](#page-11-35), [74\]](#page-12-0), proposals suggesting the application of these methods for determining cumulative tissue damage and assessing injury risk within a sporting context [[5\]](#page-10-5) are most appropriate. One of the earliest examples of such a method is the Palmgren–Miner rule [[71,](#page-11-33) [72\]](#page-11-34). As recently empha-sized by Edwards and others [[5](#page-10-5), [9](#page-10-6), [10](#page-10-7), [71](#page-11-33)], an important feature of commonly used damage accumulation estimation methods, such as the Palmgren–Miner rule, is that they recognise the non-linear relationship between load magnitude and damage [[5](#page-10-5), [71](#page-11-33), [72](#page-11-34)], depicted in Fig. [3.](#page-5-0) Such is the infuence of this relationship, a 10% reduction in stress generally is associated with a corresponding 100% increase, or more, in the number of cycles to failure [[13\]](#page-10-10). It follows that cumulative damage can vary substantially depending on the loading pattern experienced (exact combination of loading magnitude and number of loading cycles), even when cumulative loads are similar [[5](#page-10-5), [71,](#page-11-33)

Fig. 3 Theoretical stressed-life plot (S–N curve) for a material subjected to cyclic loading demonstrating the non-linear relationship between load magnitude and damage. Fatigue life is defned as the number of cycles to failure N_f at a particular stress magnitude σ . Reproduced from Edwards with permission [\[5](#page-10-5)]

[72\]](#page-11-34). This concept holds particular relevance to athletic injury risk determination considering the variable loading regimens (combinations of loading magnitudes and loading cycles) typically experienced by athletes.

Notably, the non-linear relationship between peak stress magnitude (induced by an applied load) and the number of cycles to failure is well described by an inverse power law, which describes the stress-life relationship of a material using a power function (Eq. [1](#page-5-1)). Within this function, N_f is the number of cycles to failure, *A* is a proportionality constant, σ is the stress magnitude, and *b* is the slope of the S–N curve

$$
N_{\rm f} = A \cdot \sigma^{-b}.\tag{1}
$$

While the inverse power law model and damage accumulation estimation methods are useful for the approximation of fatigue damage, there are limitations, such as the inability to account for localised stress concentrations or changes in molecular orientation [[75,](#page-12-1) [76](#page-12-2)]. Considering many of these challenges, engineers typically do not seek to determine an exact point of failure but commonly attempt to determine a failure range and the probability of failure which may be most appropriate for athletic injury risk determination. For a more detailed examination of damage accumulation estimation methods within biological tissues and athletic specifc contexts, the reader is directed to an article by Edwards [[5\]](#page-10-5) on modelling overuse/gradual onset injuries as a mechanical fatigue phenomenon.

2.3 Measures of Internal Load and the Psycho‑Physiological Load‑Response Pathway

Although mechanical loading and the mechanical loadresponse pathway hold considerable conceptual relevance to tissue damage accumulation and injury, an additional pathway that requires attention is the psycho-physiological load-response pathway. This pathway is concerned with the psycho-physiological responses of an athlete to a given training stimulus, which may be related to injury. Measures of physiological (e.g., heart rate, lactate concentrations, etc.) and psycho-physiological [e.g., rating of perceived exertion (RPE)] internal loads are commonly used across the sporting landscape to assess the internal psycho-physiological responses of an athlete to an applied training stimulus [[1,](#page-10-0) [77](#page-12-3)]. Although stress and strain are internal to an athlete and can therefore be categorized as a measure of internal load, in both training and research settings, internal load measures typically refer to those that are psycho-physiological in nature. Accordingly, within this section, internal load primarily refers to the psycho-physiological stress experienced by an athlete. However, considering that there are interrelations between psycho-physiological functioning, tissue properties, and mechanical loading [[6](#page-10-3)], both the mechanical load-response and psycho-physiological load-response pathways are, indeed, somewhat interrelated. Despite this, it is worth noting that psycho-physiological responses to any given external stimulus are highly variable and individualised. Accordingly, despite the interrelation between the relevant pathways, external training load metrics and mechanical loading do not necessarily refect internal psycho-physiological loads and should not be used to assess the psycho-physiological load-response pathway. Despite this limitation, psycho-physiological responses to external training loads may conceptually be related to injury outcomes based on activity intensity and a range of factors potentially related to the relative psycho-physiological stress experienced, such as psycho-physiological fatigue and alterations to psycho-physiological functioning. These relationships are based on an increased risk of a sudden traumatic injury event occurring or an increase in various tissue loadings due to a range of potential psycho-physiological fatigue-related factors, such as those related to neuromuscular functioning, i.e., impairments in technique [[78,](#page-12-4) [79\]](#page-12-5), motor coordination [\[78](#page-12-4)], muscle activation timing [[78,](#page-12-4) [80](#page-12-6)], muscle functioning [78, [79](#page-12-5)], as well as other factors such as changes in psychological state [\[81](#page-12-7)]. Despite these conceptual links, the evidence supporting current measures of internal load as acceptable proxies of these factors is scarce and the contributions of many of these factors to injury incidence remain uncertain and likely highly variable.

The aforementioned concerns are augmented when considering the growing body of research contesting the relationship between certain injury types, such as anterior cruciate ligament injuries, and psycho-physiological fatigue [[80](#page-12-6)–[85](#page-12-8)]. Despite this, the relevance of the relationship between psycho-physiological fatigue and injury likely varies between specifc injury types and may, therefore, be more applicable and causally related to certain types of injury [[78\]](#page-12-4) compared to others [\[82,](#page-12-9) [85\]](#page-12-8). Of further importance, although measures of internal load may act as acceptable proxies of psycho-physiological load, they are not actual measures of psycho-physiological fatigue [\[86](#page-12-10)] nor do they accurately refect the mechanical load experienced since the same psycho-physiological loads can be associated with different mechanical stimuli. It follows that, for the above-mentioned reasonings, current measures of psycho-physiological load cannot account for mechanically induced tissue fatigue damage, negating its relevance to tissue deterioration and particularly overuse/gradual onset injuries. Considering the aforementioned concerns, metrics that utilise these measures would expectedly show inconsistent and unreliable fndings with injury, which certainly appears to be the case [\[85](#page-12-8)[–91](#page-12-11)].

2.4 Association is Not Causation: Cumulative Load and Exposure Time

An important consideration when examining relationships between certain risk factors and injury is that the association of variables does not necessarily imply causation [[92,](#page-12-12) [93](#page-12-13)]. This is a commonly reiterated mantra within the scientifc community [\[92,](#page-12-12) [93](#page-12-13)] which holds considerable relevance to the training load–injury relationship, especially when considering the underlying relationship between exposure time and cumulative load. Within this context, exposure time refers to the length of time that an athlete is exposed to a particular activity that puts them at risk of injury i.e., matches, training, sprinting, etc. and which is also used as the denominator when calculating the risk of injury. This should not be confused with previous exposure time to an activity and the accumulation of chronic loads, which may infuence the injury risk for subsequent exposures [\[94](#page-12-14), [95](#page-12-15)]. It is well established that injury risk increases with exposure time [\[96](#page-12-16)]. This is a logical, positive relationship as the longer the exposure time, the longer an athlete's exposure to the very activity and environment that puts the athlete at risk of injury [[97](#page-12-17)]. Accordingly, injury risk is commonly expressed relative to exposure time which sets a time paradigm within which risk can be assessed [\[97](#page-12-17)]. It follows that altering the associated time period would inevitably modify the risk.

Similar to the injury risk–exposure time relationship is the inherent positive relationship between cumulative load and exposure time. Considering that training loads are accumulated over time, the longer the exposure time to a given activity the more time is aforded for load accumulation. The acknowledgement of this relationship is of high importance as the associations between training load and injury would expectedly be infuenced and strengthened by exposure time acting as a confounder. Accordingly, it follows that the associations established between injury and training load are not necessarily causal for many injury types, and signifcant associations and alterations to injury risk may arise as a mere refection of the exposure time–injury relationship, depending on the analysis conducted. This holds particular relevance considering the growing body of recent literature challenging fatigue as an important risk factor for certain injury types [[80–](#page-12-6)[85](#page-12-8)] that have commonly been associated with training loads.

Establishing causal relationships between cumulative load, fatigue (mechanical and psycho-physiological) and injury is critical to the formation of appropriate injury risk mitigation strategies, as per the popularised 'sequence of prevention' for sports injuries [\[98\]](#page-12-18). Importantly, if training load is not causally related to certain injuries, injury risk mitigation strategies that are based on managing training loads may be simply infuencing injury risk by manipulating exposure times. Although managing injury risk based on exposure time management may still be appropriate within specifc contexts and circumstances, such strategies may also be harmful to the performance, training, and developmental goals of the athlete and must therefore be implemented with caution. To determine whether training load is indeed causative to injuries, the contributions of fatigue, both mechanical and psycho-physiological, to specifc types of injuries needs to be explored in detail and their contributions to injury aetiology established and not just assumed. Such causal understandings will assist in the determination of the appropriateness of specifc injury risk mitigation strategies and training load metrics, such as the ACWR which will be discussed in the following section, to inform injury risk mitigation strategy.

3 The Acute:Chronic Workload Ratio: A Case Study

Of recent interest in sport science is the application of a training load metric called the "acute:chronic workload ratio" (ACWR) which has been proposed as a 'valid' measure for quantifying and reducing the risk of athletic injury [\[8](#page-10-4)]. Notably, the ACWR has gained substantial traction within the sport science literature with over 100 studies existing on the topic [\[99](#page-12-19)]. Although recent studies and articles have highlighted a number of computational concerns [\[98–](#page-12-18)[102](#page-12-20)], and the relationships exhibited between this metric and injury have recently been revealed to be caused by statistical artefact [\[99](#page-12-19)], the widespread popularity and application of this metric $[8, 101-105]$ $[8, 101-105]$ $[8, 101-105]$ $[8, 101-105]$ $[8, 101-105]$ as well as the spurious etiological foundations that underpin it, justify the ACWR as an ideal, topical case study from which more advanced, conceptually sound measures of injury risk assessment may be developed.

The ACWR metric was created with the intention of quantifying injury risk based on the efect of acute changes in athletic workloads and was based on Banister's Fitness-Fatigue concept [[106\]](#page-12-23). However, although there is some evidence to support certain facets of the ACWR, even when considering more recent variations such as the use of exponentially weighted moving averages, a number of major conceptual faws exist. Some of these include the interchangeable use of a variety of training load measures and metrics as input variables into the ratio [[107,](#page-12-24) [108](#page-12-25)], the limitations of these current inputs, the inability to account for mechanical loading, the lack of tissue-specifc measures of strength or loading, the absence of the non-linear relationship between load magnitude and damage, as well as more generally, the questionable relationship between training load, fatigue and the causal mechanisms of many types of injury.

3.1 Conceptual Basis of the Acute:Chronic Workload Ratio

Evaluating the conceptual strengths, limitations, and overall viability of the ACWR is benefcial to the development of understandings regarding training load and injury, and serves as an example of the need for a detailed reasoning when proposing a metric or any measure as a proxy of causal mechanisms of injuries. Fundamentally, this ratio was not proposed as an indication of long-term undertraining or overtraining, but rather as an indication of excessive acute (e.g., 1-week) changes in load relative to an athlete's chronic (3–6 weeks) load exposure [\[8](#page-10-4)]. Within this interpretation, the ACWR was proposed as a tool to assess the injury risk associated with acute changes in workload. Specifcally, when the acute load rises relative to the chronic load, a higher value presents, bestowing an increased risk of injury. For a given acute load, an athlete with a high chronic load yields a lower score, which has been suggested to be indicative of a lower injury risk [\[103](#page-12-26), [104,](#page-12-27) [109](#page-12-28)]. Although it has been suggested that a "sweet spot" exists, whereby one can maximise net performance potential by having an appropriate training load, while limiting the negative consequences of training [[8\]](#page-10-4), typically, heightened chronic loads are considered to have a protective efect on an athlete. If the chronic load is low, the athlete is less resilient and presents with a heightened score for a given acute load. An excessive rise in acute load has been termed a 'spike' in workload, which has been associated with an increased injury risk [[55,](#page-11-16)

[103,](#page-12-26) [109\]](#page-12-28). While this rationale appealed to practitioners, when examined from a conceptual perspective, taking into account many of the concepts presented earlier within this article, considerable weaknesses emerge.

3.2 Deconstructing the Acute:Chronic Workload Ratio

3.2.1 Chronic Load: A Proxy for Athlete Resilience?

Within the ACWR, chronic workload represents the rolling average of the most recent 3–6 weeks of training. In this respect, it is maintained that chronic training loads are analogous to a state of 'ftness' which may protect against injury [\[8\]](#page-10-4), and is the sole protective input into the ratio. Accordingly, for chronic loads to be the sole protective input for the ratio, one must assume that 3–6 weeks of chronic load data acts as a viable proxy measure of the multitude of factors contributing to athlete resilience. Although some authors have suggested that chronic loads are protective for an athlete [[95,](#page-12-15) [110\]](#page-12-29), and there is evidence to support that adequate physical preparation reduces injury risk [\[94](#page-12-14), [95,](#page-12-15) [110,](#page-12-29) [111](#page-13-0)], there are a number of limitations related to this concept that warrant attention. At the forefront, chronic loads are not a measure of the myriad of physical competencies that may have protective effects on athletes, nor are chronic loads a measure of tissue resilience. Of particular concern regarding this is the "one size fts all" approach highlighted by the variety of external and internal training load measures that have commonly been used as input measures and metrics into the ACWR, such as various GPS-derived measures [\[103,](#page-12-26) [105](#page-12-22), [112](#page-13-1)] and RPE [[87,](#page-12-30) [90](#page-12-31), [109\]](#page-12-28). As explored previously, the various training load measures and metrics have difering purposes and are not interchangeable. Furthermore, their contributions to athlete and tissue resilience range from nil to variable. These concerns are further reinforced when considering that recovery, strength training, mechanical loadings and inter-athlete diferences are not properly accounted for or excluded entirely from the ACWR and the various input variables.

Although its simplicity is enticing and within certain sporting contexts chronic load data may act as a tentative quasi-indicator of athlete resilience, it must be acknowledged that athlete resilience is a complex phenomenon that is often developed over large periods of time [\[113](#page-13-2), [114](#page-13-3)] and incorporates a number of tissues and risk factors [\[14](#page-10-12), [115,](#page-13-4) [116](#page-13-5)] that may have weak or non-existent relationships with the variety of training load measures and metrics available. While a simple surrogate measure of athlete resilience is attractive, careful metric input selection is stressed and caution surrounding the limitations of chronic loads acting as a valid proxy measure of athlete resilience are emphasized.

3.2.2 Acute Load

Within the calculation of the ACWR, acute training loads can be as short as one session or as long as one week. In this respect, it has been suggested that acute training loads are analogous to a state of 'fatigue' that, when excessive relative to chronic load, leads to injury. Accordingly, for acute load to be the sole negative function of the ACWR calculation is to imply that acute training loads act as the primary stimulus for injury occurrence. This is a most concerning assumption as although acute loads may act upon the psycho-physiological and mechanical load-response pathways, a rigorous causal explanation of justifcation for this assumption does not exist. Of immediate concern, the metric is not tissue or injury specifc, and tissue loading is not accounted for in any valid capacity within any of the input variables currently available. Furthermore, the relationship between the psycho-physiological load-response pathway and injury remains ambiguous. When also considering that tissue damage accumulation is not estimated in any capacity, acute loads are not a measure of psycho-physiological fatigue, and many injuries appear to occur in a manner that is largely independent of prior training load and psycho-physiological fatigue [\[80](#page-12-6)[–84](#page-12-32)], it is evident that any potential causal explanation is not only speculative but also unsupported by a sound rationale. Of additional concern, the "appropriateness" of the time windows used to capture the acute load is often justifed by the training schedule $[117, 118]$ $[117, 118]$ $[117, 118]$ $[117, 118]$ $[117, 118]$, with it having been suggested that in team sports, 1 week of training appears to provide a logical and convenient unit [\[8](#page-10-4)]. Such justifcations are, therefore, based on convenience as opposed to physiological, mechanical or mechanistic reasoning. Accordingly, considerable doubts regarding the relevance of acute training loads to injury causality are evident in the current application of the ACWR. There is currently no evidence or even conceptual framework supporting acute training loads as being causal to injury and minimal evidence exists supporting the inclusion of acute training loads as the main negative causal factor determining injury risk.

3.2.3 ACWR Summary

When proposing and selecting a metric as a proxy of factors involved in a causal pathway, the examination of its plausibility from a mechanistic perspective is crucial. From this perspective, it is evident there are substantial conceptual shortcomings of the ACWR and similar metrics, which are simplistic attempts to capture injury risk for a number of injury types with varying mechanisms, across a variety of tissues, and within diferent sporting contexts. When considering that there is no attempt to estimate cumulative tissue damage, and neither tissue strength, mechanical loads, recovery, or the plethora of other factors contributing to a given injury event appear to be quantifed in any meaningful capacity in the ratio, the ACWR (or similarly developed metrics) appears to be a poor proxy of the mechanisms of injury. Furthermore, the relationship between the psycho-physiological load-response pathway and injury remains uncertain, and many injuries may occur largely independent of prior training loads, proliferating the considerable limitations of this particular metric. Along with the noted statistical artefact [\[99](#page-12-19)], these aspects contribute to the diverse fndings evident with the ACWR, whereby associations with injury have been shown in a multitude of contradictory directions, or not at all [\[8](#page-10-4), [87](#page-12-30), [87](#page-12-30)[–91,](#page-12-11) [103,](#page-12-26) [109,](#page-12-28) [118](#page-13-7), [119](#page-13-8), [122](#page-13-9), [123\]](#page-13-10). Any measure or metric used as a potential causal factor for injury should be conceptually scrutinised prior to its application within applied research. This is also the case in exploratory studies when the derived associations are used to develop hypotheses. A thorough understanding of the roles of the factors related to injury risk is fundamental to develop causal structures and aetiological theories to be tested. The use of the ACWR as a case study was presented here to emphasise the need for metrics with a rigorous underlying rationale and to focus research on metrics with strong conceptual foundations.

4 Conclusion

Considering the fundamental contributions of mechanical loads to injury occurrence, attempts to relate injury and training load should seek to determine and apply appropriate measures of force and the number of load cycles, or appropriate surrogate measures of these. This approach will more closely refect the mechanical contributions to tissue fatigue and failure and may allow for the application of models that account for the mechanical load-response of specifc tissues. Internal load measures of psycho-physiological load, although somewhat conceptually related to injury, provide limited insight into tissue resilience, the loading of various tissues, specifc injury mechanisms, or the array of factors that infuence an injury event. It follows that these measures are likely too far removed from injury causation to provide meaningful, reliable relationships with injury.

When the example of the ACWR, a highly popularised method of estimating injury risk is scrutinised, it is evident that the ACWR possesses a number of limitations and conceptual faws. While a 'one size fts all' injury risk quantifcation is attractive, the multifaceted and complex occurrence of athletic injury appears to require a more detailed approach. Understanding whether the manipulation of a given variable can alter the likelihood of a future event necessitates the implementation of well conducted experimental studies or estimations from observational studies whereby causal structures are defned a priori. Forcing

explanations attempting to justify "signifcant" study results can generate involuntary HARK-ing (hypothesizing after the results are known) [\[120](#page-13-11)].

To advance injury research and understandings, it is recommended that a superior approach to quantifying tissue injury risk is undertaken. Such an approach would rely upon focussing efforts towards a tissue-specific, injury mechanism-specifc, and sport-specifc approach, basing such enquiry on the development and utilisation of detailed conceptual frameworks. This approach will encourage researchers to better understand the mechanisms and causal pathways that contribute to athlete and tissue resilience, tissue loading, and specifc types of injury within particular sporting contexts, and facilitate the investigation of various causal links and assumptions. To move training load research forward in this area, it is recommended that researchers focus efforts towards developing innovative methods to quantify the mechanical loads experienced by specifc tissues, with recent approaches potentially serving as inspiration for such endeavours [\[69](#page-11-31), [70\]](#page-11-32). Approaches such as these may encourage the development and application of computational models that accurately describe tissue behaviour and open up new possibilities regarding the accurate estimation of cumulative tissue damage. Of additional importance, it is essential that the contributions of psycho-physiological fatigue to specifc types of injury are established, while researchers must also continue to develop methods to monitor and assess tissue health and strength in applied settings.

Considering the shortcomings of currently available training load metrics and data when refecting causal pathways to injury, it is recommended that the utilisation of currently available training load metrics and data for injury risk assessment and manipulation should be avoided as such assessments have proven unreliable. Accordingly, it is recommended that training load data should be primarily utilized for monitoring whether an athlete is undertaking what is prescribed, along with contributing to the assessment of how an athlete is coping with the prescribed loads [[121](#page-13-12)]. In this respect, training load data can continue to inform applied practice and periodization. The evident limitations associated with attempting to quantify injury risk from current training load data imply that extreme caution must be exercised when considering any evident relationships with injury and when utilising such information for decision making processes.

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Declarations

Conflict of Interest Judd Kalkhoven, Mark Watsford, Aaron Coutts, W. Brent Edwards and Franco Impellizzeri declare that they have no conficts of interest.

Author contributions JTK conceived the idea for the article, wrote the frst draft of the manuscript and all versions thereafter. MLW contributed substantially to the editing and conceptual direction of the manuscript. AJC contextualised the information provided in the manuscript within the current climate of training load research. WBE contributed to the tissue engineering and mechanical load components of the manuscript. FMI contributed to the conceptual formation and editing of the manuscript as a whole with a special emphasis to Sect. [3](#page-7-0). All authors read and approved the fnal manuscript.

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References

- 1. Impellizzeri FM, Marcora SM, Coutts AJ. Internal and external training load: 15 years on. Int J Sports Physiol Perform. 2019;14(2):270–3.
- 2. Bahr R, Krosshaug T. Understanding injury mechanisms: a key component of preventing injuries in sport. Br J Sports Med. 2005;39(6):324–9.
- 3. McIntosh AS. Risk compensation, motivation, injuries, and biomechanics in competitive sport. Br J Sports Med. 2005;39(1):2–3.
- 4. Bertelsen ML, Hulme A, Petersen J, Brund RK, Sorensen H, Finch CF, et al. A framework for the etiology of running-related injuries. Scand J Med Sci Sports. 2017;27(11):1170–80.
- 5. Edwards WB. Modeling overuse injuries in sport as a mechanical fatigue phenomenon. Exerc Sport Sci Rev. 2018;46(4):224–31.
- 6. Kalkhoven JT, Watsford ML, Impellizzeri FM. A conceptual model and detailed framework for stress-related, strain-related, and overuse athletic injury. J Sci Med Sport. 2020;23(8):726–34.
- 7. Peterson R. Discussion of a century ago concerning the nature of fatigue, and review of some of the subsequent researches concerning the mechanism of fatigue. ASTM Bull. 1950;164:50–6.
- 8. Gabbett TJ. The training-injury prevention paradox: should athletes be training smarter and harder? Br J Sports Med. 2016;50(5):273–80.
- 9. Gallagher S, Heberger JR. Examining the interaction of force and repetition on musculoskeletal disorder risk: a systematic literature review. Hum Factors. 2013;55(1):108–24.
- 10. Gallagher S, Schall MC Jr. Musculoskeletal disorders as a fatigue failure process: evidence, implications and research needs. Ergonomics. 2017;60(2):255–69.
- 11. Fung YC. Biomechanics: mechanical properties of living tissues. New York: Springer-Verlag; 1981.
- 12. Fung YC. Biomechanics: mechanical properties of living tissues. 2nd ed. New York: Springer-Verlag; 1993.
- 13. Carter DR, Caler WE. A cumulative damage model for bone fracture. J Orthop Res. 1985;3(1):84–90.
- 14. Hart NH, Nimphius S, Rantalainen T, Ireland A, Siafarikas A, Newton RU. Mechanical basis of bone strength: infuence of

bone material, bone structure and muscle action. J Musculoskelet Neuronal Interact. 2017;17(3):114–39.

- 15. Lieber RL, Friden J. Muscle damage is not a function of muscle force but active muscle strain. J Appl Physiol (1985). 1993;74(2):520–6.
- 16. Garrett WE Jr. Muscle strain injuries. Am J Sports Med. 1996;24(6 Suppl):S2-8.
- 17. Crossley K, Bennell KL, Wrigley T, Oakes BW. Ground reaction forces, bone characteristics, and tibial stress fracture in male runners. Med Sci Sports Exerc. 1999;31(8):1088–93.
- Schechtman H, Bader DL. In vitro fatigue of human tendons. J Biomech. 1997;30(8):829–35.
- 19. Wang XT, Ker RF, Alexander RM. Fatigue rupture of wallaby tail tendons. J Exp Biol. 1995;198(Pt 3):847–52.
- 20. Lipps DB, Oh YK, Ashton-Miller JA, Wojtys EM. Morphologic characteristics help explain the gender diference in peak anterior cruciate ligament strain during a simulated pivot landing. Am J Sports Med. 2012;40(1):32–40.
- 21. Lipps DB, Wojtys EM, Ashton-Miller JA. Anterior cruciate ligament fatigue failures in knees subjected to repeated simulated pivot landings. Am J Sports Med. 2013;41(5):1058–66.
- 22. Thornton GM, Schwab TD, Oxland TR. Cyclic loading causes faster rupture and strain rate than static loading in medial collateral ligament at high stress. Clin Biomech (Bristol, Avon). 2007;22(8):932–40.
- 23. Bellucci G, Seedhom BB. Mechanical behaviour of articular cartilage under tensile cyclic load. Rheumatology (Oxford). 2001;40(12):1337–45.
- 24. Brinckmann P, Biggemann M, Hilweg D. Fatigue fracture of human lumbar vertebrae. Clin Biomech (Bristol, Avon). 1988;3(Suppl 1):1-S23.
- 25. Cyron BM, Hutton WC. The fatigue strength of the lumbar neural arch in spondylolysis. J Bone Joint Surg Br. 1978;60-B(2):234–8.
- 26. Gallagher S, Marras WS, Litsky AS, Burr D. Torso fexion loads and the fatigue failure of human lumbosacral motion segments. Spine (Phila Pa 1976). 2005;30(20):2265–73.
- 27. Gallagher S, Marras WS, Litsky AS, Burr D, Landoll J, Matkovic V. A comparison of fatigue failure responses of old versus middle-aged lumbar motion segments in simulated fexed lifting. Spine (Phila Pa 1976). 2007;32(17):1832–9.
- 28. Barbe MF, Gallagher S, Massicotte VS, Tytell M, Popoff SN, Barr-Gillespie AE. The interaction of force and repetition on musculoskeletal and neural tissue responses and sensorimotor behavior in a rat model of work-related musculoskeletal disorders. BMC Musculoskelet Disord. 2013;25(14):303.
- 29. Andarawis-Puri N, Flatow EL. Tendon fatigue in response to mechanical loading. J Musculoskelet Neuronal Interact. 2011;11(2):106–14.
- 30. Harris-Adamson C, Eisen EA, Kapellusch J, Garg A, Hegmann KT, Thiese MS, et al. Biomechanical risk factors for carpal tunnel syndrome: a pooled study of 2474 workers. Occup Environ Med. 2015;72(1):33–41.
- 31. Kuipers H, Drukker J, Frederik PM, Geurten P, van Kranenburg G. Muscle degeneration after exercise in rats. Int J Sports Med. 1983;4(1):45–51.
- 32. Lieber RL, Woodburn TM, Friden J. Muscle damage induced by eccentric contractions of 25% strain. J Appl Physiol (1985). 1991;70(6):2498–507.
- 33. Fung DT, Wang VM, Laudier DM, Shine JH, Basta-Pljakic J, Jepsen KJ, et al. Subrupture tendon fatigue damage. J Orthop Res. 2009;27(2):264–73.
- 34. Herman BC, Cardoso L, Majeska RJ, Jepsen KJ, Schaffler MB. Activation of bone remodeling after fatigue: diferential

response to linear microcracks and diffuse damage. Bone. 2010;47(4):766–72.

- 35. Brockett CL, Morgan DL, Proske U. Human hamstring muscles adapt to eccentric exercise by changing optimum length. Med Sci Sports Exerc. 2001;33(5):783–90.
- 36. Timmins RG, Shield AJ, Williams MD, Opar DA. Is there evidence to support the use of the angle of peak torque as a marker of hamstring injury and re-injury risk? Sports Med. 2016;46(1):7–13.
- 37. Yasui Y, Tonogai I, Rosenbaum AJ, Shimozono Y, Kawano H, Kennedy JG. The risk of Achilles tendon rupture in the patients with Achilles tendinopathy: healthcare database analysis in the United States. BioMed Res Int. 2017;2017.
- 38. Tallon C, Mafulli N, Ewen SW. Ruptured Achilles tendons are signifcantly more degenerated than tendinopathic tendons. Med Sci Sports Exerc. 2001;33(12):1983–90.
- 39. Zitnay JL, Jung GS, Lin AH, Qin Z, Li Y, Yu SM, et al. Accumulation of collagen molecular unfolding is the mechanism of cyclic fatigue damage and failure in collagenous tissues. Sci Adv. 2020;6(35):eaba2795.
- 40. Martin B. A theory of fatigue damage accumulation and repair in cortical bone. J Orthop Res. 1992;10(6):818–25.
- 41. Desmouliere A, Redard M, Darby I, Gabbiani G. Apoptosis mediates the decrease in cellularity during the transition between granulation tissue and scar. Am J Pathol. 1995;146(1):56–66.
- 42. Tomasek JJ, Gabbiani G, Hinz B, Chaponnier C, Brown RA. Myofbroblasts and mechano-regulation of connective tissue remodelling. Nat Rev Mol Cell Biol. 2002;3(5):349–63.
- 43. Komi PV, Belli A, Huttunen V, Bonnefoy R, Geyssant A, Lacour JR. Optic fbre as a transducer of tendomuscular forces. Eur J Appl Physiol Occup Physiol. 1996;72(3):278–80.
- 44. Finni T, Komi PV, Lukkariniemi J. Achilles tendon loading during walking: application of a novel optic fiber technique. Eur J Appl Physiol Occup Physiol. 1998;77(3):289–91.
- 45. Burr DB, Milgrom C, Fyhrie D, Forwood M, Nyska M, Finestone A, et al. In vivo measurement of human tibial strains during vigorous activity. Bone. 1996;18(5):405–10.
- 46. Smith DW, Rubenson J, Lloyd D, Zheng M, Fernandez J, Besier T, et al. A conceptual framework for computational models of Achilles tendon homeostasis. Wiley Interdiscip Rev Syst Biol Med. 2013;5(5):523–38.
- 47. Loundagin LL, Schmidt TA, Edwards WB. Mechanical fatigue of bovine cortical bone using ground reaction force waveforms in running. J Biomech Eng. 2018;140(3).
- 48. Amirouche F, Bobko A. Bone remodeling and biomechanical processes—a multiphysics approach. Austin J Biotechnol Bioeng. 2015;2(2):1–11.
- 49. Verheul J, Nedergaard NJ, Vanrenterghem J, Robinson MA. Measuring biomechanical loads in team sports– from lab to feld. Sci Med Football. 2020;1–7. [https://doi.](https://doi.org/10.1080/24733938.2019.1709654) [org/10.1080/24733938.2019.1709654](https://doi.org/10.1080/24733938.2019.1709654).
- 50. Seth A, Hicks JL, Uchida TK, Habib A, Dembia CL, Dunne JJ, et al. OpenSim: simulating musculoskeletal dynamics and neuromuscular control to study human and animal movement. PLoS Comput Biol. 2018;14(7):e1006223.
- 51. Petersen C, Pyne D, Portus M, Dawson B. Validity and reliability of GPS units to monitor cricket-specifc movement patterns. Int J Sports Physiol Perform. 2009;4(3):381–93.
- 52. Buchheit M, Al Haddad H, Simpson BM, Palazzi D, Bourdon PC, Di Salvo V, et al. Monitoring accelerations with GPS in football: time to slow down? Int J Sports Physiol Perform. 2014;9(3):442–5.
- 53. Johnston RJ, Watsford ML, Kelly SJ, Pine MJ, Spurrs RW. Validity and interunit reliability of 10 Hz and 15 Hz GPS units

for assessing athlete movement demands. J Strength Cond Res. 2014;28(6):1649–55.

- 54. Gabbett TJ. Quantifying the physical demands of collision sports: does microsensor technology measure what it claims to measure? J Strength Cond Res. 2013;27(8):2319–22.
- 55. Ehrmann FE, Duncan CS, Sindhusake D, Franzsen WN, Greene DA. GPS and injury prevention in professional soccer. J Strength Condition Res. 2016;30(2):360–7.
- 56. Colby MJ, Dawson B, Heasman J, Rogalski B, Gabbett TJ. Accelerometer and GPS-derived running loads and injury risk in elite Australian footballers. J Strength Cond Res. 2014;28(8):2244–52.
- 57. Kupperman N, Hertel J. Global positioning system-derived workload metrics and injury risk in team-based feld sports: a systematic review. J Athletic train. 2020;55(9):931–43.
- 58. Meyer DL. Misinterpretation of interaction efects: a reply to Rosnow and Rosenthal. Psychol Bull. 1991;110(3):571–3 (**discussion 4–6**).
- 59. Boyd LJ, Ball K, Aughey RJ. The reliability of MinimaxX accelerometers for measuring physical activity in Australian football. Int J Sports Physiol Perform. 2011;6(3):311–21.
- 60. Raper DP, Witchalls J, Philips EJ, Knight E, Drew MK, Waddington G. Use of a tibial accelerometer to measure ground reaction force in running: a reliability and validity comparison with force plates. J Sci Med Sport. 2018;21(1):84–8.
- 61. Gurchiek RD, McGinnis RS, Needle AR, McBride JM, van Werkhoven H. The use of a single inertial sensor to estimate 3-dimensional ground reaction force during accelerative running tasks. J Biomech. 2017;61:263–8.
- 62. Ancillao A, Tedesco S, Barton J, O'Flynn B. Indirect measurement of ground reaction forces and moments by means of wearable inertial sensors: a systematic review. Sensors. 2018;18(8):2564. [https://doi.org/10.3390/s18082564.](https://doi.org/10.3390/s18082564)
- 63. Hennig EM, Milani TL, Lafortune MA. Use of ground reaction force parameters in predicting peak tibial accelerations in running. J Appl Biomech. 1993;9(4):306–14.
- 64. Crowell HP, Milner CE, Hamill J, Davis IS. Reducing impact loading during running with the use of real-time visual feedback. J Orthop Sports Phys Ther. 2010;40(4):206–13.
- 65. Hamill J, Derrick TR, Holt KG. Shock attenuation and stride frequency during running. Hum Mov Sci. 1995;14(1):45–60.
- 66. Matijevich ES, Branscombe LM, Scott LR, Zelik KE. Ground reaction force metrics are not strongly correlated with tibial bone load when running across speeds and slopes: Implications for science, sport and wearable tech. PLoS ONE. 2019;14(1):e0210000.
- 67. Johnson CD, Tenforde AS, Outerleys J, Reilly J, Davis IS. Impact-related ground reaction forces are more strongly associated with some running injuries than others. Am J Sports Med. 2020;48(12):3072–80.
- 68. Sasimontonkul S, Bay BK, Pavol MJ. Bone contact forces on the distal tibia during the stance phase of running. J Biomech. 2007;40(15):3503–9.
- 69. Matijevich ES, Scott LR, Volgyesi P, Derry KH, Zelik KE. Combining wearable sensor signals, machine learning and biomechanics to estimate tibial bone force and damage during running. Hum Mov Sci. 2020;22(74):102690.
- 70. Martin JA, Brandon SCE, Keuler EM, Hermus JR, Ehlers AC, Segalman DJ, et al. Gauging force by tapping tendons. Nat Commun. 2018;9(1):1592.
- 71. Miner MA. Cumulative damage in fatigue. J Appl Mech. 1945;67:A159–64.
- 72. Palmgren AJN. Die Lebensdauer von Kugellagern. VDI-Zeitschrift. 1924;58:339–41.
- 73. Weightman B, Chappell DJ, Jenkins EA. A second study of tensile fatigue properties of human articular cartilage. Ann Rheum Dis. 1978;37(1):58–63.
- 74. Wren TA, Lindsey DP, Beaupre GS, Carter DR. Efects of creep and cyclic loading on the mechanical properties and failure of human Achilles tendons. Ann Biomed Eng. 2003;31(6):710–7.
- 75. Stephens RI, Fatemi A, Stephens RR, Fuchs HO. Metal fatigue in engineering. New York: Wiley; 2000.
- 76. Roylance DJ. Fatigue. Department of Materials Science and Engineering. Massachusetts Institute of Technology Cambridge, MA, 2139. 2001.
- 77. Drew MK, Finch CF. The relationship between training load and injury, illness and soreness: a systematic and literature review. Sports Med. 2016;46(6):861–83.
- 78. Huygaerts S, Cos F, Cohen DD, Calleja-Gonzalez J, Guitart M, Blazevich AJ, et al. Mechanisms of hamstring strain injury: interactions between fatigue, muscle activation and function. Sports (Basel). 2020;8(5): 65.
- 79. Morin JB, Samozino P, Edouard P, Tomazin K. Efect of fatigue on force production and force application technique during repeated sprints. J Biomech. 2011;44(15):2719–23.
- 80. Butterfeld TA, Herzog W. Efect of altering starting length and activation timing of muscle on fber strain and muscle damage. J Appl Physiol (1985). 2006;100(5):1489–98.
- 81. Liederbach M, Compagno JM. Psychological aspects of fatiguerelated injuries in dancers. J Dance Med Sci. 2001;5(4):116–20.
- 82. Bourne MN, Webster KE, Hewett TE. Is fatigue a risk factor for anterior cruciate ligament rupture? Sports Med. 2019;49(11):1629–35.
- 83. Doyle TLA, Schilaty ND, Webster KE, Hewett TE. Time of season and game segment is not related to likelihood of lower-limb injuries: a meta-analysis. Clin J Sport Med. 2019. [https://doi.](https://doi.org/10.1097/JSM.0000000000000752) [org/10.1097/JSM.0000000000000752](https://doi.org/10.1097/JSM.0000000000000752).
- 84. Zhou J, Schilaty ND, Hewett TE, Bates NA. Analysis of timing of secondary ACL injury in professional athletes does not support game timing or season timing as a contributor to injury risk. Int J Sports Phys Ther. 2020;15(2):254–62.
- 85. Della Villa F, Buckthorpe M, Grassi A, Nabiuzzi A, Tosarelli F, Zafagnini S, et al. Systematic video analysis of ACL injuries in professional male football (soccer): injury mechanisms, situational patterns and biomechanics study on 134 consecutive cases. Br J Sports Med. 2020. [https://doi.org/10.1136/bjsports-](https://doi.org/10.1136/bjsports-2019-101247)[2019-101247](https://doi.org/10.1136/bjsports-2019-101247).
- 86. Halson SL. Monitoring training load to understand fatigue in athletes. Sports Med. 2014;44(Suppl 2):S139–47.
- 87. Raya-Gonzalez J, Nakamura FY, Castillo D, Yanci J, Fanchini M. Determining the relationship between internal load markers and noncontact injuries in young elite soccer players. Int J Sports Physiol Perform. 2019;14(4):421–5.
- 88. Esmaeili A, Hopkins WG, Stewart AM, Elias GP, Lazarus BH, Aughey RJ. The individual and combined efects of multiple factors on the risk of soft tissue non-contact injuries in elite team sport athletes. Front Physiol. 2018;9:1280.
- 89. Jaspers A, Kuyvenhoven JP, Staes F, Frencken WGP, Helsen WF, Brink MS. Examination of the external and internal load indicators' association with overuse injuries in professional soccer players. J Sci Med Sport. 2018;21(6):579–85.
- 90. Fanchini M, Rampinini E, Riggio M, Coutts AJ, Pecci C, McCall A. Despite association, the acute: chronic work load ratio does not predict non-contact injury in elite footballers. Sci Med Football. 2018;2(2):108–14.
- 91. Lolli L, Bahr R, Weston M, Whiteley R, Tabben M, Bonanno D, et al. No association between perceived exertion and session duration with hamstring injury occurrence in professional football. Scand J Med Sci Sports. 2019;30(3):523–530.
- 92. Altman N, Krzywinski M. Association, correlation and causation. Nat Methods. 2015;12(10):899–900.
- 93. Okasha S. Philosophy of science: very short introduction. Oxford: Oxford University Press; 2016.
- 94. Myer GD, Faigenbaum AD, Cherny CE, Heidt RS Jr, Hewett TE. Did the NFL Lockout expose the Achilles heel of competitive sports? J Orthop Sports Phys Ther. 2011;41(10):702–5.
- 95. Rogalski B, Dawson B, Heasman J, Gabbett TJ. Training and game loads and injury risk in elite Australian footballers. J Sci Med Sport. 2013;16(6):499–503.
- 96. Hagglund M, Walden M, Ekstrand J. Exposure and injury risk in Swedish elite football: a comparison between seasons 1982 and 2001. Scand J Med Sci Sports. 2003;13(6):364–70.
- 97. Stovitz SD, Shrier I. Injury rates in team sport events: tackling challenges in assessing exposure time. Br J Sports Med. 2012;46(14):960–3.
- 98. van Mechelen W, Hlobil H, Kemper HC. Incidence, severity, aetiology and prevention of sports injuries. A review of concepts. Sports Med. 1992;14(2):82–99.
- 99. Impellizzeri F, Woodcock S, Coutts AJ, Fanchini M, McCall A, Vigotsky A. What role do chronic workloads play in the acute to chronic workload ratio? Time to dismiss ACWR and its underlying theory. Sports Med. 2020. [https://doi.org/10.1007/s4027](https://doi.org/10.1007/s40279-020-01378-6) [9-020-01378-6](https://doi.org/10.1007/s40279-020-01378-6).
- 100. Lolli L, Batterham AM, Hawkins R, Kelly DM, Strudwick AJ, Thorpe RT, et al. The acute-to-chronic workload ratio: an inaccurate scaling index for an unnecessary normalisation process? Br J Sports Med. 2019;53(24):1510–2.
- 101. Wang C, Vargas JT, Stokes T, Steele R, Shrier I. Analyzing activity and injury: lessons learned from the Acute:Chronic workload ratio. Sports Med. 2020;50(7):1243–54.
- 102. Lolli L, Batterham AM, Hawkins R, Kelly DM, Strudwick AJ, Thorpe R, et al. Mathematical coupling causes spurious correlation within the conventional acute-to-chronic workload ratio calculations. Br J Sports Med. 2019;53(15):921–2.
- 103. Hulin BT, Gabbett TJ, Lawson DW, Caputi P, Sampson JA. The acute:chronic workload ratio predicts injury: high chronic workload may decrease injury risk in elite rugby league players. Br J Sports Med. 2016;50(4):231–6.
- 104. Blanch P, Gabbett TJ. Has the athlete trained enough to return to play safely? The acute:chronic workload ratio permits clinicians to quantify a player's risk of subsequent injury. Br J Sports Med. 2016;50(8):471–5.
- 105. Bowen L, Gross AS, Gimpel M, Li FX. Accumulated workloads and the acute:chronic workload ratio relate to injury risk in elite youth football players. Br J Sports Med. 2017;51(5):452–9.
- 106. Banister E, Calvert T, Savage M, Bach T. A systems model of training for athletic performance. Aust J Sports Med. 1975;7(3):57–61.
- 107. Impellizzeri FM, Ward P, Coutts AJ, Bornn L, McCall A. Training load and injury part 2: questionable research practices hijack the truth and mislead well-intentioned clinicians. J Orthop Sports Phys Ther. 2020;50(10):577–84.
- 108. Impellizzeri FM, McCall A, Ward P, Bornn L, Coutts AJ. Training load and its role in injury prevention, part 2: conceptual and methodologic pitfalls. J Athl Train. 2020;55(9):893–901.
- 109. Hulin BT, Gabbett TJ, Blanch P, Chapman P, Bailey D, Orchard JW. Spikes in acute workload are associated with increased injury risk in elite cricket fast bowlers. Br J Sports Med. 2014;48(8):708–12.
- 110. Windt J, Gabbett TJ, Ferris D, Khan KM. Training load–injury paradox: is greater preseason participation associated with lower

in-season injury risk in elite rugby league players? Br J Sports Med. 2017;51(8):645–50.

- 111. Ekstrand J, Spreco A, Windt J, Khan KM. Are elite soccer teams' preseason training sessions associated with fewer in-season injuries? A 15-year analysis from the Union of European Football Associations (UEFA) elite club injury study. Am J Sports Med. 2020;48(3):723–9.
- 112. Hulin BT, Gabbett TJ, Caputi P, Lawson DW, Sampson JA. Low chronic workload and the acute:chronic workload ratio are more predictive of injury than between-match recovery time: a twoseason prospective cohort study in elite rugby league players. Br J Sports Med. 2016;50(16):1008–12.
- 113. Bailey DA, McKay HA, Mirwald RL, Crocker PR, Faulkner RA. A six-year longitudinal study of the relationship of physical activity to bone mineral accrual in growing children: the university of Saskatchewan bone mineral accrual study. J Bone Miner Res. 1999;14(10):1672–9.
- 114. Kemper HC, Twisk JW, van Mechelen W, Post GB, Roos JC, Lips P. A 15-year longitudinal study in young adults on the relation of physical activity and ftness with the development of the bone mass: the Amsterdam Growth And Health Longitudinal Study. Bone. 2000;27(6):847–53.
- 115. Kalkhoven JT, Watsford M. Mechanical contributions to muscle injury: implications for athletic injury risk mitigation. SportRxiv. 2020;15. [https://doi.org/10.31236/osf.io/a5um4.](https://doi.org/10.31236/osf.io/a5um4)
- 116. Docking SI, Cook J. How do tendons adapt? Going beyond tissue responses to understand positive adaptation and pathology development: a narrative review. J Musculoskelet Neuronal Interact. 2019;19(3):300–10.
- 117. Carey DL, Blanch P, Ong KL, Crossley KM, Crow J, Morris ME. Training loads and injury risk in Australian football-difering acute: chronic workload ratios infuence match injury risk. Br J Sports Med. 2017;51(16):1215–20.
- 118. Grifn A, Kenny IC, Comyns TM, Lyons M. The association between the acute:chronic workload ratio and injury and its application in team sports: a systematic review. Sports Med. 2020;50(3):561–80.
- 119. Suarez-Arrones L, De Alba B, Roell M, Torreno I, Strütt S, Freyler K, et al. Player monitoring in professional soccer: spikes in acute:chronic workload are dissociated from injury occurrence. Front Sports Active Living. 2020;2:75.
- 120. Kerr NL. HARKing: hypothesizing after the results are known. Pers Soc Psychol Rev. 1998;2(3):196–217.
- 121. Impellizzeri FM, Menaspa P, Coutts AJ, Kalkhoven J, Menaspa MJ. Training load and its role in injury prevention, part I: back to the future. J Athl Train. 2020;55(9):885–92.
- 122. Dalen-Lorentsen T, Bjørneboe J, Clarsen B, Vagle M, Fagerland MW, Andersen TE. Does load management using the acute: chronic workload ratio prevent health problems? A cluster randomised trial of 482 elite youth footballers of both sexes. Br J Sports Med. 2020;. [https://doi.org/10.1136/bjsports-2020-10300](https://doi.org/10.1136/bjsports-2020-103003) [3.](https://doi.org/10.1136/bjsports-2020-103003)
- 123. West SW, Williams S, Cazzola D, Kemp S, Cross MJ, Stokes KA. Training load and injury risk in elite rugby union: the largest investigation to date. Int J Sports Med. 2020;. [https://doi.org/10](https://doi.org/10.1055/a-1300-2703) [.1055/a-1300-2703](https://doi.org/10.1055/a-1300-2703).