

Assessment of Fatigue and Recovery in Sport: Narrative Review

Abstract

Fatigue is a phenomenon associated with decreases in both physical and cognitive performances and increases in injury occurrence. Competitive athletes are required to complete demanding training programmes with high workloads to elicit the physiological and musculoskeletal adaptations plus skill acquisition necessary for performance. High workloads, especially sudden rapid increases in training loads, are associated with the occurrence of fatigue. At present, there is limited evidence elucidating the underlying mechanisms associating the fatigue generated by higher workloads and with an increase in injury risk. The multidimensional nature and manifestation of fatigue have led to differing definitions and dichotomies of the term. Consequently, a plethora of physiological, biochemical, psychological and performance markers have been proposed to measure fatigue and recovery. Those include self-reported scales, countermovement jump performance, heart rate variability, and saliva and serum biomarker analyses. The purpose of this review is to provide an overview of fatigue and recovery plus methods of assessments.

Introduction

Fatigue is a phenomenon that has been identified as a significant risk factor associated with decreases in physical and cognitive performance [1] and an increase in injury incidence [2, 3]. Acute fatigue associated with physical and mental stress is expected to improve or resolve with appropriate recovery. Risk of injury is often closely associated with lower levels of endurance, fitness and strength, and good structural, biomechanical and mental adaptation to specific tasks and training environment [4, 5]. However, determining a unified definition of fatigue is not a straightforward process [6-12].

The purpose of this paper is to review definitions of fatigue and potential underlying mechanisms linking fatigue with increased injury risk and underperformance. In addition, clinically feasible markers of fatigue, methods developed to measure fatigue will be reviewed and compared.

Definitions of fatigue

Fatigue is often described as a ubiquitous [13], multifactorial [1, 8, 14], and complex phenomenon [8, 15-20], which must be studied from a collective and tissue-specific perspective [10, 11]. However, a common theme between definitions is a decline of the overall physical or specific cardiorespiratory, neuromuscular, physiological or cognitive function of performing a particular task over time due to acute or chronic exercise and/or mental load [21].

Among various definitions (see Table 1), fatigue most commonly is defined as a decline in muscular force or power during acute exercise [12, 15, 22-26], exercise-induced decline or impairment of performance [12, 21, 25, 26], or as a decrease in pre-match/baseline physiological and psychological functioning of an athlete secondary to a chronic increase of the physical and mental load [16, 27].

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Fatigue definitions in humans are closely related to patterns seen in material fatigue, where structures fail when subjected to a repetitive load and define a type of structural damage even when the material experienced stress range far below the static acute material strength [28]. It is often explained by damage that develops on the microscopic level, which grows until it reaches a critical point when it can no longer sustain the peak load [29]. This is based on the Palmgren-Miner linear damage rule used to predict component fatigue based on the summation of the repetitive cycles [30]. This level of fatigue is often described as fatigue damage [29]. It relates to the stress amplitude, but for any detailed analysis, the mean stress must be taken into account [31].

The complexity in defining fatigue in sport is related to multiple domains of recovery and adaptation processes (physical, neuromuscular control and psychological) and their interactions. This is emphasised by fatigue stemming from different or multiple factors, including prolonged physical exertion, persistent mental activity, and sleep deprivation [1]. Based on associations with various stressors, definitions for each potentially causative domain have been created. It is essential to understand that some of the domains are based on observed correlations in studies and can be either causal or non-causal. Often it is assumed that factors have prognostic value based on the potential contribution to the cause of an event (such as an injury or change in performance) through direct or indirect mechanisms[32].

Neuromuscular fatigue has been defined as a reduction in the maximal force a muscle can exert [33], or the inability to sustain exercise at a required power [33, 34]. Physiological fatigue definitions include a weakness from repeated exertion or a decreased response of cells, tissues, or organs following excessive activity, stimulation, or stress [35]. Psychophysiological changes following sustained performance [36] are often linked with a state of an organism's muscles, viscera, or central nervous system in the absence of sufficient rest. On the tissue level, the fatigue state is often defined as an insufficient restoration of cellular capacity or system-wide energy to maintain the original level of training and/or processing using typical resources [37]. Psychological (or mental) fatigue has been associated with sustained demanding cognitive activities, characterised by an increased feeling of tiredness or exhaustion, an aversion to continuing the ongoing task, plus a reduction in cognitive performance [38-40]. Mental fatigue has been linked with reduced endurance performance on cognitive and physical tasks [41-46] as well as an individual's drive to train [41, 47].

Attempts to define fatigue have resulted so far in the development of fatigue partitions, such as physical-mental [42, 48], central-peripheral [15, 21, 24, 49-51], and acute-chronic [52]. Despite partitioning being important for understanding and managing fatigue, it lacks clarity regarding the broad understanding of fatigue [9, 11]. Furthermore, distinctions between fatigue and other related concepts such as sleepiness [19], effort [53-56], exhaustion [57] and malaise [58] is still limited.

Fatigue and injury risk

Fatigue may impair the joints' kinaesthetic and proprioceptive properties [59, 60]. It increases muscle spindle discharge threshold, which interrupts afferent feedback, consequently altering joint awareness [59] affecting static postural control [61, 62], but to a lesser degree, dynamic postural control [62-70]. Effectively, fatigue can be described as an inability of the nervous system to adequately recruit motor units, the basic functional unit of the neuromuscular system associated with force production. As a result, afferent feedback is affecting efferent pathways and muscle. Gribble, Hertel, Denegar, et al. [59] suggested that fatigue and chronic ankle instability create dynamic postural control deficits, which appear to be associated with kinematic changes at the knee and hip.

Muscular fatigue has previously been shown to delay reaction time [71, 72]. With increased reaction time of the gastrocnemius and tibialis anterior muscles associated inversion ankle sprain incidence [73]. Proprioceptive capabilities [74], motor control precision [75], and movement coordination [76] are all affected by muscle fatigue. Unsurprisingly, lower limb extremity kinetics and kinematics have been shown to be affected by fatigue [2].

Fatigue is considered to be a significant risk factor in injury occurrence [2, 3]. Most injuries occur towards the end of a match or activity when individuals are fatigued [77-81]. For instance, football match injuries incidence increases towards the end of both halves [82], corresponding to 5-10% decrease in distance covered, suggesting evidence of muscular fatigue [83]. Similarly, most amateur rugby league injuries occurred in the second half of matches [77] and are likely to be associated with a fatigue-induced reduction in skill contributing to injuries in these athletes [84].

Joint instability is linked with muscle fatigue associated with muscle dysfunction [66, 85] and strength deficiencies [86-88], affecting postural control and muscles' isokinetic endurance [81, 89]. The strength reduction is related with fewer fibres being recruited [88, 90]. Hypothesis is that muscle fatigue is linked with disturbance

in joint position sense, decrease in motoneuron output [81, 91, 92] and/or desensitisation of type III and IV muscle afferents [81, 93].

Adequate measurements of players' fatigue and recovery could be a focus of injury prevention strategies in high demanding sports and optimisation of the rehabilitation process. In addition, understanding an individual's fatigue levels can facilitate optimal workload (training and match), training adaptation and performance [94].

Markers of fatigue and recovery

Research into markers of fatigue and recovery is a popular area in sports and exercise physiology [95, 96]. Those range from biochemical [97-99], hormonal [97, 100], immunological [101, 102], psychological questionnaires [103], and the assessment of neurological status, including autonomic nervous system balance [97, 104, 105]. The differing markers provide insight for athletes, coaches, and scientists into individual response to these workloads [8]. However, while a limited number of these markers have strong scientific support for their use, and there is currently no single conclusive marker of fatigue [8].

Biochemical markers

Circulating markers in the blood are particularly attractive for assessing training-induced fatigue, offering potentially objective, high accuracy and precision, a clear physiological concept concerning their connection with exercise and fatigue [99, 106], and have limited interference on training. However, there is currently no recognised single parameter that signifies fatigue and recovery changes during athletic training cycles with a satisfactory reproducibility and sensitivity [100, 106].

Urea, insulin-like growth factor 1 and creatine kinase

Urea and insulin-like growth factor 1 (IGF-1) is suggested for endurance cycling training, and creatine kinase (CK) for high intensity interval training in team sports and strength training as indicators of fatigue [106].

Increased urea and decreased IGF-1 have previously been reported during a fatigued state [107]. Urea, suggesting protein breakdown [97, 100, 106] during prolonged exercise, can also be influenced by dietary protein intake and therefore is not that reliable [108]. IGF-1 has been suggested to show a state of glucose austerity following the depletion of carbohydrate stores because of endurance training [99, 106]. Both strength and high intensity interval training are characterised by a high proportion of eccentric force production and muscle tension, which leads to muscle fibre damage, and resultingly leakage of enzyme CK from the sarcoplasm into the bloodstream [106]. This process has allowed CK to become a classic blood-borne marker of fatigue and strain in relevant disciplines [97, 100, 106]. However, it should be noted that muscle damage is only one aspect of fatigue and other aspects, such as psychological alterations or anabolic-catabolic balance, may play a role in the overall need for recovery [106]. It is also important to state that Hecksteden, Skorski, Schwindling, et al. [106] suggests that blood-borne markers are affected by the metabolic process of the activities causing fatigue, which could help explain why there is a difference in prominent markers for differing activities.

Adenosine

Adenosine concentration increases during exercise and its concentration affects blood flow in skeletal muscles [109], plus has profound tissue-protective effects in situations of ischaemia and inflammation [110]. Adenosine inhibits CD8+ T cells by activating adenosine receptors and can cause a significant immunosuppression [111]. It is a promising marker for training adaptations not associated with muscle mass [112]. However there are well recognised difficulties in measuring the adenosine concentration due to the rapid cellular uptake and degradation [113].

Lactate

Blood lactate concentration has also been shown to be sensitive to changes in exercise intensity and duration [8, 114], thus can also be used as a marker of acute fatigue. However, regularly monitoring lactate concentrations during training and competition has limitations, including inter and intra-individual differences in lactate accumulation depending on previous exercise, glycogen content, diet, hydration status, ambient temperature, amount of muscle mass used, plus the time and site of sample procedures [8].

Glutamine

Glutamine is found in various human tissues in high volumes [115, 116], as well as also being the most abundant amino acid in human muscle tissue and plasma [116-118]. Glutamine has also been stated to be the most versatile amino acid [115], this is emphasised by the differing roles glutamine has in a variety of organs and tissues [116]. These roles include maintenance of acid base balance during acidosis, the transfer of nitrogen between organs and detoxification of ammonia, a fuel for gut mucosal cells, a fuel for the immune system cells, a possible direct regulator of protein synthesis and degradation, plus as a nitrogen precursor for the synthesis of nucleotides [115-117]. Plasma glutamine has been suggested as a marker of excessive training loads [108, 115], due to abnormally low plasma glutamine levels being reported in overtrained individuals [108]. However, plasma glutamine levels do not fall after short term high-intensity exercise, but do after an acute bout of prolonged exercise [108], and decreases in plasma glutamine levels can also occur after burns, infections, inflammation, and physical trauma [108, 117, 119]. Diet can affect plasma glutamine levels, with Greenhaff, Gleeson and Maughan [120] reporting concentrations to increase temporarily following the consumption of a meal containing protein, but decrease by about 25% after several days on a low carbohydrate diet. Consequently, to use plasma glutamine as a marker of fatigue, an individual's diet plus the timing of blood sample in relation to the bouts of exercise and food intake must be standardised, it is also important to take other factors, such as infection and tissue injury, must be taken into consideration [108].

Oxidative stress

Markers of oxidative stress have been implicated in acute fatigue [121], with the total anti-oxidative activity being shown to increase following a bout of exercise [122]. Exhaustion levels correlate with isoprostanes and glutathione/oxidised glutathione [123], plus an increased 8-hydroxydeoxyguanosine being reported to continue for two to three days following sleep deprivation [124]. Consequently, biomarkers for oxidative stress and antioxidant activity measured simultaneously are implicated in fatigue conditions in healthy individuals and differentiate from chronic fatigue syndrome [121]. It is important to acknowledge that some oxidative biomarkers cannot be accurately measured in vivo due to high reactivity and short biological half-life [121].

Intracellular oxidative stress-related activities can be reduced with low Vitamin D levels, with suboptimal concentrations of serum 25-hydroxy vitamin D failing to suppress oxidative stress conditions and increase intracellular oxidative damage and can lead to apoptosis [125]. Vitamin D supports maintenance of cellular oxidation level and normal mitochondrial functions [125-128]. It has been suggested that vitamin D level can directly affect injury risk, supports recovery, bone health[129], and is essential for optimal muscle function [129-135].

Endocrine biomarkers

Thyroid

Even though energy balance is not used to diagnose chronic fatigue, a negative energy balance can affect endocrine function and influence performance and ability to adapt to training [136].

Thyroid hormones concentration can be influenced by energy balance, thus contributing to fatigue, but this relationship has not been extensively researched [136]. It is known that thyroid hormone concentrations are reduced with increased

exercise training due to negative energy imbalance and reduced energy availability [136, 137]. As a result of the reduced availability, physiological processes are downregulated to account for the increased energy expenditure from exercise [136]. Consequently, it is essential for appropriate macronutrient intake to produce positive adaptations to training and to meet the intense energy demands during competition [136]. It can be difficult for athletes completing demanding training schedules to consume the necessary amount of calories to maintain energy balance, and may lead to the physiological processes of fatigue, over training and underperformance [136]. In female athletes, thyroid hormones have been shown to be considerably lower with negative energy balance induced amenorrhea [138], while amenorrhoeic athletes have a reduced recovery capacity [139] and reduced neuromuscular performance [140]. This can have detrimental effects on athletic performance and recovery due to thyroid hormones regulating metabolism and growth [136]. Thyrotropin (thyroid stimulating hormone) and free triiodothyronine are two examples of this [136, 141] and have been found to be decreased after a prolonged, intense training cycle [136], which is magnified and extended after such a cycle has been reduced [142]. Despite this, Nicoll, Hatfield, Melanson, et al. [136] suggests that thyroid hormones change too slowly to be used as a readily available marker for fatigue. Monitoring of thyroid hormone is thought to have the potential to provide information about the overall training status of individuals, and adaptation [136].

Stress hormone responses

Increases in training loads over several weeks have been associated with repetitive large stress hormone responses, including adreno-corticotrophic hormone, catecholamines, and cortisol. It is thought that downregulation of specific hormone receptors in the target tissues occurs, which decreases the tissues' responsiveness to these hormones [108]. One of the best indicators of fatigue and overtraining is individual mood changes, as training intensifies, with increased negative moods, such as anger, confusion, depression, fatigue, and tension [108, 143]. These changes have been suggested to reflect underlying biochemical and immunological changes, which are communicated to the brain via hormones and cytokines [108].

Consequently, there appears to be various hormonal abnormalities in athletes affected by overtraining syndrome [108, 144].

Cortisol and testosterone ratio

The cortisol/testosterone ratio has been proposed as a marker of training adaptation based on the idea that cortisol is a catabolic hormone while testosterone is an anabolic hormone [108]. So far, several studies included in the review conducted by Eichner [145] did not find a significant change in the ratio in well-trained athletes during progressive increases in training loads [108].

Saliva

The cortisol and testosterone levels in saliva have been shown to provide an accurate measure of fatigue [146, 147]. This is done through previous research showing that levels of hormones taken from saliva, such as cortisol and testosterone, change during and after physical activity, and individual's biochemical response can be determined by examining these alterations [148]. This is shown by cortisol levels increasing by around 2.5 times throughout a rugby match compared to pre-match levels (baseline), and then return back to baseline within four hours [149]. Similarly, cortisol concentrations increased from baseline by ~56% and ~59% at 12 and 36 hours post rugby match, while testosterone concentrations decreased from baseline at the same time points [150].

Findings of increased amounts of cortisol maybe due to cortisol being a primary stress hormone and the post exercise rise is suggested to reflect the metabolic demands placed on the body [150, 151]. It has also been suggested that increased cortisol concentrations may reduce testosterone synthesis [152-154], which could explain the reductions in testosterone concentrations [150]. These findings could also be due to previous studies stating that fatigue may manifest it's self as alterations in hormone levels [150, 155] and increased markers of muscle damage, such as increased CK concentration [156, 157].

One benefit of saliva samples, is that they are more stable and sampled easily and repeatedly compared to blood and urine detection as they can be affected by the kidneys and other factors [1, 20]. Similarly, saliva is simpler and completely non-invasive, unlike blood and urine analysis [20, 22, 158]. Previous research has also stated how human saliva glands are innervated by both the parasympathetic nervous system and activation of the sympathetic nervous system [1, 159, 160], which is important as prolonged physical exertion triggers changes in autonomic nervous system signalled by a simultaneous withdrawal of the parasympathetic nervous system and activation of the sympathetic nervous system [1, 161].

However, despite the benefits of saliva measures it is relatively expensive and time consuming method in applied environments [162]. There is also poor temporal relationships with neuromuscular performance and the multifactorial components of fatigue cannot be assessed in a single biochemical, hormonal, or immunological measure [162]. Consequently the use of biochemical, hormonal, and immunological measures to monitor fatigue should be considered carefully [162].

Immunological markers

Immunological markers indicating fatigue are based on immune system alterations associated with training loads and other stressors [108]. Athletes suffering from overtraining syndrome are often immunosuppressed [108]. Subsequently the use of immunological markers is a logical step of assessing fatigue by assessing an individual's ability to cope with increased training and consequently provide a way of identifying an individual's fatigue status [108]. Similarly, several sections of the immune system function seem to be sensitive to both acute and chronic stress of exercise [108].

A review by Mackinnon [163] states that repeated bouts of intense, prolonged exercise might decrease the number and functional capacity of the circulating leukocytes. Immunosuppression associated with heavy training can also cause a fall in blood concentration of glutamine [108, 164]. Similarly, the circulating number of leukocytes is generally lower in athletes at rest than sedentary individuals [163, 165],

which again suggests that exercise can alter the immune function, suggesting a decrease in the immune system function, especially during high workloads [108]. A low blood leukocyte concentration could arise from the haemodilution (expansion of the plasma volume) associated with training or represent changes in leukocyte kinetics, including a decreased release from the bone marrow [108]. The large neutrophilia which comes from a prolonged exercise session could, over periods of heavy training, reduce the bone marrow stores of these crucial cells [108]. Leukocyte cells in the blood have been found to be less mature in athletes than sedentary individuals [166] has also found the leukocyte cells in the blood to be less mature in athletes than sedentary individuals. Similarly, the phagocytic activity of blood neutrophils has been reported to be significantly lower in well-trained cyclists compared to age, and body mass matched sedentary controls [165]. Mackinnon [167] also found levels of secretory immunoglobulins, such as salivary immunoglobulin A, are lower in well-trained individuals.

Several possible causes of the decreased immune function associated with heavy workloads have been proposed. One of which might simply be the cumulative effects of repeated bouts of intense exercise with the resulting increase of stress hormones, especially glucocorticoids in temporary immunosuppression [108, 168]. It is also possible that there is insufficient time for the immune system to fully recover when exercise is frequently repeated [108]. Additionally, plasma glutamine levels can alter considerably after exercise, which may become chronically depressed after repeated bouts of prolonged strenuous exercise [117, 164]. Also, through the muscle damage that occurred through repeated bouts of exercise, a decrease of serum concentration can occur, contributing to decreased immunity in athletes, and well-trained individuals have been found to have lower serum concentrations than controls [163].

Resultingly it has been suggested that possible markers of fatigue can include blood levels of stress hormones, antibodies, cytokines, and glutamine, plus the ability of leukocytes to respond to the antigens stimulation [108]. It has also been suggested that plasma glutamine/glutamate ratio changes is also a predictor of fatigue in athletes [98, 116].

Cognitive and emotional fatigue measured by psychological questionnaires

Mood changes have been significantly associated with training load and fatigue, consequently, psychological questionnaires are utilised as a method to monitor fatigue [108]. As training increases, athletes tend to develop a dose-related mood disturbance with low scores for vigour and increased scores for negative moods such as anger, confusion, depression, fatigue, and tension [108, 143]. Assessing muscle soreness and fatigue during and after each exercise session has also been suggested [169], thus could be an effective way to monitor the recovery from workloads [108]. Using psychological questionnaires as a marker of fatigue could also be useful as they are a relatively inexpensive and straightforward fatigue marker [8].

Self-reported methods

Typically, these questionnaires gather information on parameters such as muscle soreness, tiredness, sleep health and current stress levels [170]. Self-report questionnaires are widely employed to assess fatigue in sport, of which there is a plethora [12, 171-173]. This is predominantly thought to be due to their low-cost, relative ease of implementation and flexibility, enabling them to be administered across a wide range of individual and team sport settings [174].

Validated self-report questionnaires that are frequently cited in research include the Profile of Mood States (POMS), Total Quality Recovery (TQR), Daily Analysis of Life Demands for Athletes (DALDA), the Recovery Stress Questionnaire for Athletes (REST-Q), the Acute Recovery and Stress Scale (ARSS), and the Rating of Fatigue Scale (ROF) [11, 170, 175-179]. Additionally, Rating of Perceived Exertion (RPE) and session RPE (duration of training session multiplied by RPE) are frequently used to monitor both training load and expected responses to training load [11]. Table 2 shows an overview of the various questionnaires.

****Table 2 near here****

There are, therefore, a significant number of questionnaires that can reflect perceptual fatigue. Nonetheless, recent research has demonstrated that even those questionnaires explicitly developed for athletic populations have only adequate measurement properties, with weak content validity and reliability a particular concern [180, 181]. Interestingly, there is widespread evidence that sporting organisations have increasingly chosen to employ their own customised self-report questionnaires over the questionnaires published in the literature [12, 182, 183]. This divergence between research and practice likely stems from the perception that the existing questionnaires lack sports specificity and are too extensive in nature [12]. Customised questionnaires may overcome these problems but potentially raise new issues relating to even weaker validity and reliability [180, 181]. Unless the reliability and validity of custom questionnaires are discerned, conclusions drawn from the data are questionable and should therefore be interpreted with caution [180].

However, one issue with self-reported methods is that changes in perceived fatigue and muscle soreness are also known to outlast reductions in neuromuscular performance and biochemical markers [184]; however, this study was conducted only in rugby players. Similarly, changes in psychological state or mental fatigue is also known to change an individual's sense of effort forcing individuals to down-regulate their exercise capacity [42, 162]. It is supported by previous studies stating that despite self-reporting methods being frequently used to assess fatigue an individual's perception of fatigue is not a robust predictor of the ability to perform [1, 185]. Consequently, it has been stated that there is a need for objective measures of fatigue [1, 186].

Overall it is apparent that subjective self-report questionnaires can play an important role in assessing fatigue [187, 188]. However, it is prudent to interpret self-report questionnaires alongside objective measures and reference to an individual's broader training context. It is primarily due to the weak reliability and validity of self-report questionnaires [180], but also because perceptions of fatigue are not always a robust predictor of performance ability [1, 185]. Additionally, poor engagement from those completing self-report questionnaires can reduce questionnaire efficacy, with

social desirability bias potentially confounding responses further [162, 189, 190]. Previous research has also stated how subjective measures are a concern for coaches due to athletes being able to manipulate responses to produce favourable outcomes [162, 189, 190]. Another issue with questionnaires is the possibility of a lack of compliance [162].

Markers of autonomic nervous system alternations

Assessing the autonomic nervous system has also been used as a marker of fatigue. This is due to the suggestion that alterations in the autonomic nervous system status coincides with hormonal changes seen during over training and fatigue [191, 192]. However, differing methods of assessing the autonomic nervous system has been devised.

Heart rate

Monitoring heart rate during exercise is based on the linear relationship between heart rate and oxygen consumption rate during steady-state exercise [8, 193]. However, prescribing and monitoring intensity is based on a percentage of maximum heart rate, but this can be problematic because of daily differences in heart rate, which can be up to 6.5% for submaximal heart rate, and can be affected by factors such as environment, hydration, and medication [8, 194].

Heart rate recovery

Heart rate recovery assesses the rate of decline in heart rate at the end of the exercise, which has been suggested to be a marker of autonomic function and training status in individuals [8, 195]. Exercise is a consequence of increased sympathetic activity in addition to a reduction in parasympathetic activity [8]. Heart rate recovery is characterised by comparing autonomic nervous system activity, increasing parasympathetic activity and removing sympathetic nervous activity [8, 196]. Various timeframes can be used to calculate heart rate recovery, usually

between 30 seconds and 2 minutes, with the difference between heart rate at the end of exercise and 60 seconds post-exercise being the most commonly used timeframe [8, 195].

Heart rate variability

Heart rate variability has been suggested to be a practical non-invasive method for assessing cardiac autonomic nervous system [191, 197]. This is a measurement of the difference between resting and post-exercise heart rate, which has been suggested to show both negative and positive adaptations to training [8, 198]. This in part, might explain why heart rate variability has been suggested by researchers to be used to guide training on a day-to-day basis [191, 199-201]. However, scientific literature has been inconclusive about this marker, with equivocal findings from studies that examined heart rate variability and over training, with findings of increases [202], decreases [203-205], and no difference [197, 206] in cardiac autonomic system activity. Consequently, it is unknown if a relationship exists between heart rate variability and overtraining and if the marker can be used to identify the progression of overtraining before complete manifestation [191].

The equivocal findings could be due to the high day to day variability in environmental and homeostatic factors plus varying methodological approaches used [8, 198], and exercise the days before the recordings [191, 207]. These factors, individually or collectively, are likely to explain the discrepancy between studies [191, 197, 206], and thus the lack of sensitivity from heart rate variability measurements to detect fatigue could be due to type 2 errors [191, 208]. To avoid the possible inconsistencies in findings, it has been suggested that both weekly and 7 day rolling averages have validities than single day measurements [8, 191].

Neuromuscular

A decrease in maximal force capabilities of a muscle or muscle group regardless of maintaining a given task or not relates to neuromuscular fatigue [209, 210].

An objective measure of neuromuscular fatigue is the countermovement jump (CMJ), with the test being shown to be sensitive to match-induced fatigue [65, 150, 155, 184, 211]. The CMJ test is also stated to be a popular test, which is easy to administer and produces minimal amount of additional fatigue [12, 162, 212], all of which allows for repeated assessments of multiple individuals over a short time [212]. It has also been stated that jump procedures reflect the stretch-shortening cycle of lower limb musculature [162, 213]. Previous research has emphasised that athletic performances consist of stretch-shortening cycle movements [212, 214], and stretch-shortening cycle fatigue relates to numerous mechanical, metabolic and/or neural factors [212, 213, 215, 216]. Similarly, it has been stated how reduced neuromuscular function reduced conduction of afferent signals from the fatigue altered state of the muscle will lead to reduced propagation of efferent signals, which affects the ability to create compensatory movement effectively [60, 63]. However, previous research has also found no significant differences in CMJ tests pre and post-football match, when examining peak force, peak power, or rate of force development [217], and elite Australian Rules Football match, when examining mean force and mean power [148]. This could be due to the CMJ test lacking the sensitivity to detect neuromuscular fatigue from a single game [211]. It has also been stated that CMJ tests may overlook a number of fatigue-related neuromuscular changes due to the complex nature of neuromuscular fatigue [21, 212].

Summary of markers of fatigue

The importance of fatigue markers is shown through the aim of assessing fatigue in competitive athletes to individually prescribed training to achieve a balance of maximising training loads and adaptations while providing adequate recovery for the athlete [106].

Nevertheless, due to difficulties in defining fatigue, it is also challenging to measure and monitor fatigue [6, 7, 12, 218], which is emphasised by no single universally accepted measure of fatigue [1, 8, 12, 21, 219]. Consequently, several markers and/or measures are needed for early fatigue detection [116], with no single marker indicating impending fatigue. Thus, different methods are used. Similarly, Worrell

and Perrin [220] have also stated how it is challenging to study the role of muscle fatigue in sustaining injuries.

Conclusion

In conclusion, fatigue is a multifactorial and complex phenomenon. Fatigue has been suggested to be one of the most important injury risk factors. Measuring and monitoring fatigue and recovery can be difficult, emphasised by no single universally accepted definition or measure of fatigue. Consequently, differing methods are used to monitor them, including self-reported methods, heart rate variability, counter movement jump, and saliva.

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Table Captions

Table 1 Varied definitions of fatigue

Table 2 An overview of the differing self-reported methods