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INTERACTIVE PROCESSES LINK THE MULTIPLE SYMPTOMS OF FATIGUE IN SPORT COMPETITION

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ABSTRACT

Muscle physiologists often describe fatigue simply as a decline of muscle force and infer this causes an athlete to slow-down. In contrast, exercise scientists describe fatigue during sport competition more holistically as an exercise-induced impairment of performance. The aim of this review is to reconcile the different views by evaluating the many performance symptoms/measures and mechanisms of fatigue. We describe how fatigue is assessed with muscle, exercise or competition performance measures. Muscle performance (single muscle test measures) declines due to peripheral fatigue (reduced muscle cell force) and/or central fatigue (reduced motor drive from the central nervous system (CNS)). Peak muscle force seldom falls by >30% during sport but is often exacerbated during electrical stimulation and laboratory exercise tasks. Exercise performance (whole-body exercise test measures) reveals impaired physical/technical abilities and subjective fatigue sensations. Exercise intensity is initially sustained by recruitment of new motor units and help from synergistic muscles before it declines. Technique/motor skill execution deviates as exercise proceeds to maintain outcomes before they deteriorate, e.g. reduced accuracy or velocity. The sensation of fatigue incorporates an elevated rating of perceived exertion (RPE) during submaximal tasks, due to a combination of peripheral and higher CNS inputs. Competition performance (sport symptoms) is affected more by decision-making and psychological aspects since there are opponents and a greater importance on the result. Laboratory based decision-making is generally faster or unimpaired. Motivation, self-efficacy, and anxiety can change during exercise to modify RPE and hence alter physical performance.

Symptoms of fatigue during racing, team-game or racquet sports are largely anecdotal, but sometimes assessed with time-motion analysis. Fatigue during brief all-out racing is described

biomechanically as a decline of peak velocity, along with altered kinematic components. Longer sport-events involve pacing strategies, central and peripheral fatigue contributions, and elevated RPE. During match-play, the work rate can decline late in a match (or tournament) and/or transiently after intense exercise bursts. Repeated sprint ability, agility and leg strength become slightly impaired. Technique outcomes such as velocity and accuracy for throwing, passing, hitting and kicking can deteriorate. Physical and subjective changes are both less severe in real- than simulated-sport activities. Little objective evidence exists to support exercise-induced mental lapses during sport.

A model depicting mind-body interactions during sport competition shows that the RPE center-motor cortex-working muscle sequence drives overall performance levels and hence fatigue symptoms. The sporting outputs from this sequence can be modulated by interactions with muscle afferent and circulatory feedback, psychological and decision-making inputs. Importantly, compensatory processes exist at many levels to protect against performance decrements. Small changes of putative fatigue factors can also be protective. We show that individual fatigue factors including diminished carbohydrate availability, elevated serotonin, hypoxia, acidosis, hyperkalaemia, hyperthermia, dehydration, and reactive oxygen species, each contribute to several fatigue symptoms. Thus, multiple symptoms of fatigue can occur simultaneously and the underlying mechanisms overlap and interact. Based on this understanding we reinforce the proposal that fatigue is best described globally as an exercise-induced decline of performance as this is inclusive of all viewpoints.

1. INTRODUCTION

Fig.1 near here

Sport performance depends on the ability of an athlete to produce and then sustain high levels of physical, technical, decision-making and psychological skills throughout competition. Deterioration of any of these skills could appear as a symptom of fatigue, yet the manner in which fatigue is best described and measured is controversial.[1-6] The phenomenon of fatigue is complex with the underlying processes developing as exercise proceeds to ultimately manifest as a decline of performance. By incorporating a holistic approach, fatigue can be described as an exercise-induced impairment of performance during sport-events. But what exactly do we mean by impairment of performance? Figure1 shows that performance can be assessed at three different levels. At the simplest level there is a reduced force/power output by a single muscle cell or motor unit. Simultaneous detrimental effects in several motor units could impair function of a single whole-muscle, i.e. reduced *muscle performance*. A common assumption is that reduced muscle performance translates into reduced *exercise performance*. Test measures of the latter incorporate the force/power generated by several muscle groups, motor skill outcomes and fatigue sensations. A diminished exercise performance usually causes a reduced *competition performance* during sport-events, which is assessed solely by performance symptoms. The inclusion of decision-making against competitors and greater psychological aspects feature in many sport-events. Finally, the match result usually depends on the better overall competition performance on the day (Fig.1). However the result should not be used to assess competition performance, since the scoring system can have a role. Indeed, matches can be won through gaining the critical points in racket sports despite losing more points overall, or lost through a failure to convert periods of dominance into points or goals scored.

Limitations to understanding fatigue may have arisen in part from the belief that manifestations of fatigue obtained using a reductionist approach (e.g. stimulation of isolated muscles) or laboratory exercise models, relate directly to what happens in sport competition.[3,4] To enhance understanding beyond the *in vitro* and laboratory based approaches several recent reviews describe what happens during specific sports,[7-12] provide generalized fatigue mechanisms,[13-18] explore the integrative physiology of whole-body fatigue[19] or focus on mind-body interactions during voluntary exercise.[20-24] However, the symptoms and mechanisms of fatigue during sport competition still need greater understanding. Indeed, muscle physiologists may be unaware of how altered muscle function impacts sport performance, and sport scientists may be unclear about which neuromuscular processes underpin fatigue symptoms during sport-events. Hence, the purpose of this review is to take a holistic and interdisciplinary approach to: 1) describe in general terms how fatigue is assessed at muscle, exercise or competition performance levels; 2) describe specifically how fatigue is manifested during sport-events; and 3) consider whether neuromuscular, motor skill and subjective symptoms of fatigue are linked through common mechanisms/processes. Literature was sourced through databases (PubMed, Web of Science), and from reference lists in related original research and review articles.

2. QUANTIFYING THE MANIFESTATIONS OF FATIGUE

Tables1 and 2, near here

Fatigue can be quantified using performance symptoms and/or test measures (Table1). Performance symptoms are impairments of movement abilities/outcomes as they appear during sport-events. Symptoms are often obtained anecdotally from players or coaches as it is difficult to get such data without interfering with the competition. One objective approach used involves time-motion video analysis[25-31] which can reveal changes in work rate, technique, phases of play or the occurrence of errors. However, the uniqueness of each competition due to variations in the quality of opponents, match strategies, behaviors, environmental conditions, and terrain complicates interpretation of these data.[11,29] It is also unknown whether symptoms late in a match result from the physical exercise or other aspects such as anxiety due to mounting pressure.[32-34] Moreover standardized test measures are sometimes obtained before and after competitions (Table1).[8-11,27-31] These measures are commonly used to explore fatigue mechanisms but they also describe the components which determine overall performance. Interestingly, several test measures are necessary in order to describe each performance symptom (Table2).

Sport activities can be assessed in the laboratory or field settings with test measures obtained. Simulated-sport activities involve replicating an entire match,[35-39] or component of a match.[40-43] For example, hitting skills can be studied using a ball projection machine,[34,41,44-46] although this leads to modified motor skills because visual cues are absent, with normal anticipation being restricted.[45,46] Laboratory exercise on treadmill, cycle or rowing ergometers allows work intensity and conditions to be controlled, with power output measured precisely.[47-51] Exercise components such as the force applied to pedals/oars, or pedal rate can also be evaluated.[48-51] However, some laboratory tasks do not adequately replicate sport-events.[4] In particular the time-to-exhaustion tests[52-57] differ to racing sports where pacing strategies are employed.[56,57] Furthermore, test measures from exhausted individuals seldom mimic a performance symptom.[4] For example, maximum isometric voluntary contractions (MVC) do not usually occur during sport.[4,6] To investigate mechanisms, stimulation-induced models of fatigue are often used since this permits analysis of muscle test measures independently of a variable motor drive, the muscle environment is controlled, and invasive interventions/measurements can be made.[13,17,58-62] But just how closely the stimulation regimes mimic the motor activation patterns which occur during sport is questionable.[4,17]

2.1 Muscle performance: Muscle performance test measures can be obtained after repeated activation of a single muscle, exercise tasks or sport-events (Table1). When processes originate in muscle cells and directly impair muscle contractile function the phenomenon is called peripheral fatigue.[1,2,15,17] This usually involves diminished peak force measures, but when combined with a slowed shortening velocity, can manifest as a reduced muscle power (power = force x velocity). Several mechanisms are postulated to contribute to peripheral fatigue either directly or through interactive effects. These include metabolic factors (e.g. ATP, Pi, PCr, lactate),[1,2,13,17] diminished glucose or glycogen availability,[2,13,17,60,61] ionic factors (e.g. K^+, Na^+, Ca^{2+}, Cl^-),[13,17,59,62,63] acidosis,[13,17,64] hypoxia,[2,65,66] reactive oxygen species (ROS),[17,67-70] and/or ultrastructural damage.[17,71] When a reduced muscle force occurs during volitional contractions, it may also arise through a lowered drive from the motor cortex in the brain, i.e. central fatigue.[1-7] This inhibition of motor drive (reduced motoneuron firing frequency and/or de-recruitment of motor units) may be consequent to peripheral feedback from working muscles, heart or lungs and/or input from higher centers in the CNS. The presence of central fatigue is examined by superimposition of electrical stimulation on the peak force of a MVC, i.e. the twitch- or tetanus-interpolation technique,[1-4,15,16,53,72-80] or by comparing the relative decline of peak tetanic and MVC forces.[15,53,74] Superimposed stimulation is traditionally done on brief MVC[15] although more recently this has been extended to sustained or repeated voluntary contractions.[16,77-80] Direct evidence is available for reduced motor drive during hypoxia (lowered O_2),[73-76] hyperthermia (elevated core temperature),[77-79] hypoglycaemia (lowered plasma glucose),[80] or consequent to greater firing of group III and IV muscle afferents.[2,3,15,72,81-84]

A question of interest is just what is the extent of muscle force loss during exercise? Repeated electrical stimulation can reduce peak tetanic force to <40% initial, with the rate and extent depending on the muscle fiber-type/s involved.[1,17,58,59,66] This severe force loss is partially related to the high and constant stimulation frequencies used (e.g. >30Hz) which diminishes excitability.[58,59] In contrast motor unit firing in volitional contractions is slower and the rate falls to convey some protection, i.e. muscle-wisdom.[2-4,15] Fatigue during dynamic exercise tends to evoke smaller force losses than with stimulation. Brief high-intensity exercise induces a 5-15% decline of peak MVC force,[85-87] whereas prolonged cycling, running, or skiing evokes decrements of up to ~30%.[7,53] These measurements are usually made 1-2min after exercise ends which is likely to underestimate the force loss due to rapid recovery over this time period.[58,59] Prolonged isometric MVC or repeated isokinetic contractions induce larger force decrements of 50-80%[1,6,88] but these are unnatural exercise models,[4] and ischaemia during isometric contractions exacerbates force loss.[2,4] Notably, peak muscle power can decline by up to 80%,[88] hence peak force reductions alone cannot account for the entire fatigue.

2.2 Exercise performance: This is assessed with whole-body exercise tests in laboratory or field-settings, or after sport-events. Muscle performance contributes to exercise performance but there are now extra features (Fig.1). The exercise task involves: the actions of several muscle groups with intermuscular coordination influencing exercise intensity and motor skill execution; a more dominant CNS contribution where motor drive and behavior can change; a greater influence from the environment and/or fatigue sensations.

2.21 Physical abilities: Most physiological contractions appear to be submaximal. When attempting to hold a submaximal isometric contraction the force loss by some motor units does not necessarily manifest as a lowered whole-muscle force.[2,6,89,90] Indeed, recruitment of new motor units and altered firing patterns in remaining working motor units can prolong the task.[89,90] A progressive recruitment of motor units also occurs as dynamic exercise proceeds.[91] Clearly, such motor behaviors compensate for individual motor unit decrements to sustain whole muscle performance, at least for a while.

During all-out sprinting on a treadmill or cycle ergometer[47,49] the external power climbs rapidly to a peak then starts to decline after just 5s. Peak power output then falls by >50% over 30s, and during cycling the pedal rate can fall from 140 to 80rpm.[49] Repeated brief cycle sprints leads to decrements of peak power associated with reduced pedal rates/forces, and altered intermuscular coordination patterns.[92] During maximal isokinetic cycling over 30s[92] (i.e. a Wingate test) the overall muscular power falls by 60%, whilst the joint specific fatigue profiles show a reduction of 45% for hip extension power, 59% for knee extensor power, and 63% for ankle plantar flexion power. Fatigue also occurred more rapidly in distal joints. Since greater force/power loss occurs distally over the leg during cycling it seems that more studies on distal muscles/joints would enhance understanding of exercise performance.

Laboratory exercise usually involves a constant power output or incremental work until exhaustion, which noticeably differs to the pacing profile of races.[22,56,57,65] Never-the-less, these tests continue to be used because of ease to standardize the conditions. Notably, the exercised muscles still generate considerable force at exhaustion (i.e. the volitional termination of the exercise task), when peak MVC force seldom falls by >30%.[6,7,53]. The laboratory also permits controlled studies on hot or hypoxic environments,[49,54,65,73-79,93,94] where exercise tolerance is reduced, the sense of effort is elevated, and force loss exacerbated.

<u>*2.2.2 Technical abilities:*</u> Changes of technique/motor skill execution appear as exercise proceeds

but this does not necessarily imply a reduced motor skill outcome. Davids et al.[95,96] support the concept of dynamic systems theory which postulates that movement reorganization permits increased variation of skill execution in order to achieve a constant task outcome. The diversity of ways to meet task demands is possible because several muscles/joints are available to generate movements. Hence, the same result can be achieved with different patterns of movement coordination, i.e. technique deviation rather than deterioration. Royal et al.[38] provide good evidence for this concept by showing that during intense water polo drills, the technical skills fell-off yet the speed and accuracy of shots were unchanged.

Technique deviation: During locomotion impaired function by a single muscle may not reduce the overall power because synergistic muscles help sustain the work intensity.[6,72] For example, during prolonged cycling at a constant pedal rate, a reduced effective force application during the recovery phase is compensated for by greater force in the propulsive phase.[51] In this case the smaller plantar flexors appear to fatigue first leading to a greater contribution by the larger quadriceps muscles. Deviation is also seen in electromyographic data during a 6-min rowing test where elite but not weaker rowers utilized a motor behavior involving rotation between the large back and thigh muscle groups to share the workload.[97] Furthermore, when throwing skills were studied in elite handball players, after fatigue was induced with repeated forearm contractions, the successful throws showed coordination changes yet the final hand velocity remained constant.[42] A lack of a temporal delay between elbow and hand peak velocity suggests that the forearm became stiffer. A similar response occurs during high-intensity cycling where the ankle range of motion declines more than for proximal joints.[92] Such changes simplify the task coordination to help maintain accuracy.

Technique deterioration: Greater changes of motor skill execution, eventually causes diminished outcomes (Table1). Fatigue-induced reductions of accuracy for throwing,[42] passing,[37,40] hitting, [9,10,41,44] and velocity for kicking,[11,98,99] and hitting,[9,10,44,100] have been reported. In each case inappropriate movement on- and off-timing for the distal joints of arms (hitting, throwing)[41,101] or legs (cycling, kicking).[50,98,99] is associated with technique deterioration. Co-activation of antagonistic muscles may also increase during exercise,[2,50,72] to impair motor skill outcomes.

2.2.3 Subjective fatigue: The sensation of fatigue includes the conscious perception of increasing effort needed to sustain a submaximal task (i.e. the exercise feels harder), together with muscle weakness and feelings that persist at rest.[1,2,102] Although feelings of generalized fatigue/tiredness are sometimes assessed,[103-105] it is the rating of perceived exertion (RPE) which has attracted the most interest. RPE is quantified for whole-body exercise, breathing or muscles using the Borg scale.[2,6,20-23,52-57,102-116] Whole-body RPE involves awareness of sensations arising from muscle, joints, chest (i.e. labored breathing, pounding heart), skin, circulating factors and inputs from higher brain centers.[20,21,102] RPE is thought to be driven by a central feed-forward mechanism, i.e. corollary discharge associated with increased motor drive.[2,102] However, the highest RPE usually require inputs from peripheral factors including hypoglycemia,[106-108] muscle glycogen depletion,[109] systemic hypoxia[22,65] or acidosis,[110-112] high skin/core temperature,[20,54,93,94] or dehydration (loss of body water).[104,113] Notably, different combinations of these factors have the ability to evoke maximal RPE. Several findings support an intimate negative link between RPE and physical performance. First, the time-to-exhaustion is closely associated with the rate of rise of RPE.[53-55] Second, exhaustion occurs only when an individual reaches maximal RPE and exercise becomes intolerable.[2,6,22,52-57] Third, several interventions which modify higher RPE, i.e. between 15 (hard) and 19 (very very hard), also alter endurance time. For example, glucose,[106-108] bicarbonate,[110-112] or fluid ingestion,[104,113] O_2 supplementation[65] or psychological interventions[114-116] dampen RPE and prolong exercise. In contrast, hyperthermia,[54,93,94] hypoxia,[22,65] serotonin agonists,[52] or mental fatigue,[55] exacerbate RPE and abbreviate exercise. Clearly, one should avoid these higher RPE in order to sustain exercise. Several other sensations change during fatiguing exercise (Table1)[1,2,15,72,102,110] but whether/how they influence physical performance is unclear.

The subjective phenomenon of mental fatigue, whose characteristics include lowered attention (concentration), working memory, vigor, decision-making and feelings of mental tiredness, occurs with long periods of challenging cognitive activity.[1,5,6,117] But does mental fatigue also occur with physical exercise? Many studies show that psychomotor test scores are unchanged or improve after dynamic laboratory exercise, e.g. choice reaction times become faster,[117,118] although some tests of prefrontal-dependent cognition reveal impairment.[119] It appears that in most cases physical exercise does not push an athlete over the crest of the traditional,[32] yet outdated, inverted-U curve for cognitive performance. However, when there is also heat stress,[117,120,121] dehydration,[117,120,121] or hypoglycemia,[105,117,122] some cognitive test responses become impaired. Importantly, none of these tests relate closely to the mental activities performed during sport-events. Recently, Marcora et al.[55] addressed the related question of whether prior mental fatigue influences subsequent physical performance? They provide the intriguing finding that mental fatigue, induced with a challenging computer task, elevates RPE throughout exercise and hastens exhaustion. Hence mental fatigue processes interact with processes that limit physical abilities.

2.3 Competition performance: This involves a large input from exercise performance (Fig1) but a distinction is that it is now about winning against opponents rather than just the absolute performance level. It is only assessed by performance symptoms although muscle/exercise test measures are sometimes obtained after sport-events. The sport-setting differs to the laboratory since competitors, team-mates and audiences are involved, and there is importance (i.e. monetary, prestige) on the result. Consequently, in many sports decision-making and psychological aspects (Fig.1) have a greater impact on competition performance.

2.3.1 Decision-making: Match-play requires fast and accurate decisions, i.e. rapidly choosing the best movement response from several possibilities. Unfortunately, most psychomotor tests of decision-making[117,118] appear to only remotely mimic sport-related decisions. Using an improved testing approach, McMorris and colleagues[123,124] studied anticipatory decision-making using tachistoscopically presented static images of soccer match-play following intense cycling. They found that visual search and information processing were faster, and accuracy improved, but only after maximal exercise. However, the non-specific exercise task used and inability of static slides to provide full lead-in information makes extrapolation of these findings to soccer match-play uncertain. Royal et al.[38] took another approach with a video-based temporal occlusion decision-making task and examined elite water polo players undertaking intensive drills in the pool. Anticipation accuracy fell slightly with low exertion relative to rest, but when intensity increased towards maximal RPE, there was ~20% improvement. These improved decisions may relate to greater exercise-induced arousal[53,117] possibly mediated via catecholamines.[117,122] Although studies on anticipation are mounting[34,38,45,46] the issue of whether anticipatory skills are impaired by fatigue processes during sport-events warrants greater investigation.

2.3.2 Psychological aspects: Motivation, self-efficacy, and anxiety are psychological constructs that may change during exercise and influence physical performance.[32,34,54,115,116,125,126] Motivation, or the willingness to exert effort, may counteract any negative influence of high RPE.[2,6,22,54,125] In fact, verbal encouragement is a motivational requirement for valid laboratory assessments of central fatigue and time-to-exhaustion.[2,15] Wilmore[125] demonstrated that enhancing motivation by the presence of competitors increased cycle time-to-exhaustion in the laboratory. Also, in a sport-setting a supportive crowd, team-mates, and/or the coach can enhance motivation and exercise intensity,[32,126] whereas a hostile audience can dampen motivation, especially with non-athletes.[32] Any influence of motivation may occur via attenuating RPE[126] or direct effects on the motor cortex.[122] Self-efficacy is an individuals' subjective belief concerning their ability to succeed at a specific task. It is suggested that fatigue sensations are interpreted differently according to the degree of self-efficacy that an individual maintains during a specific task. Indeed, subjects with lower self-efficacy tend to have higher RPE, although this relationship declines at higher exercise intensities.[115,116] Other psychological interventions, such as hypnosis, can also alter RPE but only at lower work loads.[114-116] Moreover, a recent wall-climbing study[127] showed that repeated climbs leading to high RPE was associated with a decline of perceived maximum reach, i.e. reduced self-efficacy. However, it was only with exhaustive climbs and maximal RPE that the maximum height reached actually declined. Anxiety may increase during match-play especially when a result is eminent. This may in turn reduce self-efficacy, motor skill execution[32] and/or anticipatory decision-making.[34] A related observation is that the stress hormone, adrenaline, is higher in plasma prior to and after tournament matches than practice matches.[33] The higher adrenaline concentrations or lower noradrenaline/adrenaline ratio are associated with greater nervousness.[33]

3. SPORT SPECIFIC SYMPTOMS/MEASURES OF FATIGUE

Fig.2 near here

It is widely regarded that the specific mechanisms of fatigue relate to the task-dependency of fatigue[2,3,6] or the fatigue-model employed.[3,4,17] To account for this, we now describe specifically how fatigue becomes manifested during racing, team-game or racquet sports.

3.1 Racing sports: These events require that a given distance is covered in the shortest possible time which makes the average velocity the decisive variable for performance. From the very start of short duration running events an athlete tries to operate at the maximum power and sustain this power throughout the race. However, even with world class sprinters the peak velocity falls towards the end of a race: by ~7% over 100-m and ~20% over a 400-m (Fig.2).[25,128] Of the two kinematic constituents that determine running velocity, it is the stride frequency that decreases towards the end of a race, e.g. 4.2 to 3.5 strides/s over 400-m.[128] This effect is attributed to a 35% longer ground contact time as a result of lowered moment generation about the hip and knee. A greater slowing in non-elite athletes involves decrements of both stride rate and length.[128] In longer races the pacing becomes crucial and large fluctuations of velocity occur. However a common pattern of velocity distribution involves an initial increase then a relatively constant level until the end-spurt, as shown for a 10-km race (Fig.2). This velocity profile is also observed in competitive rowing[48] and cycling time-trials,[65,75] and cannot be used to assess fatigue.

Biomechanical analyses of international swimming events show progressive impairments during sprinting.[26,129,130] For example, in the men's 200-m breaststroke,[26] the mid-pool swimming speed falls over each consecutive 50m (~7% over four lengths) along with a decline in stroke length (up to 17%). This effect is mediated by reduced propulsive forces although it is partially compensated by an increased stroke rate. Less-skilled swimmers show impairment of both kinematic variables. Moreover, the non-swimming element of turning is prolonged by ~5% in elite swimmers[26] to increase the race time. In longer races the end-spurt is also evident.[130]

<u>Table3 near here</u>

3.2 Team-game sports: A fall in work rate occurs either towards the end of a game (i.e. sustained fatigue),[8,11,27,29,131] or over several days of a tournament (i.e. cumulative/residual fatigue)[11,27,103] (Table3), and less sprinting occurs transiently after intense running in either half (i.e. temporary fatigue).[131] Tests of repeated sprint or sport-related agility times within simulated events or during breaks in matches confirm that a slowing occurs.[37,39,87,103-105,131] Ball dribbling skills are also prolonged when associated with dehydration.[104,113] Diminished isokinetic leg strength occurs after simulated soccer activities,[11,36] and reduced drive-power occurs over a simulated rugby game or between successive drives.[37] Poorer technique execution, according to standard coaching criteria, occurs during intense sport-related activities (Table3)[11,37,38,43,98] and leads to diminished outcomes.[37,98,99,103-105,132,170] For example, a slower ball speed occurs with soccer kicking along with a less coordinated kicking motion and poorer ball contact[11,98,99] Interestingly, such technique deteriorations only manifest when RPE exceeds 15 (hard).[38,43,99] Mental concentration and decision-making tests do not show impairment.[38,105,113,123,124,135]

Several mechanisms contribute to fatigue during team-game sports. The sustained fatigue towards the end of a match coincides with low muscle glycogen,[8,133,134] with half of the muscle fibers being completely depleted.[134] Furthermore, reduced pre-match muscle glycogen yields fewer sprints[132-134] and impaired kicking ability.[132] Although severe hypoglycaemia seldom occurs and plasma glucose may even rise,[8,131,132,134] glucose supplements can be helpful.[105,132] The glycolytic pathway is stressed during football[43,126,132-134] causing intramuscular acidosis[134] but this appears insufficient to lower force.[64] Changes of intramuscular phosphates, plasma K^+ and high RPE[8,11,87,134] are possible candidates for temporary fatigue. However, these factors change rapidly and the influential interstitial $[K^+]$ has not yet been measured.[134] Dehydration at moderate temperatures[8,135] can elevate RPE[104,113,135] and reduce agility.[104,113,135] Such effects may occur via raised core temperatures (>39ºC),[8,104,135] which become even higher during prolonged matches in the heat.[11,77] Finally, ROS,[136] muscle damage,[71,103] and impaired motor drive,[11] may also contribute to sustained and cumulative fatigue.

Table4 near here

3.3 Racquet sports: Many symptoms are anecdotal and still require confirmation by measurement (Table4).[9,10,12,35] Impaired movement characteristics are shown in tests with less balls being reached[44,137] or a slowing of repeated sprints.[44,100,137,142] A small decline of peak MVC force and stiffness for the quadriceps/plantar flexor muscles occurs with tennis play over several hours[30,138,139] which is primarily due to central fatigue.[30,138,139] An unchanged explosive leg strength[30] is possibly consequent to movement reorganization. Again, delays make it difficult to measure temporary fatigue following intense rallies. Unexpectedly, the RPE is lower during match-play or training,[28,30,31,44,100,139] than simulated racquet sport activities,[35,41,137] where maximal RPE and higher plasma lactates occur.[28] This may have resulted from shorter rest-periods,[35,137] whereas extended match-play shows a small decline in effective playing time.[30] Hence simulated sport-activities appear to be overly strenuous and need to better reflect match activities.

Several studies reveal impaired hitting accuracy (i.e. less ball depth on the court, increased error rates),[35,41,44,100,137,140,141] and slightly diminished ball velocity[44,100] (Table4). The latter may involve an accuracy-velocity tradeoff where ball velocity is reduced in an attempt to conserve accuracy.[41,44,100] Notably, diminished accuracy occurs with faster racquet speeds[41,137] Simulated tennis drills induce ~70% loss of ground-stroke accuracy at maximal RPE, but accuracy is unimpaired when RPE is 14, as in match-play.[35] In contrast, strenuous training for 2h caused accuracy to fall by just 6-11%,[44,100] and when fluid/carbohydrate is consumed during matches there is no loss of these skills.[100,140,142] Another study involving simulated table tennis[41] showed that in the fatigued state, forehand drive accuracy was maintained by elite players but with altered movement patterns, whereas recreational players lost precision. The latter occurred when the wrist joint was rigid and shoulder movements exaggerated.[41] These combined findings suggest that technique outcomes would be only marginally impaired with match-play. However, interventions which increase anxiety also lower hitting accuracy,[34] suggesting that psychological aspects may hinder technique during match-play

Potential mechanisms include reduced muscle performance via impaired motor drive,[9,10,138,139] excitation-contraction coupling[138] and/or lowered muscle glycogen.[140] Plasma glucose can either rise or fall[143,144] so that glucose supplementation sometimes,[95,140,141] but not always,[144] improves performance. Temporary fatigue may involve K^+,[145] phosphate metabolites,[9,10] and/or ROS.[146] Dehydration and/or hyperthermia, which reduce sprint ability are likely major contributors during exercise in the heat.[9,10,12] Also during long tournaments muscle damage and soreness[9,10,12,30] may impair on-court movements and specific skills. The moderate RPE observed in match-play[28,30,31,44,139] makes it an unlikely culprit.

3.4 Contribution of symptoms: The issue of which individual fatigue symptoms limit overall performance in sports is of interest. However, the data available on fatigue manifestations during sport competition are sparse. Also, winning a race or a phase of play can involve incredibly small differences in performance level,[25,26,129,130] so that quantitative comment on the relative importance of symptoms is of limited value. Never-the-less we make several qualitative speculations. 1) In brief all-out racing, the limitations are mainly peripheral with impairments of muscle power and coordination,[85-87,92,128] that also influence motor skills.[40] 2) In prolonged racing, decreases in muscle power involve both central and peripheral fatigue,[7,53] whilst fatigue sensations influence pacing strategies.[56,57] 3) Team-game and racquet sports (prolonged matches or tournaments) involve the same components as 2, but high RPE can be dampened by match behaviors including longer rest periods. Fine motor skills have an important (team-game sport) or very large involvement (racquet sport), with deterioration likely to impact the result. 4) High levels of decision-making (anticipation skills) are especially important for elite performance in interceptive (team-game/racquet) sports,[34,45,46,123] but not with sprint racing. However, to date there is no evidence supporting impaired decision-making in sport-events.[38,105,123,113,124] 5) Competitions in harsh environments can potentially bring on a full range of fatigue symptoms.[9-12] 6) Psychological aspects, especially motivation, are important for all sports. Just how/whether psychological aspects deteriorate is debatable, although they may contribute more in prolonged events. We next focus on fatigue mechanisms, noting that fatigue factors contribute to several fatigue symptoms. Therefore, it may be more useful to evaluate these factors, and potential methods to eradicate such changes, since this could enhance performance by attenuating multiple fatigue symptoms.

4. ARE THE SYMPTOMS OF FATIGUE LINKED BY COMMON MECHANISMS?

Fig.3 near here

4.1 Model to explain fatigue symptoms: Several physiological and psychophysical processes potentially interact to evoke multiple symptoms of fatigue (Fig.3). Muscle performance declines either directly through peripheral fatigue processes and/or indirectly by central fatigue processes. As exercise proceeds a rising corollary discharge associated with recruitment of more motor units to working and/or synergistic muscles,[2,6,89-91] leads to elevated RPE.[2,6,20] Afferent feedback from working muscles and circulating factors also elevate RPE.[20,72,102] These combined inputs to the perceived exertion area in the brain may then interact with the motor cortex leading to altered motor drive/behavior or the cessation of exercise. We reinforce the recent proposal[147] that the RPE area-motor cortex-working muscle sequence is a central feature for exercise (Fig.3). However, high RPE may not impair motor output,[72] and altered motor drive may benefit[1,2,15,72] rather than hinder the working muscles. That is, the events may not always act sequentially to lower performance, e.g. maximal RPE need not cause central fatigue.[15]

Modifying influences which impinge on the above sequence arise from exercise-induced peripheral changes, environmental and psychological effects, which together may elicit central fatigue, altered motor skills and fatigue sensations. Noakes and colleagues[20,22,23,54-56,102] have re-introduced interest on mind-body interactions through the central governor hypothesis for prolonged racing. Whilst the brain is undoubtedly the central integrator (Fig.3), arguments have appeared opposing any need for an independent subconscious central governor area.[24,147,148] Our model expands on the earlier schemes suggesting interplay between peripheral aspects and the CNS during exercise.[1,10,16,21,72,147] We include psychological and decision-making inputs, and emphasize that overall physical performance involves muscle force/power along with motor skills. A high positive motivation may counteract effects of raised RPE[22,55] via the RPE center[147] or indirectly via the motor cortex.[147] Improved decision-making may have a positive influence via the motor cortex. In contrast, mental fatigue,[55] lowered self-efficacy,[115,116] or anxiety may augment the exercise-induced rise of RPE to interfere with decision-making and/or directly reduce motor output.

4.2 Protective/compensatory mechanisms: The presence of safety margins, resisting and compensatory processes gives protection against catastrophic events such as rigor, severe muscle, brain or cardiac damage, or mechanical injury.[1,22] Indeed, the notion of compensatory effects within the neuromuscular system has been emphasised previously.[2] Muscle cells have several processes available to maintain ion[17,63] and metabolite levels,[13,17] and fuel supply.[13,17] Moreover, large perturbations of these factors to critical levels are needed before force declines, i.e. considerable safety margins exist.[17,63] At the single whole-muscle level changes of motoneuron firing pattern and recruitment of fresh motor units help to sustain submaximal forces.[2,6,15,89,90] At the limb or whole-body level, the activation of synergists and a sharing of workload between muscle groups can delay loss of technique or power.[2,3,6,51,97] Even when motor skills change there may still be successful technique outcomes.[38,95,96] Hence muscle function can become impaired but without consequences. Moreover, a rising RPE provides a conscious warning to alter exercise behavior.[24] Also, elevated hormonal levels during intense or prolonged exercise, e.g. catecholamines or endorphins, can improve mood,[16] decision-making[117,124] and attenuate several fatigue symptoms.[62,149,150] Even with physical deterioration a faster decision-making and behavioural changes may still permit a winning performance.

Table5 near here

4.3 Linking fatigue factors: It has been suggested that different fatigue measures involve quite distinct mechanisms.[6,24] Importantly, we now confirm that individual fatigue factors can actually evoke multiple fatigue symptoms (Table5). During brief sprints these factors are thought to be peripheral in origin,[17,47,49] without impaired motor drive,[85,86] whilst noting that coordination can worsen.[50,128] Many of the factors implicated in longer sport-events (Table5) have been shown to influence the CNS, or this can be inferred through effects via afferent feedback.[15,16,72,83,84] Some factors also modulate levels of other factors.[16,64,108,112]

4.3.1 Carbohydrate availability: A declining plasma glucose level during prolonged exercise leads to exhaustion when <3mM,[1,16,106-109] yet severe hypoglycaemia is uncommon in sport[8-

12,134,143,144] Interestingly, glucose administration protects against stimulation-induced peripheral fatigue,[61,105] along with other fatigue symptoms mediated via the CNS[80,100,105-109,122,140,141] (Table5). The latter is not surprising since carbohydrate is the preferential fuel for the CNS.[16,151] Moreover, glucose supplementation is effective only when glucose falls considerably,[105-109,144] presumably by restoring plasma/cerebral glucose,[106-108] cerebral glycogen[16,108] or stimulating afferent feedback from the mouth.[152] Low muscle glycogen is a common feature in sports exceeding 60min,[8-14,153] where it impairs intramuscular calcium release,[17,60] elevates RPE,[22,109,132] and may indirectly influence the CNS (Table5). Such effects via the CNS may involve interleukin-6, a cytokine released from glycogen depleted muscle,[154,155] and which stimulates afferent feedback or circulates to the CNS.[156] This hypothesis is strengthened by the finding that interleukin-6 injection promotes fatigue sensations and diminishes 10-km running performance in humans.[157] Alternatively, muscle/plasma carbohydrate deficiency may act through elevated brain serotonin.[14,16,158] Despite considerable indirect evidence and a sound rationale supporting a role for serotonin in fatigue processes,[14,16,52,159,160] its contribution in humans remains unclear.[14,160]

4.3.2 Hypoxia and acidosis: High-intensity exercise by athletes or exercise at altitude leads to systemic hypoxemia.[3,18,73] The resulting tissue hypoxia exacerbates force loss during fatiguing stimulation especially in fast-twitch fibers,[66] and alters afferent feedback.[76,161] In addition, the occurrence of cerebral hypoxia[16,76,162,163] impairs CNS function (Table5). Importantly, O_2 supplementation improves cycling or rowing performance over 5-8 min.[18,65,76,162,164] An intramuscular acidosis during intense exercise[1,13,17,64] only marginally depresses muscle force.[17,64] However, a large extracellular acidosis[64] stimulates group III and IV muscle afferents[83] and desaturates haemoglobin to induce cerebral hypoxia,[16,18,162] and therefore may cause central fatigue.[64] Evidence that a severe systemic acidosis increases RPE and reduces exercise tolerance comes from protection seen with bicarbonate.[64,110-112,165]

4.3.3 Hyperkalaemia: Potassium efflux from working muscle fibers increases [K^+] firstly in the interstitium,[63,166,167] and then plasma,[63] where increases have been shown during sport.[134,145] Unfortunately interstitial changes have not been measured during sport for technical reasons.[8,134] High interstitial [K^+] depresses muscle force,[58,59,62,63] stimulates group III and IV muscle afferents,[83,166,167] and may sensitize other sensory receptors.[72,166,167] This neural feedback along with raised cerebral [K^+][16] leads to the intriguing postulate that K^+ may influence the CNS.

4.3.4 Dehydration and hyperthermia: Effects of dehydration on muscle function are inconsistent,[168] although impairments have been observed.[168,169] Other symptoms appear when dehydration exceeds ~2% body weight (Table5). For example, dehydration sometimes causes deterioration of motor skills.[104,113,140,170] and elevates RPE.[113,135] Fluid ingestion can improve function directly via the water level or by lowering core temperature,[11,104,135] but it is often hard to distinguish between effects of dehydration and hyperthermia. When core temperature reaches a critical level of ~40°C, exercise is usually terminated.[16,77,93,94] The multitude of symptoms brought on by hyperthermia (Table5) may be consequent to high cerebral temperatures,[16,93,94] reduced cerebral blood flow,[16,93,94] and/or cerebral hypoxia following desaturation of haemoglobin.[16,18]

4.3.5 Reactive oxygen species: There is mounting interest in ROS during sport[136,146] since it impairs muscle function,[17,70] stimulates muscle afferents[156,171] and changes occur in the brain.[172] Interestingly, some antioxidants attenuate force loss during either repeated stimulation[67] or submaximal voluntary contractions,[68] and increase exercise tolerance.[69] Muscle damage following eccentric contractions in sport,[71] has been linked to ROS but this is usually treated as a separate phenomenon to fatigue.[17,71]

4.3.6 Protective aspects: Several factors initially exert protective before detrimental effects. For example, small increases of extracellular [K^+] or muscle temperature improve muscle/exercise performance.[8,63,70] Lactate has long been considered a villain yet it protects muscle function[17,64,173,174] and substitutes for glucose as a cerebral fuel substrate.[16,151] Also when factors change simultaneously during exercise (e.g. K^+, H^+, lactate) they sometimes counteract the detrimental effects imposed by each other factor in isolation.[17,63,173] Several factors activate group III and IV muscle afferents[2,6,83,156,161,171] to stimulate cardio-respiratory reflexes[166,167] or directly increase blood flow to working muscle,[166,167] and these are all beneficial responses.

5. CONCLUSION AND RECOMMENDATIONS

Table6 near here

The number and extent of the fatigue symptoms, and the fatigue factors involved, depends on the characteristics of the sporting task, the individual, and the environment. Manifestations of central and peripheral fatigue, impaired technique, and fatigue sensations often occur simultaneously, and psychological aspects may modify these symptoms. In many sports the decline of peak MVC force is moderate compared with stimulation-induced fatigue or some laboratory exercise. Moreover, central fatigue can account for much of the depressed MVC in prolonged races and match-play. Anecdotal suggestions of impaired mental function during sport are generally unsupported by testing. Instead, decision-making is unchanged/improves unless associated with hyperthermia, dehydration or hypoglycaemia. We suggest that the main factors causing fatigue symptoms include: i) diminished carbohydrate availability, increased brain serotonin, and dehydration during prolonged sports, ii) hyperkalaemia, systemic acidosis and hypoxia during high-intensity sports, and iii) hyperthermia, dehydration, and hypoxia during sport-events in hostile environmental conditions. In order to understand both compensatory and detrimental processes during fatiguing activities more studies should make use of an expansionist approach. This involves starting from a reduced muscle model then systematically incorporating exercise features in a stepwise manner, to eventually resemble a real-sport competition. Furthermore, the processes involved in mind-body interactions during sport, warrants greater investigation. Other recommendations are given in Table6.

On returning to our introductory comments, part of the reason for contradictions in the fatigue literature arise from a lack of clarity about how to describe fatigue[1,5,6,24] and from a belief that it is necessary to use a precise definition of fatigue. We reveal multiple symptoms of fatigue and show that single fatigue factors can influence several symptoms (Table5), hence overlapping and interacting mechanisms are involved. Therefore it makes sense to use a global definition of fatigue such as *an exercise-induced decline of performance*. However, when undertaking mechanistic studies to explain components of the fatigue phenomenon, precise details on the performance symptoms or test measures to be quantified, are required. Thus, descriptions of fatigue which specify the performance components such as any reduction in the capacity to exert muscle force/power during voluntary exercise,[3] or the inability to sustain a task,[1,2,6] or the occurrence of fatigue sensations,[102] can all justifiably be included.

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FIGURE LEGENDS

Figure 1. The assessment of fatigue can be made at three different levels - muscle, exercise or competition performance. Performance symptoms can only be obtained during sport competition. Muscle and/or exercise test measures can be made after sport-events or laboratory exercise tasks. Stimulation-induced fatigue or prolonged activation of a single muscle group is only assessed with muscle performance test measures. The dotted boxes indicates aspects that may change and influence performance, and the levels where they can influence performance measures.

Figure 2: Running velocity as a percentage of race distance covered for 100-m (●), 400-m (○) and 10-km (■) events by elite male athletes. Velocity was averaged over each 10 m, 50 m or 1 km for the respective events. Data are mean values (±SD) from the finals at the 1997 World Athletic Championships.[25] For the 100-m race the peak velocity fell after 5-6 s to the finish line by 3.7-10.1%. For the 10-km race the velocity increased on average by 4.7% from the 9th to 10th km.

Figure 3: Model depicting the perceived exertion-motor cortex-working muscle sequence responsible for symptoms of fatigue during exercise/sport. Inputs to this sequence (dotted lines) from higher brain centers (psychological, decision-making), afferent feedback (from working muscle, other peripheral sites), and circulating feedback (from body environment) act through perceived exertion and/or motor areas to modulate motor drive/behavior. Dotted boxes have potential to interact (not indicated above).

Table 5: Do individual fatigue factors contribute to multiple fatigue symptoms? The selected references provide supporting evidence. Some aspects are unknown, unclear (inconsistent findings), or likely (there is suggestive/indirect support). Note: factors must change considerably before significant symptomatic changes occur. Peripheral fatigue: direct impairment of muscle force either at rest or during fatiguing stimulation. Central fatigue: impairment of muscle force through reduced motor drive (shown by twitch- or tetanus-interpolation during brief or sustained MVC). Time-to-exhaustion is assessed with submaximal, maximal and/or incremental exercise tests.

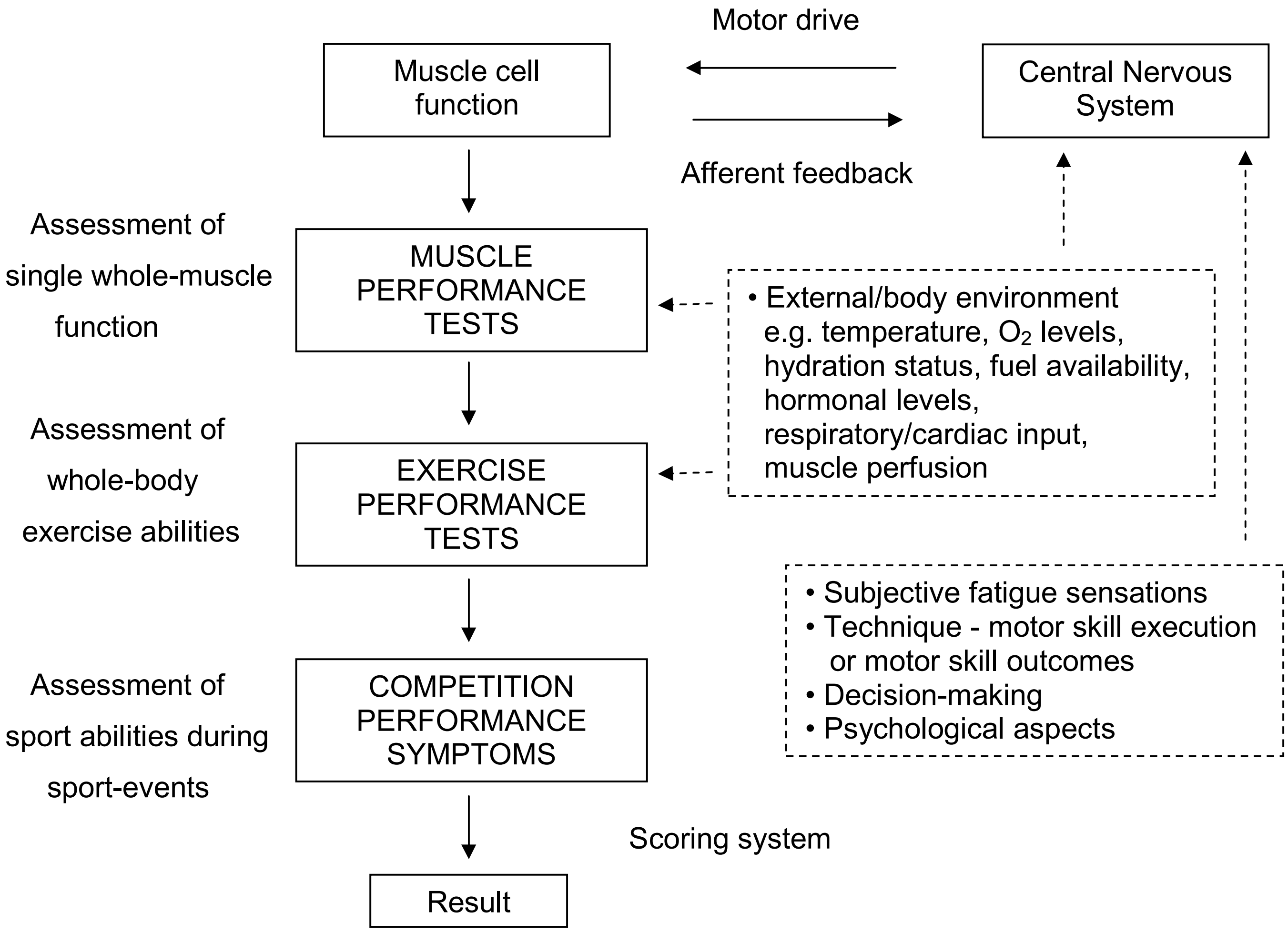

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Table 1: Common manifestations of fatigue during exercise or sport competition

Performance symptoms

- ↓ whole body work rate/velocity, inability to surge, ↑ rest periods, cessation of exercise
- ↓ technique execution (tired looking movements)
- ↓ hitting, kicking or throwing velocity
- ↑ error rate (e.g. ↓ accuracy of hitting, kicking or passing, missed catches or tackles)
- ↑ mental lapses (i.e. ↓ concentration, ↑ tiredness, slower/inaccurate decisions) *

Test measures

Physical exercise:

- ↓ muscle force (e.g. peak MVC, isokinetic, or tetanic forces)
- ↓ muscle, limb/joint, or whole-body power
- ↑ time (endurance, sprint, agility, repeated sprint)
- ↓ stride frequency, ↓ stroke length, ↓ pedal rate, ↓ range of motion

Technique:

- ↓ motor skill execution (e.g. ↓ foot or hand speed)
- ↓ motor skill outcome (e.g. ↓ ball velocity or accuracy)

Subjective sensations: **

- ↑ sense of effort (i.e. ↑ rating of perceived exertion)
- ↑ sense of generalized fatigue/tiredness
- ↑ sense of force (includes ↑ sense of heaviness)
- ↑ ratings of muscle soreness, ↑ discomfort, ↑ pain

Decision-making: ***

- simple and choice reaction times, visual recognition tests, Stroop test, mental concentration tests
- sport-related anticipation tests

Psychological aspects: **

- motivation, self-efficacy, anxiety

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*anecdotal only. **assessed using rating scales or questionnaires. ***are often improved or unchanged. Note: the subjective sensations, decision-making, or psychological aspects may also influence other measures. MVC - maximal voluntary isometric contraction.

Table 2: Selected performance symptoms are associated with several component test measures.

Performance symptoms		**Test measures**								
1. ↓ locomotor speed	←	↓ limb power	←	↓ muscle power	←	↓ muscle force	←	↓ motor drive	←	↑ RPE
↑				↓ coordination		↓ muscle velocity				↓ self-efficacy
(↓ stride/pedal rate, ↓ stroke length)										↓ motivation
2. ↓ speed to the ball/tackle	←	↓ locomotor speed								
		↓ interceptive skills (i.e. ↓ anticipatory speed/accuracy)	←	↓ concentration						
3. ↓ hitting/kicking accuracy	←	↓ speed to the ball								
		↓ technique execution	←	Δ muscle power	←	↑ muscle velocity	←	Δ motor drive	←	↑ RPE
				↓ coordination		Δ muscle force				↓ self-efficacy
										↑ anxiety
										↓ concentration
4. ↓ number high-intensity bursts	←	↑ RPE	←	physiological/psychological inputs						
(or cessation of exercise)		↑ sense of generalized fatigue/tiredness								
		↑ ratings of muscle soreness, ↑ discomfort, ↑ pain								
		↓ motivation								

Some links (and changes) depicted above are speculative.

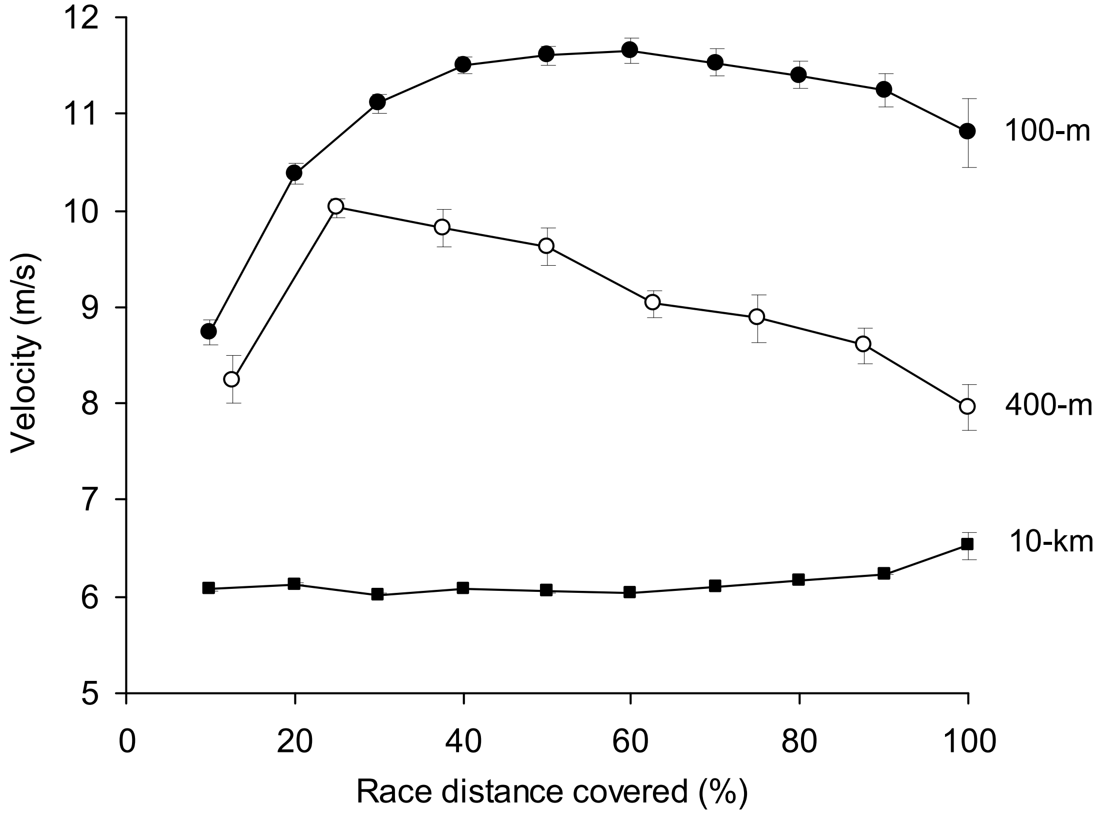

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Table 3: Symptoms/measures of fatigue during team-game sports

	References
- reduced work rate (1st vs. 2nd half, quarters, consecutive days): #	[8,11,27,29,131]
• ↓ total distance covered	
• ↓ proportion of time spent sprinting (↓ 40% in last 15 min)	
• ↑ proportion of time spent standing or walking	
• ↓ tackles in 2nd half, less work off the ball	
- ↑ repeated sprint/sport-related agility time	[37,39,87,103-105,113,134,135]
- ↓ peak isokinetic force (↓ 5-20% quadriceps, hamstring strength)	[11,36]
- ↓ leg/whole body power (↓ vertical jump height, ↓ 10% rugby drive-power)	[37,103]
- ↓ joint range of motion	[39,99]
- impaired technique execution:	[11,37,38,43,98,99]
• ↓ rugby tackling skills	
• ↓ water polo shooting skills	
• ↓ soccer kicking motion	
- reduced technique outcome:	[37,98,99,103-105,132,170]
• ↓ soccer kicking speed (24.7 to 21.8 m/s)	
• ↓ soccer passing/shooting accuracy	
• ↓ rugby passing accuracy	
• ↓ basketball shooting accuracy	
• ↓ cricket bowling accuracy	
- ↑ RPE (15-19)	[38,43,113,132,135]
- ↑ sense of generalized fatigue	[103-105]
- ↑ muscle soreness	[103]
- unchanged mental concentration	[113,135]
- improved decision-making	[38,105,123,124]

From time-motion analysis of soccer, hockey, basketball matches. All other data including whole-body RPE (15-point Borg scale) are from sport-related activities.

Table 4: Symptoms/measures of fatigue during racquet sports

	References
Anecdotes	[9,10,12,35]
- impaired on-court movement characteristics:	
• ↓ speed to ball, poor body position relative to ball (i.e. a late hit)	
• ↓ ability to reach wide ball	
- diminished stroke quality/mistimed strokes:	
• ↓ ball accuracy or velocity	
• ↑ error rate (hit net or out)	
• ↓ racquet grip firmness, poor position of racquet head	
- ↓ aggression, ↑ lethargy/tiredness, ↑ mental lapses	
Measurements	
- ↓ effective playing time	[30]
- ↑ single or shuttle sprint time	[44,100,137,142]
- ↓ peak MVC force (↓ 10-13%), ↓ 9% leg stiffness	[30,138,139]
- unchanged leg power (standing, counter-movement jumps)	[30]
- unchanged peak tetanic force (80 Hz)	[30,138]
- low-frequency fatigue (↓ 20/80 Hz force ratio)	[138]
- ↓ joint range of motion:	[41]
- impaired technique outcome:	[35,41,44,100,137,140,141]
• ↓ service, ground stroke/driving accuracy (includes ↑ 9% error rate)	
• ↓ service, ground stroke/driving velocity (forehand 116 to 111 km/h)	
- ↑ RPE (13-16) *	[28,30,31,44,100,139]
- ↑ RPE (17-20) **	[35,41,137]
- ↑ muscle soreness	[30]

Data are from tennis, table tennis and squash. * from match-play/practice. ** from simulated racquet sport activities. Effective playing time = (ratio of real to total playing time). RPE is whole-body or limb ratings assessed with the 15-point Borg scale.

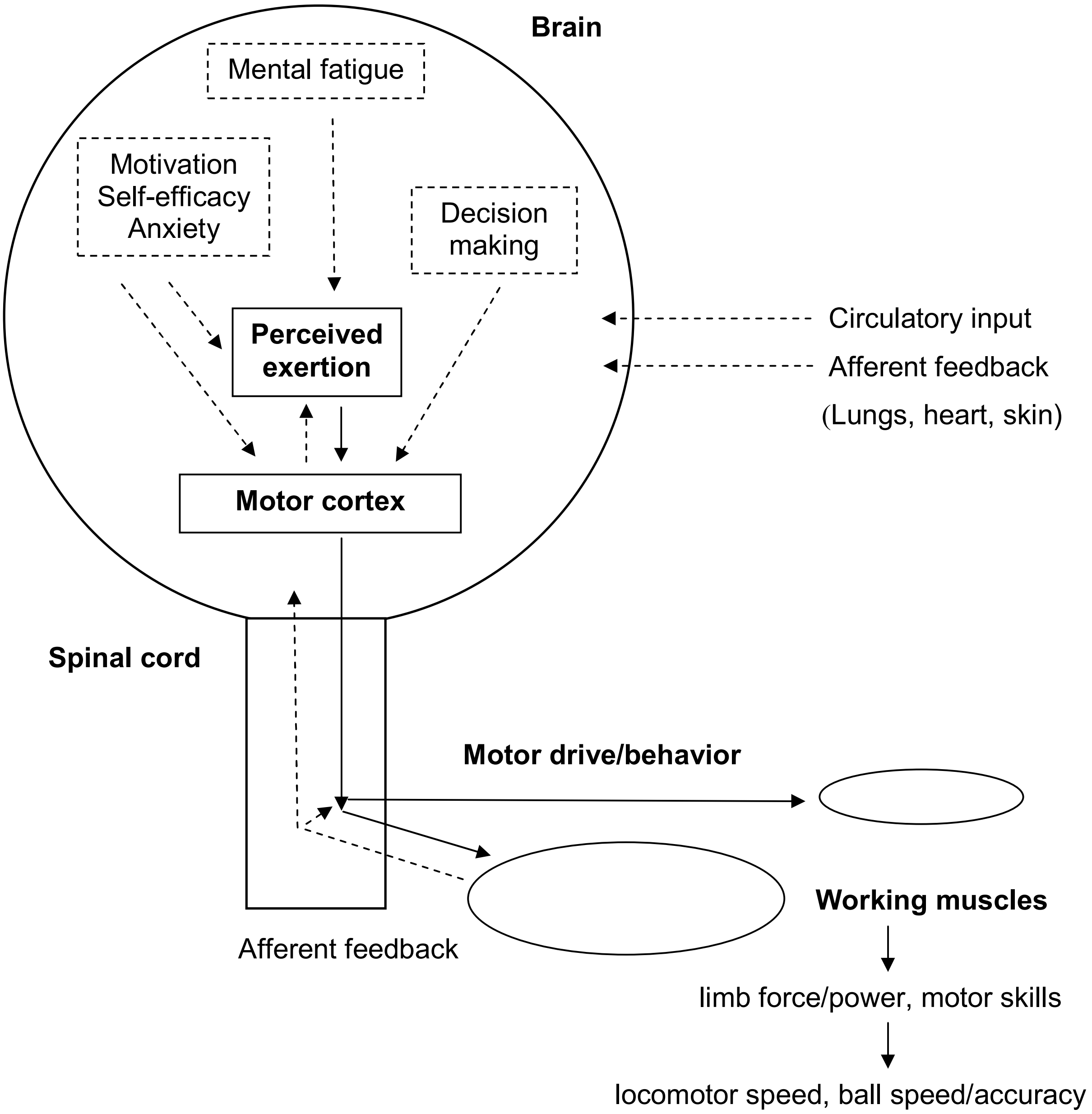

Figure 3: Model depicting the perceived exertion-motor cortex-working muscle sequence responsible for symptoms of fatigue during exercise. Inputs to this sequence (dotted lines) from higher brain centers (psychological, decision-making), afferent feedback (from working muscle, other peripheral sites), and circulating feedback (from body environment) act through perceived exertion and/or motor areas to modulate motor drive/behavior. Dotted boxes have potential to interact (not indicated above).

Fatigue Factor	Peripheral fatigue	Afferent feedback	Central fatigue	↓ Time to exhaustion	↑ RPE	↓ Motor skill outcome	↓ Decision making
↓ glucose	Yes [61,105]	Yes [152]	Yes [16,80]	Yes [105-109,152]	Yes [106,107,120]	Yes [100,132,140,141]	Yes [117,122]
↓ glycogen	Yes [60]	Likely [156,171]	Likely [53,157]	Yes [109,153]	Yes [22,109,132]	Yes [132]	Likely
↑ serotonin	No	No	Likely [14,16,159,160]	Yes [14,52,159,160]	Yes [52,153]	Unknown	Unknown
↓ O_2	Yes [18,65,66,73]	Yes [76,161]	Yes [18,73-76]	Yes [22,65,162,164]	Yes [22,65,164]	Unknown	Unknown
↑ H^+	Yes [13,17,64]	Yes [83]	Likely [64]	Yes [64,111,165]	Yes [64,110-112]	Unknown	Unknown
↑ K^+	Yes [58,59,62,63]	Yes [83,166,167]	Likely [16]	Yes [69]	Likely	Unknown	Unknown
↓ H_2O	Unclear [168,169]	Unknown	Unclear [168,169]	Yes [168]	Yes [113,135]	Yes [104,113,140,170]	Yes [117,120]
↑ temperature	No [8,70]	No	Yes [16,77-79]	Yes [54,93]	Yes [16,54,93,94]	Yes [104,135]	Yes [120,121]
↑ ROS	Yes [17,67-70]	Yes [156,171]	Unknown	Yes [69]	Unknown	Unknown	Unknown

Table 5: Do individual fatigue factors contribute to multiple fatigue symptoms?

Table 6: Recommendations for future work

1. Performance symptoms/test measures need to be assessed during the actual sport competition. In particular the temporary fatigue within a match and cumulative fatigue after a tournament need greater attention. Development of techniques for rapid measurements and which do not interfere with the sport are required.
2. More realistic sport simulations are needed to satisfy external validity since the severity of fatigue measures obtained after simulated-sport activities can be excessive compared with sport competition.
3. Many muscle function tests have utilized the quadriceps muscle. Other sport-relevant muscle groups, especially those of the distal limbs should receive greater attention.
4. Whether and how high RPE influences physical and technical abilities rather than just the time-to-exhaustion should be addressed.
5. Greater understanding of the compensatory processes during fatigue in sport is needed.
6. Enhanced understanding of the factors and processes which link neuromuscular, motor skill, subjective and psychological aspects during exercise is required.