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Multiple Sprint Work

Physiological Responses, Mechanisms of Fatigue and the Influence of Aerobic Fitness

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Abstract

The activity patterns of many sports (e.g. badminton, basketball, soccer and squash) are intermittent in nature, consisting of repeated bouts of brief (≤6-second) maximal/near-maximal work interspersed with relatively short (≤60-second) moderate/low-intensity recovery periods. Although this is a general description of

the complex activity patterns experienced in such events, it currently provides the best means of directly assessing the physiological response to this type of exercise. During a single short (5- to 6-second) sprint, adenosine triphosphate (ATP) is resynthesised predominantly from anaerobic sources (phosphocreatine [PCr] degradation and glycolysis), with a small (<10%) contribution from aerobic metabolism. During recovery, oxygen uptake ($\dot{V}O_2$) remains elevated to restore homeostasis via processes such as the replenishment of tissue oxygen stores, the resynthesis of PCr, the metabolism of lactate, and the removal of accumulated intracellular inorganic phosphate (P_i). If recovery periods are relatively short, $\dot{V}O_2$ remains elevated prior to subsequent sprints and the aerobic contribution to ATP resynthesis increases. However, if the duration of the recovery periods is insufficient to restore the metabolic environment to resting conditions, performance during successive work bouts may be compromised. Although the precise mechanisms of fatigue during multiple sprint work are difficult to elucidate, evidence points to a lack of available PCr and an accumulation of intracellular P_i as the most likely causes. Moreover, the fact that both PCr resynthesis and the removal of accumulated intracellular P_i are oxygen-dependent processes has led several authors to propose a link between aerobic fitness and fatigue during multiple sprint work. However, whilst the theoretical basis for such a relationship is compelling, corroborative research is far from substantive. Despite years of investigation, limitations in analytical techniques combined with methodological differences between studies have left many issues regarding the physiological response to multiple sprint work unresolved. As such, multiple sprint work provides a rich area for future applied sports science research.

1. Activity Profiles of Multiple Sprint Sports

The activity patterns of many sports are intermittent in nature, fluctuating randomly from brief periods of maximal or near maximal work to longer periods of moderate- and low-intensity activity. The duration of these events is often >1 hour and in the case of team sports (e.g. basketball, hockey, rugby and soccer), activity patterns are considerably influenced by player position.[1-6]

In field sports (e.g. hockey, rugby and soccer), distances covered during games range from 5000 to 11 000m depending on player position, skill level and game duration.[1,2,7] The percentages of gametime spent in various forms of locomotion are difficult to quantify due to methodological differences between studies. However, the mean duration of high-intensity efforts is reported to be approximately 4–7 seconds,[1,3,6,8] of which approximately 2 seconds is attributed to all-out sprinting.[1,3,9] Although the ratio of high- to low-intensity activities ranges from 1 : 6 to 1 : 14,[2,6,10-12] values are clouded by limitations in the various methods used to determine these intensities.

In contrast to field sports, racquet sports (e.g. badminton, squash and tennis), due to the nature of the games, display much more consistent activity patterns. In general, high-intensity efforts (rallies) are on average 5–10 seconds in length depending on playing ability,[13-19] with work to rest ratios ranging from 1 : 1 to 1 : 5. A summary of the results of several time-motion analyses of racquet sports is presented in table I.

2. Physiological Demands of Multiple Sprint Sports

Research into the physiological demands of multiple sprint sports indicates that these events place considerable demands on both aerobic and anaerobic pathways, although the relative contribution from

Table I. Typical work to rest ratios experienced in racquet sports

Sport	Playing level	Mean rally time (sec)	Work : rest ratio	Reference
Squash	Range of abilities	4.4–8.8	1 : 1	14
		6.9–16.6	1 : 1	19
Badminton	Range of abilities	4.2–4.9	1 : 2	14
	National level	7.4	1 : 2	16
		4.6	1 : 2	18
Tennis	State level	10.2	1 : 1.7[a]	13
	Range of abilities	4.0–4.3	1 : 5	14
	College level	10.0	1 : 1.8[a]	15

a Does not include time spent changing ends.

each of these sources is an issue of some controversy.[8,15,20-22] The average physiological response to intermittent sporting events is reported to be similar to that of prolonged continuous exercise, with mean exercise intensities of 60–75% maximum oxygen uptake ($\dot{V}O_{2max}$),[10,12,13,16,19,23] and mean heart rates of 70–90% of maximum.[4,13-17,19] However, expressing intensity as an average value during a game is likely to mask the complexity of the physiological processes that regulate this type of activity. Moreover, field-based physiological assessments of multiple sprint sports have several limitations. For instance, direct field-based assessments of oxygen uptake ($\dot{V}O_2$) are confounded by the inhibitory effects of the portable devices currently available for this type of assessment. Furthermore, this type of assessment is only feasible in simulated match-play. One way to address this problem has been to predict $\dot{V}O_2$ from heart rate data using laboratory-determined submaximal heart rate/$\dot{V}O_2$ relationships. However, heart rate/$\dot{V}O_2$ relationships can be compromised during intermittent work due to factors such as emotional stress, elevated levels of catecholamines, and the accumulation of various metabolic by-products.[10,13,16,24]

Field-based assessments of blood lactate have often been used to indicate anaerobic lactacid adenosine triphosphate (ATP) production. However, blood lactate levels are only a reflection of the balance between lactate production and clearance. Furthermore, sampling times are restricted to natural breaks in matches or disruptions to standard match conditions and only reflect the level of activity during the few minutes prior to sampling. Although field-based assessments of blood lactate during multiple sprint sports generally report relatively low mean values of between 2 and 5 mmol/L,[1,3,10,16,18,19,25] peak values as high as 10 mmol/L have been recorded.[12]

The limitations associated with field-based physiological assessments of multiple sprint sports have led many researchers to investigate this type of work in a laboratory setting.[26-31] These studies have typically examined brief (≤6-second) bouts of maximal work interspersed with relatively short (≤60-second) stationary recovery periods. Although laboratory-based investigations of intermittent work differ considerably from the activity patterns experienced in the field, they currently provide the best means of directly assessing the physiological response to this type of activity. Before reviewing research into the metabolic factors that may limit performance, it is important to consider the complex energetics associated with this type of work.

3. The Energetics of Brief Maximal Work

3.1 Adenosine Triphosphate

Energy for muscular work is obtained from the hydrolysis of ATP (equation 1).

$$ATP \underset{ATPase}{\rightleftharpoons} ADP + P_i + energy \quad \text{(Eq. 1)}$$

where ADP is adenosine diphosphate and P_i is inorganic phosphate. Within muscle, the human body typically stores approximately 20–25 mmol/kg dry muscle (dm) of ATP, which with peak ATP turnover

rates of approximately 15 mmol/kg dm/sec, is enough to fuel 1–2 seconds of maximal work.[29,32,33] As the store of ATP becomes depleted, ATP for continued muscular work is resynthesised by the integration of various metabolic processes.

3.2 Phosphocreatine

Phosphocreatine (PCr) is particularly important during explosive activities when a high rate of energy release is required (equation 2). The resynthesis of ATP is driven by the reaction between PCr and ADP. The reaction is catalysed by the enzyme creatine kinase and results in the formation of ATP and free creatine (Cr).

$$PCr + ADP + H^+ \underset{\text{Creatine kinase}}{\rightleftharpoons} ATP + Cr$$

(Eq. 2)

Intramuscular PCr stores total approximately 80 mmol/kg dm.[29,32-34] During maximal work, PCr degradation follows an exponential pattern of decay (figure 1) with maximal turnover rates of approximately 9 mmol ATP/kg dm/sec,[35] largely depleting stores within 10 seconds.

3.3 Anaerobic Glycolysis

Anaerobic glycolysis involves the breakdown of glucose, mainly in the form of muscle glycogen, to ATP and lactate (equation 3).

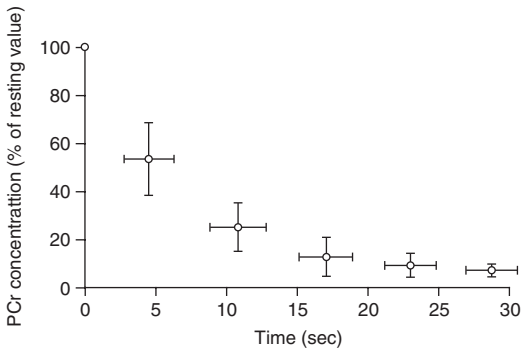

Fig. 1. Phosphocreatine (PCr) kinetics of the medial gastrocnemius during 30 seconds of repeated maximal plantar flexions of the foot determined from localised nuclear magnetic resonance imaging. Open circles represent PCr as a percentage of resting values; bars represent standard deviations (reproduced from Walter et al.,[36] with permission).

$$\text{Glycogen} + 3\ ADP + 3\ P_i \longrightarrow 3\ ATP + 2\ \text{lactate}^- + 2\ H^+$$

(Eq. 3)

ATP production from anaerobic glycolysis is activated rapidly at the onset of maximal work reaching peak rates of around 6–9 mmol ATP/kg dm/sec[33,35,37,38] after approximately 5 seconds.[39,40]

3.4 Aerobic Metabolism

During maximal work, aerobic ATP resynthesis is achieved primarily through the oxidation of glucose (equation 4).[34,41]

$$C_6H_{12}O_6\ (\text{glucose}) + 6O_2 + 38\ ADP + 38\ P_i \longrightarrow 6\ CO_2 + 6\ H_2O + 38\ ATP$$

(Eq. 4)

It is difficult to accurately assess the aerobic contribution to a short bout of maximal work due to methodological problems associated with: (i) assessing the $\dot{V}O_2$ of the working muscles; (ii) determining the size of the active muscle mass; and (iii) evaluating the contribution of oxygen released from myoglobin. However, during the first 6 seconds of a 30-second maximal sprint, the mean rate of aerobic ATP turnover has been estimated at 1.32 mmol ATP/kg dm/sec (approximately 9% of the total energy produced).[33]

3.5 The Adenylate Kinase Reaction

During intense periods of work, when the required rate of ATP provision cannot be maintained by the above energy pathways, ATP can be generated from pairs of ADP molecules. The reaction is catalysed by the enzyme adenylate kinase and results in the formation of ATP and adenosine monophosphate (AMP) [equation 5].

$$ADP + ADP \underset{\text{Adenylate kinase}}{\rightleftharpoons} ATP + AMP$$

(Eq. 5)

AMP is further deaminated to inosine monophosphate (IMP) and ammonia in a reversible reaction catalysed by the enzyme AMP deaminase (equation 6).

$$AMP + H^+ \underset{\text{AMP deaminase}}{\rightleftharpoons} IMP + NH_4^+$$

(Eq. 6)

Although these reactions may temporarily reduce the availability of adenine nucleotides for phosphorylation, the majority are resynthesised during recovery via the purine nucleotide cycle. Moreover, high-intensity training is reported to reduce the loss of adenine nucleotides during intense exercise.[42]

3.6 Summary

During brief periods of maximal work, ATP provision is maintained through the complex integration of various metabolic processes. These processes work together to achieve peak ATP turnover rates of around 15 mmol ATP/kg dm/sec. However, as work bouts are repeated, as in many team sports, the metabolic response to subsequent work bouts is determined by the duration of the intervening rest periods.

4. The Physiology of Multiple Sprint Work

Early investigations into the energetics of short (≤10-second) bouts of intermittent work suggested that the ATP required to fuel contractile activity was derived predominantly from aerobic metabolism.[43,44] The theoretical basis for this conclusion was that oxygen bound to myoglobin offset the usual oxygen deficit that occurs at the onset of a bout of exercise. This store would subsequently be replenished during each recovery period, thereby providing a large aerobic contribution to overall energy production. However, the intensities of the work bouts used in these investigations were considerably less than maximal. In contrast, Margaria et al.,[45] using intensities sufficient to exhaust subjects within 30–40 seconds of continuous treadmill running, suggested that with sufficient recovery (≥25 seconds) the ATP required to fuel 10-second bouts of 'heavy' intermittent work was derived predominantly from the degradation of PCr. However, this conclusion was highly speculative, as PCr was not measured in the study. It is now accepted that intermittent bouts of brief maximal work are fuelled by the integration of the aforementioned metabolic pathways. The role of these pathways during multiple sprint work will be the focus of the next section of this article.

4.1 Anaerobic Energy Provision During Multiple Sprint Work

4.1.1 Phosphocreatine

During a single short (5- to 6-second) maximal sprint, PCr degradation is reported to account for approximately 50% of the total anaerobic ATP provision.[29,33,46] However, the PCr contribution during repeated sprints is largely determined by the extent to which PCr stores are replenished during intervening recovery periods. The recovery kinetics of PCr have been examined *in vivo* (using ^{31}P magnetic resonance spectroscopy) and *in vitro* (using muscle biopsies) in several investigations.[36,47-57] The consensus of opinion appears to be that PCr recovery kinetics are extremely complex, as reflected by large individual and between-protocol differences.

Analyses of PCr recovery kinetics under ischaemic conditions have demonstrated that PCr resynthesis is achieved exclusively via aerobic ATP resynthesis.[48,51,53,55] Moreover, PCr recovery kinetics have been shown to be sensitive to manipulations of oxygen availability (figure 2).[52,58] After submaximal work, with minimal disruption to pH, PCr follows a monoexponential pattern of resynthesis (figure 2), the time/rate constants of which are re-

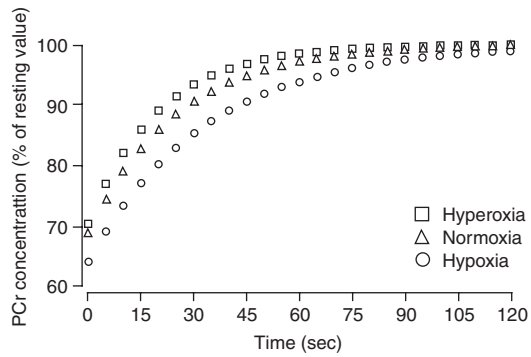

Fig. 2. The influence of oxygen availability on phosphocreatine (PCr) recovery kinetics of the gastrocnemius following 5 minutes of repeated submaximal plantar flexions of the foot determined from localised nuclear magnetic resonance imaging.[52]

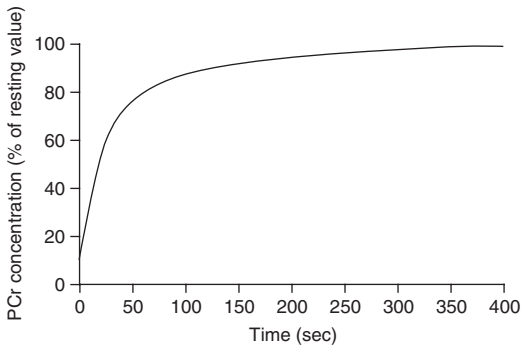

Fig. 3. Phosphocreatine (PCr) recovery kinetics of the medial gastrocnemius following 30 seconds of repeated maximal plantar flexions of the foot determined from localised nuclear magnetic resonance imaging (reproduced from Walter et al.,[36] with permission).

ported to provide an index of oxidative capacity.[56,57] However, following maximal work, PCr recovery kinetics are best described by a biexponential pattern of resynthesis (figure 3), the initial fast phase of which is reported to be largely unaffected by the concomitant drop in pH.[36,54,55]

Information on the influence of recovery duration on PCr resynthesis during short-duration maximal intermittent work is sparse due to the invasive nature of muscle biopsy procedures and the fact that ^{31}P magnetic resonance spectroscopy techniques cannot as yet be used to examine the large muscle masses involved in sprint work. However, using 10×6-second maximal sprints (cycle ergometer), Gaitanos et al.[29] reported that 30-second recovery periods enabled PCr to make a substantial contribution (≥50% of the total anaerobic ATP provision) to ATP resynthesis throughout each sprint. Furthermore, despite a progressive decline in the pre-sprint concentration of PCr throughout each trial, it is likely that with resynthesis rates of around 1.3 mmol/kg dm/sec, 30-second recovery periods would have enabled PCr to continue to make a substantial contribution to total ATP resynthesis beyond the final sprint.

4.1.2 Glycolysis

During a brief maximal sprint, the rapid drop in PCr concentration is offset by the increased activation of glycolysis with the two processes combining to maintain ATP turnover at a rate of 11–14 mmol ATP/kg dm/sec.[29,46] At high glycolytic rates, the concentration of muscle lactate increases to extremely high levels and the associated increase in hydrogen ion (H^+) concentration has often been implicated as a cause of fatigue.[59-61] During recovery, glycolysis is reportedly switched off[53,62,63] and the return of pH to resting levels follows a monoexponential pattern of resynthesis (figure 4) with a half-time of approximately 9 minutes.[60,64]

The rate of glycolytic ATP provision is regulated by the intricate interplay between many metabolic factors (figure 5). During maximal intermittent work, progressive changes in the metabolic environment lead to a gradual inhibition of glycolysis with repeated sprints.[29,33,65,66] For example, in the study by Gaitanos et al.,[29] glycolysis accounted for 44% of the total anaerobic ATP provision during the first sprint, whilst the corresponding value for the tenth sprint was 16% (figure 6). Moreover, in four of the subjects (n = 7), the glycolytic contribution to total anaerobic ATP production during the tenth sprint was estimated to be zero.

Various mechanisms have been postulated to account for the inhibition of glycolysis with repeated sprints.[65] One suggestion is that glycolysis is impaired by the progressive depletion of muscle glycogen stores that accompanies this type of work.[29,67] Several studies have reported altered glycolytic rates following glycogen manipulation.[68-70] In contrast, other investigations report contradictory findings.[71-75] Another suggestion is that glycolysis is impaired by the aforementioned progressive drop in pH. An accumulation of H^+ is known to inhibit

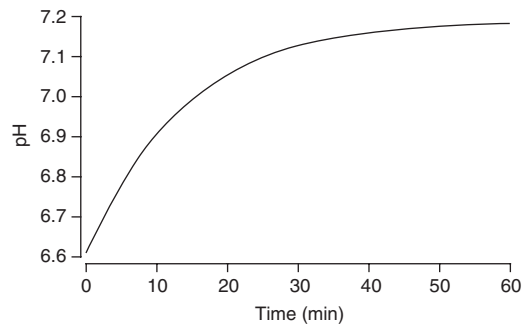

Fig. 4. Time course of muscle pH during passive recovery from 6 minutes of exhaustive dynamic exercise.[64]

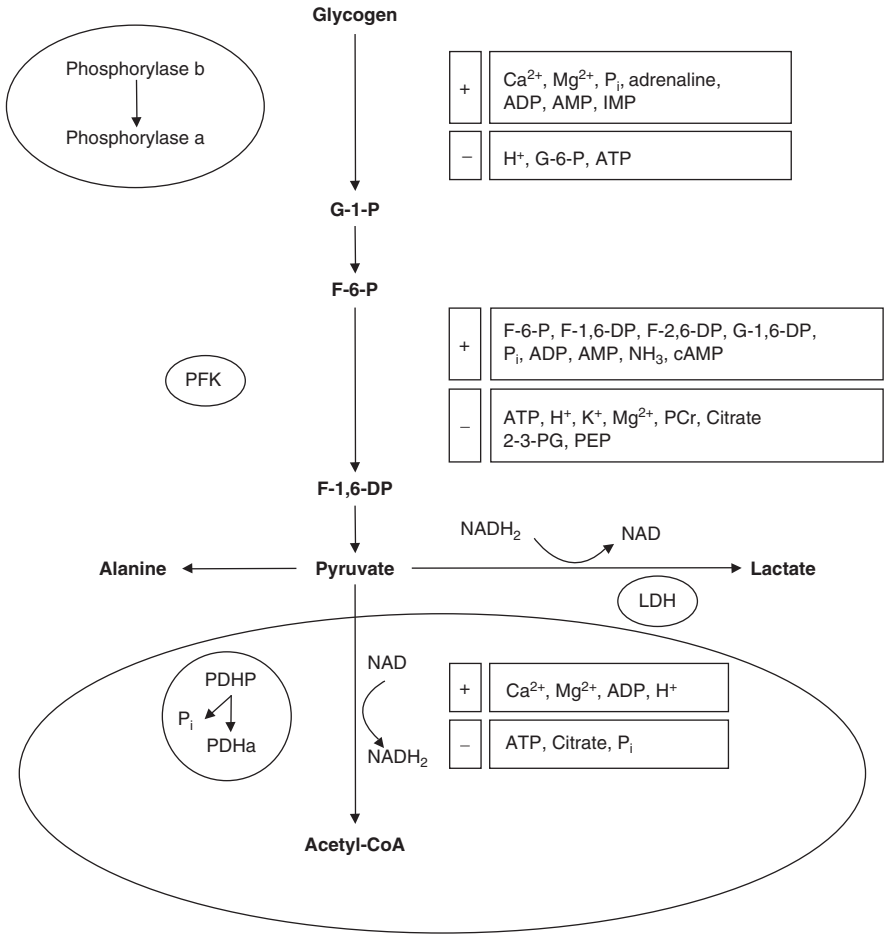

Fig. 5. Schematic representation of the anaerobic metabolic pathways of glycogenolysis/glycolysis and a number of potential regulators (reproduced from Bangsbo,[65] with permission). **2-3-PG** = 2-3-phosphoglycerate; **ADP** = adenosine diphosphate; **AMP** = adenosine monophosphate; **ATP** = adenosine triphosphate; **cAMP** = cyclic adenosine monophosphate; **CoA** = coenzyme A; **F-1,6-DP** = fructose-1,6-diphosphate; **F-2,6-DP** = fructose-2,6-diphosphate; **F-6-P** = fructose-6-phosphate; **G-1,6-DP** = glucose-1,6-diphosphate; **G-1-P** = glucose-1-phosphate; **G-6-P** = glucose-6-phosphate; **IMP** = inosine monophosphate; **LDH** = lactate dehydrogenase; **NAD** = nicotinamide-adenine dinucleotide; **$NADH_2$** = the reduced form of NAD; **PCr** = phosphocreatine; **PDHa** = active form of pyruvate dehydrogenase; **PDHP** = pyruvate dehydrogenase phosphatase; **PEP** = phosphoenolpyruvate; **PFK** = phosphofructokinase; **P_i** = inorganic phosphate; **+** indicates positive regulators; **–** indicates negative regulators.

phosphorylase and phosphofructokinase (PFK), the key regulatory enzymes of glycogenolysis and glycolysis.[76] However, the influence of pH on PFK is reported to be negligible under normal physiological conditions ($pH \geq 6.4$).[77,78] A third possibility is that glycolysis is inhibited by an accumulation of cytosolic citrate, since citrate also exerts an inhibitory effect on PFK.[76,79-82] However, the influence of citrate on PFK is reportedly small within the normal physiological range of 0.1–0.3 mmol/L.[83] Although the progressive impairment of glycolysis during repeated maximal sprints may result from the interplay between several regulatory processes, further investigations are required before the precise mechanisms of glycolytic inhibition can be identified.

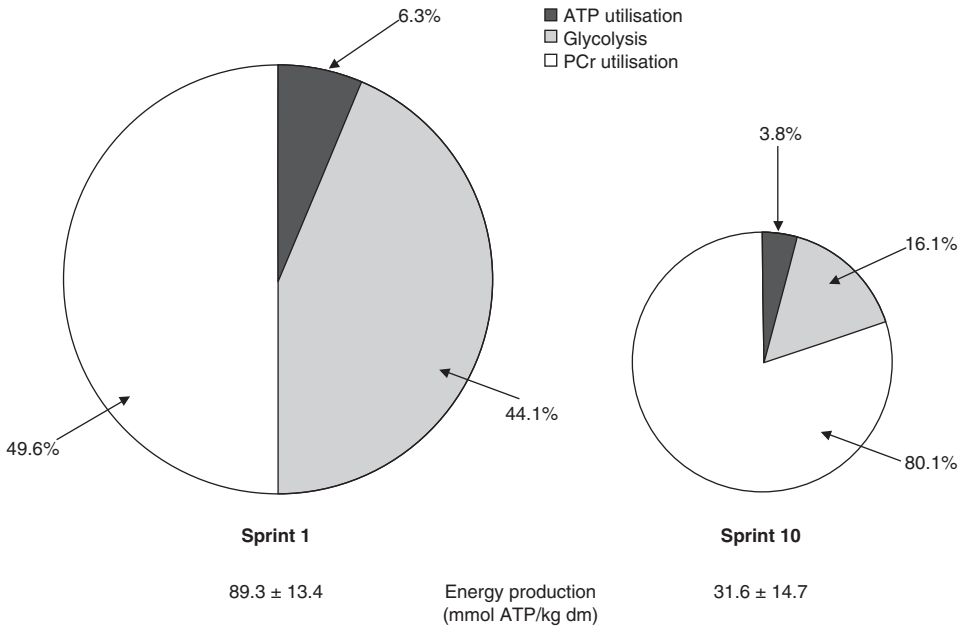

Fig. 6. Anaerobic adenosine triphosphate (ATP) production (excluding energy provision related to lactate efflux) during the first and tenth sprints of 10 × 6-second maximal sprints interspersed with 30-second recovery periods (reproduced from Gaitanos et al.,[29] with permission). **PCr** = phosphocreatine.

4.2 Aerobic Energy Provision During Multiple Sprint Work

At the onset of a bout of intense exercise there is a delay in $\dot{V}O_2$ by the working muscles (figure 7). However, if the duration of the work period is limited to a few seconds, oxygen bound to myoglobin (MbO_2) may buffer the initial oxygen demand of the exercise.[84-86]

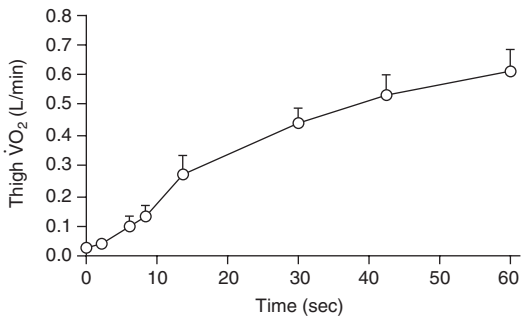

Fig. 7. Thigh oxygen uptake ($\dot{V}O_2$) during the first minute of a 3-minute bout of intense (~120% maximal $\dot{V}O_2$) knee-extensor exercise. Values are corrected for blood transit times (reproduced from Bangsbo et al.,[87] with permission).

The MbO_2 content of human skeletal muscle is approximately 2 mmol O_2/kg dm.[88,89] This store of oxygen is rapidly desaturated at the onset of exercise in response to a rapid drop in the intracellular partial pressure of oxygen.[90,91] At an intensity sufficient to elicit $\dot{V}O_{2max}$, MbO_2 is desaturated to approximately 50% of resting values within 20 seconds.[90,91] However, the sensitivity of MbO_2 desaturation to exercise intensity is an issue of some controversy.[90,91]

During recovery, MbO_2 stores are fully replenished within 20 seconds of the cessation of exercise.[91] With such a rapid rate of resaturation, it is unlikely that the availability of oxygen from myoglobin would be a limiting factor during repeated sprints. However, *in vivo* examinations of myoglobin function by means of ^1H magnetic resonance spectroscopy are a recent development and clearly more research is required to fully establish the role of myoglobin during single and repeated bouts of maximal work.

Based on the above findings, Bangsbo et al.[34] estimated the mean rate of aerobic ATP turnover

during the first 5 seconds of a 3-minute bout of intense (~120% $\dot{V}O_{2max}$) exercise to be 0.7 mmol ATP/kg dm/sec. This value compares well with the value of 1.3 mmol ATP/kg dm/sec calculated by Parolin et al.[33] during the first 6 seconds of a 30-second maximal sprint and substantiates the small (<10%) aerobic contribution to overall ATP resynthesis during a single short maximal sprint. However, as sprints are repeated, the level of aerobic ATP provision is reported to increase progressively due to elevated and possibly accelerated $\dot{V}O_2$ kinetics.[29,33,66,92,93] For instance, during recovery from a bout of high-intensity work, $\dot{V}O_2$ remains elevated for some time in order to restore the metabolic environment to resting conditions through processes such as the replenishment of MbO_2 stores, the resynthesis of PCr, the metabolism of lactate, and the removal of accumulated intracellular P_i.[94-97] If subsequent sprints are performed before $\dot{V}O_2$ has returned to resting levels, then the $\dot{V}O_2$ of successive sprints will be elevated (figure 8).

The elevation in $\dot{V}O_2$ with repeated sprints appears to be accompanied by an accelerated $\dot{V}O_2$ at the onset of each work bout (figure 9). Although the mechanisms responsible for this effect are poorly understood, corroborative research supports a pH-mediated response leading to an increased Bohr shift of the oxygen-haemoglobin dissociation curve, increased vasodilation in the working muscles, increased recruitment of motor units, and increased activity of pyruvate dehydrogenase.[34,98-101] However, the issue of accelerated $\dot{V}O_2$ kinetics is a complex and controversial one, which has at present only been examined during prolonged (≥180-second) bouts of submaximal work.[102] Moreover, with the exception of Bangsbo et al.,[34] investigations have relied on pulmonary measurements to establish muscle $\dot{V}O_2$ kinetics with a tendency to focus on 'primary' and 'slow' components of $\dot{V}O_2$, rather than the initial (0- to 20-second) 'cardiodynamic' phase. Whilst the modulation of muscle $\dot{V}O_2$ kinetics associated with limb-lung transit effects has been shown to be negligible during moderate-intensity exercise,[103] the same may not be true during maximal work. Clearly, further investigations are required to establish the kinetics of $\dot{V}O_2$ during multiple sprints.

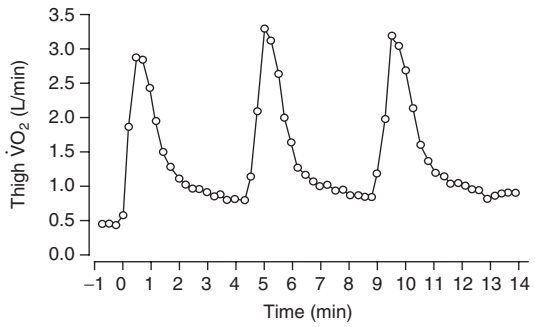

Fig. 8. Oxygen uptake ($\dot{V}O_2$) during 3 × 30-second bouts of maximal isokinetic cycling separated by 4-minute recovery periods (reproduced from Putman et al.,[66] with permission).

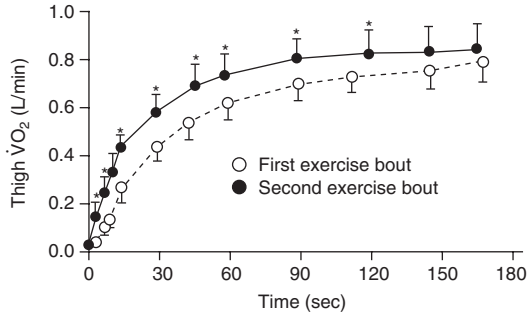

Fig. 9. Thigh oxygen uptake ($\dot{V}O_2$) during 2 × 3-minute bouts of intense (~120% maximal $\dot{V}O_2$) knee-extensor exercise separated by a 6-minute period of passive rest. Values are corrected for blood transit times (reproduced from Bangsbo et al.,[34] with permission). * indicates significantly ($p < 0.05$) different from first exercise bout.

Although the above investigations support a progressive increase in aerobic ATP production during repeated sprints, the level of aerobic ATP provision will still be considerably less than the overall energy demand.[29] As such, the major role of aerobic metabolism during multiple sprint work appears to lie in its exclusive contribution to the restoration of homeostasis during intervening recovery periods.

5. Fatigue During Multiple Sprint Work

Muscular fatigue has been the focus of numerous scientific investigations. At a recent symposium on the subject, McCully et al.[104] defined fatigue as "the development of less than the expected amount of force as a consequence of muscle activation".

During multiple sprint work, fatigue is manifested as a progressive decline in power output, the magnitude of which is largely determined by the duration of the intervening recovery periods (figure 10).[31,105,106] However, during the first few bouts of brief maximal intermittent work, fatigue can often be masked by a potentiation effect (figure 11). This effect is apparent in a number of investigations,[27,30,31,107,108] the mechanisms of which remain largely unresolved.[109-112]

5.1 Mechanisms of Fatigue

During repeated bouts of maximal work, fatigue is associated primarily with changes in the intramuscular environment.[111,113-115] Although the precise aetiology of muscular fatigue remains an issue of much conjecture, causative factors include:

- a lack of available ATP for actin-myosin coupling, Na^+/K^+ pumping, and Ca^{2+} uptake by the sarcoplasmic reticulum (SR);
- an inhibition of any of the above by various metabolic by-products;
- alterations of excitation-contraction coupling, from the action potential to Ca^{2+} release from the SR.[37]

5.2 Energy Metabolism and Fatigue

The idea that muscular fatigue may be due to a failure of the metabolic processes to resynthesise

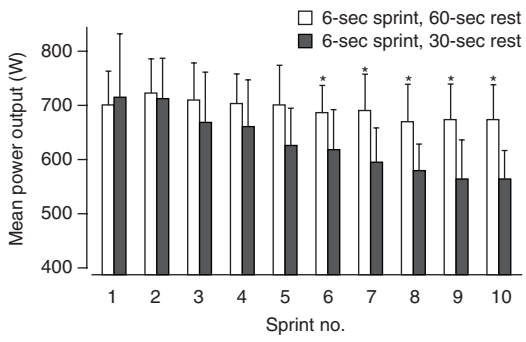

Fig. 10. Mean power output data during 10 × 6-second maximal treadmill (non-motorised) sprints interspersed with either 30- or 60-second recovery periods (reproduced from Holmyard et al.,[31] with permission). * indicates significantly ($p < 0.05$) different from 30-second recovery trial.

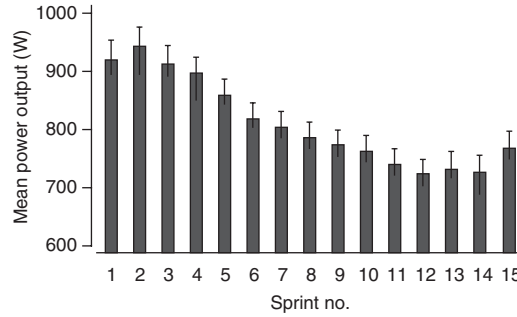

Fig. 11. Mean power output data during 15 × 5-second bouts of maximal sprint cycling interspersed with 50-second stationary rest periods (reproduced from Robinson et al.,[107] with permission).

ATP at the required rate is supported by the fact that fatigue during multiple sprint work is associated with signs of energy deficiency, i.e. increased concentrations of IMP and hypoxanthine.[42,105,116] Since energy provision during brief maximal sprints is maintained predominantly by anaerobic sources (PCr degradation and glycolysis), deficiencies in energy provision are likely to be associated with limitations in anaerobic metabolism.

5.2.1 Phosphocreatine Availability

After a bout of intense/maximal work, the recovery of force or power output follows a time-course similar to that of PCr resynthesis (figure 12).[49,59,114,117-120] As such, PCr availability is likely to be a major limiting factor in the development of fatigue during multiple sprint work. The link between PCr availability and fatigue is reinforced by the fact that a number of investigations into multiple sprint work have reported reductions in fatigue following a period of creatine supplementation (figure 13).[121-126] Although there are a number of conflicting reports,[127-131] the above findings suggest that the link between PCr availability and fatigue may be more than just coincidental.

5.2.2 Glycogen Availability

In contrast to PCr, with a normal resting intramuscular concentration of approximately 300 mmol/kg dm,[29,37] glycogen availability is unlikely to be a major limiting factor in the ability to maintain ATP provision during multiple sprint work. This is particularly so given the glycolytic inhibition

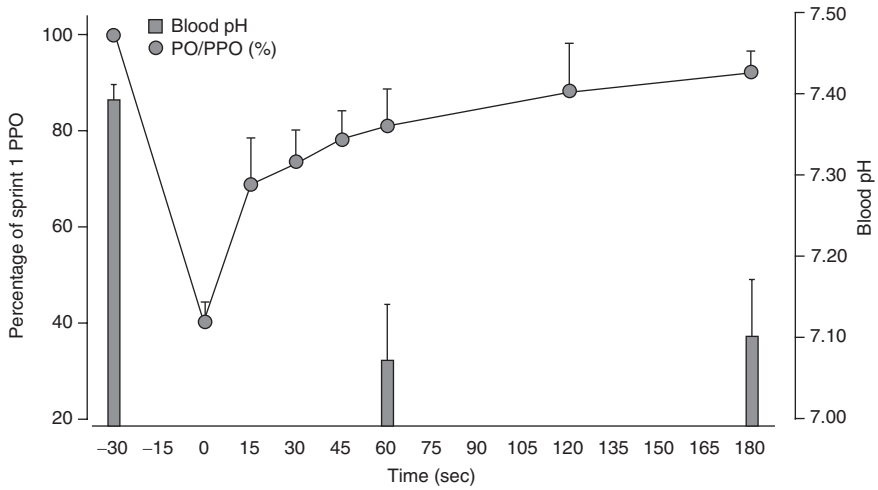

Fig. 12. Power output (expressed as a percentage of peak power output) and blood pH at rest, and during 3 minutes of stationary recovery following a 30-second maximal sprint on a non-motorised treadmill.[118] **PO** = power output; **PPO** = peak power output.

that appears to accompany this type of activity.[29,33,65,66] However, alterations in glycogen availability via dietary manipulation have been shown to have a pronounced effect on the ability to maintain high power outputs during the latter stages of repeated bouts of brief (6-second) high-intensity (>300% $\dot{V}O_{2max}$) work (figure 14).[67] Although under normal circumstances glycogen availability appears to have little influence on the ability to maintain high power outputs during short periods of brief maximal intermittent work, the drop in pH associated with anaerobic glycolysis has often been implicated as a causative agent of muscular fatigue.

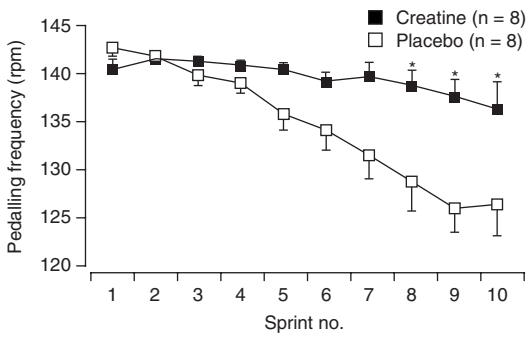

Fig. 13. Pedalling frequencies during the last 2 seconds of 10 × 6-second bouts of high-intensity cycling interspersed with 30-second stationary rest periods following a 6-day period of either creatine or placebo administration. Subjects were instructed to try to maintain a pedalling frequency of 140 revolutions per minute (rpm) throughout each sprint (reproduced from Balsom et al.,[122] with permission). * indicates significantly ($p < 0.05$) different from placebo.

5.3 Metabolite Accumulation and Fatigue

5.3.1 Acidosis

Several studies have shown strong correlations between the decline in intramuscular pH and the reduction in force or power output.[132-134] Moreover, a number of *in vitro* studies on skinned skeletal muscle fibres have reported reductions in isometric force and shortening velocity as a result of acidosis.[135-139] However, early investigations using skinned fibre preparations were conducted under relatively low temperatures (≤15°C) in an attempt to maintain intracellular mechanical stability. In contrast, recent investigations using more advanced techniques report that pH has little effect on contractile function under physiological temperatures.[140-143] This lack of association between pH and impaired contractile function is reinforced by the fact that the time-course of the recovery of force or power output following a bout of intense/maximal work is much faster than that of pH (see figure 12). Moreover, high power outputs have been obtained under acidic conditions.[49,117-119] Although fatigue during multiple sprint work cannot be ex-

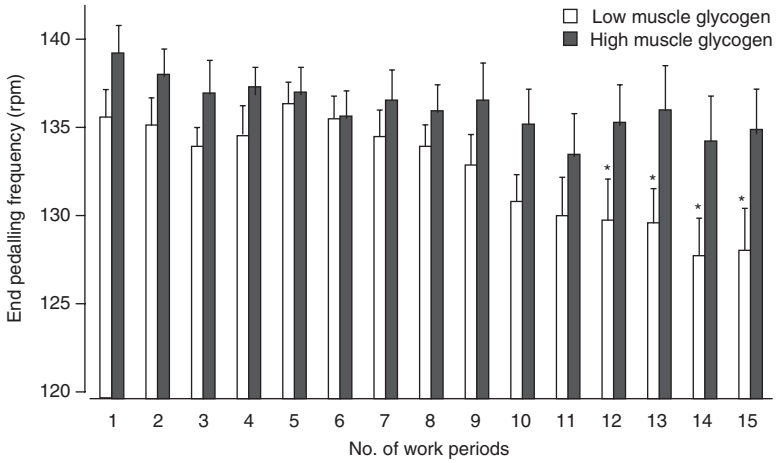

Fig. 14. The influence of glycogen availability on end pedalling frequency during the last 3 seconds of 15 × 6-second bouts of high-intensity cycling interspersed with 30-second stationary rest periods. All subjects were instructed to try to maintain a pedal frequency of 140 revolutions per minute (rpm) [>300% maximal oxygen uptake] throughout each work period (reproduced from Balsom et al.,[67] with permission). * indicates significantly ($p < 0.05$) different from high muscle glycogen trial.

plained by a direct influence of acidosis on the contractile machinery, acidosis may still impair performance through indirect mechanisms such as its potential role in glycolytic inhibition.

The uncertainty regarding the extent to which acidosis impairs multiple sprint performance is reflected in the results of investigations into the ergogenic effects of sodium bicarbonate ($NaHCO_3$) ingestion. $NaHCO_3$ has been used in a number of studies in an attempt to increase extracellular buffering capacity and thereby reduce H^+ accumulation in muscle.[144] Using 10 ×10-second sprints (50-second rest periods), Lavender and Bird[145] reported a significant reduction in fatigue following $NaHCO_3$ administration, the magnitude of which increased with successive sprints (figure 15). More recently, Bishop et al.[146] reported similar effects using 5 × 6-second sprints (24-second rest periods). In contrast, Gaitanos et al.[147] reported that $NaHCO_3$ ingestion, despite causing a shift in the acid-base balance of the blood, had no significant effect on multiple sprint (10 × 6-second sprint, 30-second rest) performance. Whilst various methodological differences may have contributed to the disparities between these results, further investigations are clearly required to fully establish the precise role, if any, of acidosis in the development of muscular fatigue.

5.3.2 Inorganic Phosphate Accumulation

Although early research focused on acidosis as the most likely cause of muscular fatigue, recent findings have led the focus of attention to switch to that of intracellular P_i accumulation.[148-152] The principle mechanism by which P_i appears to interfere with muscle function is by inhibiting Ca^{2+} release from the SR. SR Ca^{2+} release controls actin-myosin cross-bridge interactions and thereby regulates force production. The link between SR Ca^{2+} release and fatigue has been observed in a number of

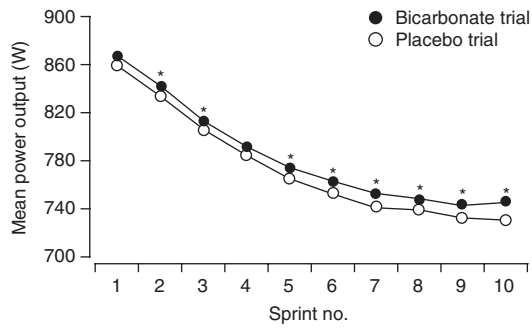

Fig. 15. The influence of sodium bicarbonate ingestion on mean power output data during 10 × 10-second bouts of maximal sprint cycling interspersed with 50-second stationary rest periods (reproduced from Lavender and Bird,[145] with permission from the BMJ Publishing Group). * indicates significantly ($p < 0.05$) different from placebo.

investigations,[153-156] potential mechanisms of which include a precipitation of calcium phosphate within the SR and an inhibition of the SR Ca^{2+} release mechanism.[148,157-161] Although a P_i-linked impairment of SR Ca^{2+} release is currently considered to be the major cause of high-intensity muscular fatigue, further research is required to establish the mechanism(s) of this response.

5.4 Summary

This section has described how performance during multiple sprint work can be influenced by many factors associated with energy metabolism and metabolite accumulation. All in all, it appears that fatigue during multiple sprint work is likely to be the result of a spectrum of events rather than a single causative factor, with metabolites such as Na^+ and K^+ also having potential roles to play in its aetiology. The final section of this article will focus on the influence of another potential performance modulator during multiple sprint work, namely oxygen availability, with particular focus on the influence of aerobic/endurance training.

6. The Influence of Oxygen Availability on Multiple Sprint Work

The influence of oxygen availability on performance during both submaximal and maximal workloads has been extensively studied using a wide range of methodologies.[162-167] In general, hypoxic conditions are associated with increased rates of fatigue, whilst hyperoxic conditions have a contrasting effect. These same effects are also evident in studies that have examined the influence of oxygen availability on multiple sprint work.[168,169] For example, under conditions of enhanced oxygen availability (achieved via erythropoietin administration), Balsom et al.[168] reported that the ability to maintain performance during 15 × 6-second treadmill sprints (~250% $\dot{V}O_{2max}$) interspersed with 24-second rest periods, was associated with a reduced accumulation of anaerobic metabolites (blood lactate and hypoxanthine). In contrast, under hypoxic conditions (hypobaric chamber), the ability to perform 10 × 6-second cycle sprints (~350% $\dot{V}O_{2max}$) interspersed with 30-second rest periods, was associated with an increased accumulation of blood lactate, a reduced $\dot{V}O_2$, and an increased rate of muscular fatigue (figure 16).[169] The authors hypothesised that oxygen availability mediated its effect on multiple sprint performance by influencing: (i) the magnitude of the aerobic contribution to ATP resynthesis during work periods; and/or (ii) the rate of PCr resynthesis during intervening rest periods.

The idea that oxygen availability may have influenced the aerobic contribution to each sprint is supported by evidence from a number of studies that oxygen availability has a significant influence on the rate of $\dot{V}O_2$ at the onset of high-intensity exercise.[170-173] Specifically, hyperoxic conditions result in a speeding of $\dot{V}O_2$ kinetics at the onset of exercise, whilst hypoxic conditions have the opposite effect. A faster on-transient $\dot{V}O_2$ response, as a result of enhanced oxygen availability, would reduce the magnitude of the oxygen deficit incurred during each sprint and thereby place less demand on anaerobic sources to maintain the required rate of ATP provision.

Although a modified aerobic contribution to ATP resynthesis during each sprint provides a possible explanation for the findings of Balsom et al.,[168,169] the results can also be reconciled by the fact that oxygen availability may have influenced the magnitude of the contribution to ATP resynthesis made by

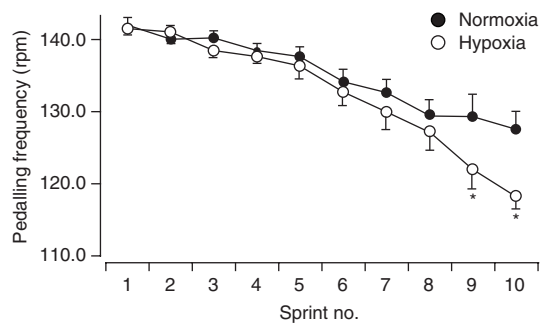

Fig. 16. Pedalling frequencies during the final second of 10 × 6-second bouts of high-intensity (~350% maximal oxygen uptake) cycling (30-second stationary rest periods) under hypoxic and normoxic conditions. Subjects were instructed to try to maintain a pedalling frequency of 140 revolutions per minute (rpm) throughout each sprint (reproduced from Balsom et al.,[169] with permission). * indicates significantly ($p < 0.05$) different from normoxic condition.

PCr. In effect, the link between oxygen availability and PCr recovery kinetics observed by Haseler et al.[52] and Idström et al.[58] (see figure 2) is likely to have influenced the magnitude of the PCr contribution to ATP turnover during each sprint. A higher PCr availability at the onset of each sprint as a result of hyperoxia would reduce the demand on anaerobic glycolysis to maintain the required rate of ATP turnover.

In addition to the hypotheses put forward by Balsom et al.,[168,169] oxygen availability may have influenced multiple sprint performance via its influence on P_i accumulation. Oxygen availability has been shown to influence the rate of P_i accumulation during exercise (figure 17) and recovery.[58,166] As such, the increased rate of fatigue observed by Balsom et al.[169] under hypoxic conditions may have been the result of a more rapid accumulation of P_i during each sprint, and a reduced rate of removal during recovery.

Although the investigations by Balsom et al.[168,169] provide a valuable insight into the influence of oxygen availability on multiple sprint performance, the intensities used were less than the maximal intensities often experienced in many sporting activities. Nevertheless, the influence of oxygen availability on multiple sprint performance has led several authors to suggest that aerobic/endurance training may convey an enhanced ability to resist fatigue during this type of work.[26,50,92,174-177] Although the theoretical basis for this assumption is compelling, corroborative scientific evidence is far from substantive.

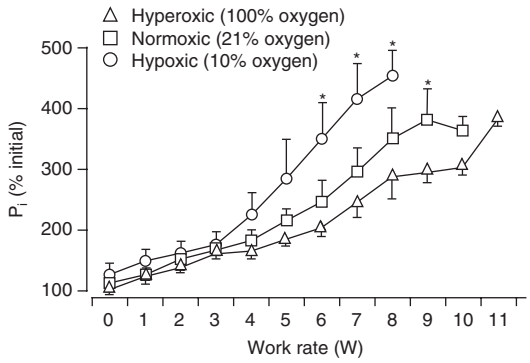

Fig. 17. The relationship between muscle inorganic phosphate (P_i) concentration and work rate for each of three different fractions of inspired oxygen during repeated plantar flexion exercise using 31P-magnetic resonance spectroscopy (reproduced from Hogan et al.,[166] with permission). * indicates significantly ($p < 0.05$) different from other oxygen availability conditions at this work rate.

6.1 Endurance Training and On-Transient Oxygen Uptake Kinetics

The influence of endurance training on $\dot{V}O_2$ kinetics at the onset of exercise has been the focus of a number of investigations.[178-183] Although findings are once again limited by a lack of experimentation using maximal workloads and the use of pulmonary gas exchange data to determine the $\dot{V}O_2$ response, research to date suggests that endurance training leads to an elevation in $\dot{V}O_{2max}$ and a possible speeding of on-transient $\dot{V}O_2$ kinetics.

6.2 Endurance Training and Phosphocreatine Recovery Kinetics

In contrast to the above, information on the influence of endurance training on PCr recovery kinetics is sparse. However, McCully and Posner[184] reported enhanced PCr recovery kinetics following 2 weeks of endurance training. Moreover, a number of investigations have reported enhanced PCr recovery kinetics in endurance-trained athletes compared with sprinters and untrained controls.[56,185-188] Despite the considerable amount of evidence supporting a link between endurance training status and PCr recovery kinetics, attempts to establish a relationship between $\dot{V}O_{2max}$ and PCr recovery kinetics show some conflicting results. For example, Cooke et al.[50] reported no significant differences in PCr resynthesis rates between individuals grouped on the basis of whether or not they possessed a high (mean $\dot{V}O_{2max}$: 64.4 ± 1.4 mL/kg/min) or a low (mean $\dot{V}O_{2max}$: 46.6 ± 1.1 mL/kg/min) $\dot{V}O_{2max}$. In contrast, Takahashi et al.[56] reported significant negative correlations between $\dot{V}O_{2max}$ and the time-constants for PCr resynthesis following light, moderate, severe, and exhausting exercise. Moreover, Bogdanis et al.[92] reported that the resynthesis of PCr was strongly correlated ($r = -0.89$; $p < 0.01$) with endurance fitness as determined from the percentage of $\dot{V}O_{2max}$ corresponding to a blood lactate

concentration of 4 mmol/L. Individual differences in PCr recovery kinetics combined with the use of relatively low subject numbers may account for many of the discrepancies between the results of these investigations.

6.3 Endurance Training and Lactate Clearance

One of the ways in which endurance training could potentially enhance multiple sprint performance is by increasing the rate of lactate clearance during intervening rest periods. However, whilst some cross-sectional studies report that endurance-trained athletes possess an enhanced blood lactate clearance capacity,[189-191] others have yielded conflicting results.[192,193] Methodological differences such as the timing of the lactate samples, and the use of monoexponential rather than biexponential curves to describe lactate recovery data may account for some of these discrepancies. Moreover, in most cases, differences in lactate clearance capacities between endurance-trained and untrained individuals have been assessed during recovery from exercise at the same relative intensity, rather than from the same level of blood lactate accumulation. Although Bassett et al.[192] attempted to address this issue by adjusting individual workloads to produce the same level of blood lactate, subtle differences in peak lactate between the groups (figure 18) supports the need for further research.

In contrast to the number of cross-sectional studies on the influence of endurance training on lactate

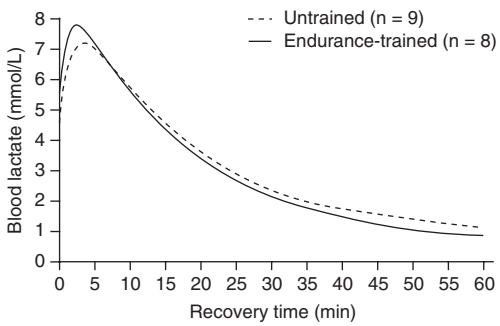

Fig. 18. Blood lactate in endurance-trained and untrained subjects following 3 minutes of continuous cycling (reproduced from Bassett et al.,[192] with permission).

recovery kinetics, longitudinal investigations on the topic are sparse yet nonetheless confusing. For example, Evans and Cureton[194] reported that 6 weeks of endurance training had no significant effect on the rate of blood lactate clearance during passive recovery from exhaustive exercise. In contrast, Fukuba et al.[195] reported that 13 weeks of endurance training improved lactate clearance capacity as determined from the 'slow' rate constant (γ_2) of the biexponential blood lactate recovery curve. Moreover, Donovan and Pagliassotti[196] reported that endurance-trained rats achieved higher rates of blood lactate clearance following exogenous lactate infusion. Although the results of Evans and Cureton[194] are potentially flawed by the use of monoexponential rather than biexponential curves to describe blood lactate recovery kinetics, the precise influence of endurance training on blood lactate clearance remains equivocal.

6.4 Endurance Training and Inorganic Phosphate Kinetics

A final way in which endurance training could potentially enhance multiple sprint performance is by speeding off-transient P_i kinetics. However, whilst P_i accumulation is currently considered to be one of the major causes of muscular fatigue, research into the influence of endurance training on P_i accumulation is sparse. In fact, the only study to date that appears to have investigated this topic is a cross-sectional study by Yoshida and Watari[188] that examined differences between endurance-trained athletes and untrained controls in their metabolic responses to repeated bouts of work. Although the authors reported no significant between-group differences in on-transient P_i kinetics, off-transient P_i kinetics were significantly faster in endurance-trained athletes than in untrained controls (figure 19).

6.5 Endurance Training and Multiple Sprint Performance

Although the results of investigations into the mechanisms by which endurance training may enhance multiple sprint performance are far from con-

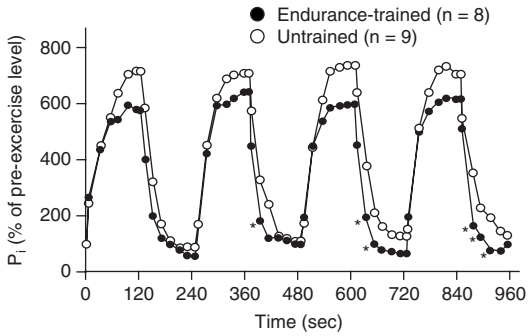

Fig. 19. Inorganic phosphate (P_i) kinetics during 4 × 2-minute bouts of repeated knee flexion exercise (20 kg/min) interspersed with 2-minute stationary rest periods in endurance-trained runners and untrained controls (reproduced from Yoshida and Watari,[188] with permission). * indicates significantly ($p < 0.05$) different from untrained controls.

clusive, there is some direct evidence to support the idea that endurance training may enhance performance during this type of work. For example, Hamilton et al.[30] reported that despite lower measures of peak power output, compared with games players (mean $\dot{V}O_{2max}$: 52.5 ± 4.9 mL/kg/min), endurance-trained athletes (mean $\dot{V}O_{2max}$: 60.8 ± 4.1 mL/kg/min) had an enhanced ability to resist fatigue during 10 × 6-second maximal sprints interspersed with 30-second rest periods. Moreover, this enhanced ability to resist fatigue was associated with higher rates of $\dot{V}O_2$ and lower peak blood lactate concentrations. More recently, Helgerud et al.[197] examined the effects of 8 weeks of aerobic interval training on soccer performance. As a result of the training, total match distance increased by 20%, number of sprints increased by 100%, involvement with the ball increased by 24%, and average work intensity increased from 82.7 ± 3.4% to 85.6 ± 3.1% of maximum heart rate. Although these investigations add further support to the idea that aerobic/endurance training may enhance the ability to perform multiple sprint work, direct evidence of the precise influence of endurance training on multiple sprint performance is lacking. Moreover, attempts to relate various multiple sprint performance indices with one of the key parameters of aerobic fitness, namely $\dot{V}O_{2max}$, reveal conflicting results.[174,176,198,199] For example, correlations between relative $\dot{V}O_{2max}$ and fatigue range from −0.16 to −0.56.[174,176,198,199] Although methodological differences may account for many of the discrepancies, the influence of protocol variation on the magnitude of those discrepancies is at present unknown.

7. Conclusions

The term 'multiple sprint work' provides a general description of the complex activity patterns experienced in many field and court sports. Research into the energetics of this type of activity supports a predominantly PCr-mediated ATP provision during work periods and an exclusively aerobic process of recovery. Whilst the ability to maintain multiple sprint performance may be attributed to a multitude of factors, PCr availability and intracellular P_i accumulation appear the most likely determinants. Moreover, the fact that both PCr resynthesis and intracellular P_i removal (via ADP phosphorylation) are oxygen-dependent processes suggests that a high level of aerobic fitness may convey an enhanced ability to resist fatigue during this type of work. However, whilst there is some evidence to suggest that endurance-trained athletes display an enhanced ability to maintain multiple sprint performance, further research is required to confirm the mechanisms of this response. Despite over 40 years of research, many issues regarding the physiological response to multiple sprint work remain unresolved. In particular, mechanisms of fatigue and the factors that regulate the same require further investigation. A greater understanding of the physiological response to multiple sprint work is likely to help athletes and coaches improve performance in many sports.

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References

1. Bangsbo J, Nørregaard L, Thorsø F. Activity profile of competition soccer. Can J Sports Sci 1991; 16 (2): 110-6

2. Brewer J, Davis J. Applied physiology of rugby league. Sports Med 1995; 20 (3): 129-35
3. Docherty D, Wenger HA, Neary P. Time-motion analysis related to the physiological demands of rugby. J Hum Mov Stud 1988; 14 (6): 269-77
4. Ekblom B. Applied physiology of soccer. Sports Med 1986; 3 (1): 50-60
5. Reilly T, Thomas V. A motion analysis of work-rate in different positional roles in professional football match-play. J Hum Mov Stud 1976; 2: 87-97
6. Withers RT, Maricic Z, Wasilewski S, et al. Match analyses of Australian professional soccer players. J Hum Mov Stud 1982; 8: 159-76
7. Reilly T, Borrie A. Physiology applied to field hockey. Sports Med 1992; 14 (1): 10-26
8. Mayhew SR, Wenger HA. Time-motion analysis of professional soccer. J Hum Mov Stud 1985; 11 (1): 49-52
9. Brodowicz GR, Schatz JC, Svoboda MD. Frequency, intensity and duration of locomotion of semi-professional soccer players. J Hum Mov Stud 1990; 18: 63-71
10. Bangsbo J. The physiology of soccer: with special reference to intense intermittent exercise. Acta Physiol Scand Suppl 1994; 619: 1-155
11. Nicholas CW. Anthropometric and physiological characteristics of rugby union football players. Sports Med 1997; 23 (6): 375-96
12. Reilly T. Energetics of high-intensity exercise (soccer) with particular reference to fatigue. J Sports Sci 1997; 15 (3): 257-63
13. Christmass MA, Richmond SE, Cable NT, et al. Exercise intensity and metabolic response in singles tennis. J Sports Sci 1998; 16: 739-47
14. Docherty D. A comparison of heart rate responses in racquet games. Br J Sports Med 1982; 16 (2): 96-100
15. Elliott B, Dawson B, Pyke F. The energetics of singles tennis. J Hum Mov Stud 1985; 11: 11-20
16. Faccini P, Dal Monte A. Physiologic demands of badminton match play. Am J Sports Med 1996; 24 (6): S64-6
17. Liddle SD, Murphy MH, Bleakley W. A comparison of the physiological demands of singles and doubles badminton: a heart rate and time-motion analysis. J Hum Mov Stud 1996; 30: 159-76
18. Majumdar P, Khanna GL, Malik V, et al. Physiological analysis to quantify training load in badminton. Br J Sports Med 1997; 31: 342-5
19. Montpetit RR. Applied physiology of squash. Sports Med 1990; 10 (1): 31-41
20. Fox EL. Sports physiology. Philadelphia (PA): Saunders, 1984
21. Maud PJ. Physiological and anthropometric parameters that describe a rugby union team. Br J Sports Med 1983; 17: 16-23
22. Seliger V, Ejam M, Pauer M, et al. Energy metabolism in tennis. Int Z Angew Physiol 1973; 31: 333-40
23. Boyle PM, Mahoney CA, Wallace WFM. The competitive demands of elite male field hockey. J Sport Med Phys Fit 1994; 34: 235-41
24. Ballor DL, Volovsek AJ. Effect of exercise to rest ratio on plasma lactate concentration at work rates above and below maximum oxygen uptake. Eur J Appl Physiol 1992; 65: 365-9
25. Bergeron MF, Maresh CM, Kraemer WJ, et al. Tennis: a physiological profile during match play. Int J Sports Med 1991; 12 (5): 474-9
26. Balsom PD. High intensity intermittent exercise: performance and metabolic responses with very high intensity short duration work periods [dissertation]. Stockholm: Karolinska Institute, 1995
27. Brooks S, Nevill ME, Meleagros L, et al. The hormonal responses to repetitive brief maximal exercise in humans. Eur J Appl Physiol 1990; 60 (2): 144-8
28. Christmass MA, Dawson B, Passeretto P, et al. A comparison of skeletal muscle oxygenation and fuel use in sustained continuous and intermittent exercise. Eur J Appl Physiol 1999; 80: 423-35
29. Gaitanos GC, Williams C, Boobis LH, et al. Human muscle metabolism during intermittent maximal exercise. J Appl Physiol 1993; 75 (2): 712-9
30. Hamilton AL, Nevill ME, Brooks S, et al. Physiological responses to maximal intermittent exercise: differences between endurance-trained runners and games players. J Sports Sci 1991; 9 (4): 371-82
31. Holmyard DJ, Cheetham ME, Lakomy HKA, et al. Effect of recovery duration on performance during multiple treadmill sprints. In: Reilly T, Lees A, Davids K, et al. editors. Science and football. London: F & N Spon, 1988: 134-42
32. Bogdanis GC, Nevill ME, Lakomy HKA, et al. Power output and muscle metabolism during and following recovery from 10 and 20 s of maximal sprint exercise in humans. Acta Physiol Scand 1998; 163: 261-72
33. Parolin ML, Chesley A, Matsos MP, et al. Regulation of skeletal muscle glycogen phosphorylase and PDH during maximal intermittent exercise. Am J Physiol 1999; 277: E890-900
34. Bangsbo J, Krustrup P, González-Alonso J, et al. ATP production and efficiency of human skeletal muscle during intense exercise: effect of previous exercise. Am J Physiol 2001; 280 (6): E956-64
35. Hultman E, Sjöholm H. Energy metabolism and contraction force of human skeletal muscle in situ during electrical stimulation. J Physiol 1983; 345: 525-32
36. Walter G, Vandenborne K, McCully KK, et al. Noninvasive measurement of phosphocreatine recovery kinetics in single human muscles. Am J Physiol 1997; 272: C525-34
37. Hultman E, Bergström M, Spriet LL, et al. Energy metabolism and fatigue. In: Taylor A, Gollnick P, Green H, et al. editors. Biochemistry of sport and exercise VII. Champaign (IL): Human Kinetics, 1990: 73-92
38. Jones NL, McCartney N, Graham T, et al. Muscle performance and metabolism in maximal isokinetic cycling at slow and fast speeds. J Appl Physiol 1985; 59 (1): 132-6
39. Gastin PB. Energy system interaction and relative contribution during maximal exercise. Sports Med 2001; 31 (10): 725-41
40. Greenhaff PL, Bodin K, Casey A, et al. Dietary creatine supplementation and fatigue during high-intensity exercise in humans. In: Maughan RJ, Shirreffs SM, editors. Biochemistry of sport and exercise IX. Champaign (IL): Human Kinetics, 1996: 219-42
41. Bangsbo J, Graham T, Johansen L, et al. Elevated muscle acidity and energy production during exhaustive exercise in humans. Am J Physiol 1992; 263: R891-9
42. Hellsten-Westing Y, Norman B, Balsom PD, et al. Decreased resting levels of adenine nucleotides in human skeletal muscle after high-intensity training. J Appl Physiol 1993; 74 (5): 2523-8
43. Astrand I, Astrand PO, Christensen EH, et al. Myohemoglobin as an oxygen-store in man. Acta Physiol Scand 1960; 48: 454-60
44. Christensen EH, Hedman R, Saltin B. Intermittent and continuous running. Acta Physiol Scand 1960; 50: 269-86

45. Margaria R, Oliva RD, Di Prampero PE, et al. Energy utilisation in intermittent exercise of supramaximal intensity. J Appl Physiol 1969; 26 (6): 752-6
46. Boobis L, Williams C, Wootton SA. Human muscle metabolism during brief maximal exercise. J Physiol 1982; 338: 21P-2P
47. McMahon S, Jenkins D. Factors affecting the rate of phosphocreatine resynthesis following intense exercise. Sports Med 2002; 32 (12): 761-84
48. Blei ML, Conley KE, Kushmerick MJ. Separate measures of ATP utilization and recovery in human skeletal muscle. J Physiol 1993; 465: 203-22
49. Bogdanis GC, Nevill ME, Boobis LH, et al. Recovery of power output and muscle metabolites following 30 s of maximal sprint cycling in man. J Physiol 1995; 482 (2): 467-80
50. Cooke SR, Petersen SR, Quinney HA. The influence of maximal aerobic power on recovery of skeletal muscle following anaerobic exercise. Eur J Appl Physiol 1997; 75 (6): 512-9
51. Harris RC, Edwards RHT, Hultman E, et al. The time course of phosphorylcreatine resynthesis during recovery of the quadriceps muscle in man. Pflugers Arch 1976; 367: 137-42
52. Haseler LJ, Hogan MC, Richardson RS. Skeletal muscle phosphocreatine recovery in exercise-trained humans is dependent on O_2 availability. J Appl Physiol 1999; 86 (6): 2013-8
53. Quistorff B, Johansen L, Sahlin K. Absence of phosphocreatine resynthesis in human calf muscle during ischaemic recovery. Biochem J 1992; 291: 681-6
54. Roussel M, Bendahan D, Mattei JP, et al. 31P magnetic resonance spectroscopy study of phosphocreatine recovery kinetics in skeletal muscle: the issue of intersubject variability. Biochim Biophys Acta 2000; 1457: 18-26
55. Sahlin K, Harris RC, Hultman E. Resynthesis of creatine phosphate in human muscle after exercise in relation to intramuscular pH and availability of oxygen. Scand J Clin Lab Invest 1979; 39: 551-8
56. Takahashi H, Inaki M, Fujimoto K, et al. Control of the rate of phosphocreatine resynthesis after exercise in trained and untrained human quadriceps muscles. Eur J Appl Physiol 1995; 71 (5): 396-404
57. Thompson CH, Kemp GJ, Sanderson AL, et al. Skeletal muscle mitochondrial function studied by kinetic analysis of postexercise phosphocreatine resynthesis. J Appl Physiol 1995; 78 (6): 2131-9
58. Idström JP, Subramanian VH, Chance B, et al. Oxygen dependence of energy metabolism in contracting and recovering rat skeletal muscle. Am J Physiol 1985; 248 (17): H40-8
59. Bergström M, Hultman E. Relaxation and force during fatigue and recovery of the human quadriceps muscle: relations to metabolite changes. Pflugers Arch 1991; 418: 153-60
60. Metzger JM, Fitts RH. Role of intracellular pH in muscle fatigue. J Appl Physiol 1987; 62 (4): 1392-7
61. Sahlin K. Metabolic factors in fatigue. Sports Med 1992; 13 (2): 99-107
62. Sahlin K, Gorski J, Edström L. Influence of ATP turnover and metabolite changes on IMP formation and glycolysis in rat skeletal muscle. Am J Physiol 1990; 259: C409-12
63. Taylor DJ, Bore PJ, Styles P, et al. Bioenergetics of intact human muscle: a 31P nuclear magnetic resonance study. Mol Biol Med 1983; 1 (1): 77-94
64. Sahlin K, Harris RC, Nylind B, et al. Lactate content and pH in muscle samples obtained after dynamic exercise. Pflugers Arch 1976; 367: 143-9
65. Bangsbo J. Regulation of muscle glycogenolysis and glycolysis during intense exercise: in vivo studies using repeated intense exercise. In: Maughan RJ, Shirreffs SM, editors. Biochemistry of exercise IX. Champaign (IL): Human Kinetics, 1996: 261-75
66. Putman CT, Jones NL, Lands LC, et al. Skeletal muscle pyruvate dehydrogenase activity during maximal exercise in humans. Am J Physiol 1995; 269: E458-68
67. Balsom PD, Gaitanos GC, Söderlund K, et al. High-intensity exercise and muscle glycogen availability in humans. Acta Physiol Scand 1999; 165: 337-45
68. Asmussen E, Klausen K, Nielsen LE, et al. Lactate production and anaerobic work capacity after prolonged exercise. Acta Physiol Scand 1974; 90: 731-42
69. Greenhaff PL, Gleeson M, Maughan RJ. The effects of a glycogen loading regimen on acid-base status and blood lactate concentration before and after a fixed period of high-intensity exercise in man. Eur J Appl Physiol 1988; 57: 254-9
70. Maughan RJ, Poole DC. The effects of a glycogen-loading regimen on the capacity to perform anaerobic exercise. Eur J Appl Physiol 1981; 46: 211-9
71. Bangsbo J, Graham T, Kiens B, et al. Elevated muscle glycogen and anaerobic energy production during exhaustive exercise in man. J Physiol 1992; 451: 205-27
72. Jacobs I. Lactate concentrations after short, maximal exercise at various glycogen levels. Acta Physiol Scand 1981; 111: 465-9
73. Ren JM, Broberg S, Sahlin K, et al. Influence of reduced glycogen level on glycogenolysis during short-term stimulation in man. Acta Physiol Scand 1990; 139: 467-74
74. Spencer MK, Katz A. Role of glycogen in control of glycolysis and IMP formation in human muscle during exercise. Am J Physiol 1991; 260: E859-64
75. Symons JD, Jacobs I. High-intensity exercise performance is not impaired by low intramuscular glycogen. Med Sci Sports Exerc 1989; 21 (5): 550-7
76. Boscá L, Aragón JJ, Sols A. Modulation of muscle phosphofructokinase at physiological concentration of enzyme. J Biol Chem 1985; 260 (4): 2100-7
77. Dobson GP, Yamamoto E, Hochachka PW. Phosphofructokinase control in muscle: nature and reversal of pH-dependent ATP inhibition. Am J Physiol 1986; 250: R71-6
78. Spriet LL, Söderlund K, Bergström M, et al. Skeletal muscle glycogenolysis, glycolysis, and pH during electrical stimulation in men. J Appl Physiol 1987; 62 (2): 616-21
79. Parmeggiani A, Bowman RH. Regulation of phosphofructokinase activity by citrate in normal and diabetic muscle. Biochem Biophys Res Commun 1963; 12 (4): 268-73
80. Passonneau JV, Lowry OH. P-fructokinase and the control of the citric acid cycle. Biochem Biophys Res Commun 1963; 13 (5): 372-9
81. Taylor WM, Halperin ML. Regulation of pyruvate dehydrogenase in muscle. J Biol Chem 1973; 248 (17): 6080-3
82. Wu TFL, Davis EJ. Regulation of glycolytic flux in an energetically controlled cell-free system: the effects of adenine nucleotide ratios, inorganic phosphate, pH, and citrate. Arch Biochem Biophys 1981; 209 (1): 85-99
83. Peters SJ, Spriet LL. Skeletal muscle phosphofructokinase activity examined under physiological conditions in vitro. J Appl Physiol 1995; 78 (5): 1853-8
84. Conley KE, Ordway GA, Richardson RS. Deciphering the mysteries of myoglobin in striated muscle. Acta Physiol Scand 2000; 168: 623-34
85. Richardson RS, Newcomer SC, Nnoyszewski EA. Skeletal muscle intracellular PO_2 assessed by myoglobin desaturation: re-

sponse to graded exercise. J Appl Physiol 2001; 91 (6): 2679-85
86. Wittenberg BA, Wittenberg JB, Caldwell PRB. Role of myoglobin in the oxygen supply to red skeletal muscle. J Biol Chem 1975; 250 (23): 9038-43
87. Bangsbo J, Krustrup P, González-Alonso J, et al. Muscle oxygen kinetics at onset of intense dynamic exercise in humans. Am J Physiol 2000; 279: R899-906
88. Akeson A, Biörck G, Simon R. On the content of myoglobin in human muscles. Acta Med Scand 1968; 183: 307-16
89. Harris RC, Hultman E, Kaijser L, et al. The effect of circulatory occlusion on isometric exercise capacity and energy metabolism of the quadriceps muscle in man. Scand J Clin Lab Invest 1975; 35 (1): 87-95
90. Molé PA, Chung Y, Tran TK, et al. Myoglobin desaturation with exercise intensity in human gastrocnemius muscle. Am J Physiol 1999; 277: R173-80
91. Richardson RS, Noyszewski EA, Kendrick KF, et al. Myoglobin O_2 desaturation during exercise. J Clin Invest 1995; 96 (4): 1916-26
92. Bogdanis GC, Nevill ME, Boobis LH, et al. Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. J Appl Physiol 1996; 80 (3): 876-84
93. Trump ME, Heigenhauser GJF, Putman CT, et al. Importance of muscle phosphocreatine during intermittent maximal cycling. J Appl Physiol 1996; 80 (5): 1574-80
94. Bahr R, Grønnerød O, Sejersted OM. Effect of supramaximal exercise on excess postexercise O_2 consumption. Med Sci Sports Exerc 1992; 24 (1): 66-71
95. Bangsbo J, Hellsten Y. Muscle blood flow and oxygen uptake in recovery from exercise. Acta Physiol Scand 1998; 162: 305-12
96. Børsheim E, Knardahl S, Høstmark AT, et al. Adrenergic control of post-exercise metabolism. Acta Physiol Scand 1998; 162: 313-23
97. Gaesser GA, Brooks GA. Metabolic bases of excess post-exercise oxygen consumption: a review. Med Sci Sports Exerc 1984; 16 (1): 29-43
98. Bohnert B, Ward SA, Whipp BJ. Effects of prior arm exercise on pulmonary gas exchange kinetics during high-intensity leg exercise in humans. Exp Physiol 1998; 83: 557-70
99. Gausche MA, Harmon T, Lamarra N, et al. Pulmonary O_2 uptake kinetics in humans are speeded by a bout of prior exercise above, but not below, the lactate threshold [abstract]. J Physiol 1989; 417: 138P
100. Gerbino A, Ward SA, Whipp BJ. Effects of prior exercise on pulmonary gas-exchange kinetics during high-intensity exercise in humans. J Appl Physiol 1996; 80 (1): 99-107
101. Rossiter HB, Ward SA, Kowalchuk JM, et al. Effects of prior exercise on oxygen uptake and phosphocreatine kinetics during high-intensity knee-extension exercise in humans. J Physiol 2001; 537 (1): 291-303
102. Jones AM, Koppo K, Burnley M. Effects of prior exercise on metabolic and gas exchange responses to exercise. Sports Med 2003; 33 (13): 949-71
103. Grassi B, Poole DC, Richardson RS, et al. Muscle O_2 uptake kinetics in humans: implications for metabolic control. J Appl Physiol 1996; 80 (3): 988-98
104. McCully KK, Authier B, Olive J, et al. Muscle fatigue: the role of metabolism. Can J Appl Physiol 2002; 27 (1): 70-82
105. Balsom PD, Seger JY, Sjödin B, et al. Maximal-intensity intermittent exercise: effect of recovery duration. Int J Sports Med 1992; 13 (7): 528-33
106. Wootton S, Williams C. The influence of recovery duration on repeated maximal sprints. In: Knuttgen HG, Vogel JA, Poortmans J, editors. Biochemistry of exercise, international series in sports science, vol. XIII. Champaign (IL): Human Kinetics, 1983: 269-73
107. Robinson JM, Stone MH, Johnson RL, et al. Effects of different weight training exercise/rest intervals on strength, power and high intensity exercise endurance. J Strength Cond Res 1995; 9 (4): 216-21
108. Stone MH, Sanborn K, Smith LL, et al. Effects of in-season (5 weeks) creatine and pyruvate supplementation on anaerobic performance and body composition in American football players. Int J Sport Nutr 1999; 9 (2): 146-65
109. Abbate F, Sargeant AJ, Verdijk PW, et al. Effects of high-frequency initial pulses and posttetanic potentiation on power output of skeletal muscle. J Appl Physiol 2000; 88 (1): 35-40
110. Güllich A, Schmidtbleicher D. MVC-induced short-term potentiation of explosive force. N Stud Athletics 1996; 11 (4): 67-81
111. MacIntosh BR, Rassier DE. What is fatigue? Can J Appl Physiol 2002; 27 (1): 42-55
112. Smith JC, Fry AC, Weiss LW, et al. The effects of high-intensity exercise on a 10-second sprint cycle test. J Strength Cond Res 2001; 15 (3): 344-8
113. Bigland-Ritchie B, Woods J. Changes in muscle contractile properties and neural control during human muscular fatigue. Muscle Nerve 1984; 7 (9): 691-9
114. Cherry PW, Lakomy HKA, Boobis LH, et al. Rapid recovery of power output in females. Acta Physiol Scand 1998; 164: 79-87
115. Duchateau J, Hainaut K. Electrical and mechanical failures during sustained and intermittent contractions in humans. J Appl Physiol 1985; 58 (3): 942-7
116. Balsom PD, Seger JY, Sjödin B, et al. Physiological responses to maximal intensity intermittent exercise. Eur J Appl Physiol 1992; 65: 144-9
117. Hitchcock HC. Recovery of short-term power after dynamic exercise. J Appl Physiol 1989; 67 (2): 677-81
118. Holmyard DJ, Nevill ME, Lakomy HKA, et al. Recovery of power output after maximal treadmill sprinting [abstract]. J Sports Sci 1994; 12 (2): 140
119. Sahlin K, Ren JM. Relationship of contraction capacity to metabolic changes during recovery from a fatiguing contraction. J Appl Physiol 1989; 67 (2): 648-54
120. Sargeant AJ, Dolan P. Effect of prior exercise on maximal short-term power output in humans. J Appl Physiol 1987; 63 (4): 1475-80
121. Aaserud R, Gramvik P, Olsen SR, et al. Creatine supplementation delays onset of fatigue during repeated bouts of sprint running. Scand J Med Sci Sports 1998; 8 (5): 247-51
122. Balsom PD, Ekblom B, Söderlund K, et al. Creatine supplementation and dynamic high-intensity intermittent exercise. Scand J Med Sci Sports 1993; 3: 143-9
123. Bogdanis GC, Nevill ME, Lakomy HKA, et al. The effects of oral creatine supplementation on power output during repeated treadmill sprinting. J Sports Sci 1996; 14: 65-6
124. Jones AM, Atter T, Georg KP. Oral creatine supplementation improves multiple sprint performance in elite ice-hockey players. J Sport Med Phys Fitness 1999; 39 (3): 189-96
125. Mujika I, Padilla S, Ibanéz J, et al. Creatine supplementation and sprint performance in soccer players. Med Sci Sports Exerc 2000; 32 (2): 518-25
126. Yquel RJ, Arsac LM, Thiaudière E, et al. Effect of creatine supplementation on phosphocreatine resynthesis, inorganic

phosphate accumulation and pH during intermittent maximal exercise. J Sports Sci 2002; 20 (5): 427-37
127. Barnett C, Hinds M, Jenkins DG. Effects of oral creatine supplementation on multiple sprint cycle performance. Aust J Sci Med Sport 1996; 28 (1): 35-9
128. Dawson B, Cutler M, Moody A, et al. Effects of oral creatine loading on single and repeated maximal short sprints. Aust J Sci Med Sport 1995; 27 (3): 56-61
129. Delecluse C, Diels R, Goris M. Effect of creatine supplementation on intermittent sprint running performance in highly trained athletes. J Strength Cond Res 2003; 17 (3): 446-54
130. Leenders NM, Lamb DR, Nelson TE. Creatine supplementation and swimming performance. Int J Sport Nutr 1999; 9 (3): 251-62
131. McKenna MJ, Morton J, Selig SE, et al. Creatine supplementation increases muscle total creatine but not maximal intermittent exercise performance. J Appl Physiol 1999; 87 (6): 2244-52
132. Cady EB, Jones DA, Lynn J, et al. Changes in force and intracellular metabolites during fatigue of human skeletal muscle. J Physiol 1989; 418: 311-25
133. DeGroot M, Massie BM, Boska M, et al. Dissociation of [H+] from fatigue in human muscle detected by high time resolution 31P-NMR. Muscle Nerve 1993; 16 (1): 91-8
134. Miller RG, Boska MD, Moussavi RS, et al. 31P nuclear magnetic resonance studies of high energy phosphates and pH in human muscle fatigue: comparison of aerobic and anaerobic exercise. J Clin Invest 1988; 81 (4): 1190-6
135. Chase PB, Kushmerick MJ. Effects of pH on contraction of rabbit fast and slow skeletal muscle fibers. Biophys J 1988; 53 (6): 935-46
136. Cooke R, Franks K, Luciani GB, et al. The inhibition of rabbit skeletal muscle contraction by hydrogen ions and phosphate. J Physiol 1988; 395: 77-97
137. Godt RE, Nosek TM. Changes of intracellular milieu with fatigue or hypoxia depress contraction of skinned rabbit skeletal and cardiac muscle. J Physiol 1989; 412: 155-80
138. Kentish JC, Palmer S. Effect of pH on force and stiffness in skinned muscles isolated from rat and guinea-pig ventricle and from rabbit psoas muscle [abstract]. J Physiol 1989; 410: 67P
139. Metzger JM, Moss RL. Greater hydrogen ion-induced depression of tension and velocity in skinned single fibres of rat fast than slow muscles. J Physiol 1987; 393: 727-42
140. Bruton JD, Lannergren J, Westerblad H. Effects of CO_2-induced acidification on the fatigue resistance of single mouse muscle fibers at 28 degrees C. J Appl Physiol 1998; 85 (2): 478-83
141. Pate E, Bhimani M, Franks-Skiba K, et al. Reduced effect of pH on skinned rabbit psoas muscle mechanics at high temperatures: implications for fatigue. J Physiol 1995; 486 (3): 689-94
142. Westerblad H, Bruton JD, Lännergren J. The effect of intracellular pH on contractile function of intact, single fibres of mouse muscle declines with increasing temperature. J Physiol 1997; 500 (1): 193-204
143. Wiseman RW, Beck TW, Chase PB. Effect of intracellular pH on force development depends on temperature in intact skeletal muscle from mouse. Am J Physiol 1996; 271 (3): C878-86
144. Linderman JK, Gosselink KL. The effects of sodium bicarbonate ingestion on exercise performance. Sports Med 1994; 18 (2): 75-80
145. Lavender G, Bird SR. Effect of sodium bicarbonate ingestion upon repeated sprints. Br J Sports Med 1989; 23 (1): 41-5
146. Bishop D, Edge J, Davis C, et al. Induced metabolic alkalosis affects muscle metabolism and repeated-sprint ability. Med Sci Sports Exerc 2004; 36 (5): 807-13
147. Gaitanos GC, Nevill ME, Brooks S, et al. Repeated bouts of sprint running after induced alkalosis. J Sports Sci 1991; 9 (4): 355-70
148. Allen DG, Kabbara AA, Westerblad H. Muscle fatigue: the role of intracellular calcium stores. Can J Appl Physiol 2002; 27 (1): 83-96
149. Dahlstedt AJ, Katz A, Wieringa B, et al. Is creatine kinase responsible for fatigue? Studies of isolated skeletal muscle deficient in creatine kinase. FASEB J 2000; 14 (7): 982-90
150. Dahlstedt AJ, Westerblad H. Inhibition of creatine kinase reduces the rate of fatigue-induced decrease in tetanic Ca^{2+} in mouse skeletal muscle. J Physiol 2001; 533 (3): 639-49
151. Fryer MW, Owen VJ, Lamb GD, et al. Effects of creatine phosphate and Pi on Ca^{2+} movements and tension development in rat skinned skeletal muscle fibres. J Physiol 1995; 482 (1): 123-40
152. Kabbara AA, Allen DG. The role of calcium stores in fatigue of isolated single muscle fibres from the cane toad. J Physiol 1999; 519 (1): 169-76
153. Allen DG, Lee JA, Westerblad H. Intracellular calcium and tension during fatigue in isolated single muscle fibres from Xenopus laevis. J Physiol 1989; 415: 433-58
154. Baker AJ, Kostov KG, Miller RG, et al. Slow force recovery after long duration exercise: metabolic and activation factors in muscle fatigue. J Appl Physiol 1993; 74: 2294-300
155. Gyorke S. Effects of repeated tetanic stimulation on excitation-contraction coupling in cut muscle fibres of the frog. J Physiol 1993; 464: 699-710
156. Westerblad H, Lee JA, Lamb AG, et al. Spatial gradients of intracellular calcium in skeletal muscle during fatigue. Pflugers Arch 1990; 415 (6): 734-40
157. Duke AM, Steele DS. Mechanisms of reduced SR Ca^{2+} release induced by inorganic phosphate in rat skeletal muscle fibers. Am J Physiol 2001; 281: C418-29
158. McLester Jr JR. Muscle contraction and fatigue: the role of adenosine 5'-diphosphate and inorganic phosphate. Sports Med 1997; 23 (5): 287-305
159. Posterino GS, Dutka TL, Lamb GD. L(+)-lactate does not affect twitch and tetanic responses in mechanically skinned mammalian muscle fibres. Pflugers Arch 2001; 442 (2): 197-203
160. Stackhouse SK, Reisman DS, Binder-Macleod SA. Challenging the role of pH in skeletal muscle fatigue. Phys Ther 2001; 81 (12): 1897-903
161. Westerblad H, Allen DG, Lännergren J. Muscle fatigue: lactic acid or inorganic phosphate the major cause? News Physiol Sci 2002; 17: 17-21
162. Cymerman A, Reeves JT, Sutton JR, et al. Operation Everest II: maximal oxygen uptake at extreme altitude. J Appl Physiol 1989; 66 (5): 2446-53
163. Eiken O, Tesch PA. Effects of hyperoxia and hypoxia on dynamic and sustained static performance of the human quadriceps muscle. Acta Physiol Scand 1984; 122 (4): 629-33
164. Fulco CS, Lewis SF, Frykman PN, et al. Muscle fatigue and exhaustion during dynamic leg exercise in normoxia and hypobaric hypoxia. J Appl Physiol 1996; 81 (5): 1891-900
165. Hogan MC, Kohin S, Stary CM, et al. Rapid force recovery in contracting skeletal muscle after brief ischemia is dependent on O_2 availability. J Appl Physiol 1999; 87 (6): 2225-9
166. Hogan MC, Richardson RS, Haseler LJ. Human muscle performance and PCr hydrolysis with varied inspired oxygen

fractions: a 31P-MRS study. J Appl Physiol 1999; 86 (4): 1367-73
167. Peltonen JE, Rantamaki J, Niittymaki SP, et al. Effects of oxygen fraction in inspired air on rowing performance. Med Sci Sports Exerc 1995; 27 (4): 573-9
168. Balsom PD, Ekblom B, Sjödin B. Enhanced oxygen availability during high intensity intermittent exercise decreases anaerobic metabolite concentrations in blood. Acta Physiol Scand 1994; 150 (4): 455-6
169. Balsom PD, Gaitanos GC, Ekblom B, et al. Reduced oxygen availability during high intensity intermittent exercise impairs performance. Acta Physiol Scand 1994; 152 (3): 279-85
170. MacDonald M, Pedersen PK, Hughson RL. Acceleration of $\dot{V}O_2$ kinetics in heavy submaximal exercise by hyperoxia and prior high-intensity exercise. J Appl Physiol 1997; 83 (4): 1318-25
171. Hughson RL, Kowalchuk JM. Kinetics of oxygen uptake for submaximal exercise in hyperoxia, normoxia, and hypoxia. Can J Appl Physiol 1995; 20 (2): 198-210
172. Linnarsson D, Karlsson J, Fagraeus L. Muscle metabolites and oxygen deficit with exercise in hypoxia and hyperoxia. J Appl Physiol 1974; 36 (4): 399-402
173. Pedersen PK. Oxygen uptake kinetics and lactate accumulation in heavy submaximal exercise with normal and high inspired oxygen fractions. In: Knuttgen HG, Vogel JA, Poortmans J, editors. Biochemistry of exercise, international series in sports science, vol. XIII. Champaign (IL): Human Kinetics, 1983: 415-20
174. Aziz AR, Chia M, Teh KC. The relationship between maximal oxygen uptake and repeated sprint performance indices in field hockey and soccer players. J Sport Med Phys Fitness 2000; 40: 195-200
175. Bell GJ, Snydmiller GD, Davies DS, et al. Relationship between aerobic fitness and metabolic recovery from intermittent exercise in endurance athletes. Can J Appl Physiol 1997; 22 (1): 78-85
176. Dawson B, Fitzsimons M, Ward D. The relationship of repeated sprint ability to aerobic power and performance measures of an aerobic work capacity and power. Aust J Sci Med Sport 1993; 25 (4): 88-93
177. Tomlin DL, Wenger HA. The relationship between aerobic fitness and recovery from high intensity intermittent exercise. Sports Med 2001; 31 (1): 1-11
178. Carter H, Jones AM, Barstow TJ, et al. Effect of endurance training on oxygen uptake kinetics during treadmill running. J Appl Physiol 2000; 89 (5): 1744-52
179. Chilibeck PD, Paterson DH, Petrella RJ, et al. The influence of age and cardiorespiratory fitness on kinetics of oxygen uptake. Can J Appl Physiol 1996; 21 (3): 185-96
180. Demarle AP, Slawinski JJ, Laffite LP, et al. Decrease of O_2 deficit is a potential factor in increased time to exhaustion after specific endurance training. J Appl Physiol 2001; 90 (3): 947-53
181. Norris SR, Petersen SR. Effects of endurance training on transient oxygen uptake responses in cyclists. J Sports Sci 1998; 16 (8): 733-8
182. Phillips SM, Green HJ, MacDonald MJ, et al. Progressive effect of endurance training on $\dot{V}O_2$ kinetics at the onset of submaximal exercise. J Appl Physiol 1995; 79 (6): 1914-20
183. Yoshida T, Udo M, Ohmori T, et al. Day-to-day changes in oxygen uptake kinetics at the onset of exercise during strenuous endurance training. Eur J Appl Physiol 1992; 64 (1): 78-83
184. McCully KK, Posner JD. Measuring exercise-induced adaptations and injury with magnetic resonance spectroscopy. Int J Sports Med 1992; 13: S147-9
185. Laurent D, Reutenauer H, Payen JF, et al. Muscle bioenergetics in skiers: studies using NMR spectroscopy. Int J Sports Med 1992; 13 (1): S150-2
186. McCully KK, Boden BP, Tuchler M, et al. Wrist flexor muscles of elite rowers measured with magnetic resonance spectroscopy. J Appl Physiol 1989; 67 (3): 926-32
187. McCully KK, Vandenborne K, De Meirleir K, et al. Muscle metabolism in track athletes, using 31P magnetic resonance spectroscopy. Can J Physiol Pharmacol 1992; 70 (10): 1353-9
188. Yoshida T, Watari H. Metabolic consequences of repeated exercise in long distance runners. Eur J Appl Physiol 1993; 67 (3): 261-5
189. Freund H, Lonsdorfer J, Oyono-Enguelle S, et al. Lactate exchange and removal abilities in sickle cell patients and in untrained and trained healthy humans. J Appl Physiol 1992; 73 (6): 2580-7
190. Oyono-Enguelle S, Marbach J, Heitz A, et al. Lactate removal ability and graded exercise in humans. J Appl Physiol 1990; 68 (3): 905-11
191. Taoutaou Z, Granier P, Mercier B, et al. Lactate kinetics during passive and partially active recovery in endurance and sprint athletes. Eur J Appl Physiol 1996; 73 (5): 465-70
192. Bassett Jr DR, Merrill PW, Nagle FJ, et al. Rate of decline in blood lactate after cycling exercise in endurance-trained and untrained subjects. J Appl Physiol 1991; 70 (4): 1816-20
193. Oosthuyse T, Carter RN. Plasma lactate decline during passive recovery from high-intensity exercise. Med Sci Sports Exerc 1999; 31 (5): 670-4
194. Evans BW, Cureton KJ. Effect of physical conditioning on blood lactate disappearance after supramaximal exercise. Br J Sports Med 1983; 17 (1): 40-5
195. Fukuba Y, Walsh ML, Morton RH, et al. Effect of endurance training on blood lactate clearance after maximal exercise. J Sports Sci 1999; 17 (3): 239-48
196. Donovan CM, Pagliassotti MJ. Enhanced efficiency of lactate removal after endurance training. J Appl Physiol 1990; 68 (3): 1053-8
197. Helgerud J, Engen LC, Wisloff U, et al. Aerobic endurance training improves soccer performance. Med Sci Sports Exerc 2001; 33 (11): 1925-31
198. Bishop D, Lawrence S, Spencer M. Predictors of repeated-sprint ability in elite female hockey players. J Sci Med Sport 2003; 6 (2): 199-209
199. Wadley G, Le Rossignol P. The relationship between repeated sprint ability and the aerobic and anaerobic energy systems. J Sci Med Sport 1998; 1 (2): 100-10

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