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 (˙VO2) 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 (Pi). If recovery periods are relatively short,
˙VO2 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, perform-
ance 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 Pi
as the most likely causes. Moreover, the fact that both PCr resynthesis and the
removal of accumulated intracellular Pi 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 intermit-
tent in nature, fluctuating randomly from brief peri-
ods 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 influ-
enced by player position.\textsuperscript{[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.\textsuperscript{[1,2,7]} The percentages of game-
time spent in various forms of locomotion are diffi-
cult to quantify due to methodological differences
between studies. However, the mean duration of
high-intensity efforts is reported to be approximate-
ly 4–7 seconds,\textsuperscript{[1,3,6,8]} of which approximately 2
seconds is attributed to all-out sprinting.\textsuperscript{[1,3,9]} Al-
though the ratio of high- to low-intensity activities
ranges from 1 : 6 to 1 : 14,\textsuperscript{[2,6,10-12]} values are
clded 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,\textsuperscript{[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 mul-
tiple sprint sports indicates that these events place
considerable demands on both aerobic and anaerob-
ic pathways, although the relative contribution from
Table I. Typical work to rest ratios experienced in racquet sports

<table>
<thead>
<tr>
<th>Sport</th>
<th>Playing level</th>
<th>Mean rally time (sec)</th>
<th>Work : rest ratio</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Squash</td>
<td>Range of abilities</td>
<td>4.4–8.8</td>
<td>1 : 1</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6.9–16.6</td>
<td>1 : 1</td>
<td>19</td>
</tr>
<tr>
<td>Badminton</td>
<td>Range of abilities</td>
<td>4.2–4.9</td>
<td>1 : 2</td>
<td>14</td>
</tr>
<tr>
<td>National level</td>
<td>7.4</td>
<td>1 : 2</td>
<td></td>
<td>16</td>
</tr>
<tr>
<td>Tennis</td>
<td>State level</td>
<td>10.2</td>
<td>1 : 1.7*</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>Range of abilities</td>
<td>4.0–4.3</td>
<td>1 : 5</td>
<td>14</td>
</tr>
<tr>
<td>College level</td>
<td>10.0</td>
<td>1 : 1.8*</td>
<td></td>
<td>15</td>
</tr>
</tbody>
</table>

* Does not include time spent changing ends.

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).

\[
\text{ATP} \xrightarrow{\text{ATPase}} \text{ADP} + P_i + \text{energy}
\]  
(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. 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).

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

(Eq. 2)

Intramuscular PCr stores total approximately 80 mmol/kg dm. 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, 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).

$$\text{Glycogen} + 3 \text{ADP} + 3 \text{Pi} \rightarrow 3 \text{ATP} + 2 \text{lactate}^- + 2 \text{H}^+$$

(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 after approximately 5 seconds.

3.4 Aerobic Metabolism

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

$$\text{C}_6\text{H}_{12}\text{O}_6 (\text{glucose}) + 6\text{O}_2 + 38 \text{ADP} + 38 \text{Pi} \rightarrow 6 \text{CO}_2 + 6 \text{H}_2\text{O} + 38 \text{ATP}$$

(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 $\dot{V}_\text{O}_2$ 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).

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).

$$\text{ADP} + \text{ADP} \rightleftharpoons \text{ATP} + \text{AMP}$$

(Eq. 5)

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

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 nmol 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. 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. 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. 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 31P magnetic resonance spectroscopy) and in vitro (using muscle biopsies) in several investigations. 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. Moreover, PCr recovery kinetics have been shown to be sensitive to manipulations of oxygen availability (figure 2). 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.](image-url)
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. During recovery, glycolysis is reportedly switched off and the return of pH to resting levels follows a monoexponential pattern of resynthesis (figure 4) with a half-time of approximately 9 minutes.

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 PCr concentration (% of resting value) at one minute following 60 seconds of repeated maximal work being approximately 50% of resting levels. 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.

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 31P 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. 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. 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. During recovery, glycolysis is reportedly switched off and the return of pH to resting levels follows a monoexponential pattern of resynthesis (figure 4) with a half-time of approximately 9 minutes.

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. For example, in the study by Gaitanos et al., 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. One suggestion is that glycolysis is impaired by the progressive depletion of muscle glycogen stores that accompanies this type of work. Several studies have reported altered glycolytic rates following glycogen manipulation. In contrast, other investigations report contradictory findings. Another suggestion is that glycolysis is impaired by the aforementioned progressive drop in pH. An accumulation of H+ is known to inhibit...
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; PEP = phosphoenolpyruvate; PFK = phosphofructokinase; Pi = 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 ≥ 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.
4.2 Aerobic Energy Provision During Multiple Sprint Work

At the onset of a bout of intense exercise there is a delay in VO₂ by the working muscles (figure 7). However, if the duration of the work period is limited to a few seconds, oxygen bound to myoglobin (MbO₂) may buffer the initial oxygen demand of the exercise.\[^{84-86}\]

The MbO₂ content of human skeletal muscle is approximately 2 mmol O₂/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 VO₂max, MbO₂ is desaturated to approximately 50% of resting values within 20 seconds.\[^{90,91}\] However, the sensitivity of MbO₂ desaturation to exercise intensity is an issue of some controversy.\[^{90,91}\]

During recovery, MbO₂ 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 \(^1\)H 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% VO2max) 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 MbO2 stores, the resynthesis of PCR, the metabolism of lactate, and the removal of accumulated intracellular Pi.[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.

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). 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, the mechanisms of which remain largely unresolved.

5.1 Mechanisms of Fatigue

During repeated bouts of maximal work, fatigue is associated primarily with changes in the intramuscular environment. 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 Ca2+ 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 Ca2+ release from the SR.

5.2 Energy Metabolism and Fatigue

The idea that muscular fatigue may be due to a failure of the metabolic processes to resynthesise 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. 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). 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). Although there are a number of conflicting reports, 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, 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
Physiological Response to Multiple Sprint Work

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. PO = power output; PPO = peak power output.

that appears to accompany this type of activity. 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% VO2max) work (figure 14). 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.

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. 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. 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. 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. Although fatigue during multiple sprint work cannot be ex-
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 (NaHCO₃) ingestion. NaHCO₃ 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 Bird¹⁴⁴ reported a significant reduction in fatigue following NaHCO₃ 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 NaHCO₃ 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.

While 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 Ca²⁺ release from the SR. SR Ca²⁺ release controls actin-myosin cross-bridge interactions and thereby regulates force production. The link between SR Ca²⁺ release and fatigue has been observed in a number of

![Graph showing 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,¹⁴⁶ with permission from the BMJ Publishing Group). * indicates significantly (p < 0.05) different from placebo.](image_url)
investigations, potential mechanisms of which include a precipitation of calcium phosphate within the SR and an inhibition of the SR Ca$^{2+}$ release mechanism. 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. 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. For example, under conditions of enhanced oxygen availability (achieved via erythropoietin administration), Balsom et al. reported that the ability to maintain performance during 15 × 6-second treadmill sprints (~250% VO$_{2\text{max}}$) 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% VO$_{2\text{max}}$) interspersed with 30-second rest periods, was associated with an increased accumulation of blood lactate, a reduced VO$_2$, and an increased rate of muscular fatigue (figure 16). 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 VO$_2$ at the onset of high-intensity exercise. Specifically, hyperoxic conditions result in a speeding of VO$_2$ kinetics at the onset of exercise, whilst hypoxic conditions have the opposite effect. A faster on-transient VO$_2$ 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., 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...
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 Pi accumulation. Oxygen availability has been shown to influence the rate of Pi accumulation during exercise.[158,166] 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 Pi 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.

6.1 Endurance Training and On-Transient Oxygen Uptake Kinetics

The influence of endurance training on VO2 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 VO2 response, research to date suggests that endurance training leads to an elevation in VO2max and a possible speeding of on-transient VO2 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 VO2max 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 VO2max: 64.4 ± 1.4 mL/kg/min) or a low (mean VO2max: 46.6 ± 1.1 mL/kg/min) VO2max. In contrast, Takahashi et al.[56] reported significant negative correlations between VO2max 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 VO2max corresponding to a blood lactate.

Fig. 17. The relationship between muscle inorganic phosphate (Pi) 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.[169] with permission). * indicates significantly (p < 0.05) different from other oxygen availability conditions at this work rate.
Physiological Response to Multiple Sprint Work

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, others have yielded conflicting results. 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. 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 recovery kinetics, longitudinal investigations on the topic are sparse yet nonetheless confusing. For example, Evans and Cureton 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. reported that 13 weeks of endurance training improved lactate clearance capacity as determined from the ‘slow’ rate constant of the biexponential blood lactate recovery curve. Moreover, Donovan and Pagliassotti reported that endurance-trained rats achieved higher rates of blood lactate clearance following exogenous lactate infusion. Although the results of Evans and Cureton are potentially flawed by the use of monoexponential rather than biexponential curves to describe 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 Pi kinetics. However, whilst Pi accumulation is currently considered to be one of the major causes of muscular fatigue, research into the influence of endurance training on Pi 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 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 Pi kinetics, off-transient Pi 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-
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 Pi accumulation appear the most likely determinants. Moreover, the fact that both PCr resynthesis and intracellular Pi 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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