Fatigue during intermittent-sprint exercise

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SUMMARY

1. There is a reversible decline in force production by muscles when they are contracting at or near their maximum capacity. The task-dependent nature of fatigue means that the mechanisms of fatigue may differ between different types of contractions. This paper examines how fatigue manifests during whole-body, intermittent-sprint exercise and discusses the potential muscular and neural mechanisms that underpin this fatigue.

2. Fatigue is defined as a reversible, exercise-induced reduction in maximal power output (e.g., during cycling exercise) or speed (e.g., during running exercise), even though the task can be continued.

3. The small changes in surface electromyogram (EMG), along with a lack of change in voluntary muscle activation (estimated from both percutaneous motor nerve stimulations and trans-cranial magnetic stimulation), indicate that there is little change in neural drive to the muscles following intermittent-sprint exercise. This, along with the observation that the decrease in EMG is much less than that which would be predicted from the decrease in power output, suggests that peripheral mechanisms are the predominant cause of fatigue during intermittent-sprint exercise.

4. At the muscle level, limitations in energy supply, including phosphocreatine hydrolysis and the degree of reliance on anaerobic glycolysis and oxidative metabolism, and the intramuscular accumulation of metabolic by-products, such as hydrogen ions, emerge as key factors responsible for fatigue.

Key words: energy supply, metabolite accumulation, multiple sprint work, muscle activation, neuromuscular fatigue.

INTRODUCTION

There is a reversible decline in force production by muscles when they are contracting at or near their maximum capacity. This has been classically demonstrated in isolated muscle cells stimulated to give maximal isometric tetani. Such experiments have provided valuable insights into the potential determinants of fatigue. Nonetheless, such conditions are obviously artificial and the application of such findings to dynamic exercise has been questioned. Although a similar pattern for the decline in muscle performance is observed when athletes are asked to perform intermittent-sprint exercise (Fig. 1), the task-dependent nature of fatigue means that the mechanisms of fatigue may differ. In particular, there may be a role for neural/brain factors on the fatigue process during intermittent-sprint exercise (factors obviously omitted from isolated muscle preparations). The present paper examines how fatigue manifests during whole-body, intermittent-sprint exercise and discusses the potential muscular and neural mechanisms that underpin this fatigue.

DEFINITIONS

Intermittent-sprint exercise

There are many disciplines in which athletes are required to repeatedly produce maximal or near-maximal efforts (i.e., sprints), interspersed with brief recovery intervals (consisting of complete rest or low- to moderate-intensity activity), over an extended period (1–4 h). Although some authors have used the word ‘sprint’ to describe exercise lasting 30 s or more, for the purposes of the present paper ‘sprint’ activities are constrained to brief exercise, in general ≤ 10 s. Longer-duration, maximal-intensity exercise, in which there is a considerable decrease in performance, is referred to as ‘all-out’ exercise, but is not discussed in this paper.

Fatigue

For the purpose of the present paper, ‘fatigue’ is defined as a reversible, exercise-induced reduction in maximal power output (e.g., during cycling exercise) or speed (e.g., during running exercise), even though the task can be continued. Fatigue during intermittent-sprint exercise typically develops rapidly after the first sprint (Fig. 1). It is now accepted that exercise-induced fatigue can be caused by a variety of factors, ranging from the generation of an inadequate motor command in the motor cortex (i.e., neural factors) to factors related to metabolite accumulation or...
energy supply (i.e. muscular factors). The aim of this paper is to
discuss evidence for the contribution of these mechanisms to fati-
gue during intermittent-sprint exercise.

**FACTORS INFLUENCING FATIGUE DURING
INTERMITTENT-SPRINT EXERCISE**

**Influence of initial sprint performance**

An important factor influencing fatigue during intermittent-sprint
exercise is the initial (i.e. first sprint) mechanical score, which
has been reported to be correlated with the decrement in perfor-
ma ince over subsequent sprints.7,8 This is probably because sub-
jects with a greater initial sprint performance will produce
greater changes in muscle metabolites, arising secondary to a
higher anaerobic contribution, which have been related to larger
performance decrements.10 In support of this, individuals with
lower anaerobic power reserves, implying less reliance on anaer-
obic metabolism, show a higher fatigue resistance during inter-
mittent-sprint exercise.7 This suggests that the metabolic
pathways supporting force production and not the absolute force
generated per se may explain power decrements during intermit-
tent-sprint exercise. Therefore, initial sprint mechanical output
per se cannot solely account for performance decrements during
intermittent-sprint exercise. Indeed, previous fatiguing muscle
contractions (i.e. a prior set of intermittent sprints) exacerbate
the rate of fatigue during subsequent sprints, despite being
matched for initial sprint power.11 Similarly, there is greater
fatigability (i.e. larger work decrement) across five 6 s cycling
sprints repeated every 30 s in low compared with moder-
ately aerobically trained women matched for single-sprint
performance.12

**Task dependency**

The mechanisms of fatigue vary with intensity, duration and type
of contraction. This task-dependent nature of fatigue is also
apparent with different types of intermittent-sprint exercise. For
example, the exercise mode (e.g. cycling vs running) has been
reported to affect the development of fatigue;3 decrement scores
during intermittent-sprint cycling protocols (10–25%) are gener-
ally greater than those for running protocols (5–15%). The type
of resistive load (e.g. mechanically, wind or electromagnetically
resisted) also appears to affect fatigue development during
intermittent-sprint exercise. Moreover, fatigue resistance during
intermittent-sprint exercise depends on the distribution (e.g. number
of repetitions) and duration of the work periods,16 as well as the
recovery pattern (i.e. the nature,17–19 duration20–23 and intensity24
of the recovery between sprints). Although there is some conflict
within the literature,24 performing active versus passive recovery is
generally associated with a higher degree of fatigue develop-
ment.17,19,24 Compared with a passive recovery, low- and moderate-
intensity active recoveries (approximately 20% and 35% maximal
oxygen uptake (VO2max), respectively) have similar effects on inter-
mittent-sprint exercise performance and muscle metabolism.24

![Graph showing typical mechanical work profile during intermittent-sprint exercise](image_url)

**Determinants of fatigue during intermittent-sprint exercise**

As described above, there is a reversible decline in power output
when athletes are asked to repeat short-duration sprints (< 10 s)
terspersed with brief recovery.3 This is typically accompanied
by a decrease in maximal isometric contraction (MVC) force.26
Although a concern with most studies is the time taken to trans-
fer from the cycle ergometer to the isometric ergometer (to per-
form the MVC), using a specially constructed cycle ergometer
that allows an MVC to be performed directly on the cycle ergom-
eter we have unpublished data that there is little effect of the
short time that it takes to transfer from one ergometer to the other
(D Bishop, unpubl. obs., 2010). This decrease in the ability of
muscle to produce force has typically been attributed to the gen-
eration of an inadequate motor command in the motor cortex (i.e.
nerval factors) and/or factors related to metabolite accumulation
or energy supply (i.e. muscular factors).

**Neural factors (‘central fatigue’)**

Because maximal sprint exercise demands high levels of neural
drive,27 failure to fully activate the contracting musculature
should decrease force production and reduce intermittent-sprint
performance. Potential changes in muscle activation during and
following intermittent-sprint exercise have traditionally been
assessed by surface electromyogram (EMG), percutaneous motor

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**List of abbreviations:**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>βm</td>
<td>Muscle buffer capacity</td>
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<tr>
<td>EMG</td>
<td>Electromyogram</td>
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<td>MN</td>
<td>Motor nerve</td>
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<td>MCT</td>
<td>Monocarboxylate transporters</td>
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<td>M-wave</td>
<td>Muscle compound action potential</td>
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<td>MVC</td>
<td>Maximal isometric contraction</td>
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<td>PCR</td>
<td>Phosphocreatine</td>
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<td>TMS</td>
<td>Trans-cranial magnetic stimulator</td>
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<tr>
<td>VO2max</td>
<td>Maximal oxygen uptake</td>
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Figure 1: Typical mechanical–work profile during intermittent-sprint exercise. The data are for ‘all-out’ sprints, interspersed with brief recovery periods, performed on a cycle ergometer.
nerve (MN) stimulations and, more recently, trans-cranial magnetic stimulation (TMS).

Changes in EMG

In conjunction with the decrease in maximum force/power production, a concurrent decline in the amplitude of EMG signals (integrated EMG values and root mean square) has been reported in several,\(^7,11,26,28-30\) although not all.\(^21,31-33\) Studies. Although further research is required, the changes in EMG appear to depend on the magnitude of fatigue reported. With mild fatigue (power decrement score < 10%), a steady level of neural activation during intermittent-sprint exercise has usually been reported.\(^21,31-33\) However, when there is greater fatigue (> 10%), a concurrent decline in mechanical performance and the amplitude of the EMG signals have consistently been reported across sprint repetitions.\(^7,11,26\) These changes in surface EMG activity suggest a suboptimal motor unit activity (i.e. a decrease in recruitment, firing rate or both).

Although many studies have used changes in EMG as a proxy for changes in neural drive, difficulties in interpreting EMG data need to be acknowledged (e.g. amplitude cancellation phenomena, excessive sweat, changes in fibre membrane and motor unit properties).\(^34\) Another confounding factor when interpreting changes in EMG during intermittent-sprint exercise is the concurrent reduction in power output that makes it difficult to determine whether the decrease in EMG activity is the consequence, or the cause, of the reduced power output. In an effort to resolve this issue, we recently used the EMG collected during three warm-up sprints to establish the power–EMG relationship and to determine whether the subsequent decrease in EMG previously observed during intermittent-sprint exercise was consistent with the decrease in power output (O Girard et al., unpubl. obs., 2010). Interestingly, there was only a 5.5% decrease in EMG, which was significantly less than that estimated from the power–EMG relationship established during the warm up (approximately –23%; Fig.2). These changes suggest that the predominant cause of fatigue during intermittent-sprint exercise is not a decrease in neural drive (as inferred from changes in surface EMG).

![Fig. 2](image_url) Changes in real (○) and predicted (●), as estimated from the pre-exercise power-electromyogram (EMG) relationship, surface EMG during an intermittent-sprint test. The data are from 10 × 6 s ‘all-out’ sprints on a cycle ergometer interspersed with 30 s of passive recovery. RMS, root mean square of the EMG signal.

Change in voluntary activation (estimated from percutaneous MN stimulations)

Traditionally, MN stimulations applied mainly during isometric contractions (i.e. the twitch interpolation method) have been used to measure how much of the muscle’s possible force is produced by a voluntary contraction. Using this approach, we and others have established that there is only a small decrease in voluntary activation from the pre- to post-exercise MVC.\(^26,31\) This suggests that, under conditions of considerable fatigue, failure to fully activate the contracting musculature may only make a small contribution to fatigue during intermittent-sprint exercise. This is consistent with the small changes in muscle activation inferred from changes in surface EMG activity.

Changes in voluntary activation (estimated from TMS)

A decrease in voluntary activation can theoretically arise from changes at the spinal level and/or supraspinal factors. Potential supraspinal factors include disturbances in brain electrical activity, cortical excitability and/or brain neurotransmitter (e.g. serotonin, dopamine and acetylcholine) concentrations.\(^35,36\) By applying TMS stimulations (together with conventional MN stimulation) during MVC before and after intermittent-sprint exercise, it is possible to assess the completeness of cortically and peripherally derived estimates of voluntary activation. Using this method, we have recently found that there is no change in voluntary activation (as estimated from TMS) following intermittent-sprint exercise (O Girard et al., unpubl. obs., 2010).

Conclusions

The small changes in surface EMG, along with the lack of change in voluntary muscle activation (estimated from both percutaneous MN stimulations and TMS), indicate that there is little change in neural drive to the muscles following intermittent-sprint exercise. This, along with the observation that the decrease in EMG is much less than that which would be predicted from the decrease in power output, suggests that peripheral mechanisms are the predominant cause of fatigue during intermittent-sprint exercise.

MUSCULAR FACTORS (PERIPHERAL FATIGUE)

Muscle excitability

Following intense dynamic contractions there are marked ionic disturbances at the skeletal muscle level, arising secondary to decreases in Na+/K+-ATPase activity.\(^37,38\) In such cases, the Na+/K+ pump is not able to readily re-accumulate the K+ efflux from the muscle’s cells, resulting in at least a doubling of muscle extracellular [K+]\(^+\).\(^39\) Although these modifications impair cell membrane excitability and depress force development, probably by slow inactivation of Na+ channels,\(^40\) our unpublished observations have shown that plasma [K+], when corrected for changes in plasma volume, does not change following intermittent-sprint exercise (F Serpiello et al., unpubl. obs., 2010). However, further research is required because: (i) interstitial [K+] is considerably higher than venous plasma [K+] at similar work intensities; and
(ii) venous [K\(^+\)] values may not reflect the concentration in the interstitium (i.e. the site where K\(^+\) may have its effects).\(^{39}\) An indirect measure of muscle excitability can be obtained by applying an electrical stimulus to peripheral nerves. Decreased muscle compound action potential (M-wave) amplitude, but not duration, has been reported after an intermittent-sprint running protocol, suggesting that action potential synaptic transmission, rather than propagation (i.e. impulse conduction velocity along the sarcolemma), may be impaired during such exercise.\(^{51}\) However, a potentiation of the M-wave response has also been reported following intermittent-sprint exercise.\(^{26}\) Thus, further research is needed to determine whether impairments in muscle excitability contribute to muscle fatigue induced by intermittent sprints.

**Limitations in energy supply**

*Phosphocreatine availability*

Total intramuscular phosphocreatine (PCr) stores are approximately 80 mmol/kg dry muscle. Because maximal rates of PCr breakdown can approach 9 mmol/kg dry muscle per s,\(^{41}\) maximal sprinting results in a severe reduction in intramuscular PCr content. For example, the PCr concentration after a 6 s sprint has been reported to be approximately 35–55% of resting values.\(^{10,42–44}\) Because recovery times during intermittent-sprint exercise generally do not exceed 60 s, the ATP/PCr stores are likely to be only partially restored at the onset of each subsequent sprint.\(^{5,43}\) Coupled with the fact that the resynthesis of PCr and the recovery of power output follow similar time-courses, it has been proposed that performance during intermittent-sprint exercise may become increasingly limited by PCr availability.\(^{45,46}\) Consistent with this hypothesis, significant correlations have been reported between the resynthesis of PCr and the recovery of power output in the first 10 s of a second 30 s sprint \((r = 0.84; \ P < 0.05)^{5}\) and the partial restoration of intermittent-sprint performance (i.e. total work done; \(r = 0.67, \ P < 0.05; \) A Mendez-Villanueva, unpubl. obs., 2012). Furthermore, most research indicates that short-term creatine supplementation can improve intermittent-sprint performance.\(^{47}\) Collectively, these results suggest that PCr availability is an important determinant of intermittent-sprint performance.

**Anaerobic glycolysis**

Anaerobic glycolysis supplies approximately 40% of the total energy to a single 6 s sprint, with a progressive decrease in glycolysis as sprints are repeated.\(^{10,48}\) As a consequence, there is an eightfold decrease in the absolute ATP production from glycolysis from the first to the last sprint of 10 × 6 s maximal sprints interspersed with 30 s of recovery.\(^{10}\) Although the reduction in the rate of ATP production by anaerobic processes is greater than the decrements in power output, these results nonetheless suggest that reduction in anaerobic glycolysis contributes to fatigue during intermittent-sprint exercise.

**Oxidative metabolism**

There is a perception that the capacity for oxidative metabolism is not an important determinant of intermittent-sprint performance because the contribution of oxidative phosphorylation to total energy expenditure during a single short sprint is quite small (< 10%).\(^{49,50}\) However, as sprints are repeated, the contribution of aerobic metabolism increases progressively such that aerobic metabolism may contribute as much as 40% of the total energy supply during the final sprints of an intermittent-sprint exercise.\(^{49}\) Surprisingly, subjects may even reach their \(\text{VO}_{2\text{max}}\)\(^{51,52}\) during the latter sprints.\(^{57,59}\) This suggests that the contribution of aerobic metabolism during intermittent-sprint exercise may be limited by \(\text{VO}_{2\text{max}}\) and that increasing \(\text{VO}_{2\text{max}}\) may allow for a greater aerobic contribution during the latter sprints, potentially minimizing fatigue. This hypothesis is supported by the observation that subjects with a greater \(\text{VO}_{2\text{max}}\) are better able to maintain power output/sprint times during intermittent-sprint exercise and is supported by significant correlations \((r = –0.45 \text{ to } –0.75)\) between \(\text{VO}_{2\text{max}}\) and fatigue indices.\(^{12,54–57}\) However, it should be noted that not all studies have reported significant correlations between \(\text{VO}_{2\text{max}}\) and fatigue indices during intermittent-sprint exercise \((r = –0.20 \text{ to } –0.30).^{8,58–63}\) The absence of stronger correlations between \(\text{VO}_{2\text{max}}\) and intermittent-sprint performance may be related to the belief that the primary factor limiting \(\text{VO}_{2\text{max}}\) is the ability of the cardiorespiratory system to deliver \(\text{O}_2\) to the contracting muscles, whereas intermittent-sprint performance may be limited primarily by peripheral muscle factors.\(^{54}\) This is supported by the observation that fatigue during intermittent-sprint exercise is correlated with maximal ADP-stimulated mitochondrial respiration measured directly in muscle fibres.\(^{65}\) Thus, although perhaps not the most important factor, oxidative capacity does appear to be a determinant of fatigue during intermittent-sprint exercise.

**Metabolite accumulation**

*Acidosis*

Many findings suggest that the considerable increases in muscle\(^{1,2,4,6–67}\) and blood\(^{21}\) H\(^+\) accumulation that occur during intermittent-sprint exercise may affect sprinting performance. For example, correlations have been observed between the level of fatigue during intermittent-sprint exercise and both muscle buffer capacity \((\beta m)\) and changes in blood pH\(^{8,12,66}\) Furthermore, the content of skeletal muscle monocarboxylate transporters (i.e. MCT1), which facilitate the intramuscular lactate and H\(^+\) removal process, has been inversely correlated with fatigue during intermittent-sprint exercise.\(^{69}\) Although the mechanisms remain contentious, it is possible that H\(^+\) accumulation affects intermittent-sprint performance via adverse effects on the contractile machinery and/or through the inhibition of ATP derived from glycolysis, possibly via negative effects on phosphofructokinase and glyco-gen phosphorylase.\(^{70}\)

In contrast with the above hypothesis, acidification as a direct cause of muscle fatigue has been challenged on at least three accounts: (i) the time-course of the recovery of force/power is much faster than that of pH; (ii) high power outputs have been obtained under acidic conditions; (iii) the ingestion of sodium bicarbonate (known to increase extracellular buffering capacity) has, in some cases, been reported not to affect intermittent-sprint performance.\(^{33,71}\) Furthermore, researchers have failed to observe
a relationship between the recovery of muscle pH and the recovery of either 30 s ‘all-out’ performance or intermittent-sprint performance (A Mendez-Villanueva, unpubl. obs., 2012). Therefore, further research is needed to clarify whether H⁺ accumulation is an important determinant of fatigue during intermittent-sprint exercise.

INFLUENCE OF OTHER FACTORS

Other factors, such as age, training status and sex, have been reported to influence intermittent-sprint performance. In general, being young, female or aerobically trained has typically been associated with a smaller fatigue score. However, further research is required to establish whether these differences can be attributed to differences in fatigability or can be explained largely by differences in initial sprint performance.

CONCLUSIONS

During intermittent-sprint exercise, the inability to reproduce performance in subsequent sprints (fatigue) is manifested by a decline in sprint speed (running) or peak/mean power output (cycling). Although not extensively studied, failure to fully activate the contracting muscle does not appear to be a major determinant of fatigue during intermittent-sprint exercise. To date, the principal factors proposed to be responsible for fatigue include limitations in energy supply (e.g. PCR content and oxygen consumption) and the accumulation of by-products of metabolism (e.g. H⁺).

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