REVIEW ARTICLE

Sports Med 2009; 39 (4): 257-278
0112-1642/09/0004-0257/$49.95/0

Muscle Fatigue in Males and Females during Multiple-Sprint Exercise

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Abstract

Females have often been reported to have a greater muscle fatigue resistance than males, especially during exercise at low-to-moderate intensities. Differences in muscle mass, muscle metabolism and voluntary activation patterns have been the primary explanations for the differences in performance and physiological responses to exercise between sexes. However, while ample data are available for isometric contractions, dynamic activity is a less studied mode of exercise, and there is even less information regarding multiple-sprint exercise (MSE). This is surprising given that MSE places unique demands on metabolic processes in the muscle where energy supply oscillates between fuelling contractile activity and restoring homeostasis. As such, MSE provides a rich area for future applied research. This review examines the limited data available concerning the physiological responses of males and females to sprint exercise, and discusses the methodological confounds arising from non-appropriate comparison methods. Based on original findings,

we highlight that sex differences in the absolute mechanical work performed during a given task might explain a significant part of the differences in physiological responses of males and females to sprint exercise. We therefore suggest that future studies using male and female subjects to answer basic physiological questions use mechanical work as a covariate.

Human skeletal muscle fatigue can be defined as a transient, exercise-induced reduction in the maximal force capacity of the muscle.[1,2] Several mechanisms have been proposed that contribute concurrently to the fatigue exhibited by a muscle or muscle group following exercise, and the classic approach used to identify the cause of muscle fatigue has been to distinguish between 'central' and 'peripheral' mechanisms. Typically, peripheral skeletal muscle fatigue involves processes occurring at or distal to the neuromuscular junction, in the presence of unchanged or increasing central motor output.[3-6] On the other hand, central fatigue is due to failure at a site within the CNS.[2,7,8] Studies applying an electrical stimulus to peripheral nerves and/or a magnetic stimulus to the motor cortex have demonstrated that both 'central' and 'peripheral' mechanisms are involved during fatiguing contractions, and a number of good scientific reviews on this topic are available to the reader.[1,2]

It has also been demonstrated that human skeletal muscle fatigue is influenced by the biological sex of the individual.[9-11] Studies on the physiological function of females have mostly concentrated on isolated muscle exercise (e.g. isometric and isokinetic contractions of a single muscle group). This work has provided tremendous advances in our understanding of possible mechanisms for these specific tasks. In the last decade, however, the popularity of team and court sports, which require the athlete to sprint intermittently over the course of the game, has increased.[12-14] Consequently, many authors have explored the physiology of multiple-sprint exercise (MSE). This particular pattern of activity involves repeated bouts of short duration (≤8 seconds), high-intensity (>300% $\dot{V}O_{2max}$) exercise, separated by short rest duration (≤30 seconds). Such short rest periods have been shown to negatively affect subsequent sprint performance.[15,16] Current knowledge of MSE physiology is based largely on the responses of young adult males, and this is somewhat surprising since in most countries team and court games are also popular sports among females. More importantly, there is now strong evidence that the mechanisms underlying force decline are highly task specific.[1,2] This means that muscle fatigue can be induced by a combination of processes contributing in different ways to the decline in force, according to the details of the task (intensity, duration, mode of contraction, muscle, etc.). As such, one cannot rely on data arising from isometric contraction research to explain sex differences in performance and muscle fatigue during MSE. Such whole-body tasks (e.g. running and cycling) need to be further explored to achieve greater understanding of female physiology.

This paper begins with an updated review of general physiological sex differences that could potentially contribute to the sex differences observed during MSE. Then we discuss the relative importance of these factors in the fatigue processes during sprint exercise. Finally, we summarize the limited number of studies that have investigated the physiological responses to MSE in females versus males, and evaluate the evidence for and against the existence of a sex-related difference in the manifestation of skeletal muscle fatigue in response to MSE. In particular, we question whether males and females display different degrees of fatigue during MSE.

1. General Physiological Sex Differences

It is well accepted that males possess greater absolute muscle strength and produce greater power output scores than their female counterparts in a variety of muscles and in a variety of exercise conditions.[17-30] Research has also fairly well documented the influence of sex on muscle

fatigue, typically reporting that a female's muscle is capable of longer endurance times (i.e. greater resistance to fatigue) and faster recovery (i.e. ability to restore force or power output) than a male's muscle.[23,29,31-35] This has been observed with the use of various fatiguing, isometric protocols at low to moderate intensities, and during sprint exercise, where females have been observed to maintain the initial absolute power output for a longer time than males.[19,36,37]

Several mechanisms may explain the effect of sex on performance and fatiguability. Evidence supporting body composition (e.g. lean and fat mass), muscle metabolism (e.g. hormonal regulation, enzymatic activities and substrate utilization), muscular characteristics (e.g. typology) and motor unit discharge rate as factors accounting for the sex differences are discussed below.

1.1 Morphology and Body Composition

Differences in morphology and body composition are the most visible and obvious differences when one first compares the two sexes. On average, and at the same age, males are taller (+11 cm), heavier (+13 kg) and have a greater lean mass (+18 kg) and lower fat mass (−5 kg) than their female counterparts.[28,38-41] These parameters have therefore often been suggested to explain sex differences in performance.[10,38,42,43] The primary intrinsic determinants of maximum voluntary strength include cross-sectional area (CSA) of the muscle or muscle groups, specific tension (force per unit CSA, which may be affected by the fibre type distribution and the amount of non-contractile tissue present in the muscle), and possible anatomical differences in the mechanical advantage of a muscle acting across a joint.[44-47] Males, having greater segment length and muscle mass, develop higher absolute muscle force and power output than females. For example, a significant correlation ($r = 0.91$; $p < 0.05$) between total mechanical work (during repeated cycle sprints) and body mass (BM) has been reported in athletes.[48] Thus, it is not surprising to observe smaller sex differences when indices of performance are expressed as a ratio to body mass, an index of lower limb volume, or CSA of the thigh.[27,30,41,49-52] Performances must then be scaled for body size differences to permit meaningful comparisons between males and females.

It is important, however, to point out that some studies indicate that muscle mass is not the only factor accounting for the sex difference in fatigue. In fact, while differences in performance and fatiguability are reduced, they often persist when the two sexes exercise at the same percentage of initial performance, or when the data are expressed relative to BM, lean BM (LBM), lean volume (LV) of the active limb, and when subjects are matched for strength.[16,30,37,43,50,51,53-59] For example, Fulco and colleagues[55] have shown that the fatigue rate of the adductor pollicis muscle during intermittent, isometric, submaximal contractions was still ≈2-fold slower in females than in males matched for strength. When comparing the performances of males and females during two consecutive 8-second sprints on a cycle ergometer, it has also been observed that males remained more powerful than females when data were expressed relative to LBM (+17%) and lower limb LV (+16%).[16,20] Thus, even though body dimensions explain the major discrepancies between the sexes in performances, sex differences still persist when body dimensions are appropriately controlled. Accordingly, physiological factors (as opposed to muscle mass quantity) must also contribute to the sex difference in performance.

1.2 Endocrine Status

The secretion of sex hormones is another difference between males and females. Androgens (e.g. testosterone) increase protein synthesis and lead to muscle hypertrophy.[60,61] The higher androgen concentration found in males is therefore likely to contribute to some sex differences (e.g. muscle mass, selective hypertrophy of type II fibres). On the other hand, estrogens (i.e. estrone, estradiol and estriol) increase growth hormone (GH) concentration, which is known to stimulate lipolysis and to reduce glycogenolytic activity by reducing plasma adrenaline (epinephrine) secretion.[62,63] However, although the higher estrogen

concentration in females does indeed increase GH release at rest in young and adult females compared with males of the same age,[64-67] exercise seems to evoke a similar incremental GH response in both sexes.[67-69]

Sex also appears to affect the sympathetic responses to supramaximal exercise,[56,70,71] lowering plasma catecholamine levels (and subsequently blood lactate) in females during exercise at the same relative intensity compared with males with similar fitness levels. While some have attributed these differences to a direct inhibitory effect of estradiol on the sympathetic nervous system,[72] Sandoval and Matt[68] concluded that these differences were most likely due to differences in the absolute workload performed by males and females. Less absolute work would lead to less lactate production and glycogen use, and thus less glucose would be taken up by the muscle for refuelling glycogen stores after exercise in females.[68] This study therefore demonstrates indirectly the importance of matching subjects for total work performed before attempting to draw any conclusion regarding sex differences.

Researchers continue to debate whether the different phases of the menstrual cycle affect athletic performance and fatiguability,[73] and possibly modify the magnitude of sex-related differences.[74,75] Some authors have reported higher voluntary muscle strength and total work in a short-term, all-out performance during the luteal phase.[76-78] There is also evidence of enhanced blood lactate removal at high intensities,[79,80] greater O_2 consumption during recovery from repeated cycle sprints,[76] and increased excess post-exercise O_2 consumption after prolonged exercise[81] during the luteal phase relative to the follicular phase. While these metabolic changes could improve multiple-sprint performance by enhancing recovery between sprints,[76] such findings need to be balanced by the many studies that suggest that hormonal fluctuations throughout the cycle do not contribute to sex difference in performance and fatiguability.[82-85] In summary, the multiple contrasting findings clearly demonstrate that there is currently no consensus about the impact of the monthly hormonal fluctuations upon sex differences observed in performance and muscle fatigue.

1.3 Enzyme Activities

In vitro measurements of muscle enzyme activities are related to whole-body energy metabolism,[86] and can provide an insight into the relative contribution of the different energy production pathways in males and females.[70] Very few studies have analysed the impact of sex on the activity of enzymes involved in the phosphagen (or alactic) energy systems. However, activities of myosine adenosine triphosphatase (ATPase) and creatine phosphokinase have been found to be higher in males than females,[87,88] suggesting a greater potential phosphagen energy provision in males to support performance.

The maximal activities of glycolytic and glycogenolytic enzymes (glycogen phosphorylase, phosphofructokinase [PFK] and lactate dehydrogenase) are also lower in females.[19,42,62,87-92] Interestingly, Jaworowski et al.[42] reported that the higher maximal activities of glycolytic enzymes in males were related to the height, mass, muscle CSA and relative area of type II fibres, which were all significantly larger in males than in females. As males participate more in intense activities than females, particularly during childhood,[93-96] the greater glycolytic enzyme activities reported in males may then be caused by daily activity patterns rather than intrinsic physiological sex differences. An increased muscle mass associated with a greater muscle recruitment is likely to contribute to the greater anaerobic potential in males.[36,38,55,62,97]

Assessment of oxidative enzyme activities of the Kreb's cycle has been contradictory; higher values for males[87,90,92] or no sex differences[19,42,91,98] have been reported for various enzymes. For enzymes involved in lipid oxidation, no sex differences have been reported.[19,42,92] Again, however, when sex differences have been reported it is difficult to establish whether these are attributable to training or actual sex differences. Evidence against the existence of sex differences is provided by the observation that oxidative enzyme activities and mitochondrial volume density increase have been found to adapt in a similar manner (maximal activity and

magnitude) following endurance exercise training in the two sexes.[98-100]

1.4 Substrate Utilization

Sex differences in muscle metabolism during exercise have been investigated quite thoroughly.[19,62,63,101,102] Overall, muscle characteristics account for an energetic balance in which the aerobic contribution is more important in females during prolonged sprints[19,39,101] and during submaximal, isometric contractions.[62,97] For example, Hill and Smith[39] estimated the aerobic contribution to total work during a 30-second cycle sprint to be 25% in females and 20% in males. According to Fulco et al.[55] and Russ and Kent-Braun,[103] this predisposition to oxidative phosphorylation would allow a faster ATP resynthesis during recovery. On the contrary, a greater reliance on the anaerobic glycolytic pathway in males would induce a greater fatiguability and a slower recovery.[23,97]

The reduced reliance on glycolytic energy in females may be related to other factors in addition to the lower maximal activation velocity of glycolytic enzymes and the greater reliance on fat metabolism. For example, the lower increase in plasma catecholamine concentration during sprints in females may also influence the stimulation of glycolytic enzymes.[54,56,104,105] Therefore, a reduced maximal activation velocity of glycolytic enzymes, a reduced stimulation of glycolytic pathways, and greater reliance on fat oxidation are all likely to contribute to muscle glycogen sparing and the lower blood lactate concentration reported in females after a 30-second sprint on a cycle ergometer.[54,56,106]

While a lack of sex difference in ATP or phosphocreatine (PCr) reduction has been reported in type I and type II fibres after a 30-second Wingate test,[54] one study has demonstrated a smaller ATP reduction in females than in males in type II fibres among three Wingate tests separated by 20 minutes of recovery.[36] The authors explained these differences via a faster ATP resynthesis in females via a greater inosine monophosphate (IMP) reamination during the recovery phases.[36] On another occasion, the same protocol (three 30-second Wingate tests interspaced with 20 minutes of recovery) resulted in a similar ATP and PCr content decrease and alactic ATP turnover rate in males and females.[107] Males, compared with females, have also been found to exhibit a greater decrease in ATP and PCr concentrations after MSE consisting of 5×6-second sprints every 30 seconds.[108] This lack of consistency for exercise-induced metabolism changes is likely to be related to biopsy timing and the protocols used rather than actual sex differences.

1.5 Muscle Fibre Properties

Based on the histochemical staining properties for the myofibrillar myosin ATPase, different fibre categories can be distinguished in human skeletal muscle. It is generally accepted that untrained females have smaller fibre CSA in all fibre types than untrained males in the muscles of the upper and lower limbs, as do female athletes and bodybuilders compared with their male counterparts.[19,29,38,42,54,62,91,92,98,109-112]

Studies in which fibre percentage has been estimated are less consistent. While several authors failed to observe any differences in the mean proportion of type I, IIA and IIX fibres (% number) between males and females from similar sport specialties and fitness level,[19,36,38,42,98,101,110,111,113] others have reported a greater distribution of type I fibres and lower distribution of type IIX fibres in females.[29,91,92,109] Such discrepancies may be related to sampling bias in subject selection and/or problems with the accurate estimation of fibre percentage.[29] Nonetheless, fibre size and property differences between sexes may have an impact on the peak power output and on the subsequent ability to maintain power output.

1.6 Neural Activation

Neuromuscular activation patterns are increasingly described in males and females during maximal tasks, but it is difficult to form precise conclusions about a typical trend in neuromuscular responses to exercise, as results are highly dependent upon the nature of the task.[103] Further difficulty arises from the controversial

use of electromyogram (EMG) recordings. Indeed, while useful information (e.g. the net motor unit activity) can be extracted from an appropriately recorded surface EMG, there remains a complex mismatch between the spinal cord output and both EMG amplitude and frequency parameters, which limits the interpretation of EMG data when recorded alone.[114,115]

Nonetheless, the maximum voluntary EMG has been found to decrease only in males, after performing 20 repetitions of a maximal squat-lift with a load of 100% of the one repetition maximum.[116] This suggests an attenuation of skeletal muscle recruitment after a strenuous heavy-resistance exercise in males compared with females. Failure in voluntary activation during maximal tasks has also been directly examined between sexes. For example, Russ and Kent-Braun[103] observed a greater neural activation deficit (i.e. reduction in voluntary activation as assessed from a supramaximal train of stimuli superimposed onto a maximal voluntary contraction [MVC] without any concomitant change in the compound muscle action potential nor muscle twitch characteristics) in males than in females during maximal, intermittent, isometric contractions of the dorsiflexor muscles (5-second contraction, 5-second rest conducted to exhaustion). More recently, transcranial magnetic stimulation of the motor cortex to examine the contribution of supraspinal fatigue in performance decrement[8,117,118] has been applied to males and females during a maximal task (six 22-second MVC of the elbow flexors, separated by 10 seconds).[119] The authors concluded that the greater muscle fatigue (i.e. torque reduction) in males than in females was not explained by a difference in supraspinal fatigue but rather involved mechanisms located within the muscles.

A greater peripheral fatigue (as assessed via *M*-wave amplitude alterations with no concomitant reduction in EMG amplitude) for males than females has also been reported during intermittent MVCs of the adductor pollicis muscle (5-second contraction, 2-second rest conducted for 3 minutes).[53] Even though males were stronger than females in this study (mean MVC force: males 10.0 ± 2.7 kg vs females 6.6 ± 1.1 kg; $p < 0.05$), the fatigue index did not show a significant sex difference (males 45% vs females 38%; $p > 0.05$), which suggests a similar amount of fatigue in both sexes. These data indicated that males were more susceptible to transmission failure at the neuromuscular junction and/or decreases in muscle membrane excitability.[53] Interestingly, during a submaximal contraction of the lumbar musculature sustained to exhaustion, a faster compression of the median frequency has been reported in males than in females,[31] which also suggests higher fibre conductibility impairments[114,120-122] in males compared with females.

Overall, the clear distinction of neuromuscular activation patterns in males versus females in exercise physiology is not an easy task, as they are underpinned by the task characteristics. Therefore, it is likely that the central motor output to locomotor muscles will differ during MSE, as well as the 'central' and/or 'peripheral' nature of fatigue mechanisms. Nonetheless, such differences in neuromuscular physiology between the sexes could contribute to the difference in fatigue resistance observed during repeated sprints.

1.7 Summary

Morphological, metabolic and neuromuscular properties of the muscle tissue are different between males and females, and predominately explain the differences in strength, power output and fatigue resistance between the sexes. Historically, body composition has been the principal factor used to account for sex differences in performances, but research has demonstrated that enzyme activities, substrate use and central motor output also contribute. Muscle fatigue in males and females during sprint exercise has not been studied extensively in the literature, and as a consequence, the influence of the above-discussed factors is not well understood. The final sections of this review focus on male versus female performance and muscle fatiguability during sprint exercise, with particular emphasis on analysing the appropriateness of current methods used to compare the two sexes.

2. Sex Differences in Sprint-Induced Muscle Fatigue

The introduction and section 1 of this article have highlighted the importance of sprinting and repeated sprinting to team-sport performance, and the origins of sex differences in terms of general performance and fatiguability. An understanding of sprint metabolism is, at this point, necessary to comprehend the demands placed on team-sport athletes during competition, and the reader is referred to the detailed reviews of Glaister[123] and Spencer and colleagues.[124] Although studies have shown that females have a greater muscular endurance than males during isometric exercises,[31,55,57,103,119] there is a lack of information on the sex difference in sprint-induced fatigue, considering the amount of information documenting strength and power output differences.[19,28,29,33,35,51,101,125-127] After brief synopses of the determinants of fatigue during sprint exercise, this section focuses on the sex difference in fatigue during single and multiple sprints of varied duration.

2.1 Single-Sprint Exercise

2.1.1 Determinants of Fatigue

Many studies have examined the participation of the energy-producing systems during maximal sprinting exercise of varying duration (10-, 20- and 30-second sprints). During such sprints, peak power is quickly reached in 2–3 seconds (before peak pedal speed), and thereafter power declines.[20,128-131] This implies a very high energy need from the very beginning of the sprint. Overall, during a single, short-duration sprint (≤6 seconds), the rate of ATP utilization is extremely high, with a mean value of ~15 mmol/kg/sec dry muscle (d.m.).[132] Approximately 50% of the ATP is supplied by the degradation of PCr, while intramuscular ATP stores, anaerobic glycolysis and aerobic energy provide the remainder.[106,132-134] As the sprint duration increases, energy system contribution is modified, and one notes a progressively greater participation of anaerobic glycolysis and oxidative metabolism (figure 1).[128,135-138] For all sprint durations, it has been demonstrated that ATP depletion is minimal and is unlikely to constitute a limiting factor of performance.[130,132,133,137,140] However, such intensities result in a severe reduction in intramuscular PCr concentration. Indeed, PCr depletion after 6 seconds of sprinting has been reported to be around 35–55% of resting values.[132,133,140,141] The study of muscle metabolic responses to 10 and 20 seconds of cycle ergometer sprinting demonstrated that PCr was reduced by about 55% after 10 seconds and about 73% after 20 seconds.[135,136,142] Following a 30-second sprint, the depletion is even greater (i.e. up to 80%).[130,133,135,143-146] Maximal sprinting activity thus requires considerable contribution of PCr to provide energy, and it is likely that the ability to sustain sprint exercise will be affected by PCr availability in the working muscles. This is supported by the direct relationship ($r^2 = 0.74$; $p < 0.05$) between the percentage recovery of PCr following a recovery period and the subsequent recovery of performance, expressed as percentage of mean power output (figure 2).[143] These data were later confirmed by high correlations between %PCr resynthesis and the percentage recovery of mean power ($r = 0.84$; $p < 0.05$) and mean pedalling speed ($r = 0.91$; $p < 0.05$) during the initial 10 seconds of a second 30-second sprint.[135]

From muscle lactate concentrations, it has been estimated that >40% of total anaerobic

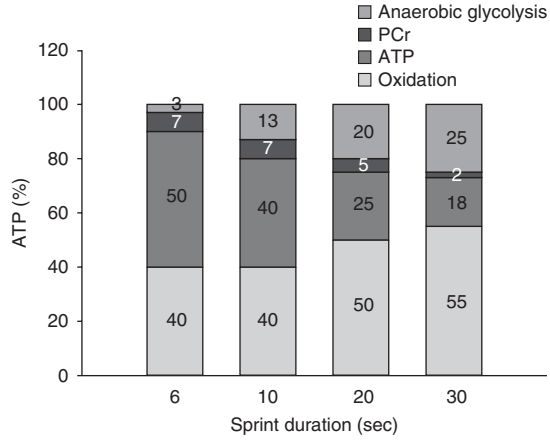

Fig. 1. Schematic illustration of relative energy system contribution to ATP resynthesis (in percentage of total energy) during sprint of varying duration (adapted from Bogdanis et al.,[128,135,136] Gaitanos et al.,[132] Medbø and Tabata[139] and Spriet et al.[138]). **PCr** = phosphocreatine.

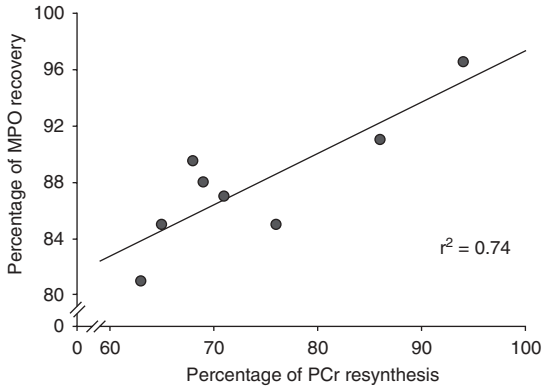

Fig. 2. Relationship between the percentage of phosphocreatine (PCr) resynthesized during the 3-minute recovery period and the mean power output (MPO) achieved during the 6-second sprint (relative to resting value) performed 3 minutes after a 30-second cycle sprint (reproduced from Bogdanis et al.[143] with permission).

energy during a single 6-second sprint bout is provided via anaerobic glycolysis.[132,133] The subsequent decline in muscle glycogen that occurs during repeated, maximal sprints could theoretically contribute to impaired performance via a reduction in substrate and subsequent glycolytic flux. However, the decrease in muscle glycogen has been reported to be only ~30% during a 30-second cycle or treadmill sprint.[130,147-149] Therefore, glycogen stores do not represent a limiting factor in this type of activity.

The most significant source of anaerobic ATP during intense activities lasting at least 10–20 seconds is from glycolysis. Glycogenolysis-glycolysis has been associated with the accumulation of lactate and hydrogen (H^+) ions. Thus, high levels of power output have been associated with a decrease in blood and muscle pH on several occasions. For example, a muscle pH of 6.73 units was estimated after a maximal 30-second sprint on a non-motorized treadmill.[130] This would have, *in vitro*, inhibited PFK and glycogen phosphorylase activities, the key regulatory or rate-limiting enzymes in this pathway.[150-153] This would lead to a reduced rate of ATP production, which might set an important limitation to muscle performance.[128,135,151,152,154-156] A more important consequence of the decrease in pH may be in affecting the muscle contractile mechanism itself, by decreasing the energy available for contraction per ATP hydrolysed.[152,154] In fact, acidosis interferes with the effectiveness of calcium (Ca^{2+}) activation at many sites in the excitation-contraction process.[150,154,157,158] Finally, a decline in muscle pH may contribute to the occurrence of central fatigue. Indeed, the typical association between pH and EMG[7] is consistent with the role of pH in feedback to the CNS and a subsequent alteration in central motor drive during the development of fatigue. These metabolic perturbations have been found to act on nerve terminations of group III and IV afferents, inducing a reflex inhibition of the central drive.[3,90,159-161] Thus, H^+ accumulation may contribute to fatigue during sprint exercise.

Sprint exercise also results in other important ionic perturbations that may contribute to fatigue during sprint exercise. In particular, sprint exercise changes the extracellular potassium (K^+) ion concentration ($[K^+]$) far beyond the narrow limits seen in resting subjects. It has been suggested by some[150,162-166] that subsequent alterations in sarcolemma excitability induce muscle fatigue by preventing cell activation. For example, Medbø and Sejersted[165] reported a >200% increase in plasma $[K^+]$ after a 1-minute running sprint on a motor-driven treadmill (10.5% inclination). In muscles contracting at high workloads, inorganic phosphate (P_i) also accumulates because PCr is broken down to creatine and P_i. The $[P_i]$ increases substantially in the myoplasm during intense exercise and affects both the myofibrillar proteins and activation processes.[167-169]

Although not as extensively studied, changes in skeletal muscle recruitment may contribute to performance decrement during maximal sprinting exercise. The only two studies to have examined neuromuscular fatigue (via EMG recording) during a single sprint are those of Vandewalle et al.[170] and Hunter et al.[171] The first study observed a parallel decline in power output and integrated EMG during a 45-second cycle sprint, and suggested a progressive attenuation of spatial and/or temporal recruitment of motor units during this type of exercise. On the other hand, the EMG amplitude has been shown to remain unchanged (whereas power output declined) during a 30-second cycle sprint.[171]

The authors completed the data with an analysis of the frequency power spectrum, and demonstrated a shift of the mean power frequency towards lower values (–14.7%; $p < 0.05$). According to several authors,[1,114,121,172] this might be caused by an accumulation of metabolites and a consequent decrease in muscle pH, and/or some form of neural control through reflex regulation of muscle force to prevent muscle damage.[173-175] Clearly, more studies using electrically evoked stimulation will need to be conducted to clarify whether voluntary drive parallels the power output decline observed during maximal sprint, to ascertain if a failure of excitation is present under these conditions.

2.1.2 Fatigue during Single-Sprint Exercise in Males and Females

The few studies providing information on performances and fatiguability during sprints in males and females are shown in table I. During a 30-second, supramaximal cycling exercise (such as the Wingate anaerobic test), males develop greater absolute power output levels than females (on average, peak power and mean power are 40% and 30% greater, respectively). These discrepancies are reduced, but overall remain significant, when results are expressed relative to BM, LBM or leg LV,[37,39,51,56,58,106,178] meaning that other factors, likely related to the capacity to sustain a high ATP resynthesis rate, should account for the sexual dimorphism in prolonged sprints.[51] As previously discussed (section 1), the greater potential for anaerobic metabolism in males (due to greater glycolytic enzyme activities) and the larger muscle fibre CSA (associated with greater concentration of male sex steroids) are likely to explain the greater absolute scores in males during such sprints.

With respect to fatigue, however, it is usually accepted that, compared with males, females are capable of maintaining their peak power output for a longer time within sprints. For example, Froese and Houston[37] reported a greater fatigue index (decrement in absolute power output) in males than in females over the course of a 30-second cycle sprint. Such observations may be related to the greater reliance of females on aerobic metabolism[55,100,103,179,180] associated in turn with reduced muscle H^+ ion accumulation and reduced ionic disturbances.[97,181] Additionally, the greater ability of females to maintain motor unit activation at exhaustion[103,116] may contribute to a better maintenance of power output. However, when expressed per unit of BM or leg volume, the sex difference in muscle fatigue disappears; although males exhibited a greater absolute power decrease than females (–433 W vs –315 W, respectively; $p < 0.05$) during a 30-second cycle sprint, these discrepancies disappeared when values were related to the peak power developed during the sprint (mean fatigue index: males 47% vs females 48%; $p > 0.05$).[19] Hill and Smith[39] and Weber et al.[52] confirmed these data, showing a similar relative power output decline in males (mean 48%) and females (mean 52%). Thus, these results suggest that rather than sex differences, differences in fatiguability during 30-second sprints may actually be related to the greater initial power output of males.

When looking at the data obtained during shorter sprints (<10 seconds), leg peak power output (PPO) reached during a cycling force-velocity test was greater in boys than in girls at the same age (14–17.5 years old).[27] Moreover, for the same leg length, the optimal pedalling frequency was higher in boys than in girls, with no sex difference observed for the optimal force.[27] Better performances (+12% to +22%) have also been reported in males during 30 m and 36.5 m track sprints (≈4–6 seconds),[28,51] 5-second treadmill sprints,[59] and 8-second cycle sprints.[16,20] These results are likely to reflect the influence of androgens on qualitative muscular factors (i.e. type II muscle fibres, glycolytic ability) and then on male mechanical scores. The observed sex differences of the optimal and maximal pedalling frequency during cycle ergometer sprint[16,27] may be related to differences in proportion and/or recruitment of fast-twitch fibres. In males (in contrast to females), it has been suggested that a selective hypertrophy of type II fibres[42,182] may occur in response to greater circulating testosterone levels.[183] However, in contrast to results for the longer sprints, the sex discrepancy for short-sprint performance remained present when data

Table I. Sex differences in mechanical, metabolic and hormonal responses to sprint exercise

Study	Exercise mode	Protocol	Principal significant observations
Jacobs et al.[106] (1983)	Cycle	30 s Wingate	MPO (abs. & rel. to BM): M > F Δ [Lac-] after sprint: M > F
Murphy et al.[176] (1986)	Cycle	30 s Wingate	PPO & MPO (abs. & rel. to BM, LBM): M > F
Froese and Houston[37] (1987)	Cycle	30 s Wingate	PPO & TW (abs. & rel. to BM, LLV): M > F Fatigue index (abs.): M > F; (rel. to BM): NS
Hill and Smith[39] (1993)	Cycle	30 s Wingate	PPO, MPO, TW (abs. & rel. to BM): M > F Fatigue index: NS Anaerobic work (abs. & rel. to BM): M > F Aerobic proportion to TW: F > M
Gratas-Delamarche et al.[56] (1994)	Cycle	30 s Wingate	PPO & MPO (abs. & rel. to BM, LBM): M > F Δ [Lac-] after sprint: M > F Δ [adrenaline] after sprint: M > F
Esbjörnsson-Liljedahl et al.[54] (1999)	Cycle	30 s Wingate	PPO & MPO (abs. & rel. to BM): M > F MPO (rel. to LBM): M > F Δ [ATP] & [PCr] after sprint in type I & II: NS Δ [glycogen] after sprint in type I: M > F Δ [Lac-] after sprint in type I: M > F
Vincent et al.[58] (2004)	Cycle	30 s Wingate	PPO & MPO (abs. & rel. to BM, LBM): M > F Δ [glucose] after sprint: F > M Δ [insulin] after sprint: F > M
Weber et al.[52] (2006)	Cycle	30 s Wingate	PPO & MPO (abs. & rel. to BM): M > F PPO & MPO (rel. to LBM and LLV): NS Fatigue index: NS
Perez-Gomez et al.[51] (2008)	Cycle	30 s Wingate	PPO & MPO (abs.): M > F PPO (rel. to LLV): NS MPO (rel. to LLV): M > F
Esbjörnsson-Liljedahl et al.[19] (1993)	Cycle	3×30 s Wingate (20 min)	PPO & MPO (abs. & rel. to BM): M > F Fatigue index (abs.): M > F; (rel. to PPO): NS Total LDH activity: M > F
Bodin et al.[107] (1994)	Cycle	3×30 s Wingate (20 min)	Δ [PCr] & [ATP] after sprints: NS Alactic ATP turnover rate: M = F
Esbjörnsson-Liljedahl et al.[36] (2002)	Cycle	3×30 s Wingate (20 min)	PPO & MPO (abs.): M > F; (rel.): NR PPO decrease from sp1 to sp3: M > F MPO decrease from sp1 to sp3: M = F Δ [ATP] & [IMP] after sprint in type II: M > F Δ [glycogen] after sprint in type I: M > F
Jacobs et al.[106] (1983)	Cycle	10 s sprint	MPO (abs. & rel. to BM): M > F Δ [Lac-] after sprint: M > F
Winter et al.[30] (1991)	Cycle	4×8 s sprint (5 min)	PPO (abs.): M > F; (rel. to LLV): NS PPO (rel. to LLV-analysis of covariance): M > F
Martin et al.[27] (2004)	Cycle	2×5–8 s sprint (3 min)	V_{opt} (rel. to LL-analysis of covariance): M > F (young adults: 14–17.5 y of age)
Doré et al.[177] (2005)	Cycle	3×5–8 s sprint (4 min)	PPO (rel. to LLV-analysis of covariance): M > F (young adults: 16–20 y of age)
Mayhew and Salm[28] (1990)	Run-track	36.5 m	Sprint time: F > M

abs. = absolute value; **BM** = body mass; **F** = females; **IMP** = inosine monophosphate; **Lac-** = lactate; **LBM** = lean body mass; **LDH** = lactate dehydrogenase; **LL** = leg length; **LLV** = lean leg volume; **M** = males; **min** = minutes; **MPO** = mean power output; **NR** = not reported; **NS** = not significant; **PCr** = phosphocreatine; **PPO** = peak power output; **rel.** = relative value; **run-track** = over-ground running; **s** = seconds; **sp1** = sprint 1; **TW** = total work; **V_{opt}** = optimal velocity (i.e. velocity to reach peak power output); **[…]** indicates concentration; Δ indicates delta changes from rest to the end of the exercise.

were expressed per unit of BM (20.5%), LBM (17%), leg LV (12%) and thigh LV (16%).[16,20,59]

Comparisons of within-sprint fatigue patterns during short-duration sprints (<10 seconds) are difficult to find in the literature. We are aware of only one study that has directly investigated sex differences in fatigue pattern within a brief sprint.[16] Subjects in this study were only matched on the basis of their physical activity (males 11.8 ± 6.5 vs females 10 ± 4.2 h/week; $p > 0.05$). The authors demonstrated that during a single, all-out, 8-second cycle sprint bout against an optimal force (males 86 ± 12 N vs females 53 ± 8 N; $p < 0.05$; optimal forces corresponded to ≈10% of body mass for each sex), females had a greater decrement in relative power output than males (−31% vs −19% of PPO, respectively; $p < 0.05$). In another study conducted on ten intermittent, 6-second sprints on a non-motorized treadmill,[104] the two sexes seemed to display similar power decrement within the first sprint of the series (males −32%, females −27%), but unfortunately statistical analysis was not performed on these data.

In conclusion, more data clearly need to be collected from females to better define metabolic and neuromuscular changes, and to examine fatigue patterns during sprint exercise, especially during brief sprint exercise (<10 seconds). In light of the available results, one may imagine that males and females would exhibit similar relative performance decrements when repeated sprints are involved.

2.2 Multiple-Sprint Exercise

2.2.1 Determinants of Fatigue

There are very few data on the relative energy system contributions during MSE involving consecutive, all-out sprints of short duration. During brief periods of maximal work, ATP provision is maintained through the integration of various metabolic processes. However, as work bouts are repeated, the metabolic response to subsequent work bouts will be affected by the previous exercise and the duration of the intervening rest periods. Due to the complexity of physiological processes that regulate this type of activity, research shows that MSE places considerable demands on both aerobic and anaerobic pathways, although the relative contribution from each of these sources is still an issue of controversy.[48,123,132,184,185]

Muscular fatigue that develops during MSE is associated with signs of energy deficiency, i.e. increased concentrations of IMP, inosine, hypoxanthine and uric acid.[15,48,148,186-189] Since energy provision during MSE is maintained predominantly by anaerobic sources (PCr degradation and anaerobic glycolysis), deficiencies in energy provision are likely to be associated with limitations in anaerobic metabolism.[123,124] In particular, close relationships ($0.84 < r < 0.86$; $p < 0.05$) have been reported between PCr resynthesis and the recovery of power output in different sprinting conditions,[135,143] suggesting that the ability to reproduce high power outputs is directly related to the resynthesis of PCr. This is supported by studies showing that occlusion during recovery (and hence the prevention of PCr resynthesis) impairs the recovery of power output, while creatine supplementation improves repeated-sprint performance.[190-193] Consequently, some of the decrease in power output during MSE can probably be attributed to the decrease in the absolute contribution of PCr to the total ATP production from sprint one to sprint ten (44.3 ± 4.7 vs 25.3 ± 9.7 mmol/kg d.m., respectively).[132] In addition, a large decrease in the contribution of anaerobic glycogenolysis (11-fold reduction) and glycolysis (8-fold reduction) to energy supply has been reported from the first to tenth sprint (10×6-second sprints, 30-second recovery),[132] which is also likely to contribute to the appearance of fatigue during MSE.

The accumulation of metabolites has also been demonstrated to correlate with fatigue during MSE. In particular, the accumulation of H^+ (acidosis) may impair performance through effects on the contractile machinery and its potential role in glycolytic inhibition (through negative effects on glycolytic enzymes). This is supported by studies demonstrating a correlation between repeated-sprint ability and both muscle buffer capacity and changes in blood pH.[48,184,194] Greater improvements in repeated-sprint ability

following training have also been reported in subjects with greater improvements in muscle buffer capacity[195] and the sodium-hydrogen exchanger[196] – a ubiquitously expressed integral membrane protein that mediates the exchange of one extracellular sodium ion with one intracellular proton, which plays a central role in the regulation of intracellular pH in most cells. In addition, Bishop et al.[197] have reported a significant reduction in fatigue during 5×6-second sprints (24-second recovery) following sodium bicarbonate ($NaHCO_3$) administration. In contrast, Gaitanos et al.[198] indicated no effect of $NaHCO_3$ ingestion on performance scores and fatigue throughout ten 6-second sprints (30-second recovery). This discrepancy may be related to different exercise protocols used (cycling vs running) or the large variability that has often been observed in performance improvement in response to alkalosis.[199] Further investigations are clearly required to fully establish the role, if any, of H^+ accumulation on the development of fatigue during MSE.

Recent applied physiological findings[150,167,200] have revealed that K^+ and P_i accumulation may also have a significant role in muscle fatigue. However, even though the negative effects of a rise in interstitial $[K^+]$ and intracellular $[P_i]$ have been studied during high-intensity exercise,[165,201,202] there is no study to the authors' knowledge investigating such ionic aspects during MSE. An interesting observation, however, was provided by Mohr et al.[166] who investigated K^+ kinetics during three repeated, intense, one-legged knee extensions with 10-minute recovery (exercise protocol not specific to the activity patterns of field-based team sports). They found that, when intense exercise was repeated, the rate of K^+ accumulation in the initial phase of exercise was lowered and $[K^+]$ at exhaustion decreased, suggesting that it is not the accumulation of K^+ in the muscle interstitium *per se* that depresses performance when exercise is repeated.[166] Further research is required to investigate the consequences of K^+ and P_i accumulation during the development of fatigue when short bouts of exercise are repeated over a long period of time.

Finally, neural adjustments have been linked to fatigue occurrence during MSE. However, very few studies are available on this topic, and the uncertainty regarding the extent, if any, to which muscle recruitment impairs MSE performance is reflected in the contrasting results of investigations into the EMG signal. Observation of steady levels of EMG signal amplitude (assessed through integrated EMG or root mean square) in prime mover muscles during and after MSE[203-205] suggests that despite mechanical performance becoming progressively impaired, the neural system still recruits motor unit pools at their highest firing rate. On the other hand, proof of neural adjustments (i.e. reduction in the central neural drive to active musculature and power spectrum frequency, and inter-muscle coordination pattern changes) gathered from several studies demonstrate the progressive inability of the brain to maintain the initial pattern of motor unit activation throughout repeated sprint bouts.[204,206-209] Once again, further research is required to clarify the neural adjustments that occur during MSE.

In summary, while fatigue during MSE is likely to be the result of a spectrum of events, research supports a predominantly anaerobic ATP provision during work periods and an exclusively aerobic process of recovery.[15,48,128,132,135,186,210-215] Moreover, that both PCr resynthesis and H^+ removal 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.[123,216] This is further supported by studies showing that endurance-trained athletes are better able to resist fatigue during MSE than their sprint-trained counterparts,[198,211] and by the correlation between work decrement during MSE and the peak oxygen consumption obtained during a graded exercise test ($r = -0.62$; $p < 0.05$).[184] Therefore, if there is a sex difference in repeated-sprint ability, it is likely to be associated with sex differences in the aerobic contribution to repeated sprints, the ability to breakdown and resynthesise PCr, buffer H^+ and/or the ability to maintain an optimal muscle recruitment pattern as sprints are repeated.

2.2.2 Fatigue during Multiple-Sprint Exercise in Males and Females

Sex differences in fatiguability during MSE (i.e. the ability to recover from one sprint to the subsequent one) have been poorly examined. To the best of our knowledge, the first study to investigate sex differences during MSE was conducted in 1990, with a protocol involving a 6-second sprint on a non-motorized treadmill repeated ten times with 30 seconds of recovery between each sprint.[104] The authors demonstrated significant differences between the sexes; males had a greater peak power (+25%) and total work (+25%) than females. Once again (see section 1), the higher absolute scores in males versus females during MSE may be due to the typical sex difference in growth factors, anaerobic metabolism and the area occupied by type II muscle fibres.

Despite the greater initial sprint performance of the males (in the study by Brooks and colleagues[104] above), the fatigue index (based on work done) calculated from sprint 1 to sprint 10 was not significantly different between the sexes. One could imagine that females were less fit than males in this study, but not enough data were provided on the subjects' physical characteristics to support this assumption. However, another study using a similar protocol (10 × 5-second cycle sprints, 10 seconds of recovery) indicated that mean power decrement (absolute but not relative to leg volume) from the first to the tenth repetition was greater in teenage boys than girls (43.8 ± 7.5% vs 33.9 ± 7.9%, respectively; $p < 0.05$).[59] In addition, males were found to perform better (both absolute and relative work) than females during five 6-second cycle sprints every 30 seconds, but experienced a greater work decrement than females (13.7 ± 5.1% vs 11.0 ± 2.8%, respectively; $p < 0.05$).[108] Thus, despite the limited research, it appears that males experience greater absolute decrements in performance during MSE. This may be related to the greater involvement of anaerobic glycolysis, due to the greater initial power output in males than in females, and hence subsequent inhibition of muscle glycolysis and contractile mechanisms during later sprints (see sections 1 and 2.2.1). The sex difference in the depletion rate of high-energy phosphate stores and the reduction in central drive may also contribute to the difference in fatigue resistance in MSE, but has not yet been investigated. Finally, a greater aerobic contribution to energy supply in females would be beneficial to PCr resynthesis during recovery periods and to the maintenance of high ATP resynthesis rates during the final sprints.

Esbjörnsson-Liljedahl and colleagues[19,36,54] have also demonstrated significantly higher peak (+30%) and mean (+28%) power output and greater fatigue in males versus females during a repeated-sprint protocol consisting of repeated 30-second cycle sprints interspersed with 20 minutes of rest. Indeed, during three 30-second Wingate tests separated with 20 minutes of rest, a decline in power output among the three sprints was reported in males (8%; $p < 0.05$) but not in females (4%; p = NS). The results of this study suggest, therefore, that females have a greater ability to restore power between prolonged sprints separated by long recovery periods.

3. Sex Differences in Physiological Responses to Sprint Exercise Reanalysed

When comparing skeletal muscle fatiguability between males and females, there are some methodological confounds that may affect the interpretation of the results. For example, difficulties arise when attempting to match the sexes for absolute power output, relative power output and training background. This has contributed to the inability to definitively establish sex differences in muscle metabolism, the degree of fatigue development, and the rate of impairment of possible contributing mechanisms. The following section therefore demonstrates how this methodological issue affects sprint exercise, and proposes a different interpretation of the current sprint literature.

3.1 Methodological Concerns

Current research suggests that it is important to consider differences in absolute performance (and training background) when investigating the sex difference in metabolism, performance and fatigue. This appears particularly important during MSE, where a correlation between the initial

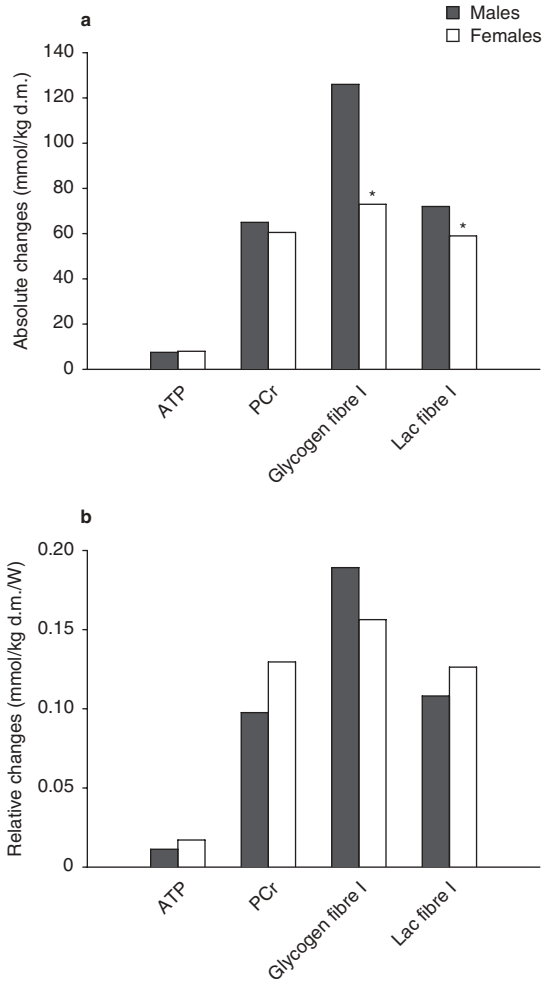

Fig. 3. (**a**) Absolute and (**b**) relative (relative to mean power output) changes in muscle metabolism during a maximal 30-second sprint on a cycle ergometer in males and females (adapted from Esbjörnsson-Liljedahl et al.,[54]). **d.m.** = dry muscle; **Lac** = lactic; **PCr** = phosphocreatinine. * significant sex difference ($p < 0.05$) as reported by the authors.

mechanical score and performance decrement over subsequent sprints has consistently been reported. Indeed, the percentage decrement in mechanical output during MSE has been reported to be positively correlated with initial sprint performance ($0.57 < r < 0.89$; $p < 0.05$).[48,59,211] Accordingly, Yanagiya et al.[59] explained the greater fatiguability in teenage boys during an MSE test (10×6 seconds, 30-second recovery) via the observation that boys developed higher power levels than age-matched girls from the beginning of the series. A deeper examination of the studies conducted by Billaut et al.[206] and Gaitanos et al.[132] also shows that the higher the performance reached in the first sprint during a series of ten all-out bouts, the greater the decline in power output across the ten sprints. Interestingly, Gaitanos and coworkers[132] additionally reported a correlation between the total work done over the first five sprints and the increase in blood lactate concentration ($r = 0.88$; $p < 0.05$), and a correlation between blood lactate concentration and power output decrement ($r = 0.82$; $p < 0.05$). Thus, the greater fatiguability reported in males during sprint tasks may be related

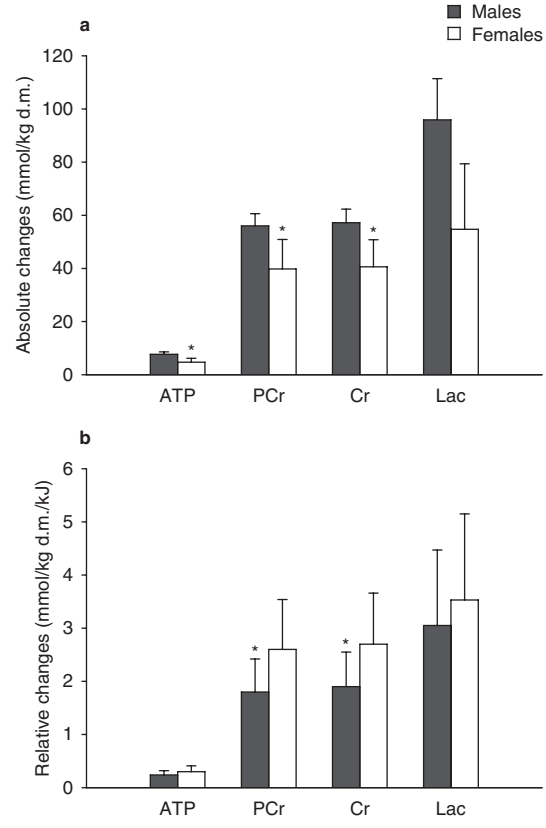

Fig. 4. (**a**) Absolute and (**b**) relative (per kJ of total mechanical work) changes in muscle metabolism of males and females during multiple-sprint exercise. When expressed per kJ of total mechanical work, the greatest metabolite changes appear in females (adapted from Bishop et al.[108]). **Cr** = creatinine; **d.m.** = dry muscle; **Lac** = lactic; **PCr** = phosphocreatinine. * significant sex difference ($p < 0.05$) as reported by the authors.

to the greater initial sprint performance rather than actual sex differences.

3.2 Influence of Total Mechanical Work

A deeper examination of the results obtained during repeated supramaximal sprints reveals that many of the differences in metabolism and performance between males and females are likely to be due to sex differences in the absolute work performed during the actual task. For example, the greater the work performed during a sprint, or a series of sprints, the greater the changes in muscle substrates and hormonal responses.[58,108] Thus, it would be logical to observe greater metabolic and hormonal disturbances in males, and hence greater performance decrements, as they typically perform more work during a given exercise. It is then conceivable that this 'methodological confound' might contribute to reported sex differences in skeletal muscle metabolism (i.e. substrate depletion and hormonal responses) and subsequent performance reported during sprint exercises.

In type I fibres, the exercise-induced glycogen reduction during a 30-second cycle sprint has been reported to be 42% smaller in females than in males, and this was associated with a 22% smaller increase in blood lactate concentration in females.[54] However, a closer look at the results of Esbjörnsson-Liljedahl et al.[54] shows that, when expressed relative to mean power output (MPO), the impact of sex on glycogen changes is strongly reduced, and relative changes in muscle lactate concentration, actually become higher in females than in males (figure 3). Interestingly, the same observations can be made from the data collected in another study by the same group.[36] The authors reported that three 30-second cycle sprints with 20 minutes of recovery induced a smaller reduction of ATP (absolute values) in females than in males in type II muscle fibres (48% vs 62%, respectively; $p < 0.05$). However, once again, males developed higher power output levels throughout the sprint bouts, and a closer examination of those data demonstrates that sex differences in ATP concentration changes after sprints are nullified when expressed relative to MPO. Thus, the higher absolute changes for ATP after exercise in males seem almost completely reduced when values integrate power output level as a covariate. These results are consistent with those of Gaitanos et al.,[132] who reported a strong correlation between work done during a 6-second sprint and changes in metabolites.

These analyses highlight the role of the work performed during exercise to account for the reported sex differences in performance, muscle metabolism and hormonal responses during repeated-sprint exercise. The greater perturbations observed in males might come from the fact that males actually perform significantly more mechanical work than females during a given sprint. This in turn is likely to lead to greater decrements in performance, as Gaitanos and colleagues[132] have shown that performance decrement is related to anaerobic metabolism during the first sprint. Furthermore, the validity of comparing powerful with less powerful subjects (males and females, respectively, in this review) has frequently been questioned, as a greater initial sprint performance is positively correlated with a greater performance decrement.[48,59,132] This raises the possibility that the often-observed sex differences in fatiguability may actually be due to inappropriate comparison methods rather than actual sex differences.

Rather, it is likely that if body dimensions and initial maximal performance are satisfactorily covaried, the changes in muscle metabolism and fatigue associated with sprint activity will largely depend on the absolute mechanical work performed by subjects. Such a methodological confound has previously been highlighted by our group in abstract form.[108] We investigated the sex difference in muscle metabolism during a MSE consisting of five all-out sprints lasting 6 seconds repeated every 30 seconds. Both absolute (kJ) and relative (J/kg) work values were greater in males than in females. As expected, the work decrement (%) over five sprints was greater in males (males 13.7 ± 5.1%, females 11.0 ± 2.8%; $p < 0.05$). The sprints were accompanied by greater absolute changes in ATP, PCr, creatine and lactate concentrations in males than in

females (figure 4). However, when expressed in relative terms (i.e. per kJ of work), only sex differences in PCr and creatine persisted – but were inverted. Sex differences in muscle metabolism then appeared to be largely due to differences in the absolute work performed by males and females.[108] Thus, these results suggest that unless males and females are matched for total work (or total work is used as a covariate), it is very difficult to compare decrements in performance during repeated sprint exercise, as differences in hormonal and metabolic responses are likely to affect the development of fatigue.

4. Summary and Future Directions

The investigation of skeletal muscle fatigue in males and females must be made with appropriate comparison methods. Studies of the fatiguability and metabolic and hormonal responses of males and females during MSE have, in our opinion, not been optimally designed to control for possible covariates. Rather than taking into account only the strength or power output capacity of the rested muscle, studies dedicated to understanding sex differences should incorporate the total mechanical work done by each sex during exercise as a covariate. Furthermore, studies are warranted where males and females are matched for both initial power and activity levels.

Although it is tempting to propose that males are more susceptible to fatigue than females for a given sprint, we must emphasize the need for more basic research comparing exercise tolerance between the sexes. In addition, greater investigation of the influence of initial force on the mechanisms of fatigue in males versus females is needed. It is encouraging to see a number of studies using advanced techniques to analyse muscle fatigue aetiology under a variety of conditions. It is probable, however, that our uncertainty surrounding the understanding of the sex difference in muscle fatigue stems from scientists from diverse specialist fields having taken a local approach, in an attempt to solve the underlying cause of fatigue. Multiple-sprint activity requires bouts of all-out intensity to be repeated several times with incomplete recovery. The potential sex difference in voluntary activation of active muscles has not been sufficiently examined during sprint activity; the greater impairment of central drive in males during discrete tasks (e.g. contraction of dorsiflexor and elbow flexor muscles) may also be found during whole-body sprinting. Furthermore, the use of transcranial magnetic stimulation would be a powerful complement for investigating the contribution of supraspinal fatigue to task failure during repeated sprints in males and females.

Acknowledgements

No sources of funding were used to assist in the preparation of this review. The authors have no conflicts of interest that are directly relevant to the content of this review.8pt? >

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