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Muscle metabolism and performance improvement after two training programmes of sprint running differing in rest interval duration

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Abstract
Repeated-sprint training often involves short sprints separated by inadequate recovery intervals. The effects of interval duration on metabolic and performance parameters are unclear. We compared the effects of two training programmes, differing in rest interval duration, on muscle (vastus lateralis) metabolism and sprint performance. Sixteen men trained three times a week for 8 weeks, each training session comprising 2–3 sets of two 80-m sprints. Sprints were separated by 10 s ($n = 8$) or 1 min ($n = 8$). Both training programmes improved performance in the 100-, 200-, and 300-m sprints, but the improvement was greater in the 10-s group during the final 100 m of the 200- and 300-m runs. Independent of interval duration, training mitigated the drop of muscle ATP after two 80-m sprints. The drop in phosphocreatine and the increases in glucose-6-phosphate and fructose-6-phosphate after two 80-m sprints were greater in the 10-s group. In conclusion, training with a limited number of repeated short sprints ($\leq$10 s) may be more effective in improving speed maintenance in 200- and 300-m runs when performed with a 1:1 rather than a 1:6 exercise-to-rest ratio. This may be due to a greater activation of glycolysis caused, in part, by the limited resynthesis of phosphocreatine during the very short rest interval.

Keywords: *ATP, carbohydrate metabolism, metabolites, phosphocreatine, speed endurance*

Introduction

Repeated-sprint training increases fatigue resistance and causes several metabolic adaptations (Iaia et al., 2009; Jacobs, Esbjörnsson, Sylvén, Holm, & Jansson, 1987; Mohr et al., 2007). Repeated-sprint training involves short sprints (up to 15 s) separated by brief rest or low-activity intervals (up to 1 min) that are inadequate for the complete resynthesis of phosphocreatine, a major energy substrate in brief high-intensity exercise (Dawson et al., 1997). The scientific rationale behind this kind of training is to cause such perturbations to the muscle metabolic milieu and ion homeostasis (Mohr et al., 2007) as to elicit favourable adaptations of the ATP-phosphocreatine and lactate systems, which provide most of the energy for sprinting (Gastin, 2001).

A possible key variable determining the adaptations caused by repeated short-sprint training is the duration of the recovery interval, or the ratio of sprint to recovery time. Most sprint training studies have employed work-to-recovery ratios ranging from 1:3 to 1:11 (Dawson et al., 1998; Ferrari Bravo et al., 2008; Hellsten-Westing, Balsom, Norman, & Sjödin, 1993a; Hill-Haas, Coutts, Rowsell, & Dawson, 2009; Linossier, Denis, Dormois, Geyssant, & Lacour, 1993). By keeping the sprint time short (<6 s), while at the same time providing a rest interval that is over ten-fold the exercise time, sprint performance can be maintained for several repetitions, and metabolic disturbances are milder (Balsom, Seger, Sjödin, & Ekblom, 1992a, 1992b).

Training for 6–8 weeks with sprints lasting 3–10 s and with work-to-recovery ratios between 1:4 and 1:11 typically increases sprint performance and fatigue resistance, accompanied by a variety of changes in muscle metabolites, proteins, and morphology (Dawson et al., 1998; Esbjörnsson, Hellsten-Westing, Balsom, Sjödin, & Jannson, 1993; Harridge et al., 1998; Hellsten-Westing et al., 1993a; Hellsten-Westing, Norman, Balsom, & Sjödin,

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1993b; Linossier et al., 1993; Linossier, Dormois, Geyssant, & Denis, 1997a; Linossier et al., 1997b; Mohr et al., 2007; Ørtenblad, Lunde, Levin, Andersen, & Pedersen, 2000; Russell et al., 2003; Thorstensson, Sjödin, & Karlsson, 1975). However, to our knowledge, no study has examined the effects of the duration of the recovery interval on metabolic and performance parameters when training with repeated short sprints. Moreover, there are no data on repeated short sprints ($\leq$10 s) with very short recovery intervals (work-to-recovery ratio of 1:1). We therefore hypothesized that a very short recovery interval would elicit greater metabolic disturbances, thus providing a stronger stimulus for adaptations, compared with a longer recovery interval during a short period of sprint training. By measuring both acute and chronic muscle metabolic responses to repeated short sprints ($\leq$10 s), we hoped to contribute to the limited knowledge regarding this issue, since only two studies that we are aware of have measured acute changes following such an exercise protocol (Linossier et al., 1993; Mohr et al., 2007). Thus, the aim of this study was to explore resting and post-exercise differences in muscle metabolism and 100–300 m sprint performance between two short-sprint training programmes, one with a very short (10 s) and one with a longer (1 min) rest interval between repeated sprints.

Methods

Participants

Sixteen healthy, physically active male physical education students, attending university classes that offered moderate sporting activity, provided written informed consent to participate in the study. The study protocol was approved by the University of Thessaloniki Medical School ethics committee, and all procedures were in accordance with the Helsinki Declaration of 1975.

Study design

The training intervention lasted 8 weeks and comprised three sessions per week (a total of 24 sessions). Sprint performance testing took place in the week before and the week after training. The pre-training performance tests were preceded by 2 weeks of pre-conditioning. Muscle biopsy was performed before and after exercise during the first and 24th training sessions. During the study period, the participants continued to attend university classes offering moderate sporting activity, as they had been doing for the previous 5 months. Other than that and the experimental protocol, they were not involved in any organized physical activity.

Pre-conditioning

In preparation for the highly demanding performance tests and training programmes, the participants underwent 2 weeks of pre-conditioning including four training sessions of similar content to the subsequent experimental sessions but at 20–30% lower intensity.

Sprint performance testing

During the week following pre-conditioning, the participants performed three running tests of 100, 200, and 300 m on an outdoor track. Run time was measured electronically using pairs of photocells placed at intervals of 50 m for the 100-m run, and 100 m for the 200- and 300-m runs. The photocells were connected to an electronic chronometer measuring thousandths of a second. The same tests were repeated during the week after training. The coefficient of variation of the measurements ranged from 1.2 to 1.9%.

Training

A randomized, parallel matched-group design was used. On the basis of performance in the initial tests, body mass, and height, the participants were divided into two equivalent groups with the following characteristics (all mean $\pm$ s_x): Group 1 – age 20.9 $\pm$ 0.5 years, body mass 70.9 $\pm$ 2.4 kg, height 1.79 $\pm$ 0.01 m; Group 2 – age 20.1 $\pm$ 0.5 years, body mass 71.3 $\pm$ 2.5 kg, height 1.80 $\pm$ 0.02 m. These groups were then randomly assigned to one of two training programmes lasting 8 weeks with three training sessions per week. Each training session included two (during the first 4 weeks) or three sets (during the second 4 weeks) of two 80-m sprints run on an indoor track. The two sprints in each set were separated by either 10 s (Group 1) or 1 min (Group 2) of rest. In both groups, sets were separated by 20 min of rest. This small number of spints in each set and long rest interval between sets was chosen to keep the contribution of the ATP-phosphocreatine and lactate systems high and minimize the aerobic contribution to the energy supply (Bogdanis, Nevill, Boobis, & Lakomy, 1996; Gaitanos, Williams, Boobis, & Brooks, 1993). By doing so, sprints were run at high speed and quality was maintained.

Dietary control

To control for the effect of diet on substrate utilization during exercise, the participants followed standard dietary plans during the 2 days preceding muscle biopsy. The plans provided 50% of energy

from carbohydrate, 35% from fat, and 15% from protein. On the morning before biopsy, the participants ate a standardized meal at least 3 h before exercising.

Muscle biopsy

Muscle samples were obtained by needle biopsy (Bergström, 1975) from the vastus lateralis muscle of a randomly selected leg before and 36 ± 2 s after the first double 80-m sprint during the first and last training sessions. Samples were immediately immersed in liquid nitrogen and stored until lyophilized within 2 days. Subsequently, samples were stored at −80°C. Two participants, one from each group, declined to provide muscle samples. Thus, muscle metabolite data are for seven participants per group.

Muscle metabolite assays

The lyophilized muscle samples were dissected free of connective tissue and blood, and they were homogenized to a fine powder, which was then extracted and analysed for ATP, ADP, phosphocreatine, creatine, glucose, glucose-1-phosphate, glucose-6-phosphate, fructose-6-phosphate, glycerol-3-phosphate, pyruvate, and lactate as described previously (Harris, Hultman, & Nordesjö, 1974). The ATP, ADP, phosphocreatine, creatine, glucose-1-phosphate, glucose-6-phosphate, fructose-6-phosphate, glycerol-3-phosphate, and pyruvate data of each participant were corrected to the individual's mean total creatine (phosphocreatine + creatine) to account for errors arising from the variable inclusion of any remaining connective tissue, fat or blood in the samples (Harris et al., 1974).

Statistical analysis

Results are reported as means ± standard errors (s_x). Performance data were compared by two-way analysis of variance [ANOVA: rest interval (10 s, 1 min) × training (pre, post)] with repeated measures on training. Muscle metabolite data were compared by three-way ANOVA [rest interval × training × acute exercise (pre, post)] with repeated measures on training and acute exercise. Statistical significance was set at $P < 0.05$. The effect size (ES) for main effects and interactions was estimated by calculating partial η^2 using SPSS v.17. Effect sizes were classified as small (0.2), medium (0.5) or large (≥0.8).

Results

Body mass did not change significantly after training (from 70.9 ± 2.4 to 70.6 ± 2.6 kg in the 10-s group and from 71.3 ± 2.5 to 71.9 ± 2.4 kg in the 1-min group).

Sprint performance

Sprint training improved performance during the 100-, 200-, and 300-m tests ($P < 0.001$, ES = 0.84–0.91, large) by an average of 5.6% in the 10-s group (from 12.55 ± 0.22 s to 11.86 ± 0.14 s, from 26.24 ± 0.47 s to 24.74 ± 0.31 s, and from 41.80 ± 0.78 s to 39.79 ± 0.66 s, respectively) and 4.9% in the 1-min group (from 12.94 ± 0.18 s to 12.31 ± 0.20 s, from 27.30 ± 0.52 s to 26.10 ± 0.60 s, and from 43.69 ± 0.95 s to 42.03 ± 0.71 s, respectively), as shown in Figure 1. The improvement was greater in the 10-s group than the 1-min group during the final 100 m of the 200-m run (1.00 ± 0.13 s vs. 0.66 ± 0.09 s $P = 0.043$; ES = 0.26, small) and during the final 100 m of the 300-m run (1.17 ± 0.15 s vs. 0.52 ± 0.12 s, $P = 0.005$; ES = 0.45, medium), as shown by the significant interval × training interactions for these split times. Training also improved performance during the first set of 80-m sprints when the muscle biopsy was performed ($P < 0.05$; Table I).

Muscle metabolites

Table II shows the muscle metabolite contents in each group before and after the first set of two 80-m sprints during the first and last training sessions. ATP decreased acutely with exercise ($P = 0.006$; ES = 0.48, medium), but training mitigated the drop in the muscle ATP content (from 17 ± 4% to 1 ± 3%, $P = 0.001$; ES = 0.79, medium) (Figure 2a). Phosphocreatine also decreased with exercise ($P < 0.001$), the decrease being greater in the 10-s than in the 1-min group (52 ± 3% vs. 35 ± 4%, $P = 0.007$; ES = 0.72, medium) (Figure 2b). Changes in creatine were the opposite of those for phosphocreatine.

Glucose tended to increase with acute exercise ($P = 0.078$; ES = 0.24, small). Glucose-1-phosphate increased significantly with exercise ($P = 0.026$; ES = 0.35, small). Glucose-6-phosphate also increased with exercise ($P < 0.001$; ES = 0.77, medium), with the increase in the 10-s group (on average, by 8.3 mmol · kg^{-1}) being higher than in the 1-min group (3.6 mmol · kg^{-1}), resulting in a significant interval × exercise interaction ($P = 0.027$; ES = 0.35, small) (Figure 3a). Frucose-6-phosphate exhibited the same response, i.e. an increase with exercise ($P < 0.001$; ES = 0.68, medium) and an interval × exercise interaction ($P = 0.017$; ES = 0.39, small) owing to the increase in the 10-s group (on average, by 1.5 mmol · kg^{-1}) being higher than that in the 1-min group (0.4 mmol · kg^{-1}) (Figure 3b).

Glycerol-3-phosphate increased acutely with exercise ($P = 0.019$; ES = 0.38, small); moreover,

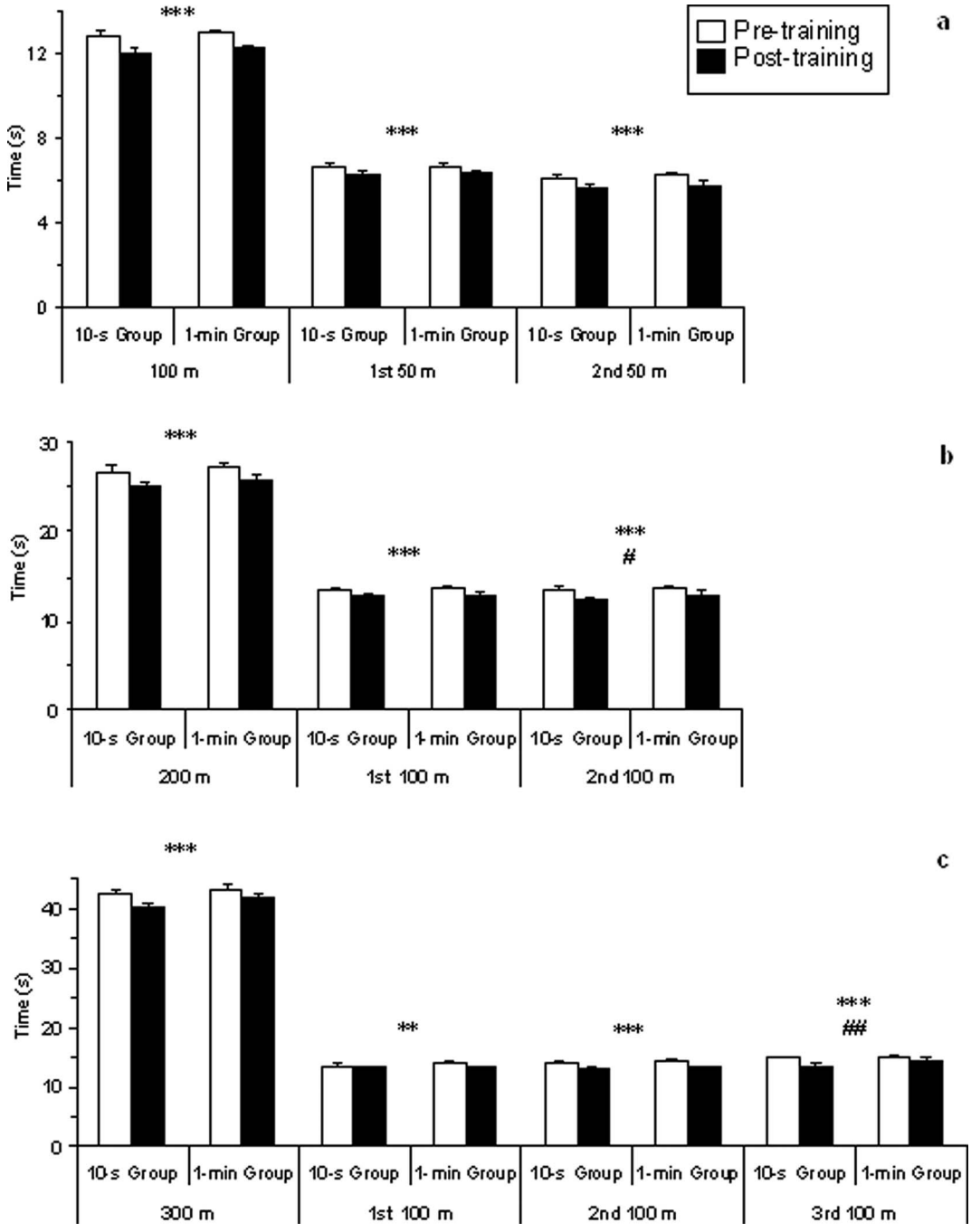

Figure 1. Run times on the 100-m (a), 200-m (b), and 300-m (c) performance tests before training and after training in the 10-s and 1-min groups. Split times are also shown for the performance tests. Error bars represent standard errors ($n = 8$ per group). $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$: main effect of training. $^{\#}P < 0.05$, $^{\#\#}P < 0.01$: interaction of rest interval and training due to a greater decrease in run time in the 10-s group than in the 1-min group with training.

Table I. Run times (seconds) for the two groups during the first set of two 80-m sprints, pre- and post-training (mean $\pm$ s_x).

	10-s group ($n = 8$)		1-min group ($n = 8$)	
	Pre-training	Post-training	Pre-training	Post-training
First 80 m*	9.83 ± 0.17	9.75 ± 0.15	10.22 ± 0.16	10.11 ± 0.12
Second 80 m***	10.98 ± 0.20	10.79 ± 0.17	10.61 ± 0.16	10.45 ± 0.15

Main effect of training: $^{*}P < 0.05$, $^{***}P < 0.001$.

Table II. Muscle metabolite contents (mmol · kg^{-1} dry muscle) for the two groups, before and after two 80-m sprints, pre- and post-training (mean ± s_x).

	10-s group ($n=7$)				1-min group ($n=7$)			
	Pre-training		Post-training		Pre-training		Post-training	
Metabolite	Pre-exercise	Post-exercise	Pre-exercise	Post-exercise	Pre-exercise	Post-exercise	Pre-exercise	Post-exercise
ATP[a, b]	19.8 ± 0.9	17.9 ± 1.2	20.9 ± 1.0	20.8 ± 1.4	22.8 ± 1.5	17.0 ± 0.6	20.8 ± 1.6	20.4 ± 1.4
ADP	3.5 ± 0.5	4.0 ± 0.4	3.4 ± 0.4	3.4 ± 0.4	2.7 ± 0.4	2.2 ± 0.2	2.7 ± 0.5	2.8 ± 0.7
Phosphocreatine[a, c]	53.9 ± 5.3	22.6 ± 2.1	58.1 ± 5.4	30.2 ± 2.7	58.6 ± 4.8	39.1 ± 4.7	59.0 ± 3.9	37.0 ± 3.2
Creatine[a, c]	48.3 ± 4.5	78.9 ± 4.1	43.5 ± 5.4	71.4 ± 4.7	48.9 ± 3.4	69.0 ± 3.3	47.9 ± 4.4	68.8 ± 5.0
Glucose	3.3 ± 0.5	4.3 ± 0.3	2.7 ± 0.5	3.4 ± 0.7	3.7 ± 0.9	5.1 ± 0.6	3.5 ± 0.5	6.4 ± 2.9
Glucose-1-phosphate[a]	0.6 ± 0.2	0.9 ± 0.2	0.6 ± 0.2	0.9 ± 0.2	0.6 ± 0.2	0.6 ± 0.1	0.4 ± 0.1	0.6 ± 0.1
Glucose-6-phosphate[a, c]	4.4 ± 1.1	12.3 ± 2.4	3.3 ± 0.7	12.1 ± 1.4	3.1 ± 0.9	7.8 ± 1.2	5.0 ± 1.5	7.5 ± 0.7
Fructose-6-phosphate[a, c]	0.7 ± 0.2	2.2 ± 0.5	0.6 ± 0.1	2.0 ± 0.3	1.0 ± 0.5	1.5 ± 0.2	1.1 ± 0.3	1.4 ± 0.1
Glycerol-3-phosphate[a, d]	2.3 ± 0.3	5.6 ± 1.0	2.7 ± 0.5	3.7 ± 0.6	1.9 ± 0.3	3.0 ± 0.3	4.0 ± 1.2	4.1 ± 0.8
Pyruvate	2.3 ± 0.3	3.0 ± 0.6	1.8 ± 0.5	3.1 ± 0.7	2.8 ± 0.9	4.2 ± 2.0	4.3 ± 2.0	3.5 ± 1.0
Lactate[a]	16.7 ± 4.4	44.2 ± 8.1	13.9 ± 3.5	46.3 ± 6.2	10.8 ± 3.4	35.2 ± 6.7	17.3 ± 4.8	38.9 ± 5.0

[a]Main effect of exercise. [b]Training × exercise interaction. [c]Interval × exercise interaction. [d]Interval × training interaction (all $P < 0.05$).

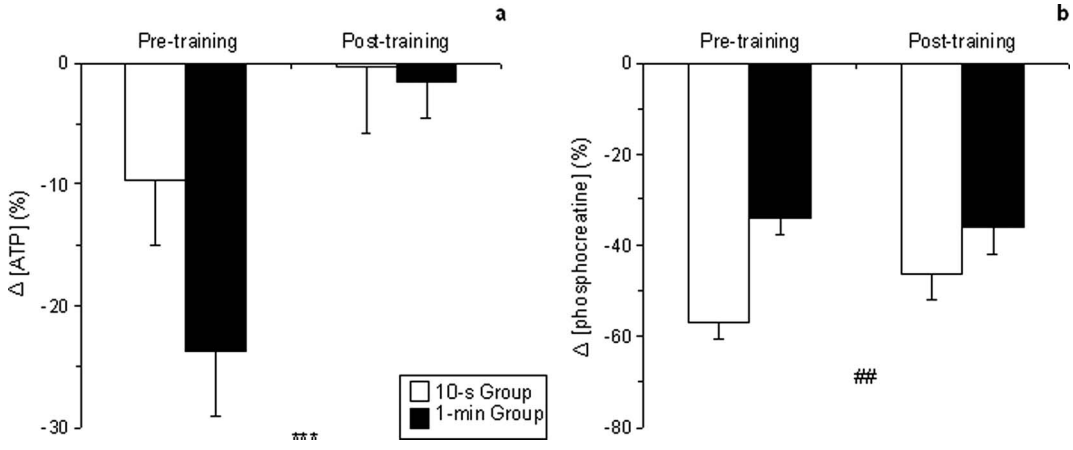

Figure 2. Percentage changes in the muscle ATP (a) and phosphocreatine (b) contents after two 80-m sprints in the 10-s and 1-min groups, pre- and post-training. Error bars represent standard errors ($n=7$ per group). ***Main effect of training ($P=0.001$). ##Main effect of rest interval ($P=0.007$).

it exhibited an interval × training interaction ($P=0.038$; ES = 0.31, small), which is explained by the fact that training caused an overall decrease in the 10-s group (on average, from 4.0 to 3.2 mmol · kg^{-1}), as opposed to an overall increase in the 1-min group (from 2.5 to 4.0 mmol · kg^{-1}). Finally, lactate increased with exercise ($P < 0.001$; ES = 0.85, large). Although the post-exercise values in the 10-s group were numerically higher than those in the 1-min group, this difference did not reach statistical significance.

Discussion

We have presented performance and metabolic responses to two repeated short-sprint training programmes differing in exercise-to-rest ratio, i.e. 1:1 (in the 10-s group) versus 1:6 (in the 1-min group). The originality of our study lies primarily in that previous studies examining repeated short-sprint training (≤10 s) have employed recovery intervals 3- to 11-fold greater than the exercise time (Dawson et al., 1998; Esbjörnsson et al., 1993; Ferrari Bravo et al., 2008; Harridge et al., 1998; Hellsten-Westing et al., 1993a, 1993b; Hill-Haas et al., 2009; Linossier et al., 1993, 1997a, 1997b; Mohr et al., 2007; Ørtenblad et al., 2000; Russell et al., 2003; Thorstensson et al., 1975). In fact, given the importance of the duration of recovery between repeated sprints for determining the kind of adaptations to training (Billat, 2001; Ross & Leveritt, 2001), it was rather surprising to find a lack of research on short, repeated-sprint

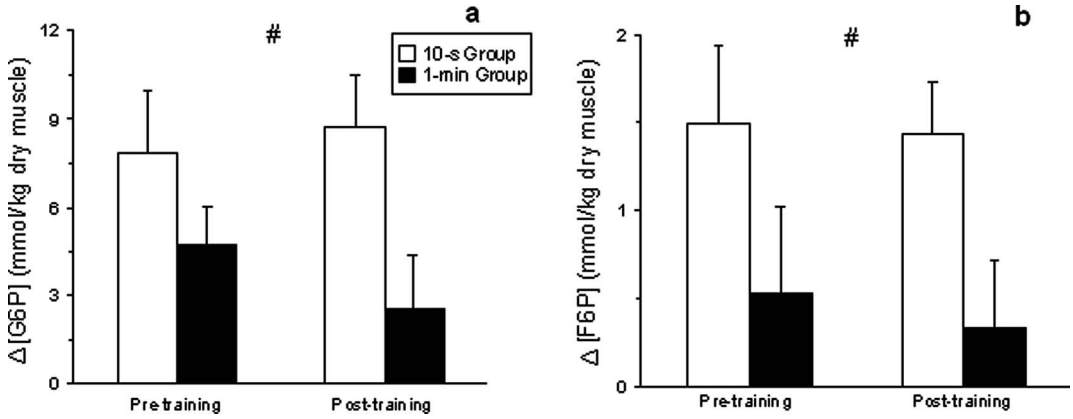

Figure 3. Changes in muscle glucose-6-phosphate, G6P (a) and fructose 6-phosphate, F6P (b) after two 80-m sprints in the 10-s and 1-min groups before and after training. Error bars represent standard errors ($n = 7$ in each group). #Main effect of rest interval ($P < 0.05$).

training modes such as the ''1:1'', which is applied in sprint training to improve speed endurance maintenance.

The finding of increased sprint performance after training in the 1-min group is in accordance with the majority of studies on intermittent sprint training with long recovery intervals (Dawson et al., 1998; Esbjörnsson et al., 1993; Harridge et al., 1998; Hellsten-Westing et al., 1993a, 1993b; Linossier et al., 1993, 1997a, 1997b; Mohr et al., 2007; Ørtenblad et al., 2000; Russell et al., 2003; Thorstensson et al., 1975). However, the present study showed that training with short ($\leq$10 s) sprints was more effective in improving speed maintenance when performed with a very short exercise-to-rest ratio (1:1) than with a longer ratio (1:6). A major difference of this study compared with all previous ones was the very low training volume. In this sense, it is remarkable that a total sprint distance of 9.6 km and a time of 20 min over a period of 8 weeks were so effective in increasing sprint performance by about 5%, at least in physically active individuals. This increase is comparable to that found in similar studies with short ($\leq$10 s) sprints (Dawson et al., 1998; Mohr et al., 2007; Russell et al., 2003; Thorstensson et al., 1975), in which improvements in sprint time ranged from 1 to 6%. Unfortunately, the demands of this study were such that they prohibited the recruitment of athletes (as in most studies of this type), in whom the results could have been slightly different. The greatest effect of training with the very short compared with the longer interval was not on acceleration or peak speed but on what coaches refer to as speed endurance, i.e. prolonging the time during which a high running speed can be maintained. This was evident by the shorter run times over the final 100 m of the 200- and 300-m sprints in the 10-s versus the 1-min group. Thus, when using very few repetitions of short repeated sprints ($\leq$10 s), short rest periods (less than three times the exercise duration) may be more beneficial than longer rest periods in maintaining speed in a 200–300 m run. On the other hand, a number of studies employing longer exercise and rest periods (Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005; Iaia et al., 2008) observed pronounced improvement in repeated high-intensity exercise and it cannot be ruled out that with a higher number of repetitions performed one after the other (as done in most similar studies), the 1:6 protocol may be as effective as the 1:1 protocol.

Possible explanations for the training-induced increase in sprint performance of both groups and for the higher speed endurance of the 10-s group can be traced to the muscle metabolite data presented in Table II, Figure 2, and Figure 3. The increase in sprint performance may be explained by the training-induced mitigation of the drop in muscle ATP content after two 80-m runs, suggesting that training augmented one or more of the mechanisms for ATP resynthesis. The better maintenance of speed after training with the 1:1 compared with the 1:6 exercise-to-rest ratio may be explained by the greater activation of glycolysis when the rest interval was short, as indicated by the larger increase in glycolytic intermediates. A greater activation of glycolysis may originate in the higher drop in muscle phosphocreatine in the 10-s than in the 1-min group after the two 80-m runs, apparently due to the very limited time available for phosphocreatine resynthesis (through aerobic ATP resynthesis from ADP and inorganic phosphate in the mitochondria) between runs. As a result, the 10-s group would have entered the second run with less phosphocreatine, less ATP, and more of ATP's degradation products (i.e. inorganic phosphate, ADP, and AMP). Higher inorganic phosphate (a substrate in glycogenolysis) and AMP (an allosteric activator of phosphorylase *b*), together with lower ATP (an inhibitor of phosphorylase *b* activation by AMP) would have resulted in higher

acceleration of glycogen breakdown throughout the second run (Greenhaff, Hultman, & Harris, 2004), and hence higher increases in its products. Indeed, all products of glycogen breakdown that were measured – glucose-1-phosphate, glucose-6-phosphate, fructose-6-phosphate, glycerol-3-phosphate (derived from the glycolytic intermediate, dihydroxyacetone phosphate), pyruvate, and lactate – had numerically higher increases with acute exercise in the 10-s group, although this was confirmed statistically only for glucose-6-phosphate and fructose-6-phosphate. It is thus possible that the greater glycolytic flow during training in the 10-s group resulted in adaptations that conferred an advantage in terms of ATP resynthesis during the final segments of the 200- and 300-m runs, when glycolysis is the predominant energy source (Gastin, 2001). However, other variables not assessed in the present study, such as an improved ion homeostasis (Iaia et al., 2008) and a difference in pH regulatory processes, may have played a role in the observed difference in performance after the two sprint training programmes assessed.

Some of the differences in muscle metabolism observed between the two groups may have been underestimated because of the time that elapsed between the cessation of exercise and the biopsy. This may have led to considerable muscle lactate release as well as ATP and phosphocreatine resynthesis.

It may be hypothesized that the improved sprint performance with intermittent sprint training is due to the increased activity of enzymes involved in at least one of the ATP-phosphocreatine, lactate, and oxygen systems. Mohr and co-workers (2007) have shown increased creatine kinase activity following sprint training. Enzymes of the lactate system, such as phosphorylase, hexokinase, phosphofructokinase and lactate dehydrogenase, increase after sprint training (Dawson et al., 1998; Hellsten-Westing et al., 1993b; Linossier et al., 1993, 1997a). However, a number of studies (Barnett et al., 2004; Iaia et al., 2008) have shown marked improvements is sprint and speed endurance performances without concomitant changes in the activity of anaerobic enzymes. Enzymes involved in aerobic energy production do not seem to increase after repeated short ($\leq$10 s) sprint training with an exercise-to-rest ratio greater than 1:4 (Dawson et al., 1998; Linossier et al., 1993, 1997a). Regarding our own research, we are currently in the process of obtaining a global view of muscle protein changes during intermittent sprint training through proteomic analysis. Preliminary findings point to changes in enzymes of all three energy systems and in structural proteins in both groups (Scigelova et al., unpublished data).

Conclusion

The main findings of the present study are as follows. First, intermittent sprint training with 2–3 sets of two very short (80 m, or 10 s) maximal runs separated by either 10 s or 1 min of rest (1:1 or 1:6 exercise-to-rest ratio), performed three times weekly for 8 weeks, increased performance in 80-, 100-, 200-, and 300-m runs in physically active young men. Second, the increase in performance with both programmes was due in part to an improvement in ATP resynthesis, as indicated by the mitigated drop in muscle ATP after a set of two 80-m runs. Third, the 1:1 training programme was more effective than the 1:6 programme in maintaining speed, since it resulted in a greater increase in running speed during the final 100 m of the 200- and 300-m runs. Fourth, this difference could be due, among other factors, to a greater activation of glycolysis caused in part by the limited resynthesis of phosphocreatine during the very short recovery period, which may have caused adaptations that enhanced the contribution of glycolysis to the energy supply during the final segments of the 200- and 300-m runs. Thus we conclude that, when employing very few repetitions of short sprints ($\leq$10 s), short rest periods (e.g. 1:1 exercise-to-rest ratio) are more beneficial than longer rest periods in improving speed maintenance during runs of 200–300 m.

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