

Review

Effects of sprint interval training on $\text{VO}_{2\text{max}}$ and aerobic exercise performance: A systematic review and meta-analysis

M. Sloth,¹ D. Sloth,² K. Overgaard,¹ U. Dalgas¹

¹Department of Public Health, Section of Sport Science, Aarhus University, Aarhus C, Denmark, ²Department of Economics and Business, Aarhus University, Aarhus C, Denmark

Corresponding author: Martin Sloth Pedersen, Department of Public Health, Section of Sport Science, Aarhus University, Dalgas Avenue 4, 8000 Aarhus C, Denmark. Tel: +45 87168614, Fax: +45 87150201, E-mail: MartinSloth25@gmail.com

Accepted for publication 21 May 2013

Recently, several studies have examined whether low-volume sprint interval training (SIT) may improve aerobic and metabolic function. The objective of this study was to systematically review the existing literature regarding the aerobic and metabolic effects of SIT in healthy sedentary or recreationally active adults. A systematic literature search was performed (Bibliotek.dk, SPORTDiscus, Embase, PEDro, SveMed+, and Pubmed). Meta-analytical procedures were applied evaluating effects on maximal oxygen consumption ($\text{VO}_{2\text{max}}$). Nineteen unique studies [four randomized controlled trials (RCTs), nine matched-controlled trials and six noncontrolled studies] were identified, evaluating SIT interventions lasting 2–8 weeks. Strong evidence support improvements of aerobic exercise performance and

$\text{VO}_{2\text{max}}$ following SIT. A meta-analysis across 13 studies evaluating effects of SIT on $\text{VO}_{2\text{max}}$ showed a weighted mean effects size of $g = 0.63$ 95% CI (0.39; 0.87) and $\text{VO}_{2\text{max}}$ increases of 4.2–13.4%. Solid evidence support peripheral adaptations known to increase the oxidative potential of the muscle following SIT, whereas evidence regarding central adaptations was limited and equivocal. Some evidence indicated changes in substrate oxidation at rest and during exercise as well as improved glycemic control and insulin sensitivity following SIT. In conclusion, strong evidence support improvement of aerobic exercise performance and $\text{VO}_{2\text{max}}$ following SIT, which coincides with peripheral muscular adaptations. Future RCTs on long-term SIT and underlying mechanisms are warranted.

Low-volume sprint interval training (SIT) is an interval training regime, which has recently received increased attention since studies have suggested that intervals of very high intensity, short duration (≤ 30 s) and relatively long recovery periods between intervals (~ 4 min) may induce improvements in aerobic power and metabolic function resembling those of traditional endurance training (Gibala et al., 2006; Burgomaster et al., 2008). These findings are of significant interest, since SIT may represent a time-efficient and alternative approach to improve aerobic performance and metabolic functioning.

SIT differs with respect to volume and intensity from more traditional approaches to aerobic training, ranging from prolonged submaximal endurance training to high-intensity interval training (HIT). These types of aerobic training are also well known to improve aerobic fitness and overall health through a range of both central and peripheral adaptations (Laursen & Jenkins, 2002). In particular, HIT has become a popular exercise mode since it effectively challenges the aerobic energy system eliciting rapid aerobic improvements even in highly trained people. HIT can be performed in many ways but is usually defined as “Brief to moderate duration inter-

vals with high intensity (above anaerobic threshold) separated by brief periods of low intensity exercise or rest to allow some but normally not full recovery” (Laursen & Jenkins, 2002). It could be argued that SIT can be classified as a form of HIT training in the highest end of the intensity spectrum. However, in the present review, we will only address SIT defined as training bouts with short duration (10–30 s) and all-out effort interspersed with long recovery periods.

The main adenosine triphosphate (ATP) resynthesis during SIT exercise bouts would be expected to rely on predominantly anaerobic metabolism (McCartney et al., 1986), whereas traditional endurance training and most forms of HIT predominantly relies on aerobic metabolism. Nonetheless, it seems that SIT induces many of the same aerobic and metabolic adaptations as traditional endurance exercise.

Many of the muscle adaptations involved in improved aerobic and metabolic function (increased enzyme capacity, mitochondrial biogenesis, and increased glucose transporter GLUT4, etc.) have been associated with improved glycemic control and insulin sensitivity and therefore, the potential effect of SIT on these metabolic adaptations will also be addressed in this review

(Hughes et al., 1993; Simoneau et al., 1995; Simoneau & Kelley, 1997; Heilbronn et al., 2007).

In order to provide a systematic review meta-analysis establishing general patterns of the literature on the effects of SIT, the purpose of the present study was to (i) systematically review the results of the existing scientific literature concerning aerobic performance and metabolic adaptations to SIT, (ii) apply meta-analytical procedures for evaluation of possible effects of SIT on $\text{VO}_{2\text{max}}$ and (iii) identify possible underlying mechanisms responsible for these adaptations.

Methods and materials

The present report includes both a systematic literature review on the aerobic effects of SIT and a meta-analysis evaluating the effect on $\text{VO}_{2\text{max}}$.

Systematic literature search

Before performing the systematic literature search, a definition of SIT and some further inclusion criteria were defined.

Definition of SIT

The term sprint training covers many different types of training interventions applying various exercise intensities, durations of bouts, work/recovery relationships, and total work volumes (Ross & Leveritt, 2001). However, for this review, a definition of a SIT intervention was formulated and served as inclusion criteria.

In terms of duration of each bout, intensity, recovery between bouts, and total volume of the SIT intervention (per session) had to meet the following definition:

- Duration of bouts: 10–30 s.
- Intensity: maximal, “all-out.”
- Volume: ≤ 12 repetitions.
- Recovery: ≥ 5 times the duration of work.

Bout durations of less than 10 s were excluded based on the fact that bouts lasting less than 10 s are considered more anaerobic dependent (Linossier et al., 1993, 1997; Bogdanis et al., 1998) compared to the longer bouts (Bogdanis et al., 1995, 1996) and studies using very brief sprint bouts have reported no change in mitochondrial enzymes (Linossier et al., 1993; Dawson et al., 1998). An upper cutoff of 30 s was based on the assumption that longer duration bouts would result in substantially increased aerobic contribution to the ATP turnover (Withers et al., 1991) and be associated with a marked drop in intensity. Thus, it could not in our opinion be categorized as low-volume SIT. Intensity was defined based on the general conception that a sprint is an “all-out effort (or near)” (Ross & Leveritt, 2001). Volume and recovery were defined to ensure low total training volume and ability to maintain high power output during all sprint bouts in a training session.

Inclusion criteria

To focus the literature selection according to the research questions and to ensure comparability among the included studies, some inclusion criteria regarding study design, study duration, and fitness level of the participants were formulated before the search.

Only studies using a longitudinal design evaluating interventions consisting of solely SIT lasting of ≥ 2 weeks were included.

Only studies on humans were included and the population of interest was limited to healthy adults who were either sedentary or engaged in recreational activities 2–3 times per week having an average baseline $\text{VO}_{2\text{max}}$ of $\leq 55 \text{ mL} \times \text{kg}^{-1} \times \text{min}^{-1}$.

Finally, Randomized controlled trials (RCTs) as well as matched-controlled trials (MCTs) and noncontrolled designs were included.

Search procedure

A comprehensive literature search of six different databases (PubMed, Embase, Bibliotek.dk, SPORTDiscus, SveMed+, and PEDro) was performed on May 7, 2012. The search aimed to identify studies investigating the effects of SIT on aerobic and metabolic function published from January 1, 1995 to May 2012. The search was performed by applying the MeSH terms “exercise” or “training” in combination with “sprint training (exercise)” or “high intensity training (exercise)” or “interval training (exercise)”. Free-text searches were used in databases with no thesaurus (SPORTdiscus and Bibliotek.dk). For the exact search terms applied in the different databases, please see Table 1.

As shown in the flowchart (Fig. 1), the search procedure yielded a total of 2668 publications of which 823 were duplicates leaving 1845 unique publications for screening based on their title and abstract. This screening revealed 93 publications relevant for further reading.

Abstracts, reviews, meta-analyses, and other secondary sources were excluded ($n = 16$) as were studies using a training protocol not corresponding to the established SIT definition and studies where subjects performed other training modalities during the intervention period that could affect the results ($n = 22$). Also, studies examining the effect of a single exercise bout or a single session of SIT were excluded ($n = 21$). Furthermore, studies applying either highly trained subjects or athletes ($n = 5$) or studies applying patients having metabolic or cardiovascular diseases

Table 1. Retrieved publications and applied search terms from six different databases

Database	Articles retrieved	Search terms (MeSH, free text, etc.)
Bibliotek.dk	6	Topics: “sprinting training”; “sprinting”; interval training Free text: “sprint-interval”, “sprint træning”, “højint”
SveMed+	3	“Exercise” and (“interval training” or “high intensity exercise”)
Pubmed	1144	“Exercise” [MeSH] and (“interval training” or “sprint training” or “sprint exercise” or “high intensity training” or “high intensity exercise” or “interval exercise”) Limits: Publication Date from 1995/01/01 to 2012/12/31
Embase	1014	“Exercise” or “Training” and (“interval training” or “interval exercise” or “sprint training” or “Sprint Exercise” or “high intensity training” or “High intensity exercise”) and [embase]/lim and [humans]/lim and [medline]/lim and [1995-2012]/py
SPORTDiscus	719	Free text: “Interval training” or “Sprinting-Training” or “high intensity training” Limits: Publication Date from 1995/01/01 to 2012/12/31
PEDro	1	Search history not reported

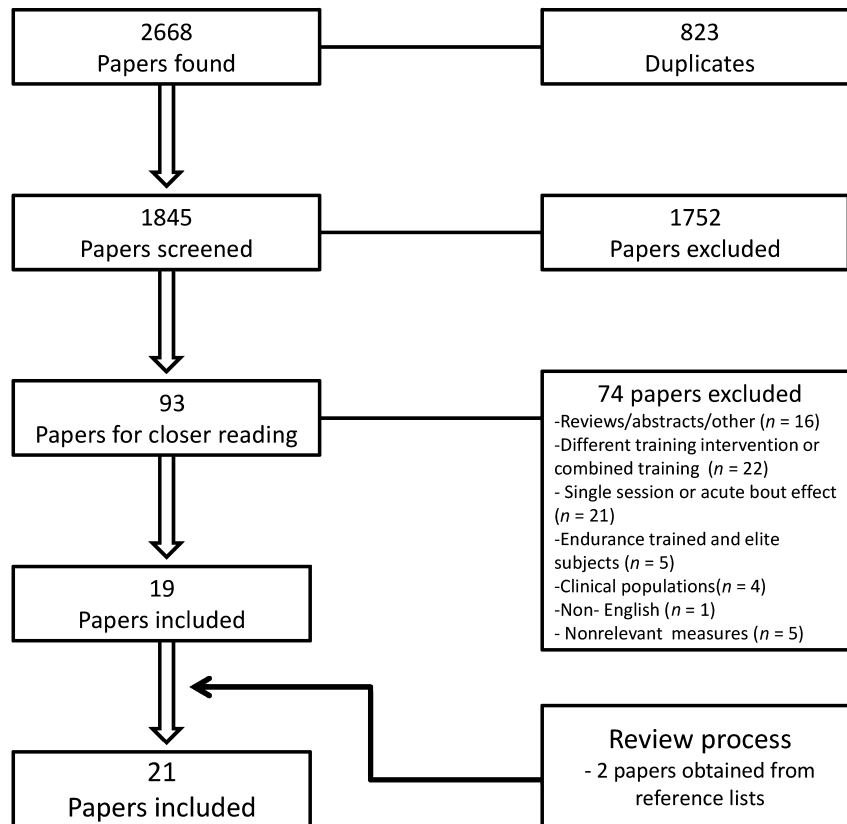


Fig. 1. Flowchart of the inclusion of literature.

were excluded ($n = 4$). Finally, non-English papers ($n = 1$) and studies with effect measures considered irrelevant for this review ($n = 5$) were also excluded.

Consequently, 74 publications not fulfilling the inclusion criteria were excluded, leaving 19 publications for inclusion. A screening of the reference lists of these 19 publications revealed two additional RCT studies (Babraj et al., 2009; Metcalfe et al., 2011), that were not captured by the original search. A total of 21 publications (19 unique studies) were, therefore, included in the review (Fig. 1).

Statistical procedures of the meta-analysis

Meta-analytical procedures were applied to further evaluate possible effects of SIT on $\text{VO}_{2\text{max}}$.

The meta-analysis was conducted consistent with the Meta-Analysis of Observational Studies in Epidemiology framework. Effect sizes (ES) were computed in one of two ways: (i) for controlled experiments, we computed ES as the mean change from before to after the intervention of the exercise group minus the mean change of the control group divided by the preintervention pooled standard deviation and adjusting for sample size; (ii) for noncontrolled experiments, ES was computed as the mean change from before to after the intervention of the exercise group divided by the preintervention standard deviation.

Within the controlled trials, a positive effect indicates a larger improvement in $\text{VO}_{2\text{max}}$ for the intervention group than the control group whereas a negative effect indicates a larger decrement in $\text{VO}_{2\text{max}}$ in the intervention group compared to the control group. Within the noncontrolled trials, a positive effect size indicates an improvement in $\text{VO}_{2\text{max}}$, whereas a negative effect size indicated a decrement. One of the studies (Hazell et al., 2010) included three treatment groups but only a single control group. In this study, a simple average of the three treatment effect sizes was computed.

The aggregated or mean effect size was computed using a random effects model and is reported as Hedges' g , which adjusts for sample size differences across studies. The underlying assumption of the random effects model is that samples are drawn from populations with different effect sizes and the true effect differs between studies.

Furthermore, based on the assumption of random effects, we computed a 95% confidence interval (CI) around the mean effect size and further tested the heterogeneity of the mean effect size. Heterogeneity is indicated if Q statistic, computed as the sum of the squares of each effect size about the weighted mean effect size, was significant (P -value < 0.05).

Results

General study characteristics

A total of 442 (150 women) subjects have been included in the selected studies with 190 subjects (69 women) enrolled in the SIT intervention groups. Subjects included are mainly characterized as healthy sedentary or recreationally active young adults ($\text{VO}_{2\text{max}}$ /peak of $< 55 \text{ mL} \times / \text{kg} \times / \text{min}$). Two studies have applied overweight/ obese men (Whyte et al., 2010) and women [Trilk et al., 2011; body mass index (BMI) ≥ 25], respectively.

The sample size for intervention groups in the selected studies range from $n = 6$ –20.

Four studies are RCTs, whereas nine studies have incorporated a control group but apply a MCT design, where subjects were matched and divided based on different parameters like baseline sprinting ability or

$\text{VO}_{2\text{max}}$. Six trials did not include a control group (non-controlled) in the design.

The most commonly used training protocol for SIT consists of repeated Wingate-based “30 s all-out” bicycle sprints performed three times per week, with training volume per session varying from three to seven repetitions, interspersed by 2–5 min of recovery between bouts (Burgomaster et al., 2005, 2006, 2008; Gibala et al., 2006; Babraj et al., 2009; Bailey et al., 2009; Richards et al., 2010; Bayati et al., 2011; Trilk et al., 2011).

Some studies have used shorter all-out sprints of 10 and 15 s (Hazell et al., 2010; Macpherson et al., 2011; Metcalfe et al., 2011) and a single study has used treadmill sprinting but an otherwise identical training intervention equal to the typical ergometer bicycle protocol (Macpherson et al., 2011).

The duration of the training intervention ranges from 2 weeks to 8 weeks of training.

A number of studies (Gibala et al., 2006; Burgomaster et al., 2008; Bailey et al., 2009; Hazell et al., 2010; Macpherson et al., 2011) also compared SIT to another intervention group with a training regime differing in terms of training type, volume, and intensity (e.g. SIT vs continuous endurance training). Some trials matched the training groups in terms of total work performed each training session (Bailey et al., 2009), while others differed significantly in total work performed (Gibala et al., 2006; Burgomaster et al., 2008; Macpherson et al., 2011). All studies included a familiarization protocol prior to the training intervention. However, there were variations in the extent and quality of these protocols.

In general, SIT was well tolerated with only few (non-serious) adverse events reported. Studies reporting sprint compliance reported values from 95% to 100% and studies reported generally low dropout rates.

Maximal aerobic power ($\text{VO}_{2\text{max}}/\text{VO}_{2\text{peak}}$)

A consistent effect of SIT was an increased aerobic power expressed as either $\text{VO}_{2\text{max}}$ or $\text{VO}_{2\text{peak}}$ (Table 2). This coincided with improvements in aerobic exercise performance. Twelve studies (McKenna et al., 1997; MacDougall et al., 1998; Barnett et al., 2004; Burgomaster et al., 2008; Bailey et al., 2009; Hazell et al., 2010; Whyte et al., 2010; Astorino et al., 2011; Bayati et al., 2011; Macpherson et al., 2011; Metcalfe et al., 2011; Trilk et al., 2011) have reported significant increases of $\text{VO}_{2\text{max}}$ or $\text{VO}_{2\text{peak}}$ in the range of ~4–13.5%, typically measured during an incremental test to exhaustion. In contrast to the majority of reports, two studies (Burgomaster et al., 2005, 2006) found no significant difference in $\text{VO}_{2\text{max}}$ following six sessions (2 weeks) of SIT despite improved performance in aerobic testing.

The meta-analysis included 13 ESs that were retrieved from the 13 selected published studies reflecting a total of 238 participants (Table 2 and Fig. 2). All 13 ESs were positive and 4 out of the 13 individual ESs reached significance (P -value < 0.05). The weighted mean ES across studies was $g = 0.63$, 95% CI (0.39; 0.87) and was statistically different from zero.

To test whether the results of the studies are sufficiently similar to warrant their combination into an overall result, we tested for homogeneity in the sample. We find that the null hypothesis of homogeneity could

Table 2. Relative changes in maximal oxygen consumption ($\Delta\text{VO}_{2\text{max}}$), effect sizes, and summary statistics

Study	Sample size	$\Delta\text{VO}_{2\text{max}}$	Duration	Effect size		95% confidence interval	
				Hedges' g	Standard error	Lower limit	Upper limit
Noncontrolled trials							
Burgomaster et al. (2008)	10	7.3%	6 weeks	0.4284	0.5792	−0.7068	1.5637
Macdougall et al. (1998)	12	6.9%	7 weeks	0.5181	0.5433	−0.5468	1.5831
McKenna et al. (1997)	8	10.8%	7 weeks	0.6976	0.6391	−0.5551	1.9503
Macpherson et al. (2011)	10	11.5%	6 weeks	0.9640	0.6106	−0.2327	2.1607
Whyte et al. (2010)	10	9.5%	2 weeks	0.6325	0.5885	−0.5210	1.7859
Mean effect, noncontrolled				0.6362	0.2637	0.1194	1.1529
Controlled trials							
Astorino et al. (2012)	20	4.7%	2 weeks	0.3482	0.6903	−1.0047	1.7011
Bailey et al. (2009)	16	7.1%	2 weeks	0.5348	0.4821	−0.4101	1.4797
Barnett et al. (2004)	16	4.2%	8 weeks	0.3362	0.4764	−0.5977	1.2700
Bayati et al. (2011)	16	9.6%	4 weeks	1.0825	0.5100	0.0829	2.0821
Burgomaster et al. (2006)	16	5.5%	2 weeks	0.3819	0.4775	−0.5540	1.3178
Hazell et al. (2010)	48	7.3%	2 weeks	0.5712	0.2331	0.1143	1.0282
Metcalfe et al. (2011)	29	13.2%	6 weeks	0.8801	0.3678	0.1592	1.6009
Trilk et al. (2011)	28	13.4%	4 weeks	0.7746	0.3813	0.0273	1.5219
Mean effect, controlled				0.6323	0.1391	0.3597	0.9049
Mean effect, total				0.6331	0.1230	0.3921	0.8742

In the controlled studies, the change in $\text{VO}_{2\text{max}}$ was determined as the mean change from pre- to post-training in the SIT group minus any change in the control group from pre to post intervention. In the noncontrolled studies, it was determined as the relative improvement from pre- to post-intervention.

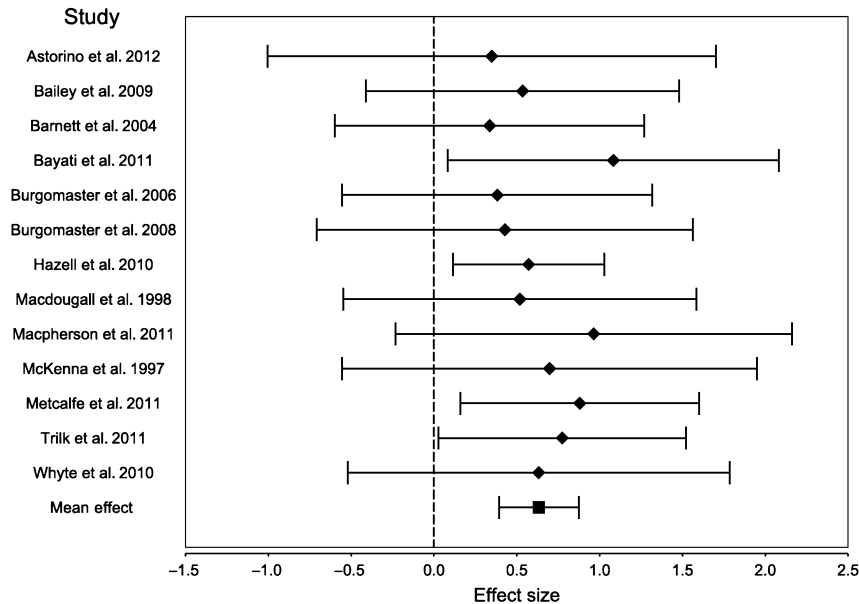


Fig. 2. Forrest plot of the effect sizes and 95% confidence intervals of the changes in maximal oxygen consumption following SIT.

not be rejected ($Q = 2.79$, d.f. = 12, P -value = 0.99). This indicates that the weighted mean ES of the sample distribution is likely to correctly describe the average effect in the population.

In summary, there is solid evidence showing that 2–8 weeks of SIT-based training improves $\text{VO}_{2\text{max}}$ in healthy sedentary or recreationally active adult men and women including untrained, overweight, and obese subjects.

Performance measures

Improved aerobic and anaerobic exercise performance following a period of SIT was consistently reported.

Anaerobic performance in terms of increased mean and peak power values during short-lasting trials (≤ 30 s) have been shown to improve following SIT. Several studies (McKenna et al., 1997; Barnett et al., 2004; Burgomaster et al., 2005, 2006, 2008; Hazell et al., 2010; Whyte et al., 2010; Astorino et al., 2011; Bayati et al., 2011) reported increases in mean and peak power during a Wingate test with improvements up to 17%.

Also, numerous studies (McKenna et al., 1997; Burgomaster et al., 2005, 2006, 2007; Gibala et al., 2006; Babraj et al., 2009; Hazell et al., 2010; Macpherson et al., 2011) reported significant improvements in exercise tests relying heavily on aerobic metabolism after as little as six sessions of SIT. A matched-controlled study of Burgomaster et al. (2005) reported a 100% increase in cycle endurance capacity at 80% of $\text{VO}_{2\text{peak}}$ in a group of recreational active men and women after six sessions of SIT, which differed significantly from the control group showing no changes in performance from pre- to post-training. A noncontrolled

study of Gibala et al. (2006) compared performance improvements during a 750-kJ cycling time trial after 2 weeks of either SIT or endurance training in two groups drawn from the same population. One group ($n = 8$) performed six sessions of SIT while another group ($n = 8$) performed the same number of sessions as high-volume endurance training (ET; 90–120 min of continuous cycling at $\sim 65\%$ $\text{VO}_{2\text{peak}}$). Significant improvements in the 750-kJ time trial was reported for both groups (SIT: 10.1% and ET: 7.5%), with no significant group differences despite the considerable differences in training volume. Total training volume (intervals only) of the SIT group expressed as total work performed during the six SIT sessions corresponded to $\sim 10\%$ of the ET group. A noncontrolled study (Macpherson et al., 2011) using treadmill training compared the effects of 6 weeks (three sessions/week) of SIT (30 s all-out treadmill) vs ET (30–60 min continuous running at $\sim 65\%$ $\text{VO}_{2\text{max}}$) on a 2000 m time-trial performance in recreationally active men and women ($n = 12$ and $n = 8$). Performance improved significantly in the SIT (4.6%) and ET (5.9%) group with no difference between groups. Hazell et al. (2010) reported improved 5-km cycling time-trial performance following three different types of SIT interventions over a 2-week period (1: 30 s all-out/4-min recovery; 2: 10 s all-out/4-min recovery; and 3: 10 s all out/ 2-min recovery) with improvements of 5.2%, 3.5% and 3.0%, respectively, and with no improvements observed in the control group. No significant differences were found between training groups.

Other studies (McKenna et al., 1997; Bayati et al., 2011) have reported performance improvements during incremental tests until exhaustion with substantial increases in the total work performed.

In summary, there is strong evidence that SIT training improves both aerobic and anaerobic performance in healthy sedentary or recreationally active men and women with some studies reporting improvements corresponding to what is seen after traditional high volume endurance training. There are reports of improved aerobic endurance performance at a submaximal workload as well as improved ability to sustain a higher mean power output during a fixed work bout dominated by aerobic metabolism.

Cardiovascular effects

Four studies (one noncontrolled (Burgomaster et al., 2008), two matched-controlled (McKenna et al., 1997; Astorino et al., 2011) and one RCT (Trilk et al., 2011)) of which three composed 4–7 weeks of SIT (McKenna et al., 1997; Burgomaster et al., 2008; Trilk et al., 2011) and one study 2 weeks of SIT (Astorino et al., 2011) reported decreased heart rate (HR) during a submaximal steady state trial following the intervention. In contrast, a RCT by Bailey et al. (2009) showed no change in moderate-intensity HR after 2 weeks of SIT. In two studies (Rakobowchuk et al., 2008; Astorino et al., 2012), no change was found in resting HR following 2 and 6 weeks of SIT, respectively.

Only two studies (Macpherson et al., 2011; Trilk et al., 2011) have measured the effects of SIT on stroke volume (SV) with conflicting results. Trilk et al. (2011; RCT) reported a significant increase in SV in a group of sedentary overweight/obese ($n = 14$, BMI >25) women during a submaximal trial ($\sim 50\%$ $\text{VO}_{2\text{max}}$) after 4 weeks of SIT (three sessions/week). Macpherson et al. (2011) reported no increase in maximal cardiac output (Q) or SV in a group of recreationally active men and women ($n = 10$) performing SIT, from pre- to post-intervention. A training group in the same study performed 6 weeks (three sessions/week) of 30–60 min continuous running at $\sim 65\%$ $\text{VO}_{2\text{max}}$ and increased SV by 9.5%, however, there was no significant difference between the two training groups. In contrast, an increased $a\text{-vO}_2$ difference of 7.1% was reported in the SIT group only, which was significantly different from the ET group. Bailey et al. 2009 also reported improved slope of slow VO_2 component, which was associated with greater O_2 extraction indicated by changes in the deoxyhemoglobin/myoglobin concentration. Lack of alteration in HR kinetics or oxyhemoglobin concentration suggested that bulk delivery to the muscles were not responsible for alterations in VO_2 kinetics. However, due to indirect measurements the authors were unable to rule out the possibility that enhanced muscle blood flow contributed in part to the alterations in VO_2 kinetics observed following SIT.

In summary, these findings suggest that SIT training may elicit changes in HR kinetics during submaximal steady state exercise, without significant changes in

resting HR. Few studies have investigated the effect of SIT on SV but findings are inconsistent. Consequently, it is not clear if improved central function (SV) and/or peripheral adaptations (increased O_2 extraction) is responsible for the observed reduction in submaximal exercise HR.

Muscle oxidative potential

Alteration in content and activity of various mitochondrial enzymes is often used as a measure of improved muscle oxidative potential. Five studies (MacDougall et al., 1998; Barnett et al., 2004; Burgomaster et al., 2005, 2006, 2008) have reported increased maximal citrate synthase (CS) activity after a period of SIT in the range of ~ 11 –42%, indicating a significant variation in the relative magnitude of CS activity up-regulation. Burgomaster et al. (2005, 2006) reported increases of 38% and 11%, respectively, after 2 weeks (three sessions/week) of SIT and Barnett et al. (2004), MacDougall et al. (1998) and Burgomaster et al. (2008) reported increases of 42%, 36%, and 25% after 8, 7, and 6 weeks (three sessions/week), respectively.

Contrasting these findings, a controlled study of Esbjörnson Liljedahl (1996) reported no change in CS activity following 4 weeks (three sessions/week) of SIT in a study applying five times longer recovery time (20 min) between each 30 s exercise bout compared to the training protocol used in the studies reporting increases in CS activity (3–4.5 min).

Beta-Hydroxyacyl-CoA-dehydrogenase (HAD), is another key enzyme related to muscle aerobic potential that is often used to reflect the maximal capacity for lipid oxidation (Burgomaster et al., 2008). HAD has been measured in a few studies following a SIT intervention, with equivocal results. Three studies showed no change in the activity of HAD (Liljedahl, 1996; MacDougall et al., 1998; Burgomaster et al., 2006) following SIT, but in a noncontrolled study of Burgomaster et al. (2008) a significant increase in HAD activity was observed after 6 weeks of SIT (three sessions/week) and the change was of the same magnitude as observed in a group who performed 6 weeks (five sessions/week) of 40–60 min of continuous cycling at $\sim 65\%$ $\text{VO}_{2\text{peak}}$ (Burgomaster et al., 2008).

Few studies have measured changes in other oxidative enzymes following SIT. Burgomaster et al. (2006) reported increased pyruvate dehydrogenase (PDH) activity during exercise after 2 weeks of SIT while Burgomaster et al. (2008) reported increased PDH content after 8 weeks of SIT. MacDougall et al. (1998) reported increased maximal activity of malate dehydrogenase (MDH) 29% and succinate dehydrogenase (SDH) of 65% after 7 weeks of training.

Gibala et al. (2006) measured changes in the maximal activity of cytochrome c oxidase (COX) and changes in protein content of COX subunit II and IV as an indicator

for increased muscle oxidative potential. Maximal COX activity and protein content of subunit II and IV increased significantly after 2 weeks of SIT, and this to the same extent as in a group performing high volume endurance training (90–120 min continuous cycling at 65% $\text{VO}_{2\text{peak}}$).

In summary, there are many reports of improved muscle oxidative potential reflected by increased oxidative enzyme activity and/or content. However, the effect on HAD is equivocal.

Changes in substrate content and utilization

A shift in substrate utilization following a period of endurance training is commonly described and represents an important adaptation for increased exercise capacity (Holloszy & Coyle, 1984; Phillips et al., 1996).

Respiratory exchange ratio and whole body carbohydrate and lipid oxidation

Only a few studies have described the effect of SIT on the respiratory exchange ratio (RER). Astorino et al. (2011) and Burgomaster et al. (2008) reported decreased RER during submaximal steady-state exercise following a period of SIT in both men and women, but a noncontrolled study of McKenna et al. (1997) reported no change in RER following 7 weeks of SIT in a group of men ($n = 8$).

In accordance with reduced RER, Burgomaster et al. 2008 also reported changes in whole-body carbohydrate (decreased) and lipid oxidation (increased) during submaximal exercise. Finally, a noncontrolled study by Whyte et al. (2010) measured decreased resting carbohydrate oxidation and increased lipid oxidation at 24 h but not 72 h after the SIT intervention when compared to baseline values.

Intramuscular substrates

Increased resting glycogen content following a period of SIT has been reported in four studies (Barnett et al., 2004; Burgomaster et al., 2005, 2006; Gibala et al., 2006). Furthermore, Burgomaster et al. (2008) reported increased glycogen content 60 min post-exercise (60 min at 65% $\text{VO}_{2\text{peak}}$) compared with pretraining levels. Also, Burgomaster et al. 2008 reported a reduction in net-glycogenolysis during submaximal exercise which was consistent with an earlier study from the same group (Burgomaster et al., 2006).

In summary, there are equivocal findings in the few studies evaluating the effects of SIT on whole-body substrate utilization. Nonetheless, there are indications of increased muscle glycogen content and reduced glycogenolysis during submaximal exercise.

Glycemic control and insulin sensitivity

Four recent studies (Babraj et al., 2009; Richards et al., 2010; Whyte et al., 2010; Metcalfe et al., 2011) have

investigated the effect of low volume SIT on glycemic control and insulin sensitivity in healthy subjects. Brabaj et al. (2009; RCT) investigated the glucose and insulin response following an oral glucose tolerance test (OGTT) pre- and post-2 weeks (six sessions, 30 s/4 min) of SIT. Post-training, an OGTT showed that both glucose and insulin area under curve (AUC) were significantly reduced. Furthermore, insulin sensitivity (determined by the Cederholm index – a measure of peripheral insulin sensitivity) was significantly increased. Applying a smaller weekly training volume, Metcalfe et al. (2011) observed no change in the glucose or insulin AUC following an OGTT in neither men ($n = 7$) nor women ($n = 8$) after training. They did, however, observe improved insulin sensitivity in men (28%), but found no significant change in women. This observation contrasts a controlled study by Richards et al. (2010) who measured changes in insulin sensitivity using the hyperinsulinemic euglycemic clamp technique (DeFronzo et al., 1979). They reported significant improved insulin sensitivity in both men ($n = 5$) and women ($n = 7$), as indicated by an increased glucose infusion rate necessary to maintain a blood glucose concentration of 5 mM. Furthermore, a single-bout control group showed no change indicating that it was a true training effect and not due to the effect of an acute exercise bout. A noncontrolled study of Whyte et al. (2010) is the only study to investigate the effect of SIT on glycemic control and insulin sensitivity in overweight/obese males (2 weeks/six sessions). They reported reduced fasting insulin concentration and insulin AUC following an OGTT and the insulin sensitivity index were significantly higher (23.3%) 24 h post-SIT. However, these values were not significantly different from baseline at 72 h post-intervention. No change in glucose AUC was reported at any time.

In summary, these findings suggest that a period of SIT may improve glycemic control and insulin sensitivity.

Discussion

A systematic literature search revealed 21 publications (19 unique studies) regarding the effects of SIT all applying healthy normal weight or overweight/obese adults as study populations. Subjects improved both aerobic performance as well as a number of metabolic functions. Furthermore, no severe side effects were observed and a high compliance and a low dropout rate were reported.

A consistent finding following 2–8 weeks of SIT performed 2–3 times a week was improvements of aerobic-based exercise performance (McKenna et al., 1997; Burgomaster et al., 2005, 2006, 2007; Gibala et al., 2006; Babraj et al., 2009; Hazell et al., 2010; Macpherson et al., 2011) and aerobic power ($\text{VO}_{2\text{max}}$; McKenna et al., 1997; MacDougall et al., 1998; Barnett et al., 2004; Burgomaster et al., 2008; Bailey et al.,

2009; Hazell et al., 2010; Whyte et al., 2010; Astorino et al., 2011; Bayati et al., 2011; Macpherson et al., 2011; Metcalfe et al., 2011; Trilk et al., 2011) despite a very low training volume. In a number of studies, the improvements were reported to be similar to those seen after traditional high-volume endurance training (Gibala et al., 2006; Burgomaster et al., 2008; Macpherson et al., 2011).

SIT may improve different muscle metabolic functions and elicit changes in substrate utilization corresponding to adaptations typically associated with high-volume endurance training relying heavily on aerobic energy turnover (Phillips et al., 1996). Finally, the results of recent studies investigating the effect of SIT on glycemic control suggest that SIT might constitute a time-efficient protocol for rapid improvement of glucose tolerance and insulin sensitivity. The fact that some studies have reported improved insulin sensitivity after a SIT intervention may in part be explained by an increase in the protein content of GLUT4 in the muscles. Burgomaster et al. reported a modestly increased GLUT 4 content (~25%) in muscle biopsies after 6 weeks of SIT and this increase persisted for 6 weeks after cessation of SIT (Burgomaster et al., 2007).

Aerobic exercise performance and $\text{VO}_{2\text{max}}$

The consistent improvements seen in exercise tolerance could be explained by increases in $\text{VO}_{2\text{max}}$; in general, however, the studies do not report a consistent correlation between improvements of performance and $\text{VO}_{2\text{max}}$, and two studies did not observe changes in $\text{VO}_{2\text{max}}$ despite improved aerobic exercise performance (Burgomaster et al., 2005, 2006). The authors hypothesized that these findings could be explained by a relatively high $\text{VO}_{2\text{peak}}$ at baseline ($48.7 \text{ mL} \times \text{kg}^{-1} \times \text{min}^{-1}$). Nevertheless, one other study used subjects with almost similar baseline $\text{VO}_{2\text{max}}$ values and found significant increases in $\text{VO}_{2\text{max}}$ after 2 weeks of SIT (Hazell et al., 2010).

$\text{VO}_{2\text{max}}$ increased from 4.2% to 13.4% in the included studies (Table 2). The improvements in $\text{VO}_{2\text{max}}$ may be explained by (1) increased oxygen availability due to central effects (cardiac output) and/or (2) as consequence of peripheral adaptations with improved ability to extract and use available oxygen due to increased muscle oxidative potential. Studies (Macpherson et al., 2011; Trilk et al., 2011; Astorino et al., 2012) on central effects are more limited and equivocal than effects on muscle oxidative capacity (MacDougall et al., 1998; Barnett et al., 2004; Burgomaster et al., 2005, 2006, 2008). A larger number of studies have reported peripheral changes with improved enzymatic adaptations and increased mitochondrial mass following SIT, suggesting significant peripheral adaptations that could explain some of the observed improvements in $\text{VO}_{2\text{max}}$ and aerobic performance. However, enzymatic changes are typically more pronounced than the related improvement

in $\text{VO}_{2\text{max}}$. Consequently, adaptations in mitochondrial enzymes of muscles cannot alone explain the changes in $\text{VO}_{2\text{max}}$, as exemplified in two studies reporting significant increases in CS activity (38% and 11%) following 2 weeks of SIT without observing improved $\text{VO}_{2\text{max}}$ (Burgomaster et al., 2005, 2006).

Time course of adaptations after SIT

SIT seem to elicit rapid changes in aerobic performance and metabolic function. Several of the selected studies have reported significant improvements in performance (Burgomaster et al., 2005, 2006; Gibala et al., 2006; Babraj et al., 2009; Bailey et al., 2009; Hazell et al., 2010), $\text{VO}_{2\text{max}}$ (Bailey et al., 2009; Hazell et al., 2010; Whyte et al., 2010; Astorino et al., 2011, 2012; Bayati et al., 2011) and muscle oxidative potential (Burgomaster et al., 2005, 2006; Gibala et al., 2006) following only 2 weeks of training (three sessions/week) applying a very low weekly training volume (≤ 12 min of exercise time). However, there seem to be no clear relationship between the magnitude of improvements in $\text{VO}_{2\text{max}}$ or muscle oxidative potential and the duration of training when, comparing the results from the studies applying two (Burgomaster et al., 2005, 2006; Gibala et al., 2006; Bailey et al., 2009; Hazell et al., 2010; Whyte et al., 2010; Astorino et al., 2011; Bayati et al., 2011) and 4–8 weeks (McKenna et al., 1997; MacDougall et al., 1998; Barnett et al., 2004; Burgomaster et al., 2007, 2008; Rakobowchuk et al., 2008; Bayati et al., 2011; Macpherson et al., 2011; Metcalfe et al., 2011; Trilk et al., 2011) of SIT, respectively. The mean improvements in $\text{VO}_{2\text{max}}$ after 2 weeks of SIT is 6.8% compared to 9.6% after 4–8 weeks. This may indicate that some of the adaptations to SIT occur at an early stage of a training period and then levels off. This pattern is supported by Burgomaster et al. (2007) who used changes in the protein content of cytochrome c oxidase subunit 4 (COX4) as a marker of changes in muscle oxidative capacity, and reported increases of ~35% after only 1 week of SIT, without further increase after 6 weeks of training.

SIT and aerobic metabolism

During an all-out 30-s cycle ergometer sprint 25–30% of the ATP resynthesized from anaerobic metabolism comes from phosphocreatine (PCr breakdown), while the major part (65–70%) comes from glycolysis (Bogdanis et al., 1995, 1996; Gastin, 2001; Withers et al., 1991). The relative contribution of aerobic metabolism have been estimated to be 25–30% of the total ATP turnover, but importantly, the contribution from aerobic pathways increases significantly with repeated trials to meet the demand for ATP resynthesis (Bogdanis et al., 1995, 1996; Gastin, 2001; Withers et al., 1991). This could explain some of the effects of

SIT on aerobic function and may also explain why the study of Liljedahl et al., who included a 20-min rest between bouts, observed no changes in CS or beta-HAD activity after 4 weeks of SIT (Liljedahl, 1996). This further suggests that despite most studies apply relatively long rest periods ($\geq 1:8$ work to rest ratio), the magnitude of the work:rest ratio may still be highly important for the aerobic gains observed after SIT. Despite the increased contribution from aerobic metabolism with successive 30 s bouts, the volume of work in the selected studies was still extremely limited. Furthermore, it does not explain why a matched-controlled study (Hazell et al., 2010) reported no difference in $\text{VO}_{2\text{max}}$ between a group performing the 30-s/4-min protocol (9.2%) and a group performing 10-s/4-min protocol (9.2%) because the contribution of aerobic metabolism in repeated 10-s sprint with the same amount of recovery (4 min) are expected to be lower than in repeated 30-s intervals. Based on the results, the authors suggested that the generation of peak power during the first few seconds of an all-out bout is more likely responsible for the adaptations to SIT, than the total work completed during a 30-s bout. If this is the case, the availability of PCr during repeated sprints might be a very important factor because it is mainly responsible for the high power output during the initial 10 s of maximal exercise (Bogdanis et al., 1996). These findings raise interesting questions for future research because the exercise time in the 10-s protocol constituted a reduction of 67% of the total training volume performed in the 30-s/4-min protocol, suggesting that an even smaller volume than the commonly used 30-s all-out protocol can induce significant aerobic and metabolic adaptations.

Underlying mechanisms for aerobic adaptations to SIT

The underlying mechanisms responsible for aerobic and metabolic adaptations to SIT are still unclear and the literature is equivocal. However, it is agreed that the rapid adaptations to SIT are somehow related to the high level of fiber recruitment that occurs during an all-out bout (Gibala et al., 2006; Burgomaster et al., 2007; Bailey et al., 2009). Especially, the potential stress of the type II muscle fibers is considered an important factor for SIT to elicit changes in oxidative capacity (Gollnick et al., 1973; Dudley et al., 1982; Gibala et al., 2006; Bailey et al., 2009). According to Henneman's size principle (Mendell, 2005), traditional endurance training would predominately be expected to recruit type I fibers and thus primarily cause specific adaptations in these fibers (Bailey et al., 2009). Moreover, studies have shown that high-intensity interval training induces greater oxidative enzyme adaptations in type II fibers than continuous endurance training (Bailey et al., 2009) and the improvements in oxidative capacity in type IIx fibers is increased when training intensity exceeds $\text{VO}_{2\text{peak}}$ (Dudley et al., 1982).

Consequently, specific oxidative adaptations in type II fibers could be partly responsible for the SIT-induced increases in $\text{VO}_{2\text{max}}$ and improved maximal aerobic performance.

The transcriptional coactivator PGC-1 α (peroxisome proliferator-activated receptor gamma coactivator 1- α) is considered a key activator of oxidative enzyme expression and mitochondrial biogenesis in a number of different cell types (Koulmann & Bigard, 2006; Liang & Ward, 2006; Burgomaster et al., 2008; Gibala et al., 2009). SIT-induced alterations related to PGC-1 α have been proposed as an important underlying mechanism related to the observed changes in aerobic function (Gibala et al., 2006; Burgomaster et al., 2008). In the study of Burgomaster et al. (2008), an increased PGC-1 α protein content following 6 weeks of SIT was reported and another study (Gibala et al., 2009) investigating the acute effect of a single SIT session (four bouts of 30-s sprint and 4-min recovery) on signaling cascades linked to PGC-1 α reported stimulation of AMPK (AMP-activated protein kinase) and p38MAPK (mitogen-activated protein kinase), which are two important signaling cascades related to PGC-1 α . Also, they reported increased PGC-1 α mRNA expression (Gibala et al., 2009). Consequently, alterations on the gene expression level and especially changes in expression of the PGC-1 α could, in part, explain the underlying regulatory mechanism responsible for the oxidative muscular adaptations induced by SIT, and are, therefore, candidate for future research. Additionally, it has been suggested that a close relationship exists between PGC-1 α function, insulin sensitivity, and type 2 diabetes, which is most likely related to the essential roles of PGC-1 α in mitochondria biogenesis and glucose/fatty acid metabolism (Liang & Ward, 2006).

Limitations and future research

Several limitations have to be kept in mind when interpreting the results of the identified studies. First, only four studies applied a RCT design while the main part of the studies applied a matched-controlled design or a noncontrolled design. Second, a general lack of blinding of assessors was evident and some of the studies lack statistical power due to small sample sizes.

Thirdly, this review was limited to investigate the effects of SIT in subjects characterized as "healthy sedentary or recreationally active adults." However, the potential for SIT to elicit physical improvements seem to be supported by the few studies investigating the effect of SIT in other populations. A randomized controlled study (Creer et al., 2004) investigated the effects of incorporating a SIT protocol in the training program of very well trained cyclists ($n = 10$) over a period of 4 weeks and they reported improvements in $\text{VO}_{2\text{max}}$ as well as neuromuscular and metabolic function following the

intervention. A control group performing endurance training did, however, also increase $\text{VO}_{2\text{max}}$, but the authors implied that the group did not represent a true control.

Data from clinical populations also offers support as shown in a study of Harmer et al. 2008, who investigated the effects of SIT (7 weeks) on aerobic and metabolic function in a group of patients with type 1 diabetes. The study showed significant improvements in both muscle oxidative potential and substrate utilization during submaximal exercise (Harmer et al., 2008). Consequently, it seems that SIT is a time-efficient training approach, which may be useful in a wider range of populations.

Future studies applying a RCT design should address the long-term effects of SIT as well as establish the time course of the adaptations to clarify whether the effects primarily occurs during the first few weeks of training. Furthermore, future investigations should try to establish the most favorable work:recovery relationship, and also, it should be established whether SIT is safe and effective for health promotion in the general population and in different groups of patients. Finally, more studies looking into cardiovascular effects of SIT and studies investigating the underlying mechanisms responsible for adaptations to SIT are warranted.

In conclusion, SIT performed by healthy sedentary/recreationally active adults is consistently reported to improve aerobic exercise performance and aerobic power in terms of improved $\text{VO}_{2\text{max}}$. Evidence supporting an increased muscle oxidative potential following SIT exists, while there is limited evidence regarding potential central cardiovascular effects. There are indications of a beneficial shift in substrate utilization and SIT may also improve glycemic control and insulin sensitivity.

Perspectives for health promotion

Typical guidelines for health promotion include a significant amount of a low- to moderate-intensity continuous exercise. Many people do not meet the recommendations (Reichert et al., 2007; Korkiakangas

et al., 2009), partly because of lack of time. Studies have shown a strong correlation between cardio respiratory fitness ($\text{VO}_{2\text{max}}$) and cardiovascular health. In this perspective, our findings regarding $\text{VO}_{2\text{max}}$ following SIT are interesting since SIT may constitute a time-efficient training protocol for improving cardiovascular health and consequently minimizing the risk of cardiovascular disease, which has a major impact on public health in the Western world (Kodama et al., 2009). The fact that SIT may induce rapid improvements in glucose tolerance and insulin sensitivity is also of importance since SIT possibly could contribute to prevention against type 2 diabetes.

However, it can be questioned whether SIT represents a realistic training modality in the general population. Even though subjects in the included studies tolerated the training very well, it was performed under controlled and supervised conditions and subjects were typically screened for any health problems prior to the training intervention. Consequently, safety issues of SIT have not been studied in (sedentary) people performing the training without supervision and/or prior medical screening. Furthermore, SIT requires a substantial amount of motivation to produce repeated “all-out” efforts. This problem has been addressed by modifying the traditional “all-out” SIT protocol toward a more tolerable training intervention using intervals of longer duration (1 min) and lower intensities $\sim 120\% \text{VO}_{2\text{max}}$ (Little et al., 2010; Reynolds, 2012).

Key words: $\text{VO}_{2\text{max}}$, aerobic power, metabolism, high-intensity interval training.

Acknowledgement

The authors would like to thank research librarian Edith Clausen for substantial contribution to the comprehensive literature search.

Funding

This research received no grant from any funding agency in the public, commercial, or not-for-profit sectors.

References

- Astorino TA, Allen RP, Roberson DW, Jurancich M. Effect of high-intensity interval training on cardiovascular function, $\text{VO}_{2\text{max}}$, and muscular force. *J Strength Cond Res* 2012; 26: 138–145.
- Astorino TA, Allen RP, Roberson DW, Jurancich M, Lewis R, McCarthy K, Trost E. Adaptations to high-intensity training are independent of gender. *Eur J Appl Physiol* 2011; 111: 1279–1286.
- Babraj JA, Vollaard NB, Keast C, Guppy FM, Cottrell G, Timmons JA. Extremely short duration high intensity interval training substantially improves insulin action in young healthy males. *BMC Endocr Disord* 2009; 9: 3.
- Bailey SJ, Wilkerson DP, Dimenna FJ, Jones AM. Influence of repeated sprint training on pulmonary O_2 uptake and muscle deoxygenation kinetics in humans. *J Appl Physiol* 2009; 106: 1875–1887.
- Barnett C, Carey M, Proietto J, Cerin E, Febbraio MA, Jenkins D. Muscle metabolism during sprint exercise in man: influence of sprint training. *J Sci Med Sport* 2004; 7: 314–322.
- Bayati M, Farzad B, Gharakhanlou R, Agha-Alinejad H. A practical model of low-volume high-intensity interval training induces performance and metabolic adaptations that resemble “all-out” sprint interval training. *J Sports Sci Med* 2011; 10: 571–576.

- Bogdanis GC, Nevill ME, Boobis LH, Lakomy HK. Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. *J Appl Physiol* 1996a; 80: 876–884.
- Bogdanis GC, Nevill ME, Boobis LH, Lakomy HK, Nevill AM. Recovery of power output and muscle metabolites following 30 s of maximal sprint cycling in man. *J Physiol* 1995; 482 (Pt 2): 467–480.
- Bogdanis GC, Nevill ME, Lakomy HK, Boobis LH. Power output and muscle metabolism during and following recovery from 10 and 20 s of maximal sprint exercise in humans. *Acta Physiol Scand* 1998; 163: 261–272.
- Burgomaster KA, Cermak NM, Phillips SM, Benton CR, Bonen A, Gibala MJ. Divergent response of metabolite transport proteins in human skeletal muscle after sprint interval training and detraining. *Am J Physiol Regul Integr Comp Physiol* 2007; 292: R1970–R1976.
- Burgomaster KA, Heigenhauser GJ, Gibala MJ. Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. *J Appl Physiol* 2006; 100: 2041–2047.
- Burgomaster KA, Howarth KR, Phillips SM, Rakobowchuk M, Macdonald MJ, McGee SL, Gibala MJ. Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. *J Physiol* 2008; 586: 151–160.
- Burgomaster KA, Hughes SC, Heigenhauser GJ, Bradwell SN, Gibala MJ. Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *J Appl Physiol* 2005; 98: 1985–1990.
- Creer AR, Ricard MD, Conlee RK, Hoyt GL, Parcell AC. Neural, metabolic, and performance adaptations to four weeks of high intensity sprint-interval training in trained cyclists. *Int J Sports Med* 2004; 25: 92–98.
- Dawson B, Fitzsimons M, Green S, Goodman C, Carey M, Cole K. Changes in performance, muscle metabolites, enzymes and fibre types after short sprint training. *Eur J Appl Physiol Occup Physiol* 1998; 78: 163–169.
- DeFronzo RA, Tobin JD, Andres R. Glucose clamp technique: a method for quantifying insulin secretion and resistance. *Am J Physiol* 1979; 237: E214–E223.
- Dudley GA, Abraham WM, Terjung RL. Influence of exercise intensity and duration on biochemical adaptations in skeletal muscle. *J Appl Physiol* 1982; 53: 844–850.
- Gastin PB. Energy system interaction and relative contribution during maximal exercise. *Sports Med* 2001; 31: 725–741.
- Gibala MJ, Little JP, van Essen M, Wilkin GP, Burgomaster KA, Safdar A, Raha S, Tarnopolsky MA. Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *J Physiol* 2006; 575: 901–911.
- Gibala MJ, McGee SL, Garnham AP, Howlett KF, Snow RJ, Hargreaves M. Brief intense interval exercise activates AMPK and p38 MAPK signaling and increases the expression of PGC-1 α in human skeletal muscle. *J Appl Physiol* 2009; 106: 929–934.
- Gollnick PD, Armstrong RB, Saltin B, Saubert CW, Sembrowich WL, Shepherd RE. Effect of training on enzyme activity and fiber composition of human skeletal muscle. *J Appl Physiol* 1973; 34: 107–111.
- Harmer AR, Chisholm DJ, McKenna MJ, Hunter SK, Ruell PA, Naylor JM, Maxwell LJ, Flack JR. Sprint training increases muscle oxidative metabolism during high-intensity exercise in patients with type 1 diabetes. *Diabetes Care* 2008; 31: 2097–2102.
- Hazell TJ, Macpherson RE, Gravelle BM, Lemon PW. 10 Or 30-S sprint interval training bouts enhance both aerobic and anaerobic performance. *Eur J Appl Physiol* 2010; 110: 153–160.
- Heilbronn LK, Gan SK, Turner N, Campbell LV, Chisholm DJ. Markers of mitochondrial biogenesis and metabolism are lower in overweight and obese insulin-resistant subjects. *J Clin Endocrinol Metab* 2007; 92: 1467–1473.
- Holloszy JO, Coyle EF. Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *J Appl Physiol* 1984; 56: 831–838.
- Hughes VA, Fiatarone MA, Fielding RA, Kahn BB, Ferrara CM, Shepherd P, Fisher EC, Wolfe RR, Elahi D, Evans WJ. Exercise increases muscle GLUT-4 levels and insulin action in subjects with impaired glucose tolerance. *Am J Physiol* 1993; 264: E855–E862.
- Kodama S, Saito K, Tanaka S, Maki M, Yachi Y, Asumi M, Sugawara A, Totsuka K, Shimano H, Ohashi Y, Yamada N, Sone H. Cardiorespiratory fitness as a quantitative predictor of all-cause mortality and cardiovascular events in healthy men and women: a meta-analysis. *JAMA* 2009; 301: 2024–2035.
- Korkiakangas EE, Alahuhta MA, Laitinen JH. Barriers to regular exercise among adults at high risk or diagnosed with type 2 diabetes: a systematic review. *Health Promot Int* 2009; 24: 416–427.
- Koulmann N, Bigard AX. Interaction between signalling pathways involved in skeletal muscle responses to endurance exercise. *Pflugers Arch* 2006; 452: 125–139.
- Laursen PB, Jenkins DG. The scientific basis for high-intensity interval training: optimising training programmes and maximising performance in highly trained endurance athletes. *Sports Med* 2002; 32: 53–73.
- Liang H, Ward WF. PGC-1 α : a key regulator of energy metabolism. *Adv Physiol Educ* 2006; 30: 145–151.
- Liljedahl ME. Different responses of skeletal muscle following sprint training in men and women. *Eur J Appl Physiol Occup Physiol* 1996; 74: 375–383.
- Linossier MT, Denis C, Dormois D, Geyssant A, Lacour JR. Ergometric and metabolic adaptation to a 5-s sprint training programme. *Eur J Appl Physiol Occup Physiol* 1993; 67: 408–414.
- Linossier MT, Dormois D, Geyssant A, Denis C. Performance and fibre characteristics of human skeletal muscle during short sprint training and detraining on a cycle ergometer. *Eur J Appl Physiol Occup Physiol* 1997; 75: 491–498.
- Little JP, Safdar A, Wilkin GP, Tarnopolsky MA, Gibala MJ. A practical model of low-volume high-intensity interval training induces mitochondrial biogenesis in human skeletal muscle: potential mechanisms. *J Physiol* 2010; 588: 1011–1022.
- MacDougall JD, Hicks AL, MacDonald JR, McKelvie RS, Green HJ, Smith KM. Muscle performance and enzymatic adaptations to sprint interval training. *J Appl Physiol* 1998; 84: 2138–2142.
- Macpherson RE, Hazell TJ, Olver TD, Paterson DH, Lemon PW. Run sprint interval training improves aerobic performance but not maximal cardiac output. *Med Sci Sports Exerc* 2011; 43: 115–122.
- McCartney N, Spriet LL, Heigenhauser GJ, Kowalchuk JM, Sutton JR, Jones NL. Muscle power and metabolism in maximal intermittent exercise. *J Appl Physiol* 1986; 60: 1164–1169.
- McKenna MJ, Heigenhauser GJ, McKelvie RS, Obminski G, MacDougall JD, Jones NL. Enhanced pulmonary and active skeletal muscle gas exchange during intense exercise

- after sprint training in men. *J Physiol* 1997; 501 (Pt 3): 703–716.
- Mendell LM. The size principle: a rule describing the recruitment of motoneurons. *J Neurophysiol* 2005; 93: 3024–3026.
- Metcalf RS, Babraj JA, Fawcner SG, Volvaard NB. Towards the minimal amount of exercise for improving metabolic health: beneficial effects of reduced-exertion high-intensity interval training. *Eur J Appl Physiol* 2011; 112: 2767–2775.
- Phillips SM, Green HJ, Tarnopolsky MA, Heigenhauser GF, Hill RE, Grant SM. Effects of training duration on substrate turnover and oxidation during exercise. *J Appl Physiol* 1996a; 81: 2182–2191.
- Rakobowchuk M, Tanguay S, Burgomaster KA, Howarth KR, Gibala MJ, MacDonald MJ. Sprint interval and traditional endurance training induce similar improvements in peripheral arterial stiffness and flow-mediated dilation in healthy humans. *Am J Physiol Regul Integr Comp Physiol* 2008; 295: R236–R242.
- Reichert FF, Barros AJ, Domingues MR, Hallal PC. The role of perceived personal barriers to engagement in leisure-time physical activity. *Am J Public Health* 2007; 97: 515–519.
- Reynolds G. How 1-minute intervals can improve your health. *Sports Med Bull* 2012; 0746-9306: 5.
- Richards JC, Johnson TK, Kuzma JN, Lonac MC, Schweder MM, Voyles WF, Bell C. Short-term sprint interval training increases insulin sensitivity in healthy adults but does not affect the thermogenic response to beta-adrenergic stimulation. *J Physiol* 2010; 588: 2961–2972.
- Ross A, Leveritt M. Long-term metabolic and skeletal muscle adaptations to short-sprint training: implications for sprint training and tapering. *Sports Med* 2001; 31: 1063–1082.
- Simoneau JA, Colberg SR, Thaete FL, Kelley DE. Skeletal muscle glycolytic and oxidative enzyme capacities are determinants of insulin sensitivity and muscle composition in obese women. *FASEB J* 1995; 9: 273–278.
- Simoneau JA, Kelley DE. Altered glycolytic and oxidative capacities of skeletal muscle contribute to insulin resistance in NIDDM. *J Appl Physiol* 1997; 83: 166–171.
- Trilk JL, Singhal A, Bigelman KA, Cureton KJ. Effect of sprint interval training on circulatory function during exercise in sedentary, overweight/obese women. *Eur J Appl Physiol* 2011; 111: 1591–1597.
- Whyte LJ, Gill JM, Cathcart AJ. Effect of 2 weeks of sprint interval training on health-related outcomes in sedentary overweight/obese men. *Metabolism* 2010; 59: 1421–1428.
- Withers RT, Sherman WM, Clark DG, Esselbach PC, Nolan SR, Mackay MH, Brinkman M. Muscle metabolism during 30, 60 and 90 s of maximal cycling on an air-braked ergometer. *Eur J Appl Physiol Occup Physiol* 1991; 63: 354–362.