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Differences in Physiological Responses to Interval Training in Cyclists With and Without Interval Training Experience

by
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The aim of this study was to determine differences in glycolytic metabolite concentrations and work output in response to an all-out interval training session in 23 cyclists with at least 2 years of interval training experience (E) and those inexperienced (IE) in this form of training. The intervention involved subsequent sets of maximal intensity exercise on a cycle ergometer. Each set comprised four 30 s repetitions interspersed with 90 s recovery periods; sets were repeated when blood pH returned to 7.3. Measurements of post-exercise hydrogen (H+) and lactate ion (LA-) concentrations and work output were taken. The experienced cyclists performed significantly more sets of maximal efforts than the inexperienced athletes (5.8 ± 1.2 vs. 4.3 ± 0.9 sets, respectively). Work output decreased in each subsequent set in the IE group and only in the last set in the E group. Distribution of power output changed only in the E group; power decreased in the initial repetitions of set only to increase in the final repetitions. H+ concentration decreased in the third, penultimate, and last sets in the E group and in each subsequent set in the IE group. LA- decreased in the last set in both groups. In conclusion, the experienced cyclists were able to repeatedly induce elevated levels of lactic acidosis. Power output distribution changed with decreased acid–base imbalance. In this way, this group could compensate for a decreased anaerobic metabolism. The above factors allowed cyclists experienced in interval training to perform more sets of maximal exercise without a decrease in power output compared with inexperienced cyclists.

Key words: *maximal glycolytic exercise, lactic acidosis, training load.*

Introduction

In recent years, there has been a great deal of research on the effects of all-out interval training (AIT) and high-intensity interval training (HIT). All-out interval training, based on glycolytic activity, consists of on repetitions of maximal intensity exercise, usually performed for about 45 s and interspaced with rest periods lasting from 30 s to 4 min (Billat, 2001; Tabata et al., 1997; Tschakert and Hofmann, 2013). Extending the recovery period may improve phosphocreatine resynthesis, allowing for increased power at the beginning of subsequent repetitions (Billat, 2001). This form of training has been found to improve indicators of physical capacity such as the removal of lactate from the blood, maximal oxygen uptake, maximal aerobic power, and submaximal exercise time at a fixed intensity (Bayati et al., 2011; Laursen and Jenkins, 2002; Tschakert and Hofmann, 2013).

Research has mostly concentrated on the effects of exercise sets composed of 6–8 repetitions (Billat, 2001; Tabata et al., 1997). During such exercise the oxygen deficit rapidly increases (Tabata et al., 1997) as do the concentrations of lactate, potassium, and hydrogen ions in the blood (Lindinger et al., 1992). The production of lactate, as well as the shift of ion concentrations between the interstitial compartment and blood

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plasma are considered the main factors underpinning long-term adaptive changes to all-out interval training (McKenna et al., 1997). However, anaerobic activity was found to decrease when an excessive number of repetitions were performed (Gaitanos et al., 1993). Therefore, it may be favorable to divide an exercise session into a series of sets composed of fewer repetitions (separated by periods of passive or active recovery) in order to repeatedly disrupt the acid–base balance during a single workout.

According to several studies, the onset of fatigue during high-intensity exercise that involves a glycolytic metabolism is the result of homeostatic imbalance (Gibson and Noakes, 2004; Hagberg, 1981; Hill and Lupton, 1923; Roelands et al., 2013). The accumulation of lactate can inhibit the release of Ca2+ from the sarcoplasmic reticulum into cytoplasm and thus decrease muscle contraction strength (Cairns, 2006). While a mild decrease in muscle pH (to 6.8–6.9 pH) would not significantly affect the amount of power muscle can generate, a severe acid–base imbalance mainly in fast twitch muscle fibers (pH below 6.7) can significantly reduce power output (Cairns, 2006). It has been theorized that hydrogen and phosphate ions weaken the mechanisms of cross-linking protein in building myofilaments (Cairns, 2006; Cooke et al., 1988). Additionally, the onset of fatigue during maximal exercise has been attributed to phosphocreatine depletion in muscle (Billat, 2001). The outflow of potassium ions from cells has also been linked to a decline in muscle power, as it causes depolarization and decreases the excitability of cell membranes (McKenna et al., 1997). Other factors influencing the onset of fatigue include the accumulation of metabolites such as adenosine monophosphate (AMP) and inosine monophosphate (IMP) (Gibson and Noakes, 2004). Since a metabolic rate and muscle power are regulated by the central nervous system (CNS), it has been suggested that the signals from peripheral receptors are integrated and provide information about the above-mentioned factors as well as the incidence of organ and tissue damage. Considering that the CNS controls the recruitment of motor units depending on the rating of perceived exertion (RPE), it may therefore reduce work output (Lambert et al., 2005; Tucker, 2009).

However, most of these peripheral factors are subject to quick recovery after exercise. The acid–base balance is restored within several minutes to approximately 1 h (Robergs et al., 2005; Yano et al., 2011), although its duration can be reduced if active recovery is used (Siegler et al., 2006). After maximal exercise, half of the phosphocreatine content is resynthesized by approximately 170 s (Hirvonen et al., 1987), although this depends on an individual's aerobic capacity (Billat, 2001). The concentrations of sodium, potassium, and chlorine ions in arterial and venous blood return to baseline values 10 min after exercise (McKenna et al., 1997). As a result, the above factors should not affect power output after cessation of a rest interval of several dozens of minutes. However, the results of previously conducted studies are lacking in data on changes in the distribution of work output and the acid–base balance when performing multiple sets of all-out interval exercise. This form of exercise generates repeated high levels of acid–base imbalance and metabolite accumulation as a result of increased glycolysis. Such physiological and biochemical changes stimulate adaptive changes in the body. However, it is important to determine the training volume of such exercise, both in terms of generated power output and the body's reaction to it.

Therefore, the aim of the present study was to determine the physiological response to subsequent sets of interval training exercise by comparing differences in cyclists experienced in all-out interval training and their peers unexperienced in AIT. It was hypothesized that, regardless of training experience, significant differences would not be found in work output or the concentrations of glycolytic metabolites between subsequent sets of interval exercise.

Material and Methods

Participants

Twenty-three mountain bike (MTB) cyclists, including members of the National Team and National Championship medalists, were recruited. They were divided into two groups depending on their experience of all-out interval training. The experienced group (E) included eleven cyclists who had been performing AIT for at least two years, whereas the inexperienced group (IE) consisted of twelve cyclists who had never trained with AIT. The study design was

approved by the Ethics Committee for Scientific Research of the University School of Physical Education in Wroclaw.

One week before the all-out interval exercise session, the participants were familiarized with the test protocol by performing one set of several repetitions of interval exercise as outlined in the Procedures section. On this day, a sample of arterialized capillary blood was drawn immediately after each repetition in order to determine blood lactate and hydrogen concentration. Participants whose lactate and hydrogen concentrations began to decrease before completing the 4th repetition were excluded from further participation.

Table 1 presents the participants' somatic characteristics and the selected variables assessing the physiological response after completing one set of interval exercise during familiarization. In this regard, no significant differences between the groups were found in any of the analyzed variables.

The cyclists had at least 4 years of training experience. Their training program was analyzed for the preceding 2 years in which both groups trained for 9 months per year. It involved three types of training composed of both aerobic and anaerobic cycling exercises.

- In the experienced cyclists these included (1) sprint interval training using several (3–6) sets of maximal intensity exercise, where each set comprised four 30 s repetitions interspersed with 90 s recovery periods. Each set was followed by 20–30 min of recovery performed at an intensity close to 60–70% of the maximal heart rate (HR_{max}). The second type of training (2) involved high intensity training of alternating exercise, with 5 min of high intensity exercise (90–100% of peak power output) followed by 15 min of medium-intensity exercise (60–70% of the HR_{max}), and (3) steady-state endurance training performed at an intensity of 80–90% power at the anaerobic threshold.
- In the inexperienced cyclists the training program involved (1) high intensity training of alternating exercise, with 5 min at a high intensity (90–100% of peak power output) followed by 15 min of medium-intensity exercise (60–70% of the HR_{max}), (2) training with alternating exercise, with 15–20 min at a high intensity (95–105% of power output at the anaerobic threshold) followed by 10–15 min of low- and medium-intensity exercise (50–70% HR_{max}), and (3) steady-state endurance training performed at an intensity of 80–90% of power at the anaerobic threshold.

HR_{max}, peak power output, and power output at the anaerobic threshold were determined for each cyclist using a progressive exercise test. Such tests were regularly performed by this group in order to determine the training load and volume. Throughout the training sessions, the heart rate and power were continually monitored using a S810 heart rate monitor (Polar Electro, Finland) and a power monitor (PowerTap System, United States). Each type of training was employed once a day in the order provided followed by one day of rest (i.e., first day – training 1, second day – training 2, third day – training 3, fourth day – day of rest, fifth day – training 1, etc.). Each training session lasted from 2 to 3 h. During the racing season all of the studied cyclists (experienced and inexperienced in interval training) participated in 17–21 races.

All of the cyclists followed a similar diet based on the recommendations of the American College of Sports Medicine (2009). Daily intake involved 6–10 g/kg of carbohydrates, 1.2–1.7 g/kg of protein, and 1.7–2.4 g/kg of fat. Additional supplements in the form of isotonic drinks, sports gels, and energy bars were used only during competition by both groups.

Procedures

The test protocol consisted of one all-out interval exercise session on an E894 cycle ergometer (Monark AB, Sweden). The load was set for each participant based on their body mass. Work and power output were controlled using MCE 2.0 software by monitoring crank speed. The session began with a 20 min warm-up, during which a load of 2 $W \cdot kg^{-1}$ of body mass was used for the first 5 min, increased to 3 $W \cdot kg^{-1}$ for the next 10 min, and then to 4 $W \cdot kg^{-1}$ for the last 5 min. Following the warm-up, an active rest was performed by cycling at a low intensity (1 $W \cdot kg^{-1}$) for 10 min. Afterwards, the all-out interval exercise session began. It was divided into successive sets of four 30 s repetitions using a load

of 7.5% body mass. Each repetition was followed by 90 s of active recovery (pedaling on the cycle ergometer without an external load). Arterial blood was drawn 3 min after the last (4^{th}) repetition in each sets and analyzed for pH using a RAPIDLab 348 blood gas system (Siemens Healthcare, Germany) and a Lactate Scout analyzer (SensLab, Germany) for lactate. After completing each set active recovery was performed by pedaling at 2.5 $W \cdot kg^{-1}$. Recovery lasted about 20–30 min and was dependent on the rate of recovery by the acid–base balance returning to baseline values (it was assumed that the pH of arterialized capillary blood should return to 7.3). Afterwards, the participant performed the next set.

The participants performed as many sets as possible until one of the following conditions was met:

– the lactate concentration in arterialized capillary blood decreased (drawn 3 min after finishing each set) by 10% compared with the highest value recorded during the training session,

– the H^+ concentration in arterialized capillary blood decreased (drawn 3 min after finishing each set) by 10% compared with the highest value recorded during the training session,

– total work output on the cycle ergometer decreased in the subsequent set by 10% compared with the highest value recorded during the training session,

– the participant refused to continue or was unable to perform all four repetitions.

The hydrogen ion concentration in arterialized capillary blood was calculated using the formula: $[H^+] = 10^{-pH}$; the result was multiplied by 10^9 in order to be presented in $nmol \cdot L^{-1}$. Total work output was based on the amount of work done in each set of the cycling exercise.

Statistical analysis

Statistical analysis was performed using Statistica 10.0 software (Statsoft). Friedman's ANOVA was applied after the arithmetic means and standard deviations were calculated. Comparisons between each successive set of interval exercise for each group were performed using the Wilcoxon test to determine the level of statistical significance. Significance was set at an alpha level of 0.01. Comparisons between the E and IE groups were made using the Mann–Whitney *U* test.

Results

The cyclists from group E performed significantly more ($p < 0.01$) sets than their peers in group IE (5.8 ± 1.2 vs. 4.3 ± 0.9 sets, respectively). Analysis was therefore performed on the data collected from the five sets completed by group E (each of the athletes performed at least this many sets) and the four sets completed by group IE (as each of the athletes performed this many sets).

In group E, work output decreased significantly only when the last set was compared with the second one. No statistically significant differences were found in the post-exercise concentration of hydrogen ions between the first and second sets, whereas a significant decrease was found for this variable in the third, penultimate, and last sets. Post-exercise lactate concentration decreased significantly only in the last set with respect to the penultimate set (Table 2).

In group IE, total work output decreased with each successive set as did post-exercise hydrogen ion concentration. Post-exercise lactate ion concentration decreased significantly only in the last set when compared with the first set (Table 2).

Significant differences between groups were found in work output performed in the last set (Table 2).

No significant differences were found in either group for pH values and hydrogen ion concentrations measured immediately before each successive set of exercise (Table 2).

In group E, work output in the first repetition of each set did not change significantly in the first three sets, but decreased in a) the penultimate set when compared with the second set and b) the last set compared with the first, second, and third sets. Work output in the second repetition of each set decreased in a) the third set relative to the second set, b) the penultimate set compared with the second set, and c) the last set compared with the second, third, and penultimate sets. Work output of the third repetition did not change in the subsequent set. Work output was larger in the fourth repetition in a) the penultimate set compared with the first and second sets and in b) the last set relative to the first and second sets (Table 3).

In group IE, work output in the first

repetition of each set decreased in a) the second and penultimate sets compared with the first set and b) the last set relative to each of the preceding sets. In the second repetition of each set, work output decreased with each subsequent set. Work output in the third repetition was significantly lower in the penultimate and last sets compared with the first and second sets. No statistically significant differences were found in the fourth repetition of each subsequent set (Table 3).

Table 1

Basic somatic and physiological characteristics of the participants experienced and inexperienced in all-out interval training

Group	Age [years]	Body height [cm]	Body mass [kg]	W_{peak} [kJ]	H^+ [nmol·l^{-1}]	LA^- [mmol·l^{-1}]
Experienced	24.6 ± 3.8	174.3 ± 8.5	67.1 ± 6.1	18.9 ± 1.8	83.5 ± 10.1	16.4 ± 2.0
Inexperienced	22.2 ± 4.5	176.4 ± 7.9	65.7 ± 7.2	18.1 ±2.3	84.9 ±11.7	16.9 ±1.6

W_{peak} – work output in the first repetition of interval exercise; H^+ – hydrogen ion concentration; and LA^- – lactate concentration measured after one set of four repetitions of interval exercise; data are presented as mean ± standard deviation

Table 2

Total work output in each set and post-exercise hydrogen and lactate ion concentration after each set of interval exercise in the experienced and inexperienced groups

	Experienced group				
	1st set	2nd set	3rd set	PU set	U set
W_{tot} [kJ]	66.9 ±9.6	67.8 ±9.5	67.1 ±9.6	67 ±9.1	66.1 ±9.6*2
H^+ [nmol·l^{-1}]	84.5 ±9	84.5 ±12.2	81.1 ±11*2	78 ±9.8*1,2	70.8 ±11.5*1,2,3,P
LA^- [mmol·l^{-1}]	16.4 ±2.1	16.6 ±2.1	15.8 ±1.4	17.1 ±2.3	15.5 ±2*P
H^+ b [nmol·l^{-1}]	40.8 ± 1.1	41.5 ± 1.8	41 ± 1.2	42.4 ± 2.5	41.9 ± 1.5
pH before	7.389 ± 0.012	7.381 ± 0.018	7.386 ±0.013	7.373 ± 0.024	7.377 ± 0.015
	Inexperienced group				
	1st set	2nd set		PU set	U set
W_{tot} [kJ]	65 ±10.8	63.3 ±11.2*1		62.1 ±11.7*1,2	59.7 ±11.4*1,2,P,E
H^+ [nmol·l^{-1}]	85.2 ±11.5	80 ±10.7*1		76.3 ±11.7*1	66.5 ±10.5*1,2,P
LA^- [mmol·l^{-1}]	16.4 ±1.2	16.2 ±0.8		15.7 ±1.8	14.4 ±2.4*1
H^+ b [nmol·l^{-1}]	41.1 ± 0.5	41.6 ± 1.7		41.9 ± 1.9	42.8 ± 2.7
pH before	7.385 ±0.005	7.381 ±0.017		7.377 ±0.019	7.369 ±0.026

PU set – penultimate set; U set – ultimate set; W_{tot} – total work output; H^+ – blood hydrogen ion concentration measured after each set; LA^- - blood lactate ion concentration measured after each set;

H^+ b – *blood hydrogen ion concentration measured immediately before a set* ;

pH before – $-\log_{10}H^+$ *measured immediately before a set*;

^{*1} – $p > 0.01$ compared with the first set;

^{*2} – $p > 0.01$ compared with the second set;

^{*3} – $p > 0.01$ compared with the third set;

^{*P} – $p > 0.01$ compared with the penultimate set;

^{*E} – $p > 0.01$ compared with the experienced group;

data are presented as mean ± standard deviation.

Table 3

Work output in the first, second, third and fourth repetition of each subsequent set of interval exercise; in the experienced and inexperienced groups

	Experienced group				
	1st set	2nd set	3rd set	PU set	U set
W1 [kJ]	19.3 ±2.3	19.3 ±2.9	18.7 ±2.7	18.4 ±2.8*2	18.1 ±2.6*1,2,3
W2 [kJ]	17.1 ±2.4	17.4 ±2.3	17.1 ±2.4*2	16.8 ±2.4*2	16.3 ±2.5*2,3,P
W3 [kJ]	15.8 ±2.6	16 ±2.4	15.9 ±2.5	15.9 ±2.2	15.9 ±2.4
W4 [kJ]	14.8 ±2.8	15.1 ±2.3	15.4 ±2.4	15.8 ±1.9*1,2	15.8 ±2.2*1,2
	Inexperienced group				
	1st set	2nd set		PU set	U set
W1 [kJ]	18.6 ±2.2	17.3 ±2.4*1		16.9 ±2.6*1	15.9 ±2.7*1,2,P
W2 [kJ]	16.5 ±2	15.9 ±2*1		15.5 ±2.2*1,2	14.7 ±2.1*1,2,P
W3 [kJ]	15.3 ±2.4	15.2 ±2.2		14.8 ±2.2*1,2	14.4 ±2.2*1,2
W4 [kJ]	14.3 ±2.3	14.6 ±2.4		14.6 ±2.2	14.4 ±2.2

*PU set – penultimate set; U set – ultimate set; W:1,2,3,4 – work output in the: first, second, third and fourth repetition of each subsequent set; *1 – p > 0.01 compared with the first set; *2 – p > 0.01 compared with the second set; *3 – p > 0.01 compared with the third set; *P – p > 0.01 compared with the penultimate set; data are presented as mean ± standard deviation*

Discussion

The results of the study showed that the group of cyclists who had trained with all-out interval training (group E) performed more sets without a significant loss in power. This may be explained by these athletes having more training experience and the ability to better anticipate fatigue, thereby allowing them to maintain muscle power levels throughout the test. Several independent research groups have demonstrated that repeating maximal efforts resulted in a change in the distribution of power output over time, with a decrease in the beginning and then an increase in the final phase of exercise (Gee et al., 2013; Lambrick et al., 2013; Thomas et al., 2012). Subsequent repetitions have been found to be performed at lower physiological cost, i.e. VO_2 and RER, allowing for greater power output at the end of exercise (Jones et al., 2013) and shorter distance times (Lambrick et al., 2013).

Several authors (Gibson and Noakes, 2004; Roelands et al., 2013) have proposed that power output is controlled by the CNS in order to protect the body from excessive homeostatic imbalance. This process was termed teleoanticipation, where the body 'anticipates' how much power can be expended depending on previous experience and a feedback mechanism in response to such afferent information as respiratory gas partial pressure, hydrogen ion and electrolyte concentrations, muscle condition, body temperature, and a subjective perception of fatigue. With regard to the experienced cyclists, this may explain their variability in work output in the subsequent set of interval exercise. With the use of 'anticipation', the first repetition in each set is performed with less work only to increase in the last repetitions of subsequent sets. Nonetheless, total work output did not change suggesting a slower onset of fatigue in group E over group IE.

Another factor that may have affected the cyclists' ability to repeat similar sets of maximal exercise was an increased tolerance to lactic acidosis. This is evidenced by the lack of differences in post-exercise hydrogen ion concentrations between the first and second sets in the experienced group of cyclists and the smaller magnitude of changes in subsequent sets when compared with group IE.

Our findings suggest that among individuals who begin interval training, the

decrease in work output may be attributed to a decrease in the glycolytic metabolism. This can be inferred by the reduced hydrogen ion concentration in subsequent sets of exercise, which may have resulted in a lower rate of glycolysis. The cause of this decline may be a reduction in the availability of glycosidic residues (Hargreaves and Richter, 1988; Jensen and Richter, 2012). A combination of short maximal workloads at 60% of VO_{2max} showed greater rates of glycogen utilization (Gollnick et al., 1974). In the present study, the decrease in H^+ ion concentration in group E was smaller than in group IE. This suggests a smaller decrease in the rate of glycolysis and phosphorolysis of glycogen, which may have been caused by greater muscle glycogen stores in the cyclists from group E as a result of regularly performed all-out interval training. Additionally, the concentration of AMP in muscle has been found to increase during maximal exercise (Weicker et al., 1990), resulting in an increase of muscle glycogen stores through the regulation of kinase activity by AMP (AMPK) (Hunter et al., 2011; McBride and Hardie, 2009).

A decrease in hydrogen ion concentration and power output was observed already in the second set in group IE, while lactate ion concentration decreased only in the last (fourth) set. A similar time course was observed in group E, in which H^+ concentration decreased in the third set well before LA^- decreased (last – fifth set). According to Brooks (2007) and Cairns (2006), the quantity of lactate produced by glycogenolysis and glycolysis is proportional to changes in hydrogen ion concentration during exercise. However, the processes behind their removal are different (Cairns, 2006; McNaughton et al., 2008). McNaughton et al. (2008) identified three basic mechanisms regulating H^+ concentration, the first involved chemical buffers, the second was by pulmonary ventilation, and the third through renal function. Whereas lactate is oxidized by the mitochondria (involving the MCT1 and CD147 proteins, lactate dehydrogenase, and cytochrome oxidase) mainly in neurons, cardiomyocytes, slow twitch muscle fibers and the liver, and is also processed into glycogen through gluconeogenesis (Cairns, 2006; Gladden, 2008; Hashimoto and Brooks, 2008). The results of the present study may indicate that the processes involved in lactate clearance require more time than those involved in H^+ recovery as evidenced by the differences in the rate of change in these two metabolites. This indicates that H^+ is a more sensitive measure of assessing interval-training loads and the metabolic cost of exercise than lactate concentration. The reasoning for this lies in the fact that hydrogen ion concentrations decreased in parallel with power output in group IE. In the experienced group, hydrogen ion concentration decreased before a drop in power output, but when H^+ concentration decreased, power distribution changed in each set.

Conclusion

Cyclists experienced in interval training were able to repeatedly induce elevated levels of lactic acidosis. Power output distribution changed with decreased acid–base imbalance: power decreased in the initial repetitions of a set, but increased in the final repetitions. In this way, this group could compensate for a decreased anaerobic metabolism. The above factors allowed cyclists experienced in interval training to perform more sets of maximal exercise without a decrease in power output when compared with cyclists of similar exercise capacity inexperienced in interval training.

Changes in power output distribution in all-out interval training can serve as a useful indicator of athletic performance in repeated high-intensity exercise. Concurrently, the observed change in power output distribution may be used as a non-invasive measure of decreased glycolytic activity.

References

American College of Sports Medicine, American Dietetic Association, Dietitians of Canada. Position of the American Dietetic Association, Dietitians of Canada, and the American College of Sports Medicine: nutrition and athletic performance. *J Am Diet Assoc*, 2009; 109: 509–527

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

Billat LV. Interval training for performance: A scientific and empirical practice. *Sports Med*, 2001; 31(2): 75-90

Brooks GA. Lactate. Link between glycolytic and oxidative metabolism. *Sports Med*, 2007; 37(4-5): 341-343

Cairns SP. Lactic acid and exercise performance. Culprit or Friend? *Sports Med*, 2006; 36(4): 279-291

Cooke R, Franks K, Luciani GB, Pate E. The inhibition of rabbit skeletal muscle contraction by hydrogen ions and phosphate. *J Physiol*, 1988; 395: 77-97

Gaitanos GC, Williams C, Boobis LH, Brooks S. Human muscle metabolism during intermittent maximal exercise. *J Appl Physiol*, 1993; 75: 712-719

Gee TI, French DN, Gibbon KC, Thompson KG. Consistency of pacing and metabolic responses during 2000-m rowing ergometry. *Int J Sports Physiol Perform*, 2013; 8(1): 70-76

Gibson AC, Noakes TD. Evidence for complex system integration and dynamic neural regulation of skeletal muscle recruitment during exercise in humans. *Br J Sports Med*, 2004; 38: 797-806

Gladden LB. A lactatic perspective on metabolism. *Med Sci Sports Exerc*, 2008; 40(3): 477-485

Gollnick PD, Piehl K, Saltin B. Selective glycogen depletion pattern in human muscle fibres after exercise of varying intensity and varying pedalling rates. *J Physiol*, 1974; 241(1): 45-57

Hagberg M. Muscular endurance and surface electromyogram in isometric and dynamic exercise. *J Appl Physiol Environ Exerc Physiol*, 1981; 51(1): 1-7

Hargreaves M, Richter EA. Regulation of skeletal muscle glycogenolysis during exercise. *Can J Sport Sci*, 1988; 13(4): 197-203

Hashimoto T, Brooks GA. Mitochondrial lactate oxidation complex and an adaptive role for lactate production. *Med Sci Sports Exerc*, 2008; 40(3): 486-494

Hill AV, Lupton H. Muscular exercise, lactic acid, and the supply and utilization of oxygen. *Quart J Med*, 1923; 16: 135-171

Hirvonen J, Rehunen S, Rusko H, Haerkoenen M. Breakdown of high-energy phosphate compounds and lactate accumulation during short supramaximal exercise. *Eur J Appl Physiol*, 1987; 56: 253-259

Hunter RW, Treebak JT, Wojtaszewski JF, Sakamoto K. Molecular mechanism by which AMP-activated protein kinase activation promotes glycogen accumulation in muscle. *Diabetes*, 2011; 60: 766–774

Jensen TE, Richter EA. Regulation of glucose and glycogen metabolism during and after exercise. *J Physiol*, 2012; 590(5): 1069-1076

Jones HS, Williams EL, Bridge CA, Marchant D, Midgley AW, Micklewright D, Mc Naughton LR. Physiological and psychological effects of deception on pacing strategy and performance. A review. *Sports Med*, 2013; 43(12): 1243-1257

Lambert EV, Gibson AC, Noakes TD. Complex systems model of fatigue: integrative homoeostatic control of peripheral physiological systems during exercise in humans. *Br J Sports Med*, 2005; 39: 52-62

Lambrick D, Rowlands A, Rowland T, Eston R. Pacing strategies of inexperienced children during repeated 800m individual time-trials and simulated competition. *Pediatr Exerc Sci*, 2013; 25(2): 198-211

Laursen PB, Jenkins DG. The scientific basis for high-intensity interval training. *Sports Med*, 2002; 32(1): 53-73

Lindinger MI, Heigenhauser GJ, McKelvie RS, Jones NL. Blood ion regulation during repeated maximal exercise and recovery in humans. *Am J Physiol*, 1992; 262: 126-136

McBride A, Hardie DG. AMP-activated protein kinase – a sensor of glycogen as well as AMP and ATP? *Acta Physiol*, 2009; 196: 99-113

McKenna MJ, Heigenhauser GJF, McKelvie RS, MacDougall JD, Jones NL. Sprint training enhances ionic

regulation during intense exercise in men. *J Physiol*, 1997; 501(3): 687-702

McNaughton LR, Siegler J, Midgley A. Ergogenic effects of sodium bicarbonate. *Current Sports Medicine Reports*, 2008: 7(4): 230-236

Robergs R, Hutchinson K, Hendee S, Madden S, Siegler J. Influence of pre-exercise acidosis and alkalosis on the kinetics of acid-base recovery following intense exercise. *Int J Sport Nutr Exerc Metab*, 2005; 14: 59-74

Roelands B, de Koning J, Foster C, Hettinga F, Meeusen R. Neurophysiological determinants of theoretical concepts and mechanisms involved in pacing. *Sports Med*, 2013; 43: 301-311

Siegler JC, Bell-Wilson J, Mermier C, Faria E, Robergs RA. Active and passive recovery and acid-base kinetics following multiple bouts of intense exercise to exhaustion. *Int J Sport Nutr Exerc Metab*, 2006; 16: 92-107

Tabata I, Irisawa K, Kouzaki M, Nishimura K, Ogita F, Miyachi M. Metabolic profile of high intensity intermittent exercise. *Med Sci Sports Exerc*, 1997; 29: 390-395

Thomas K, Stone MR, Thompson KG, Gibson AC, Ansley S. Reproducibility of pacing strategy during simulated 20-km cycling time trials in well-trained cyclists. *Eur J Appl Physiol*, 2012; 112: 223-229

Tschakert G, Hofmann P. High-intensity intermittent exercise: methodological and physiological aspects. *Int J Sports Physiol Perform*, 2013; 8(6): 600-610

Tucker R. The anticipatory regulation of performance: the physiological basis for pacing strategies and the development of a perception-based model for exercise performance. *Br J Sports Med*, 2009; 43: 392-400

Weicker H, Hageloch W, Luo J, Mueller D, Werle E, Sehling KM. Purine nucleotides and AMP deamination during maximal and endurance swimming exercise in heart and skeletal muscle of rats. *Int J Sports Med Supp*, 1990; l2: 68-77

Yano T, Matsura R, Arimistu T, Yamanaka R, Lian CS, Yunoki T, Afroundeh R. Ventilation and blood lactate levels after recovery from single and multiple sprint exercise. *Biol Sport*, 2011; 28: 233-237

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