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EFFECTS OF GLYCOLYTIC-BASED INTERVAL TRAINING ON ANAEROBIC CAPACITY IN SOCCER PLAYERS

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ABSTRACT
Purpose. The aim of this study was to determine the magnitude of changes in anaerobic endurance in response to a training protocol targeting glycolytic capacity. **Methods.** The study involved 24 soccer players from two U-18 teams. One team served as an experimental (E) group the other a control (C). Besides standard soccer practice performed by both groups, an interval training protocol was administered to the experimental group twice a week (15 sessions). One training repetition involved running a soccer-specific course. Repetition time was equal to 15 s interspersed with 45 s passive recovery. Total number of repetitions was determined by the ability to maintain target time (power) in subsequent repetitions. A 5% reduction in the distance covered (m) compared with the first repetition ended a set. The number of sets was based on the ability of player to maintain target time per repetition. Rest interval between sets was 15 min. Anaerobic performance was assessed before and after the 8-week protocol by the Wingate test in which arterial blood gases, blood lactate concentration, and respiratory variables on a breath-by-breath basis were measured. **Results.** Distance covered in group E in the first training session was 470.38 ± 77.82 m and 1182.31 ± 164.44 m in the last session. Post-intervention total work (273.63 ± 18.32 to 284.98 ± 15.76 J/kg) and maximum power (13.28 ± 1.43 to 14.14 ± 1.25 W/kg) significantly increased in the Wingate test. Statistically significant increases in lactate concentration (10.64 ± 1.54 and 12.72 ± 1.59 mmol/l) and lower blood pH (7.21 ± 0.03 and 7.19 ± 0.02) were also observed. No significant changes in any of the above variables were observed in group C. **Conclusions.** Interval training develops glycolytic capacity but with large inter-individual variability.

Key words: interval training, anaerobic endurance, glycolytic capacity, soccer

Introduction

In-game association football or soccer performance involves explosive starts, high-, moderate-, and low-intensity running, and jogging over distances of 9–13 km [1, 2]. Players also perform 150–250 intense anaerobic actions during a game [3], ranging from dribbling, kicking, sliding, or performing fakes or feints, which are crucial in deciding game outcome. Gameplay is acyclical and unstructured, entirely dependent on game laws, player positioning, tactical assumptions, and opponent dynamics [2]. This intermittent nature of soccer requires players to continually perform such actions with little to no recovery, leading to the onset of fatigue and reduced performance. The literature indicates that competitive level is associated with high-intensity activity, in which elite athletes are able to perform at a similar or even higher intensity level in the last half of a match than lower level players in the first half [1] or perform more high-intensity work in the post-season than pre-season or mid-season [2]. Additionally, better ranked players were found to perform more actions involving ball play [4]. Enhanced match performance in elite players was explained by increased endurance training later in the season, which is minimized in earlier stages due to the two games per week format typical of national cups, Champions League, or Europa League [2].

Training in sports requires constant experimentation in order to provide ever more effective methodologies to improve physiological function and therefore athletic performance [5]. The direction and magnitude of training effects are dependent on training load, itself a function of training volume and intensity. While training volume is an easily quantifiable variable, intensity is more difficult to ascertain particularly in acyclic movement structures. This is readily observant in soccer, where training volume can be controlled by training duration or number of repetitions, an objective measure of intensity in certain game-related exercises would be difficult without a highly specialized methodological approach. This makes the registration of training loads in soccer problematic, confounding the design of an empirically-based training program that should, in all cases, be based on objective data.

One of the major challenges in training is that a given external load does not induce the same physiological changes in a group of even homogeneous athletes. Individual response is grounded in not just athletic potential but factors either related or unrelated to the training process, including fitness level, previous training experience, and others. For this reason, training loads should be applied on an individual basis. Such an approach would aid monitoring the training load and minimize the effects of unwarranted stimuli [5]. Surprisingly, there is limited information on the application of individualized training loads in soccer. This is undoubtedly due to the inherent structure of soccer gameplay. However, the inclusion of cyclical isolated exercises in a training pro-

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Table 1. Literature review of glycolytic-based training effects on physiological and ergometric variables

Sample	Training protocol	Physiological response	Ergometric performance	Source
8 university students	3–5 × 30 s reps with 4 min recovery, 3 × week for 4 weeks	↑9.6% VO_2max (incremental exercise test)	↑10.3% Pmax (Wingate) ↑17.1% Pmean (Wingate) ↑16.8% Ptot (Wingate)	Bayati et al. [27]
8 recreational athletes	4–6 × 30 s maximal efforts interspersed with 4 min recovery, 3 × week for 6 weeks	↑~25% GLUT4 ↑~30–530% MCT1 ↑~15–200% MCT4	↓~13% decrease in time to pedaling 250 kJ on a cycle ergometer	Burgomaster et al. [6]
22 soccer players	12–15 × 15 s reps at 120% VO_2max with 15 s recovery, 1× week 12–15 × 40 m sprints with 30 s recovery, 1 × week for 10 weeks	↑8.1% maximal oxygen delivery	↓3.5% 40 m running time	Dupont et al. [29]
21 soccer players	6 × 40 m sprints in 3 sets, 2 × week for 7 weeks	↑3% VO_2max	↑Yo-Yo Test ↑RSA 6 × 40 m test	Ferrari Bravo et al. [35]
8 recreational athletes	4–6 × 30 s sprints at 250% VO_2max with 4 min recovery, 6 × 14 days	↑COX ↑muscle buffer capacity	↓time to complete 50 kJ and 750 kJ on a cycle ergometer	Gibala et al. [31]
7 untrained men	4–10 × 30 s pedaling bouts with 3–4 min recovery, 3 × week for 7 weeks	↑PCr (at rest) ↑glycogen (at rest) ↑blood La and H+ in 130% VO_2max efforts to exhaustion ↓muscle La and H+ in 130% VO_2max efforts to exhaustion	↑efficiency in 130% VO_2max efforts to exhaustion	Harmer et al. [13]
9 junior soccer players	4 × 4 min 90–95% HRmax sprints with 3 min recovery, 2 × week for 8 weeks	↑10.8% VO_2max ↑16% LT ↑6.7% running efficiency	↑20% distance covered ↑100% number of match sprints ↑24.1% ball play → running speed → 1RM barbell bench press → 1RM barbell squat → kicking strength	Helgerud et al. [33]

9 endurance runners	8–12 × 30 s 93% intensity runs for 4 weeks		VO_2 measured at 11, 13, 14.5, 16 km/h running speeds decreased by 6.6, 7.6, 5.7, 6.4%, respectively	Iaia et al. [30]
22 university students	4–10 × 30 s cycle ergometer bouts with 2.5–4 min recovery, 3 × week for 7 weeks	increases of ~56% Hex ~49% PFK ~36% CS ~29% MDH ~65% SDH ~8% VO_2max (incremental exercise test)	Exercise test involved 4 × Wingate tests with 4 min recovery ↑Pmax in the 2,3, and 4 Wingate repetition ↑Ptot in the 2,3, and 4 Wingate repetition	MacDougall et al. [19]
8 untrained men	4–10 × 30 s pedaling bouts with 3–4 min recovery, 3 × week for 7 weeks	↑~16% VE (incremental exercise test) ↑~9% VCO_2 (incremental exercise test) ↑~15% VO_2max (incremental exercise test) ↑~20% VO_2 at VT1 ↑~9% VO_2 at VT2 → VE (30 s sprint) → VCO_2 (30 s sprint) → VO_2 (30 s sprint) ↑VCO_2 (post-30 s sprint) ↑VO_2 (post-30 s sprint)	↑~4% Pmax (Wingate) ↑~12% Ptot (Wingate) ↓~9% FI (Wingate) ↑~10% Ptot (incremental exercise test)	McKenna et al. [20]
11 soccer players	4 × 4 min 90–95% HRmax with 3 min recovery, 2 × week for 10 weeks	↑9% VO_2max		McMillan et al. [34]
5 university students	2–7 × 15 s bouts with 45 s recovery and 2–7 × 30 s bouts with 12 min recovery, daily for 14 days	↑31% PCr ↑32% Glycogen ↓muscle La post-Wingate test ↑blood La post-Wingate test ↑44% CK activity ↑45% LDH activity ↑100% PFK activity ↑38% CS activity ↑60% HADH activity ↑11% VO_2max (incremental exercise test) ↑28% VO_2 (Wingate)	→ Pmax (Wingate) → Pmean (Wingate) ↑Pmax (incremental exercise test)	Rodas et al. [22]

↑ – increase, ↓ – decrease, → – no change

gram may provide researchers and coaches an objective and accurate means for improving performance and skills in soccer.

Glycolytic efforts are termed as such due to the energy requirements met by the anaerobic glycolysis system. This process accelerates the amount of available ADP/ATP, Ca^{+}, and AMP in muscle cytoplasm. The main energy substrate of glycolytic metabolism is naturally muscle glycogen, which may be substituted in small amounts by blood glucose [6]. The speed at which energy is delivered by the glycolytic system is less efficient than by kinase reactions but surpasses that of aerobic metabolism. However, anaerobic processes (excluding ATP resynthesis) lead to the accumulation of lactic acid and ammonia and inorganic phosphate (Pi) ions. Lactic acid is then converted to lactate (La^{-}) that, with excess hydrogen ion concentration (H^{+}), lowering pH and causing a momentary acid–base imbalance [6]. This decrease in muscle pH following high-intensity exercise contributes to the feeling of fatigue [3].

The metabolic pathways used to provide energy are both time and intensity-related and can be classified according to their production capacity. The first aspect concerns power, itself a reflection of how quickly energy can be released from outset to peak capacity (rate of ATP production), whereas the second concerns capacity outright (total ability by way of available substrates to produce ATP). In this way three metabolic energy systems can be extracted, the phosphagen, glycolytic, and aerobic systems [7].

The phosphagen system fulfills the role of providing energy in short-term, intense activities. During 30 s of maximal-intensity effort, the contribution of the phosphagen and glycolytic systems is approximately 24% and 70% [8], respectively, whereas the aerobic system is responsible for 16–40% of cellular energy [9, 10]. Continued exercise or subsequent repetitions of high-intensity activity, performed without adequate recovery, are increasingly dominated by aerobic processes, which is concomitant with a decrease in power [8]. While creatine phosphate can be resynthesized within 2 min of post-exercise recovery, a continual decrease in power is observed due to the increased levels of glycolytic metabolites. This post-exercise accumulation interferes with acid–base homeostasis.

Acid–base homeostasis is in effect a reflection of H^{+} concentration. The alkalinity or pH of blood remains relatively stable at rest, with the pH of arterialized and venous capillary blood at 7.40 and 7.35, respectively. A decrease in pH indicates an increase in H^{+} concentration and is a condition called acidosis. The condition of acidosis is associated with weakened muscle contraction strength due to impaired glycolysis, slowed calcium and troponin ion binding, reduced sarcoplasmic reticulum Ca^{2+} ATPase and calcium reuptake, and decreased enzymatic activity such as phosphofructokinase. Prolonged high-intensity exercise may decrease muscle pH to 6.2 [11] and blood pH to 6.8, whereby it has been assumed that performance at these pH is associated with high exercise capacity [5]. One of the most important factors in this regard is pulmonary ventilation (VE). In acidosis, CO_2 is eliminated by the electrolyte HCO_3^{-} and by increased VE, thereby reducing the partial pressure of CO_2 in arterial blood. This simplified explanation of respiratory compensation is not entirely complete and also should include the role of renal function in restoring acid–base balance.

Among the many goals of training, disturbing the acid–base balance is vital in order improve performance at ever-increasing levels of acidosis. One training modality that can induce this physiological response is interval training, which involves a series of short high-intensity exercises interspersed with incomplete recovery periods. Depending on the training goal, the duration of a single repetition can range from a few seconds to a few minutes and involve a wide range of repetitions [12]. Interval training was found to significantly develop both aerobic and anaerobic capacity [12, 13].

An interval training protocol targeting glycolytic capacity would need to adopt a load that could elicit maximum activation of the anaerobic pathway. Research has indicated that such an effect may be obtained by adopting 15–120 s repetitions interspersed with recovery periods at a work-to-rest ratio of 2:1 or higher [12]. The ratio is important in that 1:1 ratio was found to elicit improved oxidative capacity [12]. The exact duration of the recovery periods is dependent on how quickly the body regenerates in order to force that the next exercise repetition is dominated by glycolytic metabolism [14]. Common measures of monitoring the physiological effects of interval training are lactate concentration and blood pH level, where a plateauing of lactate level or decrease in pH is treated as a signal to interrupt subsequent exercise repetitions. This strategy is also applied in determining the number of sets performed in one training session. Another method of determining interval training load is by monitoring power output and ending exercise once a continual decrease is observed.

In line with most exercise programs, an interval training protocol targeting glycolytic capacity should be continued until no more progress is registered. For this purpose, a number of ergometric and physiological variables have been selected to study the time course of interval training effects. Among the ergometric variables, one indicator could involve maximizing the time and/or intensity of a given exercise. Among the most suitable physiological variables, Zatoń [14] showed that the increases in VE, HR, and H^{+} over subsequent training sessions could also serve as useful indicators of training effects. These measures are particularly well-suited as they can also provide information on current fitness level (aerobic or anaerobic) and are easily measurable.

The applicability of exercise testing to this effect has been successful in monitoring the training adaptations

by providing both qualitative and quantitative data. One of the most commonly applied exercises in this regard is the 30-s Wingate test performed on a cycle ergometer.

It involves measuring respiratory function and acid–base balance, the former described by analyzing the composition and volume of inspired and expired air [5]. In the assessment of anaerobic glycolytic training, variables that have been most commonly applied include VO_2, oxygen debt, HR, and recovery HR, and the increase in magnitude of these variables has diagnostic value [14].

The literature is rich with studies on the effects of various glycolytic training protocols on fitness and condition (Table 1). Physiologic responses and long-term adaptations include improvements in energy production, tolerance to exercise-induced metabolic acidosis, blood buffer capacity, and mechanical efficiency and greater activity of key enzymes that catalyze the synthesis of ATP [6, 13, 15–25].

Furthermore, current research has confirmed that glycolytic training increases energy substrate levels [15, 18, 22, 26], such as a 31% and 32% increase in PCr and glycogen stores in response to 14 days of interval training [22]. These authors suggested that the cause of increased PCr concentration may lie in the second phase of interval training (in this case repeated 30-s maximal bouts interspersed with 12-min intervals). The authors credit such a long recovery period with allowing PCr stores to be completely replenished in comparison to the first phase of training in which 15-s sprints were split up with 45-s of recovery. In turn, Gibala et al. [18] reported an increase of 28% in glycogen content following just six interval training sessions. Barnett et al. [26] observed a 17% increase in resting muscle glycogen levels as a result of an 8-week training protocol composed of 30-s efforts interspersed with 3 min rest. Other studies, such as by Burgomaster et al. [6] found an increase in insulin-regulated glucose transporter (GLUT4) levels, which was considered to be an indirect indicator of augmented glycogen concentration following sprint interval training.

Of considerable value is the evidence that the anaerobic component of interval training elicits higher VO_2max [20, 22, 27], VCO_2, and VE [28, 29] values in maximal-intensity exercise but lower in submaximal efforts [13, 20, 22, 30]. For example, Iaia et al. [30] indicated that 4 weeks of interval training resulted in lowered oxygen uptake (ml/kg/min) by 6.6, 7.6, 5.7, 6.4% at 11, 13, 14.5, 16 km/h running speeds, respectively. They also obtained reduced VE and VCO_2, in a supramaximal exercise test (130% VO_2). However, the continuation of this test until volitional exhaustion was reached resulted in increased VE compared with pre-intervention values [13]. Bayati et al. [27] documented a 9.6% increase in VO_2max after 4 weeks (3 × week) of interval training (3–5 × 30 s reps with 4 min recovery 3 × week).

Escalated VO_2 in the Wingate test was also noted by Rodas et al. [22], but this finding was not concurred by McKenna et al. [20] who also investigated the effects of repeated 30-s sprint on VO_2. Here, increased VO_2 was observed only 11.3 s after training was completed. A similar observation was made with VE and VCO_2, which was used to explained delayed blood circulation to the lungs [20].

There is overall consensus in the literature that increases in VO_2, VE, and VCO_2 are associated with improved oxidative metabolism and augmented ability to counteract acid–base imbalance, both of which directly affect athletic performance [20, 22]. McKenna et al. [20] believe these increases are the result of expanded chemoreceptor sensitivity to changes in CO_2 and H^+ levels as evidenced by elevated VE and reduced partial pressure of CO_2. Other positive training adaptation associated with enhanced respiratory function include supplemented oxygen delivery caused by higher muscle blood flow and increased capillary density as well as improved mitochondrial enzyme activity [25, 26]. For the latter factor, one of the key regulators of mitochondrial biogenesis is the proliferator-activated receptor-gamma coactivator 1-alpha (PGC-1α), which was found to propagate as a result of both aerobic and anaerobic training protocols [31].

As was mentioned, the accumulation of glycolytic metabolites (La^-, H^+, ammonia, P_i) following high-intensity exercise leads to reduced muscle contraction strength [11, 32]. Sutton et al. [32] found that a decrease in blood pH (acidosis) induced by oral delivery of ammonium chloride (NH_4CL) resulted in a significant lower exercise capacity. In their experiment, lowered blood pH co-occurred with decreased plasma La^- (explained by impaired cellular transport) which translated to a reduced glycolytic rate. This mechanism can be again explained by the limited tolerance of the body to acid–base imbalance and the post-exercise accumulation of metabolic wastes.

As was mentioned, training with a significant anaerobic component induces an increase or retention of blood La^- during maximal exercise but a decrease in blood La^- in submaximal efforts [13, 20, 22, 27]. However, there are reports of reduced as well as increased muscle La^- concentrations post-exercise [13, 22]. The conflicting findings may result from differences in training methodologies or even administered exercise tests [13, 22]. An increase in blood La^- and H^+ in exercise performed to exhaustion with a concomitant decrease in muscle may signify the effects of improved extracellular transport [13].

Nonetheless, the occurrence of acid–base imbalance, which is considered to be a significant cause of fatigue, can be negated by interval training-induced increases of blood and muscle buffer capacity. The literature has described the development of this capacity in terms of increased buffer quantity (e.g. hydrogen carbonate, phosphate) that prevents acidosis and therefore reduces the onset of fatigue [18, 24]. Edge et al. [17] showed a relationship between buffer capacity and the ability to perform repeated maximal efforts after minimal recovery.

Another factor contributing to the increase of exercise

capacity following interval training is the increase in key enzymes responsible for the resynthesis of ATP in both aerobic and anaerobic processes. Studies have observed improved muscle metabolism by increased activity of creatine kinase (CK), mitogen kinase (MK), glycogen phosphorylase (PHOS), phosphofructokinase (PFK), lactate dehydrogenase (LDH), cytochrome oxidase (COX), citrate synthase (CS), succinate dehydrogenase (SDH), malate dehydrogenase (MDH), pyruvate dehydrogenase (PDH), and hydroxyacyl-CoA dehydrogenase (HADH) [18, 26].

Helgerud et al. [33] studied the effects of interval training (4 × 4 min 90–95% HRmax sprints with 3 min active recovery) on soccer performance, finding an 8-week intervention sufficient in increasing distance covered in a match by 20%. Analysis of in-game actions showed a 100% increase in the number of sprints and a 24% increase in ball play. No change in maximum power and speed was reported. McMillan et al. [34] adopted a similar study design, finding that soccer players also presented increased ball play, although they assessed this performance variable by adopting a soccer-specific track. This track involved not just running and dribbling with the ball exercises but also jump performance. Mean VO_2max significantly improved by 9% with a concurrent rise in maximum power.

The training effects described above were based, in a large part, on interval training with a significant aerobic component due to the prevalence of oxidative ATP resynthesis. Ferrari Bravo et al. [35] compared the effects of this training modality on repeated sprint ability (RSA) with an interval-training protocol consisting of 40 m maximal shuttle runs (4 × 10m or 2 × 20 m). They observed that the increase in aerobic capacity was similar between the two conditions, but that anaerobic capacity significantly increased only in the latter group.

Thomassen et al. [36] investigated the effects of 2 min efforts at 90% HRmax and 25–30 s maximal runs in soccer players. Interestingly, 2 weeks of this protocol improved aerobic and anaerobic capacity but did not induce any changes in running speed.

Considering the structure of soccer and soccer training, interval training appears to be particularly suited towards improving soccer-specific performance. This is partly due to the critical role all three metabolic pathways serve in this sport, where interval training can significantly develop the glycolytic and aerobic systems without compromising phosphagen system [33–36].

There is still a lack of information in the literature on the effects of glycolytic-based interval training and individualized training loads on soccer performance. Data accrued on this subject may aid the training process particularly by incorporating cyclical exercises that are easy to measure and monitor. Such an approach can provide a wide range of information on training load and expand existing knowledge.

Therefore, the aim of the present study was to assess the development of anaerobic endurance in response to an interval training protocol targeting glycolytic capacity. We hypothesized that (1) such a protocol would result in improved glycolytic capacity as measured by work output across subsequent training sessions and (2) the direction of changes in anaerobic endurance would be equal across subjects with differences noted only in the amplitude of changes.

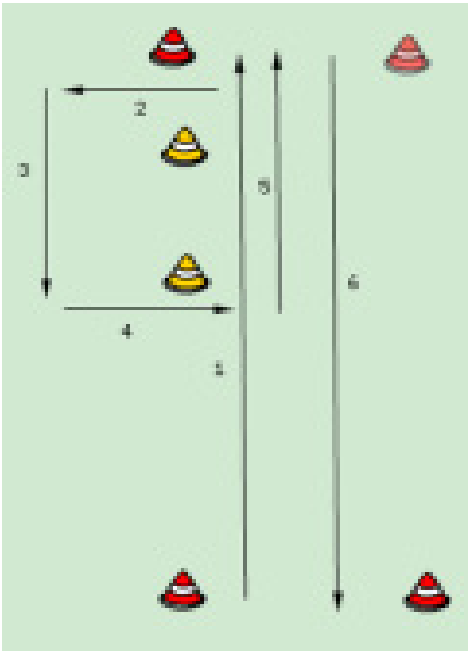
Material and methods

The study involved 24 soccer players belonging to two U-18 youth teams in the Dolnośląsk League. The members of one team were designated as an experimental group (n = 13, age 16.62 ± 0.87 years, mass 69.45 ± 5.99, height 179.08 ± 5.53 cm) and the other team as a control group (n = 11, age = 16.73 ± 1.1 years, mass = 71.08 ± 8.52 kg, body height = 176.55 ± 5.89 cm). No significant anthropometric differences were noted between the experimental (E) and control (C) groups and the mean height and mass values were similar to published height and weight norms in the appropriate age category.

While both groups attended standard soccer practice during the study duration (8 weeks), the training of group B was supplemented with glycolytic-based interval training.

Earlier research on the effects of interval training over a period of 2–10 weeks (sessions held every 24–72 h) in untrained individuals indicated that a 24 interval was too short in order to induce improved endurance capacity and that a 48 h interval was more suitable [2]. In populations with external commitments, particularly athletic, the most common solution adopted in the literature was to perform two interval training sessions per week as a complement to existing training (Table 1). This solution was credited as offering the necessary rest.

The present study adopted a similar protocol, with group E attending two interval training sessions per week with a minimum interval of 48 h that supplemented their regular soccer training. The training stimulus in group E was running a soccer-specific track as illustrated in Figure 1. The course was to be completed within 15 s and 45 s recovery was provided between each repetition. Recovery was passive as the literature shows that active recovery is not as effective when repeating maximal bouts of less than 1 min. The number of repetitions per set was dependent on the ability to maintain power (15-s time limit) in subsequent repetitions. A decrease of greater than 5% in the covered distance compared with the first repetition ended the set. Afterwards, 15 min of rest was provided before the training protocol was repeated. The number of sets, in turn, was also dependent on the ability to maintain power across subsequent sets, although they remained in the range of 2 to 4 sets. As a maximum of 3 min active recovery is known to accelerate the restitution, we included such a component in



1 – 48 m run, 2 – left shuffle, 3 – 3 m backwards run, 4 – right shuffle, 5 – 5 m run, 6 – continual run until time expired (15 s)

Figure 1. The soccer-specific track

the 15 min period of rest by administering low-intensity exercise perfecting soccer technique or coordination. The exercises were varied and modified over the 8 week period in order to negate monotony as well as provide the participants with appropriate soccer-specific training.

The design of the running protocol was based on the movement pattern present in soccer, in which players during an average match perform approximately 1100 changes in direction with and without the ball [37]. Lakomy et al. [37] indicated that stopping immediately after a sprint raised the physiological cost of a given exercise, in effect lowering the power of subsequent physical activity. They suggested that the inclusion of this type of action in ice hockey or soccer training is necessary in order to promote beneficial adaptations. The present protocol did not include running with a soccer ball in order to control for the distance covered. Almost all exercises that involve dribbling or running with a ball incur significant deviation due to imperfect technique. For this reason the present study focused only on acyclical running although actions that are commonly performed in football, including the long-distance sprint (sections 1, 6), immediately acceleration (section 5), turns in various directions (between each section), and shuffling and running backwards (sections 2, 3, 4). Such an approach would imitate in-game movement patterns while also allowing for analysis of individual development of each skill.

The anaerobic capacity of both groups was measured pre- and post-intervention using the Wingate Anaerobic Test. Blood gas was measured before and after the test as was lactate concentration (only post-test). Testing was performed in laboratory conditions at the Exercise Laboratory at the University School of Physical Education in Wrocław, Poland (PN-EN ISO 9001:2001 certified) on a Monark 894 cycle ergometer. Arterialized capillary blood from the fingertip was drawn immediately before and 3 min after the test in order to assay hydrogen ion (pH) and lactate concentrations.

Acid–base balance was assessed using a RapidLab 248 blood gas analyzer (Bayer, Germany) and included determining the partial pressures of oxygen (pO_2 – mm Hg) and carbon dioxide (pCO_2 – mm Hg), base excess (BE – mmol/l), oxygen saturation (sO_2 – %), carbon dioxide concentration in plasma ($ctCO_2$ – mmol/l), and bicarbonate concentration (HCO_3^- – mmol/l). Blood lactate (mmol/l) was measured with a LP400 photometer (Dr. Lange, Germany).

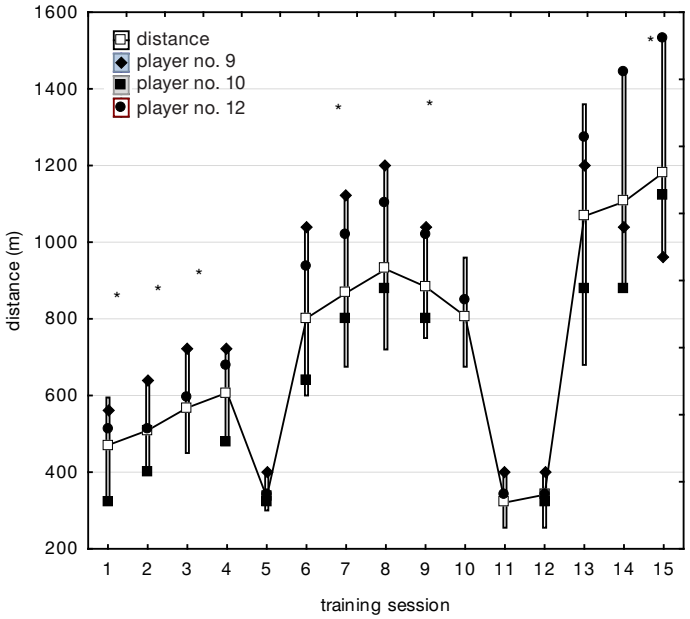
Respiratory function on a breath-by-breath basis was registered 2 minutes before beginning the Wingate test and continued for 5 min after its completion using a face mask connected to a Quark gas analyzer (Cosmed, Milan, Italy). The gas analyzer was calibrated with a reference gas mixture of: CO_2 – 5%, O_2 – 16%, and N_2 – 79%. Respiratory variables selected for analysis included oxygen uptake (VO_2 – ml/min), carbon dioxide production (VCO_2 ml/min), maximal pulmonary ventilation (VE – l/min), tidal volume (VT – l), breathing frequency (Rf – b/min), respiratory quotient (R), and the ventilatory equivalent ratios for oxygen (VE/VO_2) and carbon dioxide (VE/VCO_2).

The data set was statistically analyzed using the Statistica 9.0 package (Statsoft, USA). Means and standard deviation were calculated for the accrued data. Analysis was also performed on an individual basis for selected players. The Wilcoxon signed-rank test was used to compare the experimental (E) and control (C) groups. Significant differences between both groups were verified used the Mann–Whitney U test. An alpha level of 0.05 was accepted as statistically significant.

Results

Interval training and distance covered

In the first four training session the experimental group was able to complete only two sets in which the distance covered significantly increased in each session (Figure 2). In the fifth session the participants completed only one set, but in the sixth to tenth sessions they completed three sets. The increase in distance covered between the sixty and an eighth session was statistically significant. Distance covered decrease in the next two sessions with a significant difference between the eighth and tenth session. In the eleventh and twelfth session only one interval training set was performed, but jumped to four sets in the last three sessions. A significant increase was observed in the distance covered between the thirteenth and fifteenth and fourteenth and fifteenth sessions. Inter-individual analysis found that the smallest distance covered in the first and last training session



* statistically significant difference between training sessions

Figure 2. Mean, minimal, and maximum distance covered in each training session

was 320 m by player no. 10 and 960 m by player no. 9, respectively, who showed the best performance in the second to ninth sessions. At the study conclusion the longest distance covered was 1530 m by player no. 12.

Wingate ergometric performance

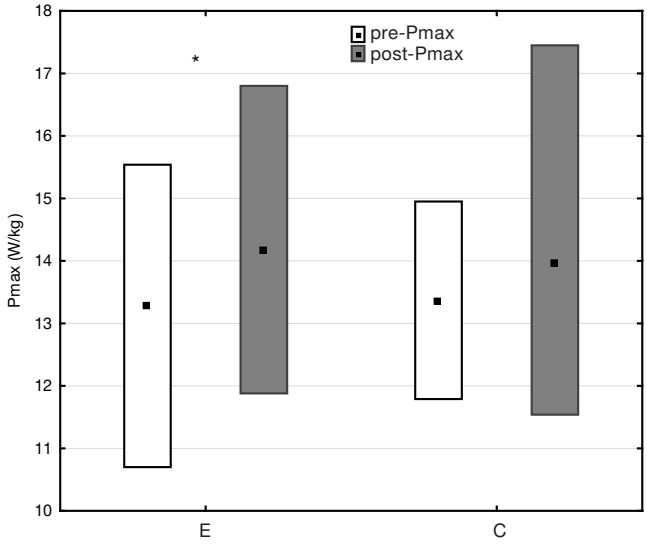
Pre- and post-intervention differences in maximal power output were significant only in group E (13.28 ± 1.43 and 14.14 ± 1.25 W/kg) (Figure 3). Similarly, differences in pre- and post-intervention work output were significant only in group E (273.63 ± 18.32 and 284.98 ± 15.76 J/kg) and not group C (273.37 ± 11.18 and 274.31 ± 12.17 J/kg) (Figure 4).

Acid–base balance

Pre- and post-intervention blood lactate levels in the experimental group were 10.64 ± 1.5 and 12.72 ± 1.6 mmol/l, respectively (Figure 5). This difference was statistically significant, unlike in the control group (11.06 ± 1.4 and 11.74 ± 1.2, respectively). The increase in work done in the experimental group was observed with a significant decrease in pH from 7.21 ± 0.029 to 7.19 ± 0.025 (Figure 6). No significant changes were observed in group C (7.21 ± 0.037 and 7.21 ± 0.033). Inter-individual analysis found that player no. 12 showed an increase from 7.188 to 7.199.

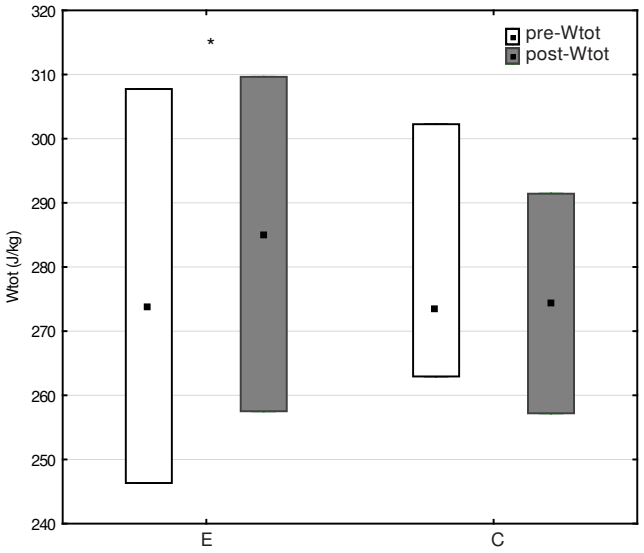
Spirometry testing

Significant decreases in oxygen uptake between the pre- and post-intervention measures in group E were noted



* statistically significant difference between training sessions

Figure 3. Mean, minimal, and maximum pre- and post-intervention maximal power (Pmax) in the experimental (E) and control (C) groups



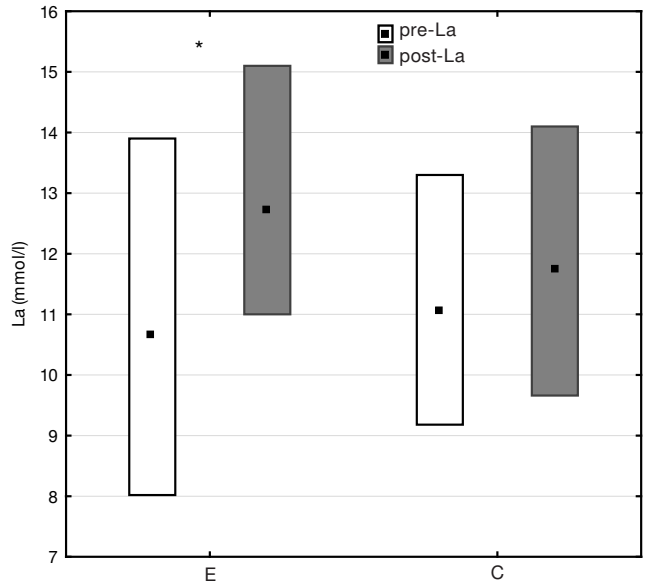
* statistically significant difference between training sessions

Figure 4. Mean, minimal, and maximum pre- and post-intervention total work (Wtot) in the experimental (E) and control (C) groups

in five recovery time points: 2'01"–2'30", 2'31"–3'00", 3'01"–3'30", 3'31"–4'00", and 4'01"–4'30". Similarly, significant decreases in group C were also observed but in seven time points: 1'01"–1'30", 1'31"–2'00", 2'01"–2'30", 2'31"–3'00", 3'01"–3'30", 3'31"–4'00", and 4'01"–4'30" (Figure 7, 8).

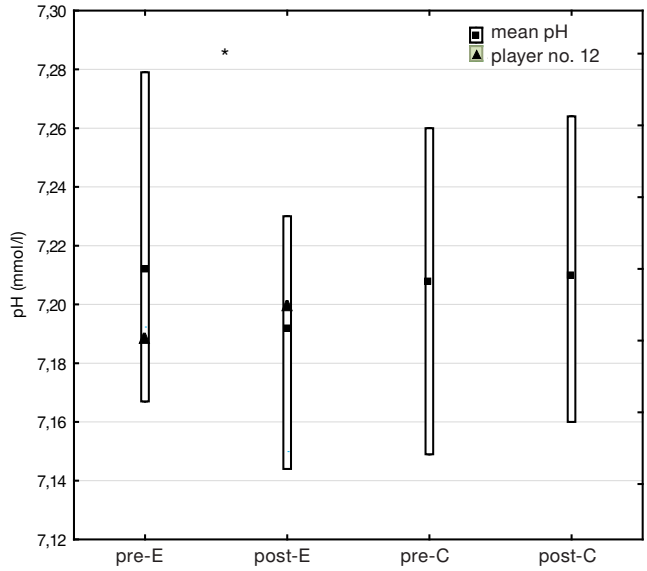
Discussion

Glycolytic capacity can be understand as the ability of the body to perform long-duration high-intensity work whose energy demands are met by the glycolytic



* statistically significant difference between training sessions

Figure 5. Mean, minimal, and maximum pre- and post-intervention blood lactate concentration (La) in the experimental (E) and control (C) groups



* statistically significant difference between training sessions

Figure 6. Mean, minimal, and maximum pre- and post-intervention blood pH in the experimental (E) and control (C) groups

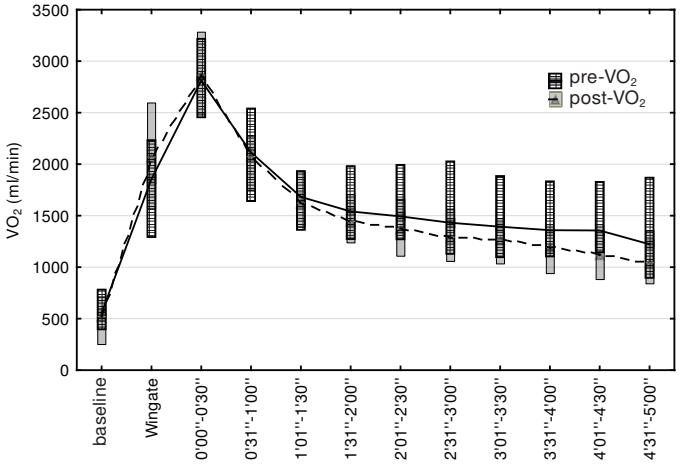


Figure 7. Mean, minimum, and maximum pre- and post-intervention oxygen uptake (VO_2) in the experimental (E) group

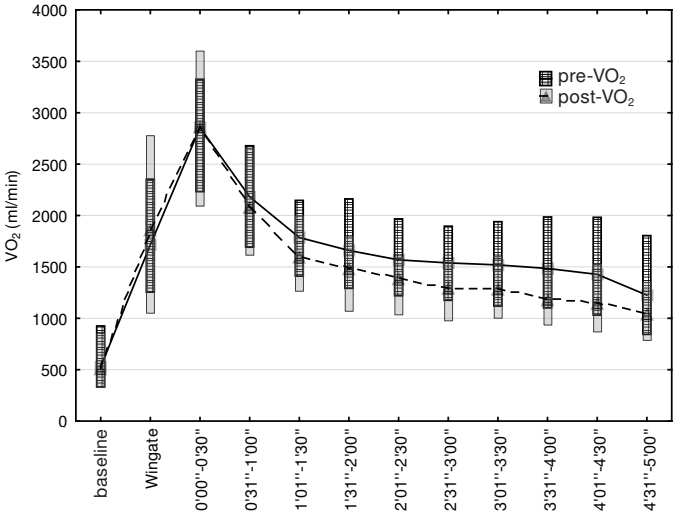


Figure 8. Mean, minimum, and maximum pre- and post-intervention oxygen uptake (VO_2) in the control (C) group

pathway [5]. A practical measure of glycolytic measure in exercise testing (Wingate) is the amount of work done. Numerous studies have confirmed an increase in work output (Wtot) following training with a large anaerobic component [22, 27, 38]. Each of the citied studies analyzed a training protocol pf 30 s pedaling bouts interspersed with 4-min recovery periods in untrained individuals. However, similar findings (improved endurance capacity) were reported in competitive athletes including soccer players [33–35]. Helgerud et al. [33] concluded that interval training is effective in improving soccer performance such as by increasing the distance covered in a match by 20% or the number of sprints by 100%. The present study confirms the positive effects of interval training, such as by an increase in total work output as measured in the Wingate test whereas no such effect was observed in the control group. While this increase was smaller in magnitude than that reported in other studies [20, 30], it should be noted that those studies were performed on untrained individuals. Additionally, it needs mention that our soccer players completed a 2-month mid-season training program prior to participating in the present study. Research shows that this period is extremely conducive to performance improvements as a result of endurance training and in fact applied by some of the best European teams [2]. Mohr et al. [2] pointed out that teams perform the greatest amount of high-intensity work during the post-season due to the amount of endurance training they perform in this period

of time. This finding is at odds with the prevailing view among Polish coaches that performance should be shaped in the pre-season and only maintained in the early season period. The result of such an approach is to improve aerobic and anaerobic capacity during the pre-season period and gradually reducing these variables as the season begins. Szmatlan-Gabryś [39] suggested that the use of anaerobic glycolytic training in the early soccer season can lead to an increase in glycolytic capacity. They described an increase in Wingate-assessed work output from 271.48 ± 13.52 to 279.19 ± 16.82 J/kg as well as improved 300 m running speed. Another work by Norkowski [38] compared the effects of running- versus cycling-based interval training by measuring anaerobic capacity via the Wingate test. No inter-group differences were observed although both showed significant improvements in glycolytic capacity. Our findings confirm that the increase in Wingate-assessed work output is a positive assertion of early season interval training, whereas normal soccer training (as performed by the control group) only maintained this ability.

Maximal power can serve as an indirect indicator of the rate of ATP production by the phosphagen system while the ability to maintain a given power level can describe the status of muscle PCr stores [5]. We found that Pmax increased in both groups although a significant difference was observed only in the experimental group and no difference in the time of sustained maximal power. The increase in maximal power with a concomitant lack of change in time of sustained Pmax in group E may indicate improved clearing and resynthesis processes as well as enhanced ATP and PCr capacity [22]. The literature is still ambiguous on the role of glycolytic training on phosphagen function. Rodas et al. [22] documented a 32% increase in muscle phosphocreatine stores following a daily 12-week workout protocol of 15 s efforts separated by 45 s of rest as well as 30 s efforts separated by 12 min of rest. An increase in phosphocreatine can increase short-term maximal effort or also contribute to increased exercise intensity as PCr serves as a buffer to excess H^+ concentrations. Other studies have reported a simultaneous increase in maximum power and total work in the Wingate test following glycolytic training [20, 27]. On the other hand, Fernandez-Fernandez et al. [40] did not observe any improvement in phosphagen-based efforts (countermovement jump and 20 m shuttle run) following two different interval training variants with a work-to-recovery design of 5 s/15 s and 90 s/3 min. Another study on the effects of a 4 min repetition of 90–95% HRmax sprints did not induce improved phosphagen function [33] but the substitution of the exercise modality with short explosive exercises did result in increased maximal power [34]. A study that analyzed the fitness of soccer players during general, specialized, and pre-season training found a progressive increase in phosphagen power and capacity as measured by running and cycle ergometer tests [39]. However, this study included both glycolytic and phosphagen-based exercise. It is possible that the improvement in Pmax we observed in both groups may be the result of phosphagen-based training at least once per week. The greater magnitude of this increase in the experimental group may be perhaps due to the addition of the interval training intervention. Additional research is warranted in understanding the precise impact of glycolytic-based interval training on phosphagen function. Nonetheless, we can conclude based on our findings and those in the literature that this training modality in conjunction with phosphagen-based training improves maximum produced power, which cannot be said in the case of a training modality combining aerobic endurance with anaerobic exercise.

The results of the present study confirm that the ability to perform high-intensity activity is subject to training effects. There are references in which a period of two or more weeks of interval training was sufficient to improve physiological function. However, we found a significant increase in work output after just two training sessions, indicating that improvement in the ability to perform glycolytic efforts in young soccer players can occur after only short-term training targeting this energy system. However, the developmental potential far exceeds such a microcycle, as we observed continual increases in the distance covered whereas changes in work output involved an increase in the number of repetitions per set resulting from the ability to perform additional repetitions without a decrease in power and an increase in the number of sets.

The receptivity of individuals to a given training load is not equivalent. The extent to which a specific load affects physiological function is grounded in number factors, among which the genetic influence is particularly noteworthy. For example, large variability was discovered after 12-weeks of progressive resistance training in muscle mass increases (from –2% to 59%), 1RM strength (0–250%), or maximum voluntary contraction strength (from –32 to 149%), [41]. However, there is no data on individual variability in interval training-induced glycolytic capacity. Inter-individual analysis of the present results finds that the training effects we observed were not equivalent across the participants. For example, the smallest increase in interval-training work output was observed in player no. 9 (71%), while an increase of 200% was recorded in player no. 12.

There is also a lack of information in the literature on the effects of repeated glycolytic training units on capacity. One study by Norkowski [38] assessed weekly changes in anaerobic capacity during an interval training mesocycle. In this investigation, athletes performed repeated bouts of 8 s exercise separated by 15 s (group 1) or 45 s (group 2) of passive recovery. However, the assessment criterion was the time to complete six repetitions and not the ability to maintain power in subsequent repetitions. In both interval modalities, capacity increased to include six microcycles but then plateaued. In the pre-

sent study, we observed first an increase (sessions 1–8), then decrease (9–11), and then again an increase (sessions 12–15) in work capacity. The outlier that was session 5 was the result of external factors and should not be considered in the normal progression of interval training. However, training sessions 11 and 12 did in fact feature reduced work output, hence the reason why another set of soccer-specific running was not performed. Subsequent training sessions concentrated on developing specific motor characteristics should involve supercompensation. The importance of recovery between each glycolytic training session was highlighted by Parra et al. [42]. These authors found that the use of uninterrupted interval training for 14 days lead to reduced exercise capacity. Hence, the introduction of a minimum 2-day recovery period between each training session may be behind the significant increase in Wingate-performance over time. Rodas et al. [22] argued that a reduction in exercise capacity due to daily interval training is associated with nervous system exhaustion. Hence, clinicians and coaches should treat a reduction in work output over time as a sign of excess load and therefore overtraining [43]. The continuation of training in such conditions is unjustified due to increasing fatigue. In fact, the continuation of training following minimal recovery can only be performed if adequate rest is later guaranteed. For this reason was training abridged following session 11 by not performing a second set of track running as was in with current training microcycle. This allowed the upward trend to continue and complete the two-training session per week schedule. Nonetheless, not all of the participants showed a continued performance improvement. Player no. 9, who presented the smallest increase in work output across the 8-week study duration in fact showed a 114% improvement up until the halfway point. From this point onwards the lack of improvement in glycolytic capacity is indicative of persistent fatigue.

As muscle fatigue increases so does the risk of injury and trauma [44]. This may explain the absence of five participants on session 10 who declared various health problems. It may be that the fatigue experienced already by session 9 was instigator of overall fatigue in the sample. However, the slight yet not significant decrease in exercise capacity between sessions 8 and 9 was not large enough to warrant the introduction of drastic change in session 10. However, the results obtained in this and later training sessions should have been treated as a clear signal to extend the rest period.

The increase in exercise capacity in sessions 1–4, 6–8, and 13–14 was directly the result of an increase in the number of repetitions performed per set without a decrease in power although a reduction in work output across work output was observed. Many of these changes were statistically significant. This finding also suggests that the recovery periods between each set were too short in duration or involved too intensive recovery. Among the various factors responsible for post-exercise fatigue, the most commonly cited are accumulations of inorganic phosphate, ammonia, ADP, IMP, lactate, and hydrogen ions. While Gupta et al. [45] claimed that 15 min of active recovery was sufficient in removing excess blood lactate after interval training performed to exhaustion, it is apparent that recover should be dependent on other factors such as training level, muscle fiber type, and the actual concentrations of post-exercise metabolites. In our study we did not individualize recovery duration, which in retrospect may have resulted in reducing the amount of work done by some of the participants.

One of the limiting factors of a glycolytic-based effort is the body's ability to function with excess waste products in muscle and blood. Our analysis of lactate and hydrogen ion concentrations found a significant difference between the pre- and post-measures in group E. Lower pH paired with increased work output may attest to improved work capacity in conditions with abnormal acid–base balance. Lutosławska et al. [46] observed a relationship between training load and blood lactate levels after the administration of the Wingate test in elite wrestlers. In that study, the inclusion of interval training within a program resulted in increased lactate concentrations, which signified that the energy demands of 30 s maximal efforts were met in a large part by anaerobic glycolysis [46]. The rise in lactate and H^+ ion concentrations in maximal efforts following an interval training intervention has been documented in other studies [13, 22]. The concomitant reduction in muscle lactate and H^+ concentrations after such training has been credited to 1) reduced glycolytic activity, 2) increased blood lactate levels, and 3) larger lactate uptake by muscle fibers. One other factor may lie in improved extracellular transport, in which the MCT protein plays an important role in La^- and H^+ clearance. There is evidence that an increase in the concentration of this protein is associated with an improved blood metabolite excretion. MCT stores, in turn, are known to increase in response to high-intensity training, providing a more efficient reduction of intracellular H^+ and lactate and thereby allowing an individual to perform more work at lower cost. Another factor that may improve La^- and H^+ transport from cells to blood may be via increased blood flow. Research had indicated that athletes show improved lactate transport than untrained individuals [47]. Furthermore, athletes whose training involves anaerobic exercise are observed with higher lactate transport values. Other positions in the literature have confirmed that the introduction of this type of exercise modality increases transport ability both in trained and untrained populations [47]. Bongaerts et al. [28] have suggested, however, that improved performance in elite athletes is more the result of efficient gluconeogenesis. As soccer is a sport consisting of high-intensity intermittent exercise, it naturally involves a significant anaerobic component to it, which may explain the increase in lactate in the control group although at a non-significant level. It can therefore be concluded that

applied training stimuli was responsible for the improvement in glycolytic metabolism in the experimental group. It appears that this change had a positive effect on the type of intermittent exercise included in the experimental protocol. Considering the literature, this change signifies enhanced metabolite removal during recovery that prevented (to a greater extent than previously) acid–base imbalance. This effect may have also contributed to the increased work output in the Wingate test. One aspect worthy of mention was the different direction of post-Wingate blood pH in player no. 12, who presented the most improved performance. Elevated pH levels with a simultaneous increase in work output and blood lactate levels may be considered to be the effect of improved ability to buffer H^+ ions. It appears likely that this improvement allowed the player no. 12 to perform more work in the last training session as acid–base balance was maintained for a longer period of time.

Among the buffering agents responsible for maintaining acid–base balance the most important are hemoglobin, bicarbonate, and phosphate. Additionally, the role of pulmonary ventilation and renal function are also important in the regulation of acidosis. Combined together these mechanisms create buffer capacity. It has been demonstrated that interval training improves buffer capacity [18, 24]. Gibala et al. [18] observed improved buffer capacity after six glycolytic and aerobic training sessions Edge et al. [16] performed a 5-week experiment to find that interval training elicited greater increased in buffer capacity (25%) endurance training (2%). We found that the Wingate concentrations of bicarbonate (HCO_3^-) and base excess (BE) significantly decreased in the experimental group. Such changes may be demonstrated in improved ability to perform in conditions of acid–base imbalance.

McKenna et al. [20] reported that interval training induced higher oxygen uptake (VO_2) and carbon dioxide production (VCO_2), whose peak values were recorded 11.3 s and 15 s after completing the Wingate test. The composition of expired air is known as a useful indirect measure of ongoing processes of muscle metabolism. The present study found that these respiratory variables increased during the test and immediately after its completion at a greater magnitude in the experimental group, although these observed differences were not statistically significant. McKenna et al. [20] have suggested that increased oxygen consumption during the Wingate test is associated with more work done, while a greater rate of carbon dioxide removal is a testament to enhanced blood homeostasis. Dupont et al. [29] in turn showed that greater VO_2 kinetics translates to improved performance in repeated short-duration exercise. Returning to our inter-individual analysis, we found that player no. 12 showed dramatically higher VO_2 levels compared with the group mean, which may have been the reason why this participant showed such significant progress. On the other hand, player no. 13, who presented the smallest increase in work output in the Wingate test, also had a considerable increase in VO_2. This may indicate that improved respiratory function in short-term high-intensity bouts is insufficient alone to cause improved performance. The study by McKenna et al. [20] found that interval training elicited increased VO_2 up to the 10^{th} min of recovery in the Wingate test compared with values recorded at baseline. This finding is contradictory to what we observed in the present study, where oxygen uptake in both groups was lower in the post-intervention measure already at 30 s recovery. This reduction was statistically significant in the control group at 1 min recovery and in the experimental group from 2 min recovery and is indicative of the oxygen debt during post-exercise recovery. The results of the control group allow for the conclusion that standard soccer training improves post-exercise recovery. However, it should be noted that no increase in the amount of work done was observed. In turn, similar changes in VO_2 were noted in the experimental group although with increased work output.

Conclusions

1. Interval training targeting glycolytic capacity enhances anaerobic capacity.
2. The ability to perform repeated high-intensity exercise improved over time but then plateaued or deteriorated at later training stages. The introduction of additional recovery reversed this trend and allowed continued improvement although not in all participants.
3. The training effects of this training modality are varied, as evidenced by the large variability of changes in work output in both the training intervention and Wingate test.

References

1. Rampinini E., Coutts A.J., Costagna C., Sassi R., Impellizzeri F.M., Variation in Top Level Soccer Match Performance. *Int J Sports Med*, 2007, 28 (12), 1018–1024, doi: 10.1055/s-2007-965158.
2. Mohr M., Krustrup P., Bangsbo J., Match performance of high-standard soccer players with special reference to development of fatigue. *J Sports Sci*, 2003, 21 (7), 519–528, doi: 10.1080/0264041031000071182.
3. Bangsbo J., Iaia F.M., Krustrup P., Metabolic Response and Fatigue in Soccer. *Int J Sports Physiol Perform*, 2007, 2 (2), 111–127.
4. Rampinini E., Impellizzeri F.M., Castagnac C., Coutts A.J., Wisløff U., Technical performance during soccer matches of the Italian Serie A league: Effect of fatigue and competitive level. *J Sci Med Sport*, 2009, 12 (1), 227–233, doi: 10.1016/j.jsams.2007.10.002.
5. Zatoń M., Jastrzębska A., Physiological-based physical fitness tests [in Polish]. PWN, Warszawa 2010.
6. Burgomaster K.A., Cermak N.M., Phillips S.M., Benton C.R., Bonen A., Gibala M.J., 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 (5), 1970–1976, doi: 10.1152/ajpregu.00503.2006.
7. Zatoń M., Hebisz R., Hebisz P., Physiological effects of training in mountain bicycling [in Polish]. AWF, Wrocław 2011.
8. Bogdanis G.C., Nevill M.E., Boobis L.H., Lakomy H.K. Nevill A.M., Recovery of power output and muscle metabolites following 30 s of maximal sprint cycling in man. *J Physiol*, 1995, 482 (2), 467–480, doi: 10.1113/jphysiol.1995.sp020533.
9. Medbo J.I., Tabata I., Relative importance of aerobic and anaerobic energy release during short-lasting exhausting bicycle exercise. *J Appl Physiol*, 1989, 67 (5), 1881–1886.
10. Smith J.C., Hill D.W., Contribution of energy systems during a Wingate power test. *Br J Sports Med*, 1991, 25 (4), 196–199, doi:10.1136/bjsm.25.4.196.
11. Fitts R.H., The cross-bridge cycle and skeletal muscle fatigue. *J Appl Physiol*, 2008, 104 (2), 551–558, doi: 10.1152/japplphysiol.01200.2007.
12. Wołkow N., Gabryś T., Szmatlatn-Gabryś U., Interval training [in Polish]. AWF, Warszawa 2007.
13. Harmer A.R., McKenna M.J., Sutton J.R., Snow R.J., Ruell P.A., Booth J. et al., Skeletal muscle metabolic and ionic adaptations during intense exercise following sprint training in humans. *J Appl Physiol*, 2000, 89 (5), 1793–1803.
14. Zatoń M., Physiological reference ranges in the monitoring and modification of training [in Polish]. *Studia i Monografie AWF we Wrocławiu*, 1990, 22.
15. Burgomaster K.A., Hughes S.C., Heigenhauser G.J.F., Bradwell S.N., Gibala M.J., Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *J Appl Physiol*, 2005, 98 (6), 1985–1990, doi: 10.1152/japplphysiol.01095.2004.
16. Edge J., Bishop D., Goodman C., The effects of training intensity on muscle buffer capacity in females. *Eur J Appl Physiol*, 2006, 96 (1), 97–105, doi: 10.1007/s00421-005-0068-6.
17. Edge J., Bishop D., Hill-Haas S., Dawson B., Goodman C., Comparison of muscle buffer capacity and repeated-sprint ability of untrained, endurance-trained and team-sport athletes. *Eur J Appl Physiol*, 2006, 96 (3), 225–234, doi: 10.1007/s00421-005-0056-x.
18. Gibala M.J., Little J.P., van Essen M., Wilkin G.P., Burgomaster K.A., Safdar A. et al., Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *J Physiol*, 2006, 575 (3), 901–911, doi: 10.1113/jphysiol.2006.112094.
19. MacDougall J.D., Hicks A.L., MacDonald J.R., McKelvie R.S., Green H.J., Smith K.M., Muscle performance and enzymatic adaptations to sprint interval training. *J Appl Physiol*, 1998, 84 (6), 2138–2142.
20. McKenna M.J, Heigenhauser G.J., McKelvie R.S., Obminski G., MacDougall J.D., Jones N.L., Enhanced pulmonary and active skeletal muscle gas exchange during intense exercise after sprint training in men. *J Physiol*, 1997,501(3),703–716,doi:10.1111/j.1469-7793.1997.703bm.x.
21. Puype J., Van Proeyen K., Raymackers J.M., Deldicque L., Hespel P., Sprint interval training in hypoxia stimulates glycolytic enzyme activity. *Med Sci Sports Exerc*, 2013, 45(11),2166–2174,doi:10.1249/MSS.0b013e31829734ae.
22. Rodas G., Ventura J.L., Cadefau J.A., Cusso R., Parra J., A short training programme for the rapid improvement of both aerobic and anaerobic metabolism. *Eur J Appl Physiol*, 2000, 82 (5–6), 480–486, doi: 10.1007/s004210000223.
23. Ross A., Leveritt M., Long-Term Metabolic and Skeletal Muscle Adaptation Short-Sprint Training: Implications for Sprint Training and Tapering. *Sports Med*, 2001, 31 (15), 1063–1082, doi: 10.2165/00007256-200131150-00003.
24. Weston A.R., Myburgh K.H., Lindsay F.H., Dennis S.C., Noakes T.N., Hawley J.A., Skeletal muscle buffering capacity and endurance performance after high-intensity interval training by well-trained cyclists. *Eur J Appl Physiol Occup Physiol*, 1996, 75 (1), 7–13, doi: 10.1007/s004210050119 .
25. Wilson D., The physiological basis of speed endurance. *Insight: FA Coaches Assoc J*, 2001, 4 (3), 36–37.
26. Barnett C., Carey M., Proietto J., Cerin E., Febbraio M.A., Jenkins D., Muscle metabolism during sprint exercise in man: influence of sprint training. *J Sci Med Sport*, 2004, 7 (3), 314–322, doi: 10.1016/S1440-2440(04)80026-4.
27. 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 (3), 571–576.
28. Bongaerts G.P.A., Wagener D.J.T., Increased hepatic gluconeogenesis: The secret of Lance Armstrong's success. *Med Hypotheses*, 2007, 68 (1), 9–11, doi: 10.1016/j.mehy.2006.04.054.
29. Dupont G., Millet G.P., Guinhouya C., Berthoin S., Relationship between oxygen uptake kinetics and performance in repeated running sprints. *Eur J Appl Physiol*, 2005, 95 (1), 27–34, doi: 10.1007/s00421-005-1382-8.
30. Iaia F.M., Hellsten Y., Nielsen J.J., Fernstrom M., Sahlin K., Bangsbo J., Four weeks of speed endurance training reduces energy expenditure during exercise and maintains muscle oxidative capacity despite a reduction in training volume. *J Appl Physiol*, 2009, 106 (1), 73–80, doi: 10.1152/japplphysiol.90676.2008.
31. Gibala M.J., McGee S.L., Garnham A.P., Howlett K.F., Snow R.J., Hargreaves M., Brief intense interval exercise activates AMPK and p38 MAPK signaling and increases the expression of PGC–1α in human skeletal muscle. *J Appl Physiol*, 2009, 106 (3), 929–934, doi: 10.1152/japplphysiol.90880.2008.
32. Sutton J.R., Jones N.L., Toews C.J., Effect of pH on muscle glycolysis during exercise. *Clin Sci*, 1981, 61 (3), 331–338, doi: 10.1042/cs0610331.
33. Helgerud J., Engen L.C., Wisløff U., Hoff J., Aerobic endurance training improves soccer performance. *Med Sci Sports Exerc*, 2001, 33 (11), 1925–1931.
34. McMillan K., Helgerud J., Macdonald R., Hoff J., Physiological adaptations to soccer specific endurance training in professional youth soccer players. *Br J Sports Med*, 2005, 39 (5), 273–277, doi: 10.1136/bjsm.2004.012526.
35. Ferrari Bravo D., Impellizzeri F.M., Rampinini E., Castagna C., Bishop D., Wisloff U., Sprint vs. Interval Training in Football. *Int J Sports Med*, 2008, 29 (8), 668–674, doi: 10.1055/s-2007-989371.
36. Thomassen M., Christensen P.M., Gunnarsson T.P., Nybo L., Bangsbo J., Effect of 2–wk intensified training and inactivity on muscle Na^+-K^+ pump expression, phospholemman ($FXYD_1$) phosphorylation, and performance in soccer players. *J Appl Physiol*, 2010, 108 (4), 898–905, doi: 10.1152/japplphysiol.01015.2009.

37. Lakomy J., Haydon D.T., The effect of enforced, rapid deceleration on performance in a multiple sprint test. *J Strength Cond Res*, 2004, 18 (3), 579–583.
38. Norkowski H., Effects of training load structure in maximal-intensity intermittent training [in Polish]. AWF, Warszawa 2003.
39. Szmatlan-Gabryś U., Endurance training. Optimizing available methods and training load [in Polish]. PWSZ, Warszawa–Oświęcim 2012.
40. Fernandez-Fernandez J., Zimek R., Wiewelhove T., Ferrauti A., High-intensity interval training vs. repeated-sprint training in tennis. *J Strength Cond Res*, 2012, 26 (1), 53–62, doi: 10.1519/JSC.0b013e318220b4ff.
41. Hubal M.J., Gordish-Dressman H., Thompson P.D., Price T.B., Hoffman E.P., Angelopoulos T.J. et al., Variability in muscle size and strength gain after unilateral resistance training. *Med Sci Sports Exerc*, 2005, 37 (6), 964–972.
42. Parra J., Cadefau J.A., Rodas G., Amigó N., Cussó R., The distribution of rest periods affects performance and adaptations of energy metabolism induced by high-intensity training in human muscle. *Acta Physiologica Scandinavica*, 2000, 169 (2), 157–165, doi: 10.1046/j.1365-201x.2000.00730.x.
43. Meeusen R., Duclos M., Foster C., Fry A., Gleeson M., Nieman D. et al., Prevention, diagnosis and treatment of the Overtraining Syndrome. *Eur J Sport Sci*, 2006, 6 (1), 100–114.
44. Duscha B.D., Annex B.H., Johnson J.L., Huffman K., Houmard J., Kraus W.E., Exercise Dose Response in Muscle. *Int J Sports Med*, 2012, 33 (3), 218–223, doi: 10.1055/s-0031-1291323.
45. Gupta S., Goswami A., Sadhukhan A.K., Mathur D.N., Comparative study of lactate removal in short term massage of extremities, active recovery and a passive recovery period after supramaximal exercise sessions. *Int J SportsMed*, 1996, 17 (2), 106–110, doi: 10.1055/s-2007-972816.
46. Lutosławska G., Hubner-Woźniak E., Sitkowski D., Borkowski L., Relationship between anaerobic capacity and blood lactate following the wingate test in elite wrestlers during an annual training cycle. *Biology of Sport*, 1998, 15 (2), 67–74.
47. Hawley J.A., Adaptations of skeletal muscle to prolonged, intense endurance training. *Clin Exp Pharmacol Physiol*, 2002, 29 (3), 218–222, doi: 10.1046/j.1440-1681.2002.03623.x.

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