



*Original Research*

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## **The Determination of Total Energy Expenditure During and Following Repeated High-Intensity Intermittent Sprint Work**

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### ABSTRACT

*International Journal of Exercise Science 10(3): 312-321, 2017.* The purpose of this study was to examine the variation in oxidative, glycolytic, and post-exercise O<sub>2</sub> kinetic contribution during two distinct high-intensity interval training (HIIT) protocols using a 1:1 work-to-rest ratio (30:30 sec) and a 2:1 work-to-rest ratio (30:15 sec). HIIT familiarized males ( $n = 6$ ) and females ( $n = 8$ ) were recruited for this study. All subject underwent 3 testing session, an incremental maximal exertion treadmill test and 30:30 and 30:15 HIIT protocols in a counterbalanced order. Each HIIT protocol measured oxygen consumption (VO<sub>2</sub>), carbon dioxide production (VCO<sub>2</sub>), and respiratory exchange ratio (RER) to represent oxidative contribution. Capillary blood lactate was also analyzed to represent glycolytic contribution during both HIIT sessions. Repeated-measures ANOVA revealed a relative and absolute significant difference between the oxidative, glycolytic, and post-exercise oxygen kinetics between 30:30 and 30:15 HIIT session. 30:30 displayed a greater contribution from the oxidative system while the 30:15 displayed an increase contribution from the glycolytic system and displayed an increase in EE during the post-exercise oxygen kinetics phase. Results also revealed no significant findings between the two HIIT sessions in regards to absolute EE (30:30 =  $258.2 \pm 43$  kcals, 30:15 =  $261 \pm 43.6$  kcals). The addition of blood lactate following exercise did display a noteworthy contribution from the glycolytic system. In conclusion, utilizing pulmonary gas exchange in conjunction with blood lactate depicts an acceptable EE estimation during a bout of HIIT.

**KEY WORDS:** High-intensity interval training (HIIT), oxidative, glycolytic, energy expenditure, lactate, anaerobic

### INTRODUCTION

High-intensity interval training (HIIT) is a well-established training modality used to improve performance in aerobic (e.g., cycling and long-distance running) and anaerobic (e.g., sprinting and team sports) competitions alike (3, 6). There are virtually endless permutations for structuring a program involving this form of training (e.g., work-to-rest ratios, intensity,

duration, and modality), which is an attractive feature leading to its ubiquitous nature in sport and exercise training. The growing attractiveness in the industry is grounded on the ability to elicit similar energy expenditure (EE) with less time commitment when compared to lower-intensity, continuous, steady-state work. (11, 22). With the increased demand for exercise programs incorporating HIIT, it seems prudent to accurately estimate the energy cost of HIIT. Traditionally, researchers have utilized indirect calorimetry to estimate EE during exercise (18). However, estimating EE by means of an indirect calorimetry tends to underestimate the energy cost when the modality of exercise has considerable contribution from the glycolytic system (17, 18). Therein lies an inherent flaw when using only indirect calorimetry to estimate EE, as this method tends to reflect only ATP-turnover from the oxidative system, to estimate total EE. When intensity exceeds oxygen uptake, substrate-level phosphorylation provides substantial contribution to overall EE (19). The anaerobic contribution to the O<sub>2</sub> deficit during high-intensity, intermittent exercise is not represented utilizing indirect calorimetry. Thus, it appears the use of indirect calorimetry to estimate EE during intermittent, high(er) intensity bouts of exercise will lead to a misrepresentation of total caloric expenditure due to the inability to measure glycolytic contribution. The glycolytic contribution has been estimated by measuring the change in blood lactate concentration following a bout exercise (14, 18, 20). This technique of estimating glycolytic contribution to EE has been implemented during various forms of resistance training (17). Surprisingly, there appears to be a paucity of data addressing the glycolytic contribution during a session of HIIT in the form of upright, repeated sprints. This is problematic as running is well-established as a common form of exercise being employed by novices to elite athletes. Therefore, it is important that exercise professionals are able to estimate the metabolic systems stressed and the energy cost from a session of HIIT. We attempted to provide a valid and reliable method of estimating EE during intermittent sprint work utilizing pulmonary gas exchange and blood lactate. Therefore, the purpose of this study was to examine the variation in oxidative and glycolytic contribution during two distinct HIIT protocols. It is hypothesized that the addition of blood lactate with O<sub>2</sub> consumption during and following exercise will improve the overall estimation of energy expenditure during high-intensity, intermittent sprint work.

## **METHODS**

### *Participants*

This study was approved by the local university Institutional Review Board for the use of human subjects and each subject's written informed consent was obtained before any testing procedures. Sixteen healthy, physically active men (n = 8) and women (n = 8) participated in this study (Table 1). To be included in this study subjects were currently performing HIIT or competing in an intermittent sport activity (e.g., soccer, rugby, basketball) at least two days per week and have been performing these sports or activities for the past three months. Exclusion criteria for this study included any musculoskeletal or orthopedic injury that may inhibit performance during the trials or if a subject was considered moderate risk or higher according to the ACSM guidelines (23). Subjects were asked to report to the Exercise Physiology Laboratory in a well-rested, hydrated state and be at least 4-h post-prandial as well as having abstained from caffeine for 4-h and alcohol for 24-h prior to testing. Each subject was also

enquired to eat a meal with similar macronutrient content 4-h before each HIIT session. All testing procedures, risks, and benefits were explained to each subject before testing sessions.

**Table 1.** Participant characteristics (n = 17).

|                                 | Mean | SD   |
|---------------------------------|------|------|
| Body fat (%)                    | 16.9 | 6.7  |
| Height (cm)                     | 174  | 7.8  |
| Weight (kg)                     | 73.2 | 12.2 |
| Age (years)                     | 23.4 | 3.7  |
| VO <sub>2peak</sub> (ml/kg/min) | 50.6 | 5.8  |
| VO <sub>2</sub> % Rank          | 86   | 12.3 |
| pVEL (mph)                      | 10.3 | 0.7  |
| VEL110% (mph)                   | 11.2 | 0.8  |

**Note:** Descriptive data is displayed as mean and standard deviation (SD) for 6 males and 8 females. VO<sub>2peak</sub>, peak oxygen consumption; VO<sub>2</sub>, oxygen consumption; pVEL, peak velocity; VEL110%; 110% of peak velocity.

### Protocol

Upon arrival to the laboratory, subject's height (cm) and body mass (kg) were measured using a stadiometer and beam scale (Detecto Scale Company, Webb City, MO, USA). Body fat percentage was estimated using a 3-site method (males: chest, abdomen, and thigh; females: triceps, iliac, and thigh) (16) using skinfold calipers (Lange, Cambridge, Maryland, USA). All anthropometric measures for a subject were performed by the same technician. Subjects were tailored with a heart rate monitor and strap (Polar Inc., Port Washington, New York, USA) worn around the subject's chest, at the level of the xyphoid process, to assess heart rate throughout each exercise session. Subjects then performed an incremental maximal exertion treadmill test (15) on a motorized treadmill (Truefitness, O'Fallon, MO). Subjects were connected via a mouthpiece to an automated metabolic measurement system (ParvoMedics TrueOne 2400, Sandy, UT) that recorded oxygen consumption (VO<sub>2</sub>, L/min), carbon dioxide production (VCO<sub>2</sub>, L/min), and respiratory exchange ratio (RER) every minute throughout the test. The metabolic cart was calibrated in accordance to the manufacture guidelines before each subject performed a maximal exertion treadmill test. Prior to the test, subjects were allowed a self-selected, 5-10 minute warm-up. Following the warm-up, subjects began running on the treadmill at a speed of 6.2 mph at 0% grade, and every minute the speed was manually increased by 0.6 mph until volitional fatigue (15). The final speed reached during the last full stage of the test determined the velocity eliciting maximal oxygen uptake (vVO<sub>2max</sub>). The vVO<sub>2max</sub> was used to control the speed at which the subjects ran during the HIIT sessions. Specifically, 110% vVO<sub>2max</sub> was the target speed during the HIIT sessions. 20 minutes following the incremental maximal exertion treadmill test, the subjects were required to run a sprint familiarization trial. During the sprint familiarization trial, pulmonary gas measures were not analyzed. The treadmill was set to 110% of the subject's vVO<sub>2max</sub>. The subject then performed one set of four sprints, at a 30:15 second (work-to-rest) ratio. The subjects were asked to straddle the treadmill belt, while the treadmill belt reached 110% of vVO<sub>2max</sub>. The subjects were instructed to paw the treadmill 5-seconds before the start of each sprint. The subjects were given a 5-second countdown before each sprint. Following the 1-second time point of the countdown, the subjects were required to be on the treadmill belt sprinting. To mitigate the effect of subject's end point knowledge of the sprint, an "off" command was

vocalized at the 30-second time point, which indicated the subjects were required to hold onto the treadmill rail and straddle the treadmill belt. This procedure was the exact same for each HIIT session.

**30:30 HIIT Session** - This session took place two to seven days after the maximal exertion treadmill test. Upon arrival to the laboratory, subjects were fit to the metabolic cart and seated in a chair for 5-10 minutes while resting  $\text{VO}_2$  was recorded. Resting measures were assessed to determine the amount of  $\text{VO}_2$  consumed above resting values. Immediately following resting  $\text{VO}_2$ , a resting capillary blood lactate (mmol) measurement was obtained and recorded using a calibrated, portable blood lactate analyzer (Lactate Plus, Nova Biomedical Corp., Waltham, Washington USA). Resting blood lactate measurements were also assessed to determine the accumulation of lactate above resting values. Subjects were tailored with a heart rate monitor and strap, to observe heart rate throughout the test. Following resting measures, subjects performed a standardized warm-up in agreement with the procedures developed by Vetter (2007) specific for sprint training. The protocol consisted of a four minute walk at 3.7 mph, a two minute run at 7.5 mph, and three rounds of a dynamic warm up (see Table 2).

**Table 2.** Standardize warm-up protocol.

| Exercise       | Repetitions | Cadence (per min) |
|----------------|-------------|-------------------|
| Toe Raises     | 10          | 30                |
| High Knee Lift | 20          | 30                |
| Buttock Kick   | 20          | 30                |

**Note.** Adapted from "Effects of six warm-up protocols on sprint and jump performance" by R. E. Vetter, 2007. *The Journal of Strength and Conditioning Research*, 21, 819-823.

Following the warm up, subjects performed a series of high-intensity intermittent sprints at 110%  $v\text{VO}_{2\text{max}}$ , established from the incremental maximal exertion treadmill test. The intervals consisted of a 30 second sprint paired with 30 seconds of passive recovery (straddling the treadmill belt). The subjects were asked to complete four sets of four 30 second sprints that were interspersed with 30 seconds of recovery (i.e., 30:30 protocol), a total of 16 sprints. Protocol procedures were the exact same as the sprint familiarization trial that was performed after the maximal exertion treadmill test. Between each set of four 30 second sprints, subjects were afforded three minutes of passive recovery (e.g., sitting in a chair). During the three minute passive recovery period, a blood lactate sample was taken at the two minute mark of the recovery phase. Scott, Croteau, and Ravlo (2009) reported that blood lactate concentration peaked two minutes after intense exercise. This process was repeated for each of the four sets of sprints. At the end of the session (completion of the four sets of sprints), participants were required to sit in a chair for 7 minutes to examine post-exercise  $\text{O}_2$  kinetics.

**30:15 HIIT Session** - This session was the same as 30:30 but the work-to-rest ratio was set at 30 seconds of sprinting and 15 seconds of passive recovery. All other measures and procedures were the same.

A metabolic cart was utilized to estimate the oxidative contribution to overall energy expenditure during the HIIT sessions. To calculate this estimation, each subject was properly

fit to a metabolic cart to measure  $\text{VO}_2$  (L/min). Net  $\text{VO}_2$ , the oxygen consumed due to exercise (i.e., oxygen consumed above rest), was calculated for every minute during both protocols by subtracting  $\text{VO}_2$  at rest from  $\text{VO}_2$  during exercise. Oxidative contribution to the overall energy expenditure during both HIIT sessions was represented as 1 L of net  $\text{VO}_2 = 21.1$  kJ. Kilojoules were further converted to kcals by dividing kJ by 4.184.

Glycolytic contribution to overall energy expenditure was estimated utilizing blood lactate concentration observed during testing. For every 1 mmol increase in blood lactate above resting was equivalent to the consumption of 3.0 ml  $\text{O}_2$  per kilogram of body weight (13; 7). Lactate samples were obtained every two minutes during the three minute passive recovery phase of each HIIT session. The lactate levels after each set were subtracted by resting values. The  $\text{O}_2$  equivalent was converted as 1 L of  $\text{O}_2 = 21.1$  kJ, to represent complete glucose oxidation. Kilojoules were further converted to kcals by dividing kJ by 4.184.

During the three minute passive recovery periods and the final 7 minute recovery period, post-exercise  $\text{O}_2$  kinetics were measured. This is done in accordance with Scott (2009) by recording the net  $\text{VO}_2$  and converting that measurement to represent energy expenditure as 1 L of net  $\text{VO}_2 = 19.6$  kJ.

During 30:30 and 30:15 total energy expenditure was calculated. Total energy expenditure was determined by summing the oxidative, glycolytic, and post-exercise  $\text{O}_2$  kinetics EE measurements.

#### *Statistical Analysis*

Relative and absolute contribution EE during the HIIT sessions was analyzed using a 2 (HIIT session)  $\times$  3 (oxidative, glycolytic, and post- $\text{O}_2$  kinetics) and a 2 (HIIT session)  $\times$  4 (oxidative, glycolytic, post- $\text{O}_2$  kinetics, and total kcals expended) repeated measures ANOVA to identify any significant main effects. When appropriate, univariate post-hoc follow-ups were analyzed to identify significant differences and 95% confidence interval for real change. All data was presented as mean  $\pm$  SD unless stated otherwise. Statistical significance is set at the 0.05 level and all data was analyzed using the statistical package for social sciences (SPSS, v 22, IBM Corporation, Armonk, NY).

## **RESULTS**

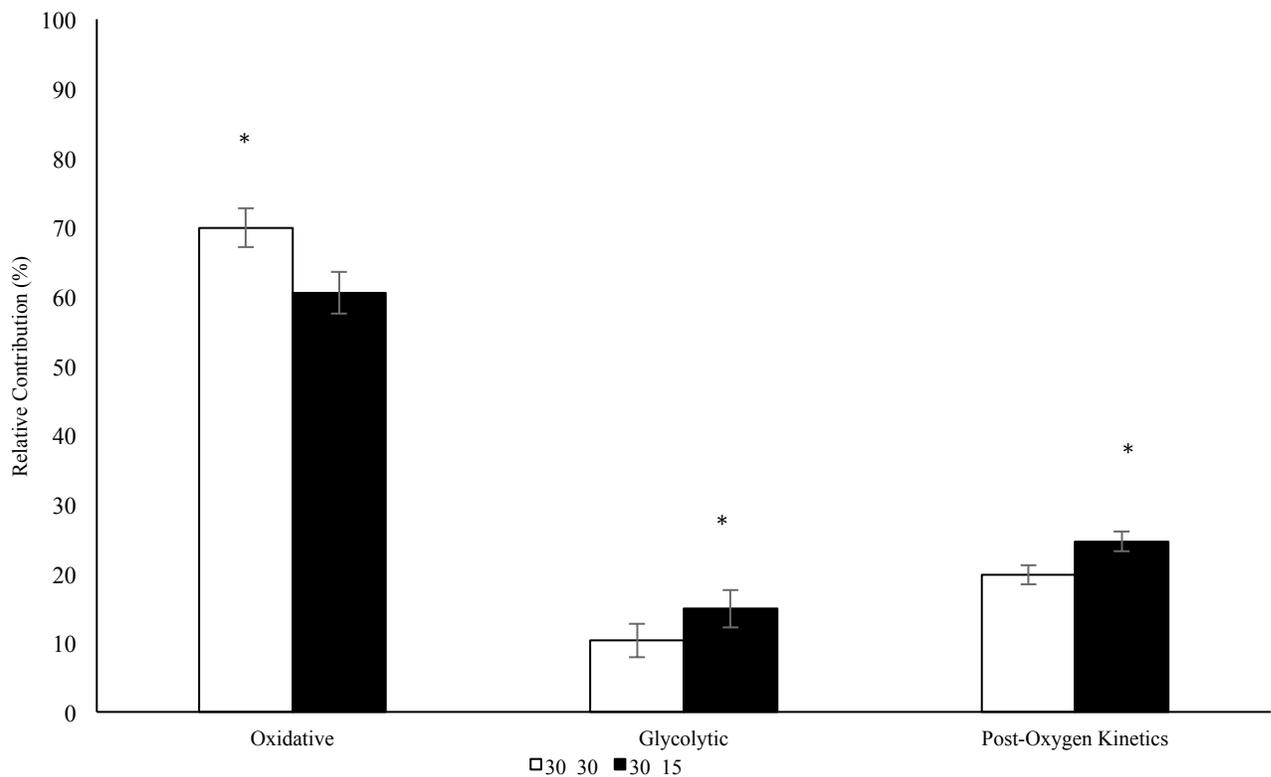
Out of the eight males that participated in the study, two were unable to complete the 30:15 HIIT session in its entirety, therefore they were excluded from the study.

Table 3 displays the differences in absolute kcal expenditure from oxidative and glycolytic system, as well as the post-exercise  $\text{O}_2$  kinetics during both HIIT sessions. When comparing total session energy expenditure, results revealed no significant difference between 30:15 and 30:30. There was a significant difference between the two sessions in regards to overall kcal expenditure from the oxidative system, glycolytic system, and post-exercise  $\text{O}_2$  kinetics (Table 3).

**Table 3.** Absolute contribution to overall EE

|                                     | 30:15        | 30:30        | Significance |
|-------------------------------------|--------------|--------------|--------------|
| Oxidative (kcal)                    | 158.2 ± 27.7 | 180.6 ± 32.5 | $p < .001$   |
| Glycolytic (kcal)                   | 39.1 ± 10.1  | 26.7 ± 7.8   | $p < .001$   |
| Post-O <sub>2</sub> Kinetics (kcal) | 64.1 ± 10.5  | 50.9 ± 8.4   | $p < .001$   |
| Total kcal                          | 261.4 ± 43.6 | 258 ± 43.4   | $p = .492$   |

Figure 1 displays the relative contribution from the oxidative and glycolytic system and post-exercise O<sub>2</sub> kinetics. When comparing relative contribution, there was a significant difference between 30:15 and 30:30 in regards to the oxidative, glycolytic, and post-exercise O<sub>2</sub> kinetics.



**Figure 1.** Relative contribution from the oxidative and glycolytic system, and post-exercise O<sub>2</sub> kinetics between both HIIT protocols. \* denotes  $p \leq .05$  when comparing 30:15 and 30:30.

## DISCUSSION

Previously, Scott (2005) affirmed that singularly relying upon pulmonary gas exchange may not be the most valid estimation of overall EE when the modality consists of high-intensity, intermittent work due to the purported glycolytic contribution. What has been shown to be a possible method of estimating EE is the integration of pulmonary gas exchange in conjunction

with change in blood lactate during high-intensity, intermittent exercise (7, 13). Therefore, we aimed to analyze the relative and absolute contribution from the oxidative and glycolytic system during two distinct HIIT protocols. The most salient finding from this study revealed 30:15 expended nearly identical kcals as 30:30. In addition, results indicated 30:15 elicited a greater glycolytic contribution and post-exercise O<sub>2</sub> kinetics influence on overall energy expenditure when compared to 30:30, while the 30:30 displayed a greater oxidative contribution to overall EE. An interesting finding from the current study revealed 30:15 resulted in a nearly identical amount of kCals expended compared to 30:30, 261 kCals vs. 258 kCals, respectively. It is important to note that total amount, as well as rate of work performed was held constant during the two HIIT sessions. The only difference between protocols was recovery after each sprint (30 seconds vs. 15 seconds). Despite the similarity in total EE between the two protocols there were significantly different contribution of kcal expenditure among the oxidative and glycolytic systems.

During 30:15 and 30:30, results revealed a noteworthy contribution from the glycolytic system, 14.9% and 10.3%, respectively. However, 30:15 exhibited greater demand from the glycolytic system during exercise when compared to 30:30. The amplified demand from the glycolytic system during 30:15 was due to increased accumulation of blood lactate across all four sets when compared to 30:30 (as reflected in greater glycolytic contribution). This finding is in agreement with those of Gosselin, Kozlowski, DeVinney-Boymel, and Hambridge (2012) that report significant increases in blood lactate concentration when increasing work-to-rest ratios from 30:30 to 60:30 ( $p < .05$ ).

Moreover, lactate is an appropriate metabolite to analyze within this paradigm as it is the end product of anaerobic glycolysis, therefore, increased blood lactate indicates increased contribution from the glycolytic system (4). It seems plausible, that if the “work” duration is consistent, and the “rest” ratio decreases (e.g., 30:30 versus a 30:15), the shorter recovery duration would elicit an increase lactate response due an insufficient recovery duration. In contrast, 30:30 allowed participants 15 additional seconds to recover after each sprint and results seem to indicate the increased recovery duration after each sprint delayed lactate accumulate in comparison to 30:15.

During a recovery period after a high-intensity effort, the oxidative system is the primary pathway to resynthesize ATP (4, 8, 24). During this study, the oxidative system contributed significantly. Indeed, results show that 30:30 displayed a greater contribution from the oxidative system when compared to the 30:15, ~70% vs. ~60%, respectively. The difference in oxidative contribution is likely attributed to the additional 15 seconds of recovery during 30:30 when compared to 30:15. The additional recovery time allowed participants to consume a greater quantity of O<sub>2</sub> and, subsequently, the increase in O<sub>2</sub> consumption may have contributed to shuttling of blood lactate via oxidative pathways (5).

The available research has shown high-intensity, intermittent exercise stimulates an increase in EPOC. (1, 12, 25). Not only does EPOC indirectly represent EE from the phosphagen system, it is also known to replenish O<sub>2</sub> saturation within the muscle, blood, and water, aid in the

removal of lactate post-exercise, repair damaged tissue, and decrease body temperature by utilizing oxidative pathways (1). During this study only a 7-minute post-exercise, along with three 3 minute bouts of post-set metabolic measurements was recorded. Due to the short time duration of post-exercise O<sub>2</sub> measurement, this was purely an analysis of post-exercise O<sub>2</sub> kinetics. However, findings within the current study are consistent with the previous literature stating high-intensity, intermittent exercise elicits a significant effect on post-exercise O<sub>2</sub> kinetics (1, 12, 25). Although this was not the primary aim of the study, results exhibited significantly greater effects on post-exercise O<sub>2</sub> consumption during 30:15 (64 kcals), compared to 30:30 (50 kcals). While the difference in kcal expenditure between the two sessions stemming from post-exercise O<sub>2</sub> kinetics was modest at 14 kcals, the larger focus should be directed towards the notion that both HIIT protocols elicited a noticeable contribution on post-exercise O<sub>2</sub> kinetics. While traditional measures of EPOC range from 1 hour post-exercise to 24 hours post-exercise, our focus centered around within session post-set O<sub>2</sub> consumption as well as immediate post-exercise O<sub>2</sub> consumption (1, 12). Post-set and post-exercise O<sub>2</sub> kinetics is an important aspect of this study because it is meant to serve as a representation of the phosphagen system (ATP and phosphocreatine) contribution to overall EE (2, 10, 21). Even within this relatively narrow frame of post-exercise O<sub>2</sub> kinetics, results indicate that post-exercise O<sub>2</sub> kinetics is valuable component to overall EE as it represented 24% (30:15) and 19% (30:30) of the total session duration.

While the findings within this study are novel and serve to further the body of knowledge around EE in HIIT, it is not without limitations. Specifically, the measurement of blood lactate to represent glycolytic contribution, while validated, has not been utilized during intermittent, treadmill sprinting, but has been shown to portray an accurate representation during running (7, 13). The current study measured blood lactate at two minutes into the three minute passive recovery phase. Scott (2006) has shown blood lactate to peak two minutes after exercise, however, this was examined during resistance training and not during intermittent sprint work. Peak lactate may occur during a different time point following HIIT. Our findings are further restricted by the small sample size, particularly within gender (8 females; 6 males).

The primary findings from this study illustrated that total energy expenditure between 30:15 and 30:30 elicited similar amounts of total kcals expended. Despite nearly identical kcal expenditure, the relative contribution between the glycolytic and oxidative system were different. Our results display the relative contribution between the primary energy systems during a bout of HIIT and the relative contribution during post-exercise O<sub>2</sub> kinetics. This study highlights the importance of utilizing pulmonary gas exchange to represent the oxidative system and EPOC and measuring the change in blood lactates to represent the overall glycolytic contribution to estimate EE during a bout of HIIT.

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## REFERENCES

1. Bahr R, Sejersted OM. Effect of intensity of exercise on excess postexercise O<sub>2</sub> consumption. *Metabolism* 40 (8): 836-841, 1991.
2. Bangsbro J, Gollnick PD, Graham TE, Juel C, Kiens B, Mizuno M, Saltin B. Anaerobic energy production and O<sub>2</sub> deficit-debt relationship during exhaustive exercise in humans. *JPN J Physiol* 442: 539-559, 1990.
3. 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. *Sports Med* 10: 751-576, 2011.
4. Brooks GA. Link between glycolytic and oxidative metabolism. *Sports Med* 37 (4-5): 341-343, 2007.
5. Brooks GA. Current concepts in lactate exchange. *Med Sci Sport* 23: 895-906, 1991.
6. Burgomaster KA, Hughes SC, Heigenhauser GJF, Bradwell SN, Gibala MJ. Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *J Appl Physiol* 98: 1985-1990, 2005.
7. di Prampero PE, Ferretti G. The energetics of anaerobic muscle metabolism: a reappraisal of older and recent concepts. *Resp Physiol* 118: 103-115, 1999.
8. Gatin PB. Energy system interaction and relative contribution during maximal exercise. *Sport Med* 31(10): 725-741, 2001.
9. Gosselin LC, Kozlowski KF, DeVinney-Boymel L, Hambridge C. Metabolic response of different high-intensity aerobic interval exercise protocols. *J Strength Cond Res* 26(10): 2866-2871, 2012.
10. Haseler LJ, Hogan MC, Richardson RS. Skeletal muscle phosphocreatine recovery in exercise-trained humans is dependent on O<sub>2</sub> availability. *J Appl Physiol* 86: 2013-2018, 1999.
11. Hazell TJ, Olver TD, Hamilton CD, Lemon PWR. Two minutes of sprint-interval exercise elicits 24-hr oxygen consumption similar to that of 30 min of continuous endurance exercise. *Int J Sport Nutr Exerc* 22: 276-283, 2012.
12. LaForgia J, Withers RT, Shipp NJ, Gore CJ. Comparison of energy expenditure elevations after submaximal running. *J Appl Physiol* 82: 661-666, 1997.
13. Margaria R, Aghemo P, Sassi G. Lactic acid production in supramaximal exercise. *Pflüg Arch* 326: 152-161, 1971.
14. Margaria R, Cerretelli P, di Prampero PE, Massari C, Torelli G. Kinetics and mechanism of oxygen debt contraction in man. *J Appl Physiol* 18: 1963.
15. Noakes TD, Myburgh KH, Schall R. Peak treadmill running velocity during the VO<sub>2</sub> max test predicts running performance. *J Sport Sci* 8 (1): 35-45, 1990.
16. Pollack ML, Schmidt DH, Jackson AS. Measurement of cardio-respiratory fitness and body composition in the clinical setting. *Compr Ther* 6 (9): 12, 1980.
17. Scott CB. Contribution of blood lactate to the energy expenditure of weight training. *J Strength Cond Res* 20(2): 404-411, 2006.

18. Scott C. Misconceptions about aerobic and anaerobic energy expenditure. *J Int Soc Sports Nutr* 2(2): 32-37, 2005.
19. Scott CB. Anaerobic and aerobic energy expenditure during heavy to severe exercise and recovery. *J Theor Biol* 7: 109-119, 2002.
20. Scott CB, Croteau A, Ravlo T. Energy expenditure before, during, and after the bench press. *J Strength Cond Res* 23(2): 611-618, 2009.
21. Scott CB, Littlefield ND, Chason JD, Bunker MP, Asselin EM. Differences in oxygen uptake but equivalent energy expenditure between a brief bout of cycling and running. *Nutr Metab* 3(1): 1-5, 2006.
22. Skelly LE, Andrews PC, Gillen JB, Martin BJ, Percival ME, Gibala MJ. High-intensity interval exercise induces 24-h energy expenditure similar to traditional endurance exercise despite reduced time commitment. *Appl Physiol Nutr Metab* 39: 845-848, 2014.
23. Thompson WR, Gordon NF, Pescatello LS. ACSM's guidelines for exercise testing and prescription. Philadelphia PA: Lippincott Williams & Wilkins; 2010.
24. Tomlin DL, Wenger HA. The relationship between aerobic fitness and recovery from high intensity intermittent exercise. *J Sport Sci Med* 31(1): 1-11, 2001.
25. Townsend JR, Stout JR, Morton AB, Jajtner AR, Gonzalez AM, Wells AJ, Mangine GT, McCormack WP, Emerson NS, Robinson IV EH, Hoffman JR, Fragala MS, Cosio-Lima L. Excess post-exercise oxygen consumption (EPOC) following multiple effort sprint and moderate aerobic exercise. *Kinesiol* 45: 1:16-21, 2013.
26. Vetter RE. Effects of six warm-ups protocols on sprint and jump performance. *J Strength Cond Res* 21(3): 819-823, 2007.

