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Research article

How to Regulate the Acute Physiological Response to “Aerobic” High-Intensity Interval Exercise

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Abstract

The acute physiological processes during “aerobic” high-intensity interval exercise (HIIE) and their regulation are inadequately studied. The main goal of this study was to investigate the acute metabolic and cardiorespiratory response to long and short HIIE compared to continuous exercise (CE) as well as its regulation and predictability. Six healthy well-trained sport students (5 males, 1 female; age: 25.7 ± 3.1 years; height: 1.80 ± 0.04 m; weight: 76.7 ± 6.4 kg; VO_{2max}: 4.33 ± 0.7 $l\cdot min^{-1}$) performed a maximal incremental exercise test (IET) and subsequently three different exercise sessions matched for mean load (P_{mean}) and exercise duration (28 min): 1) long HIIE with submaximal peak workloads (P_{peak} = power output at 95 % of maximum heart rate), peak workload durations (t_{peak}) of 4 min, and recovery durations (t_{rec}) of 3 min, 2) short HIIE with P_{peak} according to the maximum power output (P_{max}) from IET, t_{peak} of 20 s, and individually calculated t_{rec} (26.7 ± 13.4 s), and 3) CE with a target workload (P_{target}) equating to P_{mean} of HIIE. In short HIIE, mean lactate (La_{mean}) (5.22 ± 1.41 $mmol\cdot l^{-1}$), peak La (7.14 ± 2.48 $mmol\cdot l^{-1}$), and peak heart rate (HR_{peak}) (181.00 ± 6.66 $b\cdot min^{-1}$) were significantly lower compared to long HIIE (La_{mean}: 9.83 ± 2.78 $mmol\cdot l^{-1}$; La_{peak}: 12.37 ± 4.17 $mmol\cdot l^{-1}$, HR_{peak}: 187.67 ± 5.72 $b\cdot min^{-1}$). No significant differences in any parameters were found between short HIIE and CE despite considerably higher peak workloads in short HIIE. The acute metabolic and peak cardiorespiratory demand during “aerobic” short HIIE was significantly lower compared to long HIIE and regulable via P_{mean}. Consequently, short HIIE allows a consciously aimed triggering of specific and desired or required acute physiological responses.

Key words: Intermittent exercise, exercise prescription, acute physiological demand, mean load, peak workload duration.

Introduction

High-intensity intermittent exercise (HIIE) is characterized by repeated bouts of vigorous activity, interspersed by recovery phases of rest or low-intensity exercise. Due to these recovery phases, high peak workloads can be sustained for a longer accumulated time in HIIE than in one bout of continuous exercise (CE) (Gibala et al., 2012; Saltin et al., 1976).

“Aerobic” HIIE has been shown to induce similar or even superior physiological effects on oxidative capacity and endurance performance compared to traditional moderate-intensity CE in both healthy individuals (Billat, 2001; Daussin et al., 2007, 2008; Helgerud et al., 2007) and patients with chronic disease (Iellamo et al., 2013; Kemi and Wisloff, 2010; Meyer et al., 1997; Smart et al., 2013; Wisloff et al., 2007). With respect to the lactate shuttle theory (Brooks, 1986; 2009), we propose that exercise may be called “aerobic” if it provokes a systemic steady state in blood lactate concentration (LaSS) which means a balance of lactate production and elimination (Hofmann and Tschakert, 2011). In addition, even sprint intervals including short bouts of “all out” exercise for only a few seconds have been reported to enhance maximum oxygen uptake (VO_{2max}) primarily due to peripheral muscular adaptations induced by maximal workloads (Burgomaster et al., 2008; Gibala et al., 2006; 2012; Hawley, 2008).

A large part of HIIE studies referred to in literature investigated *training adaptations* such as improvements in VO_{2max}, whereas investigations of an *acute physiological response* to interval exercise are rare. However, depending on the setting of single determinants of HIIE (peak workload (P_{peak}), peak workload duration (t_{peak}), recovery load (P_{rec}), recovery duration (t_{rec}), mean load (P_{mean}), and numbers of intervals) (Buchheit and Laursen, 2013a; 2013b; Gibala et al., 2012; Tschakert and Hofmann, 2013), a broad spectrum of acute metabolic and cardiorespiratory responses can be generated by HIIE (Billat, 2001). The acute physiological response strongly determines the particular muscular and systemic training adaptations (Hawley, 2004; 2008) as well as the potential health risks during exercise, particularly in diseased populations (Meyer et al., 1997; 1998; Keteyian, 2012; Rognmo et al., 2012).

A HIIE model that has been successfully adopted in training intervention studies in both healthy individuals (Helgerud et al., 2007) and patients suffering from different chronic diseases such as heart failure (Wisloff et al., 2007) is the 4 x 4 min regime (t_{peak} = 4 min; P_{peak} = 85-95 % of maximum heart rate (HR_{max}); t_{rec} = 3 min; P_{rec} = 50-70 $\%HR_{max}$). This HIIE model has frequently been called “aerobic” (Helgerud et al., 2007; Rognmo et al., 2004; Tjonna et al., 2008; Wisloff et al., 2007); however,

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the acute physiological response induced by 4 x 4 HIIE has not yet been described.

There is still a lack of knowledge regarding the detailed acute metabolic and cardiorespiratory response to particular HIIE modes and the heterogeneity of this response due to different interval settings (Gibala et al., 2012). In addition, the *regulation* and *predictability* of desired acute physiological responses *before* HIIE remain poorly understood. The acute response to *continuous* exercise is predictable and controllable by setting the intensity and duration. Analogously, the acute physiological response induced by a particular *intermittent* exercise regime should also be controllable by means of a prescription using the aforementioned HIIE determinants. This is of high relevance particularly if HIIE is applied in training intervention studies in healthy and, more importantly, in diseased individuals. A consistent HIIE prescription model that enables the regulation and prediction of acute physiological responses to HIIE hence is needed (Tschakert and Hofmann, 2013).

The acute physiological response to interval exercise is to a large extent influenced by P_{mean} that can be calculated using the equation $P_{mean} = (P_{peak} \cdot t_{peak} + P_{rec} \cdot t_{rec}) / (t_{peak} + t_{rec})$ (equation 1). We recommended setting P_{mean} purposefully as a separate determinant since the mean load determines the acute *mean cardiorespiratory* response and additionally has a crucial impact on the acute *metabolic* response (Tschakert and Hofmann, 2013).

However, t_{peak} and P_{peak} also strongly influence the acute metabolic and peak cardiorespiratory response as emphasized by Astrand et al. (1960) and Saltin et al. (1976) decades ago. They found that HIIE with a long t_{peak} yielded higher acute metabolic and peak cardiorespiratory responses (no LaSS, greater oscillation of HR and VO_2 values around the respective mean values) compared to HIIE with a short t_{peak}. However, the authors did not further explore the relevance of P_{mean} or regulation of the acute response to HIIE.

"Aerobic" HIIE (interval exercise that leads to a LaSS) can be sustained for a longer total duration than HIIE that induces a rising blood lactate accumulation. Therefore, the question arises if the acute physiological response to interval exercise can be controlled and predicted using P_{mean} and, in particular, if HIIE is "aerobic" provided P_{mean} is set below the second lactate turn point (LTP_2) determined in an incremental exercise test (IET), the upper limit to generate "aerobic" constant load exercise (Hofmann et al., 1997; 2005).

The purpose of the present study was to determine the acute cardiorespiratory and metabolic response produced by long (t_{peak} = 4 min) and short (t_{peak} = 20 s) HIIE compared to CE matched for mean load and exercise duration in young healthy subjects. We aimed to determine which HIIE regime is "aerobic" and, in general, allows a *regulation and predictability* of the acute physiological response using P_{mean} according to equation 1. For this purpose the acute physiological responses to *CE* were used as reference values.

We hypothesized that there would be no significant difference in the acute metabolic and peak cardiorespiratory response between short HIIE and CE. However, long HIIE was hypothesized to yield a significantly higher acute physiological response than CE and short HIIE. As a consequence, we hypothesized that the regulation and prediction of the acute metabolic and peak cardiorespiratory response during HIIE using P_{mean} is possible in short rather than in long intervals and that short HIIE but not long HIIE is "aerobic". These working hypotheses were proposed despite the fact that P_{peak} in short HIIE was considerably higher compared to long HIIE and CE.

Methods

Subjects

Six young healthy well-trained sport students (5 males, 1 female; age: 25.7 ± 3.1 years; height: 1.80 ± 0.04 m; weight: 76.7 ± 6.4 kg; VO_{2max}: 4.33 ± 0.7 $l \cdot min^{-1}$) participated in this study. The experimental protocol was approved by the institutional ethical review committee. Once the test design, the experimental procedures, and associated risks had been explained, all subjects gave their written informed consent before participating in this study. They were familiar with the cycle ergometer exercise in our laboratory and were instructed to avoid strenuous exercise 24 hours before each testing session.

Experimental design

All subjects were required to report to the laboratory on four occasions separated by at least two days, and on each occasion the participants completed an exercise test conducted on an electronically controlled and mechanically braked cycle ergometer (Monark Ergomedic 839E, Monark, Sweden).

On their first visit, subjects performed an incremental exercise test (IET) until exhaustion in order to assess VO_{2max}, HR_{max}, and maximum aerobic power output (P_{max}). In addition, the first and second turn points for lactate (LTP_1, LTP_2) and for ventilation (VT_1, VT_2) were determined referring to the three phase model of metabolism (Hofmann and Tschakert, 2011) and to the Lactate Shuttle Theory by Brooks (1986; 2009). Subsequently, the participants performed three different exercise sessions matched for mean load and exercise duration in randomized order: 1) a modified version of the original Norwegian 4 x 4 HIIE model (Helgerud et al., 2007) with a t_{peak} of 4 min (long HIIE); 2) high-intensity interval exercise with a t_{peak} of 20 s (short HIIE); and 3) continuous exercise (CE) with a target workload equating to P_{mean} of both HIIE tests. The duration of each specific exercise session (without resting periods, warm up, and cool down) was 28 minutes. The participants were permitted to cycle at a cadence of 70 - 90 rpm, and each subject completed all tests with the same rpm.

Incremental Exercise Test (IET)

At the beginning of IET, subjects sat quietly on the cycle ergometer for 3 min (0 W). After this initial rest period, they completed a 3 min warm-up at 40 W for males and 20 W for the female, respectively. Then, the workload was increased by 20 W (for males) and 15 W (for female), respectively, every minute until volitional exhaustion occurred according to the standard protocol of the Austrian Society of Cardiology (Wonisch et al., 2008). In the following 3 min recovery period, the same workload as during warm-up was applied, and finally the participants had to rest for 3 min again sitting quietly on the cycle ergometer (0 W). Maximal (P_{max}, HR_{max}) and submaximal markers (LTP_1, LTP_2) were determined in order to prescribe exercise intensities of the following specific continuous and interval-type exercise tests.

High-Intensity Interval Exercise (HIIE) and Continuous Exercise (CE)

All subjects were required to perform three specific exercise sessions (long HIIE, short HIIE, and CE) matched for mean load and exercise duration in randomized order. In total, each test lasted about 47 min. It started with a 3 min resting period (sitting quietly on the cycle ergometer, 0 W), followed by a 10 min warm-up phase consisting of 3 min cycling at 40 W (males) and 20 W (female), respectively, and a 7 min adaptation phase with a workload just below the individual P_{LTP1}. This 3 + 7 min warm-up phase was conducted in order to minimize day-to-day variations in exercise performance and to prepare for the high peak workloads in HIIE. Subsequently, the specific CE or HIIE protocol of about 28 min started. Finally, a 3 min active recovery with 40 W (males) and 20 W (female), respectively, and 3 min passive recovery (0 W) on the cycle ergometer concluded the IET.

According to Helgerud et al. (2007), long HIIE in our study consisted of four intervals (4 x (4+3) min). The work periods of 4 min (t_{peak}) were performed at a P_{peak} corresponding to the power output at 95 % of HR_{max} from IET, and the recovery phases (t_{rec}) of 3 min were performed at a P_{rec} corresponding to the power output at 70 % of HR_{max} from IET. P_{mean} was calculated using the following equation: $P_{mean} = (P_{peak} \cdot t_{peak} + P_{rec} \cdot t_{rec}) / (t_{peak} + t_{rec})$ (Tschakert and Hofmann, 2013).

In short HIIE, t_{peak} was 20 s, P_{peak} corresponded to P_{max} determined in IET, P_{rec} was set just below P_{LTP1} (since P_{LTP1} was suggested to be the point of the optimal lactate clearance rate), and t_{rec} (26.7 ± 13.4 s) was calculated via equation 1. Due to the fact that t_{rec} of short HIIE was calculated for each participant, the number of intervals was different across subjects, but the total exercise trial was approximately 28 min.

CE was performed for 28 min at a target workload equating to P_{mean} of both HIIE tests.

Measurements

A 12-lead electrocardiogram (ZAN 800, ZAN, Winkling, Germany) was obtained from each subject during all tests supervised by an experienced physician. Pulmonary gas-exchange variables were collected continuously during all tests by breath-by-breath measurement and were averaged over 5 s periods (ZAN 600, ZAN, Winkling, Germany). VO_{2max} was defined as the highest 30 sec average value of oxygen uptake. HR data averaged over 5 s periods were also obtained during all tests via chest belt telemetry (PE 4000, Polar Electro, Kempele, Finland). Blood lactate and blood glucose concentrations obtained from capillary blood samples taken from ear lobes during all tests were measured via the fully enzymatic-amperometric method (Biosen S-line, EKF diagnostics, Barleben, Germany).

During IET, capillary blood samples were taken at the end of the rest and warm-up periods, at the end of each workload step, and at the end of active and passive recovery. During each of the three exercise sessions, capillary blood samples were taken accordingly at the end of the rest and 3 min warm up phase, after 1, 3, 5, and 7 min of the adaptation phase and after the 3 min active and 3 min passive recovery. During the specific exercise regimes, the times of blood collection differed: in CE after 1, 2, 3, 4, 5, 10, 15, 20, 25, and 28 min; in 4 x 4 HIIE after 2 and 4 min of the peak workload phases and after 1 and 3 min of the recovery phases; in short HIIE 4 blood collections within 7 min temporally in accordance with long HIIE.

Data analysis procedures

The determination of individual turn points during IET (LTP_1, LTP_2) was accomplished by means of a computer-supported linear regression turn point model within defined regions of interest (ROI) (Hofmann and Tschakert, 2011). ROI for LTP_1 (and VT_1) was between La (and VE) at first workload and La (and VE) at 70 % of P_{max}, ROI for LTP_2 (and VT_2) was between La (and VE) at LTP_1 and La (and VE) at P_{max} (Hofmann et al., 1997, 2001).

For long and short HIIE and CE, mean values for particular parameters were calculated by averaging the values of each subject during the specific intermittent or continuous exercise. Peak values for particular parameters represent the average of the maximum single value of each participant during the specific intermittent or continuous exercise test.

Statistical analysis

All data are presented as mean ± SD and were analyzed using SPSS (IBM SPSS Statistics 19). A one-way repeated measures ANOVA (within factors) was conducted in order to determine the effects of different exercise regimes on the acute response of metabolic and cardiorespiratory parameters. When the analysis revealed a significant difference, post-hoc paired t-tests with a Bonferroni correction were used to locate the origin of the significant difference. Statistical significance was accepted if $p < 0.05$.

Results

Incremental exercise test

The lactate performance curve and the heart rate performance curve (HRPC) are presented in Figure 1. The blood lactate curve showed three phases of blood lactate appearance and two corresponding turn points (LTP_1, LTP_2). LTP_1 and LTP_2 were significantly related to the first (VT_1) and second ventilatory threshold (VT_2) with no significant difference in power output (data not shown).

Intermittent and continuous exercise sessions

All subjects completed all exercise sessions except for one participant who terminated the 4 x 4 min HIIE two min before the end of the session due to exhaustion. P_{peak} was significantly different ($p < 0.05$) between short HIIE

Table 1. Mean and peak values of P, La, HR, and VO_2 during CE, short HIIE (20 s), and long HIIE (4 min) compared to the values at LTP_1, LTP_2, and P_{max} from IET. Data are means (±SD).

		P (W)	**La** (mmol·l^{-1})	**HR** (b·min^{-1})	**VO_2** (l·min^{-1})
Mean Values	**CE**	213.2 (42)	4.14 (1.84)	167 (8.6)	3.32 (.59)
	HIIE 20 sec	217.2 (42.2)	5.22 (1.41)	168 (5.7)	3.37 (.58)
	HIIE 4x4 min	214.5 (43.1)	9.83*‡ (2.78)	167 (4.9)	3.19 (.53)
	at LTP_1	130.0 (24.0)	1.73 (0.63)	132 (13.4)	2.30 (.55)
	at LTP_2	241.3 (36.3)	4.17 (1.33)	169 (4.8)	3.60 (.45)
Peak Values	**CE**	213.2 (42.0)	5.54 (3.45)	177 (10.7)	4.03 (.70)
	HIIE 20 sec	340.0 (47.3) *	7.14 (2.48)	181 (6.7)	4.17 (.74)
	HIIE 4x4 min	279.2 (51.8) *‡	12.37* ‡ (4.17)	188 (5.7) *‡	4.22 (.67)
	at P_{max} IET	340.0 (47.3)	12.17 (2.50)	190 (4.3)	4.33 (.71)

P, power output; La, blood lactate concentration; HR, heart rate; VO_2, oxygen uptake; CE, continuous exercise; HIIE, high-intensity interval exercise; LTP_1, first lactate turn point; LTP_2, second lactate turn point; P_{max}, maximum power output; IET, incremental exercise test. * significant ($p < 0.05$) difference to CE, ‡ significant ($p < 0.05$) difference to short HIIE

(340.0 ± 47.3 W), long HIIE (279.2 ± 51.8 W), and CE (213.2 ± 42.0 W). P_{mean} was 213.2 ± 42.0 W in all three tests corresponding to 88.0 ± 8.5 % of P_{LTP2}. Mean and peak values for La, HR, VO_2, and the respiratory exchange ratio (RER) during the three different exercise regimes are presented in Table 1.

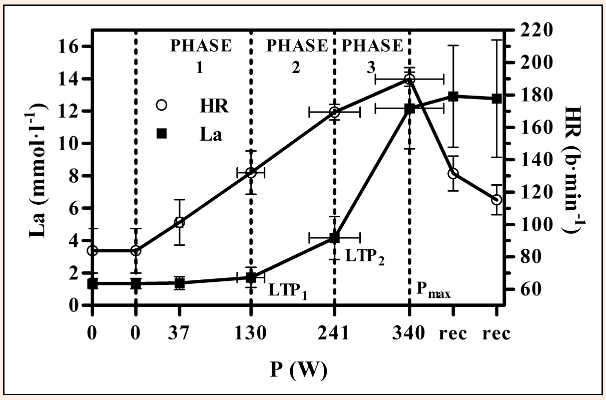

Figure 1. Performance curves for lactate and heart rate during the incremental exercise test. The three phases of lactate metabolism are separated by the first (LTP_1) and second lactate turn point (LTP_2). Values are means ± SD. La, blood lactate; HR, heart rate; P_{max}, maximum power output from the incremental exercise test.

Lactate: Since P_{mean} was below P_{LTP2} (88.0 ± 8.5 %), a lactate steady state was reached in CE as expected. Importantly, a lactate steady state was also reached in short HIIE but *not* in long HIIE (Fig. 2a). The comparison of all three exercise modes revealed significantly higher mean and peak La values in long HIIE compared to short HIIE and CE ($p < 0.05$) but no significant difference between short HIIE and CE (Figure 2a). Peak La during long HIIE was even higher than the maximal La value determined in IET.

Heart rate and oxygen uptake: HR values increased slightly during long and short HIIE and CE (Fig. 2b), but there was no significant difference for *mean* HR between the three tests. However, *peak* HR was significantly higher in long HIIE compared to short HIIE and CE ($p < 0.05$); but, there was no significant difference between short HIIE and CE (Figure 2b).

No significant difference could be found between mean and peak VO_2 values during long HIIE, short HIIE, and CE (Figure 2c).

Respiratory Exchange Ratio (RER): Our study revealed significantly higher mean and peak RER values in long HIIE than in short HIIE and CE ($p < 0.05$) with no significant difference between short HIIE and CE (Figure 2d). The mean and peak RER values decreased with time in all three exercise tests (mean: from 1.03 ± 0.04 to 0.96 ± 0.03; peak: from 1.11 ± 0.04 to 1.05 ± 0.04) (Figure 2d).

Regulation and predictability of the acute physiological response using P_{mean}: Given the acute metabolic and peak cardiorespiratory response to CE was not significantly different from short 20 s HIIE (LaSS in both exercise modes) but significantly lower than long HIIE, we demonstrated the acute physiological response to short HIIE but not to long HIIE was controllable and predictable from P_{mean}.

Discussion

This study investigated the acute metabolic and cardiorespiratory response to long and short HIIE compared to CE as well as the predictability of this acute response using P_{mean} according to equation 1 for both HIIE models in young, healthy, and well-trained subjects.

Our study revealed that short HIIE (t_{peak} = 20 s; P_{peak} = P_{max} from IET) induced an acute metabolic and peak cardiorespiratory response that was *not* significantly different compared to CE but significantly lower than in long HIIE. Short HIIE was "aerobic" in contrast to long HIIE despite a significantly higher P_{peak} in the short intervals. In addition, we found the acute physiological response to short HIIE but not to long HIIE could be controlled for using P_{mean}.

Acute metabolic response

Due to the fact that P_{mean} was lower than P_{LTP2} from IET (88.0 ± 8.5 %), "aerobic" conditions and a lactate steady state, respectively, were targeted in all tests. We found that only CE and *short HIIE* showed a LaSS and therefore were "aerobic". *Long* HIIE was not "aerobic" since it showed no LaSS and it induced blood lactate levels approximating maximal values. These data support the results of Wallner et al. (2013) who investigated the acute physiological response to short HIIE in trained runners.

As emphasized earlier by Astrand et al. (1960), Saltin et al. (1976), and recently by Tschakert and Hofmann (2013), the acute metabolic response to HIIE is

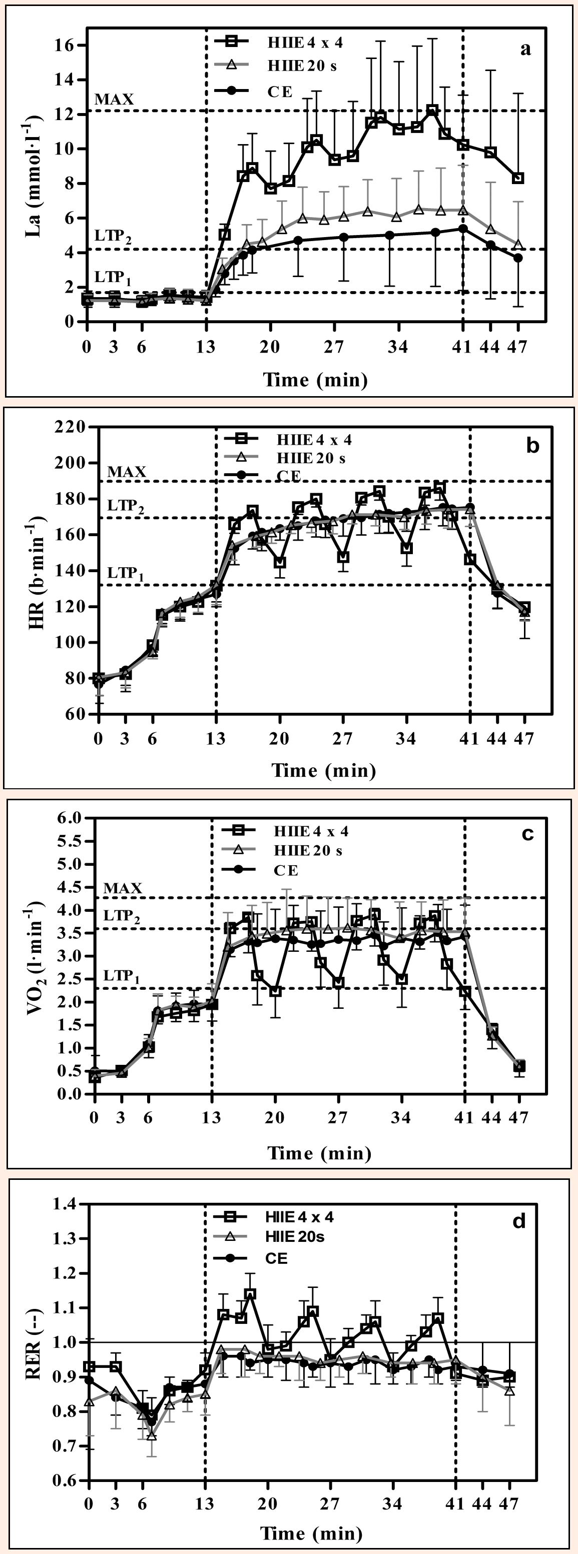

Figure 2a-d. Acute response for La (a), HR (b), VO_2 (c), and RER (d) to long HIIE, short HIIE, and CE. Values are means ± SD. The specific exercise modes were conducted from min 13 to 41 after a standardized warm up phase. In the 4 x 4 min HIIE, one subject finished the peak workload phase of the fourth interval prematurely after 2 min because of exhaustion. La, blood lactate; HR, heart rate; VO_2, oxygen uptake; RER, respiratory exchange ratio; HIIE, high-intensity interval exercise; CE, continuous exercise; P_{max}, maximum power output from the incremental exercise test; LTP_1 and LTP_2, first and second lactate turn point from the incremental exercise test

strongly influenced by the combination of t_{peak} and P_{peak}; if P_{peak} is set above P_{LTP2} (as it is usually done), blood lactate concentration increases with t_{peak} (Beneke et al., 2011; Smekal et al., 2012).

There is no doubt that combinations of t_{peak} and P_{peak} other than the combination used for our short HIIE regime may also generate a balance between lactate production and clearance. Nonetheless, one should be aware of the fact that the higher P_{peak} is, the shorter t_{peak} must be (Tschakert and Hofmann, 2013).

As illustrated in Figure 2a and 2d, the increase of net blood lactate (7.45 $mmol·l^{-1}$; 1.62 $mmol·l^{-1}$; 1.02 $mmol·l^{-1}$; 0.42 $mmol·l^{-1}$) as well as the mean and peak RER values during the four peak workload phases of 4 x 4 min HIIE decreased with each interval. Related to the findings of Parolin et al. (1999), our data suggest that the first 4 min high-intensity exercise bout induced a considerable metabolic acidosis that led to an inhibition of glycolysis and, consequently, to an increased contribution of aerobic metabolism for ATP re-synthesis during the peak workload phases of intervals 2-4. The increase of mean VO_2 from 3.30 ± 0.49 $l·min^{-1}$ during the first peak workload phase up to 3.57 ± 0.60 $l·min^{-1}$ during the last peak workload phase supports this assumption. These metabolic conditions may help explain the significant aerobic adaptations discovered in different 4 x 4 HIIE training studies (Helgerud et al., 2007; Rognmo et al., 2004; Wisloff et al., 2007).

Acute peak cardiorespiratory response

The fact that mean values for HR and VO_2 were not significantly different between all tests (Table 1) was not surprising given mean load and exercise durations were equal. However, the significantly higher *peak* HR values induced by long HIIE compared to both other tests (Table 1) reflected temporarily elevated cardiorespiratory demands during the 4 x 4 min HIIE regime. In short HIIE, the oscillation of peak (and recovery) HR values around HR_{mean} remained low because of the short t_{peak} of 20 s. As a consequence, the peak HR values of short HIIE and CE were similar. This is remarkable given the high peak workload in short HIIE corresponding to P_{max} from IET. Our results are in accordance with the fundamental findings of Astrand et al. (1960) and Saltin et al. (1976) and support the results of Meyer et al. (1997) who have successfully applied HIIE in clinical populations. Meyer et al. (1998) have also shown remarkably stable values for left ventricular function during short HIIE similar to that used in this study, even in patients with heart disease, including heart failure.

Our approach to the exercise *intensity* prescription in long HIIE (*power output at* $\%HR_{max}$) differed from that in the original Norwegian 4 x 4 HIIE model ($\%HR_{max}$) (Helgerud et al., 2007). Therefore, we compared heart rate during peak workload phases in long HIIE (power output at 95 % HR_{max} from IET) and the 95 % HR_{max} value from IET. We found average peak HR at the end of each of the four peak workload phases (180.9 ± 5.6 $b·min^{-1}$ = 95.4 $\%HR_{max}$) was not significantly different from 95 $\%HR_{max}$ from IET (180.2 ± 4.1 $b·min^{-1}$).

The peak power output at 95 $\%HR_{max}$ we used as

P_{peak} for long HIIE in our study represented the upper limit of the range of 85 - 95 $\%HR_{max}$ for the original 4 x 4 HIIE model applied in both healthy (Helgerud et al., 2007) and diseased populations (Wisloff et al., 2007). However, exercise intensities of 90 $\%HR_{max}$ - or even intensities of 85 $\%HR_{max}$, particularly in patients suffering from cardiovascular diseases treated with beta blockers (Hofmann et al., 2005; Wonisch et al., 2003) - might just as well correspond to workloads above the second turn point and induce an accordingly high acute physiological response (Hofmann et al., 2001).

Regulation and predictability of the acute physiological response by means of P_{mean}

Since all tests were matched for P_{mean} and total exercise duration, the acute physiological responses induced by *CE* were used as reference values for the regulation and predictability of the acute response to both interval tests. The acute metabolic and cardiorespiratory response to short 20 s HIIE but not to long 4 x 4 min HIIE was shown to be predictable and controllable using P_{mean}.

This indicates that, despite high peak workloads, the aerobic short HIIE regime allows a deliberately aimed triggering of specific and desired or required acute physiological responses (from very low up to markedly high mean and peak acute responses) dependent on the setting of P_{mean} and using equation 1. As a consequence, aerobic short HIIE can be applied as an endurance training strategy in order to pursue particular training goals in different training periods - both as basic endurance training with a low P_{mean} during the first preparation period and as competition-specific endurance training with a high P_{mean} close to a competition (Wallner et al., 2013). Moreover, given the marked difference in the acute physiological response between the two interval protocols, we suggest aerobic short HIIE is associated with lower health risks compared to long HIIE in patients suffering from a variety of chronic diseases. This suggestion is supported by Keteyian (2012) who pointed to the elevated health risks generated by the 4 x 4 min HIIE regime.

In contrast, long HIIE such as the 4 x 4 min model does not allow low acute metabolic and peak cardiorespiratory responses if P_{peak} is set above P_{LTP2} even if P_{mean} is low. Therefore, use of a long HIIE regime is suggested only in particular training periods and healthy subjects.

Prescription of exercise intensity

An additional aspect noticed in our study was the fact that in long HIIE, the prescription of exercise intensity by means of % HR_{max} from IET resulted in considerably different values for P_{peak}, P_{mean}, and P_{rec} across subjects with respect to their individual P_{LTP2} assessed in IET (minimum vs. maximum value for P_{peak}: 103.3 vs. 129.6 $\%P_{LTP2}$; for P_{mean}: 75.1 vs. 101.6 $\%P_{LTP2}$; for P_{rec}: 37.6 vs. 64.8 $\%P_{LTP2}$). Since CE and both HIIE modes were matched for P_{mean}, the inter-individual diversity of P_{mean} was apparent in all tests. Hence, the relative acute cardiorespiratory and metabolic response during exercise was substantially different across subjects. This inter-individual disparity was caused by different patterns of the heart rate performance curve determined in IET and, as a consequence, by markedly differing $\%HR_{max}$ values at LTP_1 and LTP_2 (minimum vs. maximum value for LTP_1: 57.07 vs. 80.53 $\%HR_{max}$; for LTP_2: 84.34 vs. 94.21 $\%HR_{max}$). This was previously shown by Hofmann et al. (2001, 2005), Tabet et al. (2006), and Wonisch et al. (2003) and recently emphasized by Hofmann and Tschakert (2011) and Tschakert and Hofmann (2013).

In addition, if exercise intensity is prescribed via $\%HR_{max}$, the time it takes for the participants to reach their individual target HR remains unspecified. No information is available about the power output and the metabolic situation until the target HR is reached. Therefore, we suggest that an accurate prescription of exercise intensities by means of $\%HR_{max}$ is inadequate particularly for intermittent exercise with short peak load durations up to 30 s.

Based on the previously established standards to prescribe exercise intensities for *continuous exercise* with respect to the lactate turn points (Hofmann and Tschakert, 2011; Smekal et al., 2012), we confirm our earlier recommendation (Tschakert and Hofmann, 2013) to use $\%P_{LTP2}$ as P_{mean}, $\%P_{LTP1}$ as P_{rec}, and $\%P_{max}$ as P_{peak} for the prescription of *aerobic HIIE*.

Limitations of the study

We must admit the number of participants (n = 6) was rather small in our study. However, the statistical power analysis revealed the statistical power was sufficient for our experimental design. Despite the small number of subjects, the difference in the acute metabolic and peak cardiorespiratory response between short and long HIIE was distinct and in accordance with the fundamental findings of Astrand et al. (1960).

A limit of this study was that P_{mean} was not deliberately set using % P_{LTP2} as recommended by the authors (see above). We wanted to use the original Norwegian 4 x 4 HIIE prescription model (Helgerud et al., 2007), which dictated the setting of the intensity and duration of peak workload and recovery phases. Therefore, P_{mean} was calculated for the 4 x 4 min HIIE regime using equation 1 and adopted for the other two tests since the intent was to match the three exercise modes for P_{mean}. However, average P_{mean} across subjects corresponded to 88.0 ± 8.46 % of P_{LTP2} from IET and, therefore, was in a desired range slightly below the power output at the maximal LaSS for CE.

Conclusion

Our study clearly revealed, in short HIIE, the acute metabolic and cardiorespiratory response is lower than in long HIIE and not different from CE, despite a considerably higher P_{peak} in short HIIE. With a P_{mean} below P_{LTP2}, a LaSS can be reached in CE and in short HIIE (“aerobic” exercise) but not using long HIIE. Moreover, the acute physiological response to short HIIE but not to long HIIE can be controllable and predictable using P_{mean}. Consequently, short HIIE can be expected to generate a longer total exercise duration than long HIIE. In addition, short HIIE is expected to allow directed triggering of specific acute physiological responses. Therefore, short HIIE is applicable to different training periods and subjects dependent on the setting of P_{mean}. In addition, data from our study suggest health risks might effectively be reduced in short HIIE compared to long HIIE for diseased populations.

Further research concerning the detailed acute physiological response and long-term effects induced by different HIIE prescriptions in different populations continues to be required (Hawley, 2008). In particular, studies are needed to investigate if "aerobic" short HIIE induces similar muscular and systemic training adaptations compared to long HIIE. Therefore, further methodological investigations including randomized controlled training intervention studies in both healthy and diseased individuals are required especially for short interval training programs.

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Key points

- High-intensity interval exercise (HIIE) with short peak workload durations (t_{peak}) induce a lower acute metabolic and peak cardiorespiratory response compared to intervals with long t_{peak} despite higher peak workload intensities (P_{peak}) and identical mean load (P_{mean}).
- Short HIIE response is the same as in continuous exercise (CE) matched for P_{mean}.
- It is possible to regulate and predict the acute physiological response by means of P_{mean} for short HIIE but not for long HIIE.
- The use of fixed percentages of maximal heart rate (HR_{max}) for exercise intensity prescription yields heterogeneous exercise stimuli across subjects. Therefore, objective individual markers such as the first and the second lactate turn point are recommend prescribing exercise intensity not only for continuous but also for intermittent exercise.

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