Can crossover and maximal fat oxidation rate points be used equally for ergocycling and walking/running on a track?

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Abstract

Aim. – To verify whether exercise intensities at the crossover point (COP) and maximal lipid oxidation (Lipoxmax) can be used interchangeably regardless of exercise mode, this study compared COP, Lipoxmax and maximal fat oxidation rate (MFO) obtained during two modes of submaximal metabolic exercise tests: stationary cycling under laboratory conditions and walking/running on a track.

Methods. – After preliminary indirect maximal progressive tests, 15 healthy subjects randomly performed submaximal exercise tests on a stationary cycle ergometer (E) and on a track (T), during which gas exchanges and substrate oxidation rates were measured.

Results. – There were no significant mean differences in COP [heart rate (HR): 149 ± 23 beats.min⁻¹ (T), 145 ± 28 beats.min⁻¹ (E); VO2: 2168 ± 896 mL.min⁻¹ (T), 2052 ± 714 mL.min⁻¹ (E)], Lipoxmax [HR: 127 ± 27 beats.min⁻¹ (T), 126 ± 23 beats.min⁻¹ (E); VO2: 1638 ± 839 mL.min⁻¹ (T), 1696 ± 656 mL.min⁻¹ (E)] or MFO [498.3 ± 192.0 mg.min⁻¹ (T), 477.7 ± 221.5 mg.min⁻¹ (E)] between the two modes of exercise. However, Bland–Altman analysis showed a clear disagreement between the two exercise modes and, in particular, a large random error [bias ± random error: for COP, −3.5 ± 53.2 beats.min⁻¹ (HR), −116.8 ± 1556.4 mL.min⁻¹ (VO2); for Lipoxmax, −0.4 ± 43.3 beats.min⁻¹ (HR), −5.7 ± 1286.4 mL.min⁻¹ (VO2); and for MFO, −20.6 ± 384.9 mg.min⁻¹].

Conclusion. – This study showed that, in young, healthy, reasonably fit subjects, exercise mode can affect intensities at the COP and the Lipoxmax. These results, which now have to be confirmed in patients with metabolic defects, suggest the need to perform specific tests to make individualized adaptations to physical activity outside of clinical settings.

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Keywords: Crossover point; Maximal fat oxidation rate; Lipoxmax; Cycling; Running; Walking

Résumé

Le point d’oxydation lipidique maximal (Lipoxmax) et le point de croisement glucidolipidique déterminés sur ergocycle en laboratoire sont-ils transférables à la marche/course sur le terrain ?.

Objectifs. – Vérifier si les intensités d’exercice au point de croisement glucidolipidique (PCGL) et d’oxydation lipidique maximale (Lipoxmax) peuvent être interchangeables en fonction du mode d’exercice. Nous avons donc comparé PCGL, Lipoxmax et le taux d’oxydation maximale lipidique (OLM) obtenus sur ergocycle dans des conditions de laboratoire à ceux obtenus lors d’un test de marche/course sur piste.

Méthodes. – Après la réalisation de tests indirects permettant de déterminer la puissance ou la vitesse maximale aérobie, 15 sujets sains ont effectué deux tests sous-maximaux sur ergocycle (E) ou sur piste (P) durant lesquels les échanges gazeux et l’oxydation des substrats ont été mesurés.

Résultats. – En moyenne, PCGL (fréquence cardiaque [FC]: 149 ± 23 [P] et 145 ± 28 battements par minute [E] et VO2: 2168 ± 896 [P] et 2052 ± 714 mL/min [E]), Lipoxmax (FC: 127 ± 27 [P] et 126 ± 23 battements par minute [E]; VO2: 1638 ± 839 [P] et 1696 ± 656 mL/min [E]) et OLM (498,3 ± 192,0 mg/min/ [P] et 477,7 ± 221,5 mg/min [E]) ne différaient pas entre les deux modalités d’exercice. Toutefois, une analyse en Bland et Altman a montré une grande variabilité individuelle (biais ± erreur aléatoire: pour PCGL, −3,5 ± 53,2 battements...
par minute [FC] et \(-116.8 \pm 1556.4\) mL/min [VO\(_2\)]; Lipox\(_{\text{max}}\), \(-0.4 \pm 43.3\) battements par minute [FC] et \(-5.7 \pm 1286.4\) mL/min [VO\(_2\)]; OLM, \(-20.6 \pm 384.9\) ng/min).

**Conclusion.** Les indicateurs métaboliques déterminés en laboratoire chez le sujet sain sur ergocycle ne sont pas applicables sur le terrain lors d’un exercice de marche/course. Ces résultats sont à confirmer chez des patients atteints de troubles métaboliques. Ils suggèrent que l’utilisation de ces indicateurs sur le terrain nécessite une détermination spécifique de l’activité physique adaptée envisagée.

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**Mots clés :** Point de croisement glucidolipidique ; Taux d’oxydation maximal lipidique ; Lipox\(_{\text{max}}\) ; Exercice sur ergocycle ; Course ; Marche

1. Introduction

Regular exercise is a key part of the treatment for individuals with metabolic defects such as obesity and type 2 diabetes [1]. Nonetheless, the usual exercise recommendations for these individuals do not fully take into account the physiological mechanisms that underlie training effectiveness at an individual level [2]. Exercise training induces metabolic changes in muscle that can restore some of the defects associated with obesity [3] and type 2 diabetes, such as muscle insulin resistance [4,5]. Accordingly, healthcare clinicians have been recommending regular physical activity to their patients [6].

Exercise intensity is one of the main factors influencing substrate utilization during exercise. During steady-state exercise performed at low intensity, a significant amount of lipids is usually oxidized. Moreover, there is an intensity of exercise that elicits the maximal oxidation of lipids (Lipox\(_{\text{max}}\)) [7]. Measurement of Lipox\(_{\text{max}}\) by graded exercise calorimetry is reproducible, but also modifiable by several physiological conditions (training, previous exercise and diet) [7]. Its measurement closely predicts what will be oxidized during 45–60 min of low- to medium-intensity training performed at the corresponding intensity. Brooks and Mercier’s ‘crossover concept’ [8] explains the balance of substrate utilization during exercise. This concept implies that, although increasing exercise intensity results in a preferential use of carbohydrate, endurance training shifts the balance of substrates during exercise towards a stronger reliance on lipids.

A recent review of studies with triathletes showed that several physiological differences are found between cycling and running, as heart rates differ between these two activities at both maximal and submaximal intensities [9]. One recent study showed a 28% higher fat oxidation rate when walking/running compared with cycling, even though the intensity eliciting the Lipox\(_{\text{max}}\) was similar between the two exercise modes [10]. However, such comparisons have been performed in particularly physically fit cyclists, but not in patients with metabolic defects or in less-fit subjects.

Recently, healthcare professionals have begun training patients with metabolic diseases (obesity and/or type 2 diabetes) at their Lipox\(_{\text{max}}\). This exercise intensity is usually determined during exercise calorimetry on a stationary cycle ergometer in a medical setting. However, when prescribing physical activity, it is crucial to take the individual’s interests into account to facilitate long-term compliance with the activity. For this reason, exercise programs that include more enjoyable and more varied outdoor physical activities (involving walking/running), as proposed for other chronic diseases [11], compared with exercising in a clinical setting could favour long-term maintenance of exercise training.

For this reason, the aim of the present study was to verify whether metabolic indicators such as the crossover point (COP), Lipox\(_{\text{max}}\) and maximal fat oxidation rate (MFO), which are determined in a laboratory setting, can be applied to a wider range of activities such as walking and jogging. Thus, the present study assessed the influence of the mode of exercise on these metabolic indicators (cycling on a stationary cycle ergometer vs running/walking on a track).

2. Methods

2.1. Subjects

Fifteen healthy subjects (aged 22–29 years) volunteered to participate in the study (10 men and five women). The study was conducted according to the Declaration of Helsinki and after approval from the local Committee on Human Research. Written informed consent was obtained from all subjects, none of whom presented with any contraindications to prolonged or heavy exercise, or any diseases. The subjects, whose characteristics are presented in Table 1, were physical education students who were relatively physically fit and mostly recreational athletes, except for two competitive cyclists.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Subjects’ characteristics.</th>
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<tr>
<td></td>
<td>Females (n = 5)</td>
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<tr>
<td>General information, anthropometric measurements</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>22 ± 1</td>
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<tr>
<td>Height (cm)</td>
<td>165 ± 7</td>
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<tr>
<td>Weight (kg)</td>
<td>62 ± 8</td>
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<tr>
<td>Body mass index (kg/m(^2))</td>
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<td>Maximal progressive exercise tests</td>
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<td>Maximal aerobic speed (MAS; km.h(^{-1}))</td>
<td>13.4 ± 2.7</td>
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<tr>
<td>VO(_2) max (mL.min(^{-1}); estimated from MAS)</td>
<td>2927 ± 856</td>
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<tr>
<td>Maximal aerobic power (MAP; W)</td>
<td>196 ± 47</td>
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<tr>
<td>VO(_2) max (mL.min(^{-1}); estimated from MAP)</td>
<td>2454 ± 519</td>
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<td>VO(_2) max (% predicted)</td>
<td>145 ± 35</td>
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Data are expressed as means ± SD.
The 13 recreational athletes trained for an average of 2.7 ± 0.5 h/week, while the two competitive cyclists trained for 6.2 ± 0.6 h/week at the time of the study.

2.2. Experimental design

Within the same week, all subjects underwent two indirect maximal progressive tests (3 days apart), one on the track and the other on an electromagnetic cycle ergometer (Ergoselect, Medisoft, Dinant, Belgium), to assess their maximal aerobic speed (MAS) and maximal aerobic power (MAP), and to estimate their VO2max. Subjects’ MAS was estimated using a modified version of the University of Montreal track test, as previously described [12]. During the test, the subjects had to run at a speed set by audible cues. By the time each cue sounded, the participant had to have reached the next cone; the cones were placed every 25 m along the edge of the track. After a 5-min warm-up, the test began at a running speed of 7 km.h⁻¹, which was increased by 0.5 km.h⁻¹ every min until exhaustion. The test ended when the subject could no longer maintain the pace imposed and/or fell behind and was unable to catch up. When the participant was farther than 5 m from a cone at the time of the cue, the test was ended. The MAS was determined as the speed maintained during the final completed stage.

The maximal progressive test performed on the electromagnetic cycle ergometer began at 40 W, and the power output increased by 20 W/min until exhaustion. MAP was determined as the power output associated with the last completed stage and when the maximal heart rate (HRmax) was close to the theoretical HRmax (220–age).

VO2max was estimated, using the MAS reached on the track [13] and the MAP achieved on the cycle ergometer [14] at the end of the maximal progressive tests, according to the following formulas: VO2max (mL.min⁻¹) = MAS × 3.5 × body weight (on the track); and VO2max (mL.min⁻¹) = MAP × 9.96 + 331 (on the cycle ergometer). Predicted VO2max values were calculated using a formula developed on a cycle ergometer [15].

The MAP and MAS values determined during these maximal progressive tests served as the maximal references on which to base protocols for the submaximal exercise tests designed to assess metabolic indices. The order in which the subjects did the submaximal exercise tests on the electromagnetic cycle ergometer (the same machine as used for MAP determination) and the track was assigned randomly.

Before the evaluations, the subjects were familiarized with the procedures and equipment. Exercise tests were always performed at the same time (between 8:00 and 10:00 AM) after at least 12 h of fasting and under similar ambient conditions (15–20°C on the track and 20°C in the laboratory). Subjects were instructed to record what they ate 24 h prior to the exercise tests and to repeat the exact same dietary pattern for all subsequent tests.

All exercise tests were conducted within 3 days and during the week following the two maximal progressive tests. The tests consisted of five 6-min workloads, as proposed by Brooks and Mercier’s crossover concept [16,17]. Gas exchanges and heart rates were continuously recorded with a portable ergospirometer (MetaMax 3B, Cortex Biophysik, Leipzig, Germany). After a 3-min rest period and an initial 3-min warm-up at 20% of MAS or MAP, four 6-min workloads, set at 30%, 40%, 50% and 60% of the MAS or MAP, were performed.

2.3. Calorimetry

Calculation of carbohydrate (CHO) and lipid oxidation rates was assessed from gas exchange measurements according to the non-protein respiratory quotient technique [18]: CHO rate of oxidation (mg.min⁻¹) = 4.585 VCO2 – 3.2255 VO2; and lipid rate of oxidation (mg.min⁻¹) = –1.7012 VCO2 + 1.6946 VO2 (with VO2 and VCO2 in mL.min⁻¹).

As recommended elsewhere [7,16], VO2 and VCO2 were averaged over the last 2 min of each exercise stage to ensure that a steady state was reached and because, at this time, CO2 production from bicarbonate buffers to compensate for the production of lactic acid becomes negligible. This technique provided CHO and lipid-oxidation rates for different levels of exercise, using the assumption that the urinary nitrogen excretion rate was negligible. These values were then converted into Kcal, with 1 g of lipids providing approximately 9 Kcal and the oxidation of 1 g of carbohydrates providing only 4 Kcal. Thus, the percentage CHO or lipids participating in total energy expenditure could be determined.

According to the crossover concept proposed by Brooks and Mercier [8], the COP of substrate utilization is defined as the intensity at which energy from CHO-derived fuels predominates over energy derived from lipids. This point can be identified when approximately 70% of energy is derived from CHO and 30% from lipids. To assess this specific point, the curve between the % oxidized CHO and power output over four points was smoothed to dampen any variations that eventually may have been linked to a point departing from the general curve fit. The MFO point is the exercise intensity at which the increase in lipid oxidation induced by the increasing intensity reaches a peak, which is then followed by a decrease as CHO becomes the predominant fuel. This was calculated from the above equations using the following empirical formula: fat = 1.6946 VO2 – 1.7012 VCO2, simplified as fat = 1.7 (1 – RER) VO2. MFO corresponds to the maximum point of the curve associated with this equation. At this point, the derived equation is equal to zero. As for COP determination, the relationship between RER (respiratory exchange ratio) and power output was smoothed. All smoothing procedures were performed with custom-made software developed by Brun et al. [19].

2.4. Statistical analysis

All parameters were expressed as means ± SD unless otherwise specified. According to their distribution, variables obtained during the track test were compared with those obtained by the cycle ergometer test using Wilcoxon’s test.

Relationships between the metabolic indices (VO2 and HR at COP, Lipoxmax, MFO) obtained from the track and cycle ergometer tests were analyzed using Spearman’s correlation coefficients. Significance was set at \( P < 0.05 \). Also,
Heart rate and oxygen uptake during the cycle ergometer and track-walking/running tests.

COP

Data are expressed as means ± SD. COP: crossover point; HR: heart rate; \( \dot{V}O_2 \): oxygen consumption; Lipox_max: exercise intensity at maximal oxidation rate; MFO: maximal fat oxidation rate.

\( ^a \) Specific to exercise mode.

Bland–Altman analysis was performed to assess, at an individual level, the agreement between the two modes of exercise.

3. Results

3.1. Subjects’ characteristics

As presented in Table 1, our study subjects had heterogeneous levels of fitness; nonetheless, on the basis of their \( \dot{V}O_2 \)max, they were all reasonably physically fit. The heterogeneity of the subjects’ aerobic aptitudes can be explained by their varied athletic backgrounds (gymnastics, basketball, cycling and track and field). However, two subjects were competitive-level cyclists, and presented MAP of 380 W and 400 W, respectively. Nevertheless, the physical fitness of all our subjects depended on exercise mode. As expected, for 11 subjects, the estimated \( \dot{V}O_2 \)max was greater for the track test whereas, in the remaining four, their estimated \( \dot{V}O_2 \)max was greater for the cycle ergometer test.

3.2. Influence of exercise mode on metabolic indices

All subjects began the track test by walking (2.0–5.0 km.h\(^{-1}\)). Fig. S1 (see supplementary material associated with this article online) shows the kinetics of oxygen consumption during the walk–run test for two subjects who had different body weights (A was lighter than B). Weight was found to be a determinant of the metabolic cost during the walk–run transition. The walk–run transition speed varied between subjects (5.0–7.0 km.h\(^{-1}\)) and was 6.2 ± 0.5 km.h\(^{-1}\) overall.

Values for the COP, Lipox_max and MFO obtained during the track and cycle ergometer tests are presented in Table 2. There was no significant difference between the HR and \( \dot{V}O_2 \) at which COP and Lipox_max occurred during both exercise modes. However, the values of COP and Lipox_max obtained on the track were moderately correlated with values obtained on the cycle ergometer (Table 2). As for MFO, there was no significant difference between exercise modes, and a fair relationship was observed between MFO obtained on the track and cycle ergometer. Similarly, substrate oxidation (CHO and lipid oxidation rates) and RER did not differ between the two exercise modes (Fig. S2; see supplementary material associated with this article online).

However, Bland–Altman analysis for COP revealed a large random error (HR: 53.2 beats.min\(^{-1}\) and \( \dot{V}O_2 \): 1556.4 mL.min\(^{-1}\)) despite a small bias (HR: \(-3.5\) beat.min\(^{-1}\) and \( \dot{V}O_2 \): \(-116.8\) mL.min\(^{-1}\); Fig. S3; see supplementary material associated with this article online). As for Lipox_max, a very small bias (HR: \(-0.4\) beats.min\(^{-1}\) and \( \dot{V}O_2 \): \(-5.7\) mL.min\(^{-1}\)) with a large random error for HR and \( \dot{V}O_2 \) measurements (43.3 beats.min\(^{-1}\) and 1286.4 mL.min\(^{-1}\), respectively) were observed (Fig. 1). Furthermore, this analysis showed a major random error with a small bias (384.9 and \(-20.6\) mg.kg\(^{-1}\), respectively) for MFO (Fig. S4; see supplementary material associated with this article online).

For all parameters, no proportional systematic errors were observed, as slopes did not differ significantly from zero (Lipox_max: HR, \( r = 0.95 \) and \( \dot{V}O_2 \), \( r = 0.73 \); COP: HR, \( r = 0.67 \) and \( \dot{V}O_2 \), \( r = 0.38 \); MFO: \( r = 0.077 \)). Examination of the individual data showed clear inconsistencies between both exercise modes, with a maximal difference of 63 beats.min\(^{-1}\) for HR and 2003 mL.min\(^{-1}\) for \( \dot{V}O_2 \). The difference in \( \dot{V}O_2 \) for the metabolic indices between exercise modes did not correlate with the walk–run transition speed (COP: \( r = 0.07 \), \( P = 0.36 \); Lipox_max: \( r = 0.10 \), \( P = 0.73 \)).

Subjects presented with COP values that were higher on the cycle ergometer (\( n = 7 \)), or lower (\( n = 8 \)) when compared with the same exercise on the track in terms of both HR and \( \dot{V}O_2 \). Similarly, Lipox_max values on the cycle ergometer were either higher (\( n = 8 \)) or lower (\( n = 7 \)), and the values for MFO were either higher (\( n = 6 \)) or lower (\( n = 9 \)) compared with the same exercise on the track.

4. Discussion

The main finding of the present study was that the intensities at Lipox_max and at the COP were indeed affected by exercise mode. Despite the lack of difference between the mean exercise intensities (assessed by HR and \( \dot{V}O_2 \)) at different metabolic indices (Lipox_max and COP), marked disagreement
was observed between walking/running and cycling at an individual level.

4.1. Determination of substrate oxidation during exercise

Carbohydrate (CHO) and fatty acids are the dominant fuels oxidized by the muscle for energy production during exercise, and the absolute and relative contributions of these fuels can be influenced by diet [20], muscle glycogen content [21], exercise intensity [22], duration [23] and training status [24].

In the present study, diet was controlled, as subjects were instructed to control and record their food intake 24 h prior to exercise testing (whether cycling or walking/running) and to repeat the exact same dietary pattern for the subsequent exercise test. Thus, although an effect of different muscle glycogen reserves cannot be excluded (a control of diet over a longer period would have been necessary), diet does not appear to account for the discrepancy between the two exercise modalities observed in some subjects.

Also, as ambient temperature can alter substrate oxidation [25], the different exercise modes were carried out under similar ambient temperatures, thus controlling potential bias induced by temperature variations.

One of the most important regulators of substrate oxidation is exercise intensity, as it has been demonstrated that increases in glycolytic flux can inhibit long-chain fatty-acid transport to mitochondria and so reduce long-chain fatty-acid oxidation [26].

In addition, to compare substrate oxidation rates between exercise modes, we selected values at similar levels of absolute oxygen uptake, as others have done previously [10]. This appears to be more relevant, as it takes into account the differences in VO2max between exercise modes. Also, it reinforces the lack of difference between metabolic indices despite greater (but not significant) fat oxidation during the track test. This also confirmed the results obtained by Achten et al. in trained cyclists [10].

Our protocols were standardized to obtain similar durations and metabolic stimulations for both exercise modes. COP and Lipoxmax are altered when theoretical values for MAP are used to plan submaximal metabolic tests [27,28], and especially when the true MAP is lower than the theoretical MAP [28]. However, as our protocols for the submaximal metabolic tests were based on true MAP or MAS values, the metabolic indices determined by each of the exercise modes appear to be valid. Nevertheless, the alteration in the linearity of metabolic cost for the submaximal metabolic progressive track protocol remains questionable. For the track test, all subjects began walking. As the energy cost is greater for running compared with walking [29], the transition between walking and running sharply increases the energy cost (Fig. S1; see supplementary material associated with this article online), impairing either more or less the linearity between oxygen uptake and exercise intensity compared with the relationship obtained on the cycle ergometer. It could be argued that a greater increment in intensity during the walk–run transition may have induced earlier reliance on CHO, thus disturbing calculation of the metabolic indices (earlier occurrence of COP and Lipoxmax). Indeed, Lipoxmax (in seven subjects) and COP (in eight subjects) occurred at a lower metabolic cost on track walking/running vs cycling. Nonetheless, as only two subjects presented RER values close to 0.91 at the end of the first stage of running, it is unlikely that a protocol bias (the walk–run transition) was responsible for the individual disagreement observed between exercise modes. Indeed, such a pattern appears to reflect a glucose-dependent profile, as previously reported in healthy subjects by Brun et al. [16]. In addition, no correlation was observed between the differences in VO2 in the two exercise modes for COP or Lipoxmax and the transition speed. This suggests that the increased metabolic cost during the transition does not explain the disagreement between exercise modes. It would be interesting to verify this aspect in overweight individuals in whom the metabolic cost associated with locomotion is increased compared with their normal-weight peers.

Furthermore, as the different exercise mode tests were progressive and the protocols used were based on a specific maximal intensity (running or cycling) reference, the individual discrepancies cannot be explained by sudden blood lactate and
4.2. H+ increases, shown to restrict carnitine palmitoyltransferase-I activity [30], a key enzyme in fatty-acid transport and, thus, fat oxidation.

Training status can be partially reflected by the level of VO2max, although it may have limited power for predicting endurance performance [31,32]. Previous studies have shown a relationship between VO2max and Lipoxmax levels [33–36]. Exercise involving a larger muscle mass (such as running) generally produces higher VO2max values in untrained individuals [9]. However, cyclists tested on a cycle ergometer can either exceed or equal the VO2max obtained by running [37]. In previous studies, a relationship between metabolic indices (Lipoxmax, MFO) and the VO2max has been found [33,34,36]. We therefore hypothesized that differences in VO2max, due to each subject’s individual preferences/specializations (running vs cycling), could be partly responsible for the differences in Lipoxmax and COP in the two exercise modes. Indeed, there was a discrepancy in estimated VO2max for the majority of subjects involved in the present study. In contrast to the above-mentioned studies, several subjects (n = 4) in the present study presented with a greater estimated VO2max during cycling vs running, implying that cycling was the easier and preferred exercise mode for these subjects. This observation suggests that the preferred exercise mode may have influenced substrate oxidation in some subjects.

A recent study by Achten et al. [10] comparing the intensity that elicits MFO, using cycle ergometer- and treadmill-based protocols, revealed that fat oxidation was higher during walking compared with cycling, although the intensity eliciting MFO did not differ between the two exercise modes. Similarly, our present study could find no significant mean difference between the two exercise modes in terms of intensities (expressed as HR or oxygen consumption) eliciting Lipoxmax and COP. However, our Bland–Altman analysis showed a large random error and, consequently, a clear disagreement between the two exercise modes. Unfortunately, as Achten et al. did not perform such an analysis, no insight was offered concerning the interindividual differences in their study.

We expected fat oxidation to be dependent on exercise mode due to the physiological differences between cycling and walking/running [9]. During running, a larger muscle mass is involved whereas, due to the stabilization of the trunk and arms while cycling, a smaller muscle mass is recruited. Nonetheless, as already mentioned, no significant mean difference was found between the two exercise modes for Lipoxmax, COP or MFO. Moreover, at an individual level, six out of 10 subjects showed greater COP, Lipoxmax and MFO during cycling compared with running. Thus, our results from a group of non-specialized athletes, with a somewhat heterogeneous aerobic capacity, challenge the current literature and suggest the need to analyze metabolic outcomes on an individual basis before opting for a specific exercise mode.

4.3. Practical implication in patients with metabolic defects

It has been clearly shown that individuals with metabolic defects have a decreased ability to oxidize fat. However, previous studies have also suggested that fat oxidation can be improved over time by regular endurance training [16]. At present in patients with metabolic diseases, however, the idea of individualized training is still not widespread. The usual exercise recommendations for type 2 diabetes do not take into account the individual patient’s metabolic background, and only indicate a broad exercise-intensity zone (% VO2max or % HRmax) that is assumed to be effective. Yet, according to Brun et al. [7], patients with type 2 diabetes have values of Lipoxmax that are markedly shifted to lower power intensities, thereby lowering the MFO. For this reason, individual evaluations of Lipoxmax have been suggested to devise an exercise program at MFO. Nevertheless, in individuals with glucose-dependent profiles, it remains a challenge to identify the precise exercise intensity that targets maximal lipid oxidation. In this case, a flattened lipid-oxidation curve should lead to a broader intensity zone, provided that the recommended exercise duration is fulfilled [38].

To prepare a personalized exercise programme, the individual’s physiological characteristics should be assessed and taken into account. However, changing physical activity behaviours in inactive individuals remains a key challenge. Many patients with metabolic disease find it difficult to undertake and adhere to an effective exercise programme, which highlights the importance of adapting the appropriate physical activity according to the patients’ interests and capabilities. For many people, outdoor walking- and running-based activities are more enjoyable and accessible than exercising in a closed environment. Together with improvements in metabolic health, adapted physical activities are more enjoyable and encourage exercise adherence [11]. This highlights the importance of verifying whether the metabolic indicators evaluated under laboratory conditions can be applied to a wider range of activities in ‘real life’.

Based on the results of our present study, Lipoxmax and COP should be evaluated under the same conditions in which the patient intends to either continue or begin an individualized exercise programme to optimize the effects on metabolic health.

5. Conclusion

The present study showed that the mode of exercise (cycling vs running/walking on a track) can affect the intensities at the COP and Lipoxmax in a young, healthy and relatively fit population. Consequently, the metabolic indices determined on cycle ergometry under laboratory conditions are not necessarily applicable in the field. This suggests that designing an individualized training programme of walking or running requires testing...
specific to the exercise mode. Finally, the results of the present study need to be confirmed in patients with metabolic diseases.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

Appendix A. Supplementary data

Supplementary data (Figs. S1–S4) associated with this article can be found, in the online version, at http://www.sciencedirect.com at doi:10.1016/j.diabet.2012.02.001.

References


