Sports Medicine and Rehabilitation

Research Article Open Access

Balance of Substrates at Exercise in Athletes: Lipodependent vs Glucodependent Sports [Version 1, 2 Approved with Reservations]

Jean-Frederic Brun[1], Emmanuelle Varlet-Marie[2,3], Marlène Fichou[1], François Bughin[1], Eric Raynaud de Mauverger[1], Christine Fédou[1] and Jacques Mercier[1]

[1]UMR CNRS 9214-Inserm U1046, «Physiopathologie & Médecine Expérimentale du Cœur et des Muscles – PHYMEDEXP», Unité d'Explorations Métaboliques (CERAMM), Université de Montpellier, Département de Physiologie Clinique, Hôpital Lapeyronie CHU Montpellier, France
[2]Institut des Biomolécules Max Mousseron (IBMM) UMR CNRS 5247, Université de Montpellier, Ecole Nationale Supérieure de Chimie de Montpellier, France
[3]Laboratoire de Biophysique & Bio-Analyses, Faculté de Pharmacie, Université de Montpellier, France

*Corresponding author: Jean-Frédéric Brun, Unité d'Explorations Métaboliques (CERAMM), Université de Montpellier, Département de Physiologie Clinique, Hôpital Lapeyronie CHU Montpellier, France, Email: j-brun@chu-montpellier.fr

Original Submission

Received: January 24, 2017
Accepted: February 24, 2017
Published: February 28, 2017
Last Updated: June 24, 2017

Open Peer Review Status: 2 Approved with Reservations

How to cite this article: Jean-Frederic Brun, Emmanuelle Varlet-Marie, Marlène Fichou, François Bughin, Eric Raynaud de Mauverger, Christine Fédou, Jacques Mercier. Balance of Substrates at Exercise in Athletes: Lipodependent vs Glucodependent Sports [Version 1, 2 Approved with Reservations]. Sports Med Rehabil. (2017) 1: 3.1

Abstract

Recent development of exercise-tests assessing the balance of substrates used for oxidation at exercise evidences various profiles among various populations, and training appears to influence these parameters. This study aimed at comparing selected groups of subjects submitted to different well-defined training protocols. We investigated the balance of substrates oxidized at exercise in 90 athletes submitted to different training protocols (28 cyclists, 32 soccer players, 19 male rugby players, 11 rugby women, compared to 41 controls) during an exercise-test consisting of five six-minute steady-state workloads designed for measuring carbohydrate and fat oxidation with indirect calorimetry. The power at which lipid oxidation reached a maximum expressed as a percentage of VO2max capacity ranked as follows: cyclists (59.5 ± 2.8) > female rugby players (45.1 ± 4.0) > male rugby players (39.5 ± 6.5) > female controls (39.6 ± 3.7) > male controls (32.5 ± 4.5) > soccer players (17.9 ± 2.0). Thus, beside the expected picture of athletes oxidizing higher quantities of lipids than controls (cyclists and male rugby players had a high ability to oxidize lipids), we evidence here, in a sample of soccer players, an opposite pattern of "early glucodependence".

Keywords

Substrate Crossover; Lipid Oxidation; Power of Maximal Lipid Oxidation; Soccer; Rugby; Cycling; Exercise; Lipid Oxidation; LIPOXmax

Introduction

While the balance of substrates utilized for oxidation at exercise has long been subject of interest [1-2], exercise-tests designed to assess this balance in athletes or patients referred for metabolic diseases have only recently been developed [3-4]. Since low intensity training increases lipid oxidation [5-6] while high intensity training improves the ability to oxidize carbohydrates (CHO) [7-8], this latter effect being reversed by overtraining [7], the individual assessment of this balance of substrates used at exercise may provide an evaluation of the metabolic effects of training protocols designed for improving lipid oxidation [4,6,9].

However, the reason for the inter-individual variability of these parameters remains poorly understood [10-13]. Clearly, energetic pathways favored by specific training programs may be markedly different among sports, some of them involving endurance activity and thus lipid oxidation and other ones involving mostly CHO oxidation. Thus, this study aimed at clarifying this issue by comparing selected groups of subjects submitted to different training protocols, in order to define which, if any, is the 'specific metabolic profile', in terms of balance of substrates, of each of these groups.

Research Design and Methods

Subjects

Subjects used in this study were 90 trained athletes: 28 cyclists, 32 male soccer players, 19 male rugby players, 11 rugby female players (national level in soccer and male rugby and regional level in cyclism and female rugby) and 41 healthy sedentary volunteers. The subjects were checked to be on good health and were free of medication. Written informed consent was obtained from subjects. The study was approved by the local medical ethics committees of Montpellier, France, in accordance with the revised Helsinki Declaration and the local regulations. All athletes had been involved in regular training for several years (>3 years), and trained 10.69 ± 0.9 hr/wk (training volumes are shown in Table 1). Their training was set by their team coaches and was not influenced by the study. The soccer team performed over the year a combination of endurance training under the form of interval training, strength training, speed training, skill and tactical training, in various proportions according to the period. Male rugby players and female rugby players underwent an heavy training mostly based on strength training. The cyclists performed 14 hours of cycling (i.e., about 450 km) per week during a nine-month training period. During the first month, training sessions were performed at low intensity with a specific target (below their ventilatory threshold: VT). During the other months, they added interval-training sessions to their endurance training, wherein they performed at high intensity with a specific target heart (above their VT).

Prior to the exercise-test, subjects' body composition was assessed with bioimpedance analysis with a six terminal impedance plethismograph BIACORPUS RX 4000 Biacorpus RX4000, (SoAGIL DEVELOPPEMENT, 8 avenue Jean-Jaurès 92130 Issy-les-Moulineaux, France) with data analysis with the software BodyComp 8.4. This device measures total resistance of the body to an alternative electric current of 50 kHz [14-15]. Body fat mass, fat-free mass were calculated in each segment of the body according to manufacturer's database-derived disclosed equations, and total water with published equations using the classical cylindric model and Hanai's mixture theory [16].

Table 1: Clinical characteristics of the 90 athletes of the study.

	Cyclists	Soccer players	Male rugby players	Female rugby players
Number	28	32	19	11
Age (yr)	30.87 ± 1.86	24.34 ± 0.63	25.91 ± 1.25	24.47 ± 0.67
Weight (kg)	70.91 ± 1.36	75.59 ± 1.04	104.45 ± 2.43	67.14 ± 1.73
Height (cm)	177.38 ± 1.28	177.75 ± 0.79	183.09 ± 3.35	165.50 ± 1.40
BMI (kg/m²)	22.74 ± 0.33	23.86 ± 0.20	31.28 ± 0.79	24.55 ± 0.67
Waist circumference	79.06 ± 1.46	79.4 ± 1.90	98.59 ± 2.43	74.53 ± 1.58
Hip circumference	94.70 ± 0.99	94.50 ± 1.39	108.50 ± 1.45	97.42 ± 1.56
Training (hr/week)	10.57 ± 0.71	13.45 ± 0.88	13.91 ± 0.84	7.16 ± 0.31
Overtraining score	10.07 ± 1.76	6.52 ±0.81	16.33 ± 3.38	7.00 ± 1.38
VO2max[a]	61.71 ± 2.94	43.36 ± 1.20	45.68 ± 3.03	37.23 ± 1.72
Crossover point[b]	47.23± 3.38%	17.33± 3.54%	47.05±4.26%	47.96±3.81%
LIPOXmax[b]	42.43%± 2.14%	19.23±2.29%	38.56±3.98%	43.33±2.84%
MFO mg/min	420.55±35.78	167.00±15.04	459.18±69.05	254.82±18.17
MFO mg/min/kg	5.94±0.48	2.43±0.21	4.38±0.65	3.87±0.31
LIPOX%Wmax	40.57±2.33	16.91±2.40	37.66±4.01	36.84±3.05
LIPOX%Wmax th	42.07±1.89	21.41±2.02	34.88±4.01	42.94±2.92
Crossover point %VO_2max	46.94±3.72	21.33±3.65	47.40±4.78	42.05±4.20
Crossover point %VO_2max th	46.39±2.93	23.13±3.00	42.59±4.35	47.59±3.92

Values are expressed as mean ± SEM; BMI = Body Mass Index;
[a] Units: VO_2max, ml/min/kg body weight. [b] Units: %VO_2max

Table 2: Clinical characteristics of the 60 control subjets of the study.

	Males	Females
Number	20	21
Age (yr)	30.13 ± 4.29	25.43 ± 1.27
Weight (kg)	77.66 ± 5.20	58.69 ± 1.18
Height (cm)	178.00 ± 3.68	164.75 ± 1.51
BMI (kg/m²)	21.22 ± 1.30	21.80 ± 0.24
Waist circumference	79.38 ± 2.82	68.22 ± 1.44
Hip circumference	93.50 ± 0.96	96.69 ± 1.10
VO_2max[a]	39.51 ± 1.60	32.05 ± 0.54
Crossover point[b]	51.98±4.38%	48.72±2.85%
LIPOXmax[b]	47.90±3.56%	48.45±3.16%
MFO mg/min	186.57±19.44	197.79±12.06
MFO mg/min/kg	3.14±0.30	3.28±0.20
LIPOX%Wmax	32.25±5.28	40.67±4.19
LIPOX%Wmax th	24.57±3.31	46.55±5.95
Crossover point %VO_2max	35.91±4.52	45.49±5.18
Crossover point %VO_2max th	26.28±2.74	50.34±6.65

Values are expressed as mean ± SEM; BMI = Body Mass Index;
[a] Units: VO_2max, ml/min/kg body weight

All sportsmen answered a standardised questionnaire developed by the French Society of Sports Medicine in order to quantify the early clinical symptoms of the overtraining syndrome [17]. This questionnaire includes 54 items; the number of positive items gives a numerical "score" that helps to evaluate the degree of exercise overload in sportsmen submitted to a heavy training program.

Exercise Testing

The subjects performed an exercise testing on an electromagnetically braked cycle ergometer (Ergoline Bosch 500) connected to a breath by breath device (COSMED Quark CPET) for gas exchange measurements. The theoretical maximal aerobic power (Wmax th) was calculated for all sportsmen using Wasserman's equations [18]. After an overnight fast, at 9 am, subjects underwent a standardized submaximal exercise-test [4-6,9] consisting of five 6-min submaximal steady-state workloads, with calculation of carbohydrate and lipid oxidation rates from gas exchange measurements at steady state at the 5th-6th min of every step according to the nonprotein respiratory quotient technique [19]. After smoothing of the curves, we calculated two parameters representative of the balance between fat and carbohydrate oxidation: the crossover point [20-22] which is the point where carbohydrate becomes the predominant fuel representing more than 70% of the total energy [20] and the point where lipid oxidation reaches a maximum [4-6,9,19].

Validity and reproducibility of this test were assessed in a previous publication. Coefficients of variation (CV) were calculated for RER, LIPOXmax and COP at four different intensities. CV of RER were between 2.8 and 4.75%. CV of LIPOXmax and COP was respectively 11.41% Pmax and 11.63%Pmax. VO2, CO2, RER, CHO and lipid oxidation rates were also compared during the incremental test and during single steady-state workloads of the same intensity performed at random order. These parameters were not significantly different.

Statistical Analysis

Significant differences among the various groups were determined with the non-parametric ANOVA (Kruskall-Wallis test). For all statistical analyses, values were expressed as mean (±SEM) and significance was accepted at $p<0.05$. All calculations were performed with the Sigmastat package (Jandel Scientific, Erkrath, Germany).

Results

Characteristics of patients are shown on tables 1 and 2, which show that there were no significant differences for age among the studied groups. Male rugby players had a higher weight and higher waist and hip circumference than both control males and other athletes. All subjects had normal blood pressure at rest and exhibited no abnormality during exercise and recovery. When expressed as raw power values, the Power of maximal lipid oxidation and the crossover point ranked as follows: male rugby players > cyclists > male controls > female rugby players > female controls > male soccer players (Figure 1). When they were expressed as percentages of theoretical maximal power this ranking became: cyclists > female rugby players > male rugby players > female controls > male controls > soccer players (Figure 2). Raw lipid oxidation rates at the level of the Power of maximal lipid oxidation ranked as follows (Figure 3): male rugby players > cyclists > female rugby players > sedentary male controls > soccer. If lipid oxidation is expressed per kg of body weight this ranking becames: cyclists > male rugby players > female rugby players > sedentary female controls > sedentary male controls > soccer players (Figure 4).

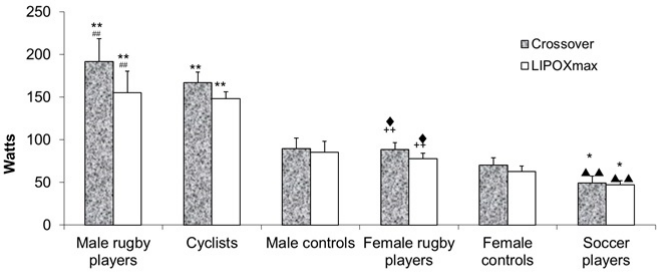

Figure 1: Comparison of the power at which occur the crossover point and the Power of maximal lipid oxidation in control subjects and in various groups of athletes. *p<0.05; **p<0.0001 (male athletes vs. male control subjects); ##p<0.0001 (male rugby players vs. soccer players); ΔΔp<0.0001 (cyclists vs. soccer players); ◊p<0.05 (female rugby players vs. female control subjects); ++p<0.0001 (female rugby players vs. male rugby players).

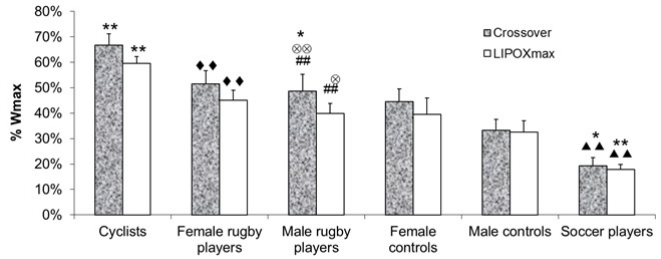

Figure 2: Comparison of the crossover point and the Power of maximal lipid oxidation, expressed in % of Wmax, in control subjects and in various groups of athletes. *p<0.05; **p<0.0001 (male athletes vs. male control subjects); ##p<0.0001 (male rugby players vs. soccer players); ΔΔp<0.0001 (cyclists vs. soccer players); ◊◊p<0.0001 (female rugby players vs. female control subjects); ⊗ p<0.05; ⊗ ⊗p<0.0001 (cyclists vs. male rugby players).

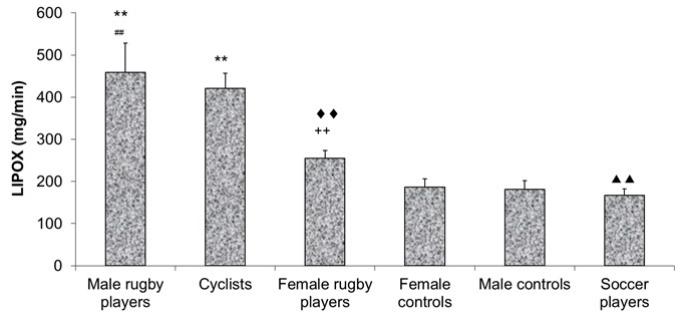

Figure 3: Lipid oxidation rates in control subjects and in various groups of athletes, expressed in mg/min. **p<0.0001 (male athletes vs. male control subjects); ##p<0.0001 (male rugby players vs. soccer players); ΔΔp<0.0001 (cyclists vs. soccer players); ◊◊p<0.0001 (female rugby players vs. female control subjects); ++p<0.0001 (female rugby players vs. male rugby players).

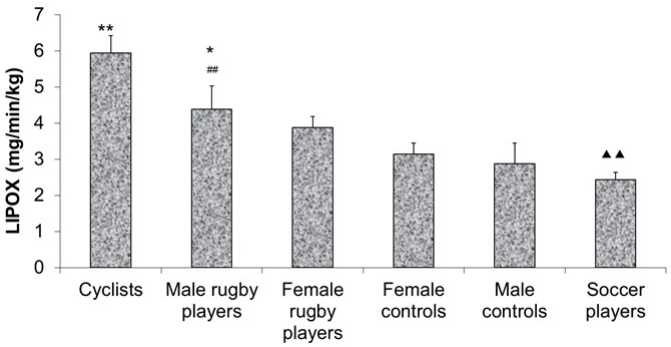

Figure 4: Lipid oxidation rates in control subjects and in various groups of athletes, expressed in mg/min/kg. *p<0.05; **p<0.0001 (male athletes vs. male control subjects); ΔΔp<0.0001 (cyclists vs. soccer players); ##p<0.0001 (male rugby players vs. soccer players).

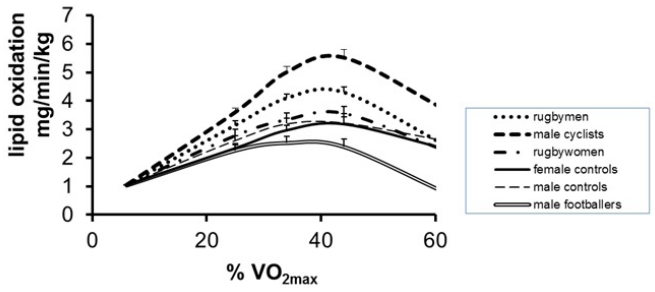

Figure 5: Lipid oxidation rates (corrected by body weight and thus expressed in mg/min/kg) in control subjects and in various groups of athletes. Male cyclists are far above the other groups (p<0.001) and football players are below the other groups and below controls (p<0.01).

Discussion

This study evidences markedly different patterns of balance of substrates among groups of athletes. Clearly, cycling and rugby are rather characterized by high rates of lipid oxidation which peaks at high exercise intensities, while in this specific sample of soccer players there is an early predominance of CHO.

Specific exercise-tests have been designed for measuring the balance of substrates at exercise in either athletes [10] or patients [4,23]. Based on our previous studies on calorimetry during long duration steady-state workloads [7-8,24] we developed a test [4] consisting of five 6-min submaximal steps, in which a steady-state for gas exchanges was obtained during the 2 last minutes. Actually, there is now a large body of literature to support the validity of such protocols of exercise calorimetry [13]. The theoretical concern was that, when exercise is performed above the lactate threshold, there is an extra CO2 production which can be assumed to interfere with the calculations [25]. In fact, below 75% of the VO2max, this increase in CO2 has no measurable effect on calorimetric calculations [26], so that these calculations closely predict oxidation rates measured by stable isotope labelling [27]. Clearly, even at high intensity exercise, respiratory gases are mostly a reflect of the balance of substrate oxidation. Even more, despite the theoretical uncertainty about the stability of gas exchanges during so short bouts, protocols of graded exercise calorimetry with only 3-min duration steps have been carefully validated and successfully used [3,11,28], further supporting the accuracy of our protocols based on 6-min steps. Interestingly, results given by the 3-min protocol and results given by the 6-min protocol are very similar, if one compares the description of the balance of substrates in endurance athletes in the current study and to that of Achten's previous paper [10]. On the whole, we think that graded exercise-tests for exercise calorimetry can nowadays be considered as validated. Two findings suggest that such explorations may provide useful information in sports medicine. First, the fact that training markedly influences the balance of substrates as assessed with this method [5-6,9]. Then, the fact that there are very different patterns of substrate oxidation among athletes, which seem to indicate that there are to some extent specific metabolic profiles.

The finding of a high ability to oxidize lipids in athletes submitted to regular endurance training, like cyclists, is consistent with previous literature [10]. There are some papers assessing the balance of substrates in healthy subjects, for example Haufe [29] reports a level of maximal lipid oxidation at 43% of VO2max in women and 42% in men (with respectively a peak value of fat oxidation at 230 mg/min and 300 mg/min). Similarly Bogadanis [30] reports a level of maximal lipid oxidation at 40.1% of VO2max in women and 39.5 % in men (with respectively a peak value of fat oxidation at 200 mg/min and 310 mg/min). In trained endurance athletes the level of maximal lipid oxidation is generally found to be higher, at 63-65 % of VO2max [3,31]. More recently Gonzalez-Haro [32] reported a level of maximal lipid oxidation at 52% of VO2max in male road cyclists (with a peak value of fat oxidation at grossly 400 mg/min). Romijn [33] reported in such athletes a level of maximal lipid oxidation ranging between 57 and 75 % of VO2max. Most of these papers employ Achten's protocol [3] with 3 min steps, which in our experience slightly overestimates exercise lipid oxidation levels [34], but it is clear that a large body of evidence shows that endurance athletes oxidize more lipids at exercise and that this oxidation peaks at a higher %VO2max than sedentary subjects.

By contrast, it is interesting to notice in soccer players, a pattern of "glucodependence" that implies a reduced reliance on lipids at exercise. Although in our study we can only present data on soccer, this pattern is likely to occur in several sports. Since exercise training at high intensity [7-8] and intermittent exercise [23] both increase the ratio between CHO and fat used for oxidation during muscular activity, this pattern may reflect an adaptation of muscle metabolism to short repeated bouts of high intensity. Daussin [35] has shown that 5 sessions per week of high intensity training increases the ability to oxidize carbohydrates with molecular adaptations at the mitochondrial level. Interestingly, such a "glucodependence" is also found in obesity [4] and type 2 diabetes [36]. In this case it can be rap-

idly reversed by a few weeks of targeted exercise training at the level of the Power of maximal lipid oxidation [5-6,37]. A recent randomized control trial on obese patients has shown that endurance training targeted at the level of maximal oxidation strongly increases the maxima lipid oxidation rate at exercise [38]. Since physical inactivity rapidly shifts the balance of substrates at rest towards a lower ratio of lipid/CHO used for oxidation [39] it can be assumed that sedentarity explains at least in part the glucodependence of these patients, while endurance training explains a reversal of this profile toward more lipid oxidation. Bruce [40] has shown that 5 sessions of one hour per week of endurance training in obese subjects induces a twofold increase in mitochondrial fat oxidation and a four-fold increase in carnitine palmitoyl transferase 1 (CPT1) activity.

Bruce [40] and Sahlin [41] have demonstrated that exercise-induced lipid oxidation is a reflect of mitochondrial fat oxidation. Accordingly, in a sample of diabetics we reported [42] that low intensity endurance training twice a week improves lipid oxidation during exercise parallel to an increase in mitochondrial fat oxidation. Therefore the bell-shaped curve of lipid oxidation at exercise is a reflect of the potential of fat oxidation by mitochondria in (mostly type-I) muscle fibers.

Although not tightly matched for age, all athletes were between 20-35 years old and thus age is not likely to interfere with our results. By contrast, there are differences in body composition, since male rugby players are both heavier and taller than the other athletes and exhibit a higher BMI and waist circumference. Actually, we found no correlation in our sample of athletes between these markers of body composition and the balance of substrates. Although it is clear that adiposity in sedentary subjects is associated with an alteration in muscular substrate metabolism, this relationship does not clearly appear in athletes. Moreover, sedentary subjects with a higher BMI exhibit a lower ability to oxidize lipids at exercise when explored by exercise calorimetry [4,9] while in this study male rugby players have both a higher BMI and a maximal lipid oxidation occurring at a higher power intensity. Thus, it is not likely that our results are explained by a higher adiposity.

Actually, concerning the ability to oxidize lipids, there is also a wide range of variability among the populations studied here, as already reported by the team of Jeukendrup [10-11,28]. These authors found an influence of two factors: fat-free mass and gender [11]. The repeatedly reported influence of gender [43-44] is not significant in our study, but Figure 2 shows that the shift towards CHO seems to occur at a slightly higher level in female than male subjects, either for sedentary controls or for rugby players. However, the magnitude of this difference does not seem to be very important, in comparison to the specific pattern of each sport, and to the marked increase in lipid oxidation induced by training in obese adults [5-6,9,45]. Our maximal lipid oxidation rates close from 500 mg/min in male rugby players and cyclists are consistent with previous reports [10] of a value of 1000 mg/min on treadmill, reduced by 50% during cycling [46]. However, there are also groups of trained subjects exhibiting a markedly lower ability to oxidize lipids, suggesting that beside the already reported factors that are gender [11-12,43-44] fat-free mass [10-12,28] and the training status [47], the variety of sport is probably an important determinant of the ability to oxidize lipids during exercise. Besides, the aging-induced decline in CHO oxidation [8] does not appear in this study on young, fit subjects. Whether our findings can be extended to larger populations of athletes trained for cycling, rugby or soccer remains of course to be demonstrated. Presumably, various training regimens may induce a different profile. We have observed that some soccer players that had initially a high ability to oxidize lipids exhibited a progressive shift toward glucodependence when submitted to the same training procedure as the others of the team presented here. Since the balance of substrates appears to be training-sensitive [6-9,24,45], it is likely that changes in training protocol will modify the profiles presented here. In addition, overreaching may reverse some training-induced modifications of the balance of substrates, as demonstrated in a follow-up study of cyclists [24].

In summary, our study shows that in some athletes, like the soccer players studied here, there is a physiological pattern of balance of substrates at exercise that mimics the “glucodependence” already reported in obese and type-2 diabetic patients. The mechanism of this adaptation remains to be further studied. However, this finding further suggests that the balance of substrates assessed with exercise calorimetry is a rather flexible physiological characteristics of an individual which may be interesting to explore in athletes submitted to various training protocols.

Acknowledgments

We are grateful to the medical team and the coaches of Montpellier-Hérault Sport Club and Mauguio cycling team for them participation in the study. The unit in which was performed the current study is endorsed by the French Society of Sports Medicine and the French Society of Physiology but not endorsed by the ACSM.

References

1. Christensen EH. Metabolism and respiratory functions during heavy physical work. Scand Arch Physiol. 1932; 91-160.
2. Edwards H, Margaria R, Dill D. Metabolic rate, blood sugar and the utilization of carbohydrate. Am J Physiol. 1934; 108-203.
3. Achten J, Gleeson M, Jeukendrup AE. Determination of the exercise intensity that elicits maximal fat oxidation. Med Sci Sports Exerc. 2002; 34: 92-97.
4. Perez-Martin A, Dumortier M, Raynaud E, Brun J-F, Fedou C, et al. Balance of substrate oxidation during submaximal exercise in lean and obese people. Diabe-

tes Metab. 2001; 27: 466-474.

5. Dumortier M, Perez-Martin A, Pierrisnard E, Mercier J, Brun J-F. Regular exercise (3x45 min/wk) decreases plasma viscosity in sedentary obese, insulin resistant patients parallel to an improvement in fitness and a shift in substrate oxidation balance. Clin Hemorheol Microcirc. 2002; 26: 219-229.

6. Dumortier M, Brandou F, Perez-Martin A, Fedou C, Mercier J, et al. Low intensity endurance exercise targeted for lipid oxidation improves body composition and insulin sensitivity in patients with the metabolic syndrome. Diabetes Metab. 2003; 29: 509-518.

7. Manetta J, Brun J-F, Maimoun L, Galy O, Coste O, et al. Carbohydrate dependence during hard-intensity exercise in trained cyclists in the competitive season: importance of training status. Int J Sports Med. 2002; 23: 516-523.

8. Manetta J, Brun J-F, Prefaut C, Mercier J. Substrate oxidation during exercise at moderate and hard intensity in middle-aged and young athletes versus sedentary men. Metabolism. 2005; 54: 1411-1419.

9. Brandou F, Dumortier M, Garandeau P, Mercier J, Brun J-F. Effects of a two-months rehabilitation program on substrate utilization during exercise in obese adolescents. Diabetes Metab. 2003; 29: 20-27.

10. Achten J, Jeukendrup AE. Maximal fat oxidation during exercise in trained men. Int J Sports Med. 2003; 24: 603-608.

11. Achten J, Jeukendrup AE. Optimizing fat oxidation through exercise and diet. Nutrition. 2004; 20: 716-727.

12. Brun J-F, Perez-Martin A, Fedou C, Mercier J. Parameters quantifying the balance of substrates at exercise in women : are they redundant, may the be predicted by -ils anthropometry? Ann Endocrinol. (Paris). 2000; 6: 387.

13. Jeukendrup AE, Wallis GA. Measurement of substrate oxidation during exercise by means of gas exchange measurements. Int J Sports Med. 2005; 26: S28-37.

14. Brun J-F, Guiraudou M, Mardemootoo C, Traoré A, Raingeard I, et al. Validation de la mesure segmentaire de la composition corporelle en comparaison avec la DEXA: intérêt de la mesure de la masse grasse tronculaire. Science & Sports. 2013; 28: 158-162.

15. Guiraudou M, Maimoun L, Dumas J-M, Julia M, Raingeard I, et al. Composition corporelle mesurée par impédancemétrie segmentaire (BIAS) et performance de sprint chez les rugbymen / Body composition measured by bioimpedance segmental (BIAS) analysis and sprint performance in rugby players, Science & Sports. 2015; 30: 298-302.

16. Jaffrin MY, Morel H. Body fluid volumes measurements by impedance: A review of bioimpedance spectroscopy (BIS) and bioimpedance analysis (BIA) methods. Medical Engineering & Physics. 2008; 30: 1257-1269.

17. Brun J-F. The overtraining: to a system of evaluation usable by routine examination. Science & Sports. 2003; 18: 282-286.

18. Wasserman K, Hansen J, Su DY, Whipp BJ. Principles of exercise testing and interpretation. 2nd edn. Philadelphia: Lea & Febiger. 1987; 274.

19. Brun J-F, Varlet-Marie E, Romain AJ, Mercier J. Measurement and physiological relevance of the maximal lipid oxidation rate during exercise (LIPOXmax). Sports Medicine and Sports Injuries. 2011.

20. Brooks GA. Mammalian fuel utilization during sustained exercise. Comp Biochem Physiol B Biochem Mol Biol. 1998; 120: 89-107.

21. Brooks GA, Mercier J. Balance of carbohydrate and lipid utilization during exercise: the "crossover" concept. J Appl Physiol (1985). 1994; 76: 2253-2261.

22. Brooks GA, Trimmer JK. Glucose kinetics during high-intensity exercise and the crossover concept. J Appl Physiol (1985). 1996; 80: 1073-1075.

23. Perez-Martin A, Raynaud E, Aïssa Benhaddad A, Fedou C, Brun J-F, et al. Balance of substrate oxidation during exercise in sedentary obeses and type II diabetics. Diabetes Metab. 2000; 26: 37.

24. Manetta J, Brun JF, Perez-Martin A, Callis A, Prefaut C, et al. Fuel oxidation during exercise in middle-aged men: role of training and glucose disposal. Med Sci Sports Exerc. 2002; 34: 423-429.

25. Macrae HS, Noakes TD, Dennis SC. Role of decreased carbohydrate oxidation on slower rises in ventilation with increasing exercise intensity after training. Eur J Appl Physiol. 1995; 7: 523-529.

26. Romijn JA, Coyle EF, Hibbert J, Wolfe RR. Comparison of indirect calorimetry and a new breath 13C/12C ratio method during strenuous exercise. Am J Physiol. 1993; 263: E64-E71.

27. Christmass MA, Dawson B, Passeretto P, Arthur PG. A comparison of skeletal muscle oxygenation and fuel use in sustained continuous and intermittent exercise. Eur J Appl Physiol Occup Physiol. 1999; 80: 423-435.

28. Venables MC, Achten J, Jeukendrup AE. Determinants of fat oxidation during exercise in healthy men and

women: a cross-sectional study. J Appl Physiol (1985). 2005; 98: 160-167.

29. Haufe S, Engeli S, Budziarek P, Utz W, Schulz-Menger J, et al. Determinants of exercise-induced fat oxidation in obese women and men. Horm Metab Res. 2010; 42: 215-221.

30. Bogdanis GC, Vangelakoudi A, Maridaki M. Peak fat oxidation rate during walking in sedentary overweight men and women. J Sports Sci Med. 2008; 7: 525-531.

31. Knechtle B, Müller G, Willmann F, Kotteck K, Eser P, et al. Fat oxidation in men and women endurance athletes in running and cycling. Int J Sports Med. 2004; 25: 38-44.

32. González-Haro C, Galilea PA, González-de-Suso JM, Drobnic F, Escanero JF, et al. Maximal lipidic power in high competitive level triathletes and cyclists. Br J Sports Med. 2007; 41: 23-28.

33. Romijn JA, Coyle EF, Sidossis LS, Rosenblatt J, Wolfe RR. Substrate metabolism during different exercise intensities in endurance-trained women. 2000; 88: 1707-1714.

34. Bordenave S, Flavier S, Fédou C, Brun J-F, Mercier J. Exercise calorimetry in sedentary patients: procedures based on short 3 min steps underestimate carbohydrate oxidation and overestimate lipid oxidation. Diabetes Metab. 2007; 33: 379-384.

35. Daussin FN, Zoll J, Ponsot E, Dufour SP, Doutreleau S, et al. Training at high exercise intensity promotes qualitative adaptations of mitochondrial function in human skeletal muscle. J Appl Physiol. 2008; 104: 1436-1441.

36. Ghanassia E, Brun JF, Fedou C, Raynaud E, Mercier J. Substrate oxidation during exercise: type 2 diabetes is associated with a decrease in lipid oxidation and an earlier shift towards carbohydrate utilization. Diabetes Metab. 2006: 604-610.

37. Venables MC, Jeukendrup AE. Endurance training and obesity: effect on substrate metabolism and insulin sensitivity. Med Sci Sports Exerc. 2008; 40: 495-502.

38. Besnier F, Lenclume V, Gérardin P, Fianu A, Martinez J, et al. Individualized Exercise Training at Maximal Fat Oxidation Combined with Fruit and Vegetable-Rich Diet in Overweight or Obese Women: The LIPOXmax-Réunion Randomized Controlled Trial. PLoS One. 2015; 10: e0139246.

39. Blanc S, Normand S, Pachiaudi C, Fortrat JO, Laville M, et al. Fuel homeostasis during physical inactivity induced by bed rest. J Clin Endocrinol Metab. 2000; 85: 2223-2233.

40. Bruce CR, Thrush AB, Mertz VA, Bezaire V, Chabowski A, et al. Endurance training in obese humans improves glucose tolerance and mitochondrial fatty acid oxidation and alters muscle lipid content. Am J Physiol Endocrinol Metab. 2006; 291: E99-E107.

41. Sahlin K, Mogensen M, Bagger M, Fernström M, Pedersen PK. The potential for mitochondrial fat oxidation in human skeletal muscle influences whole body fat oxidation during low-intensity exercise. Am J Physiol Endocrinol Metab. 2007; 292: E223-230.

42. Bordenave S, Metz L, Flavier S, Lambert K, Ghanassia E, et al. Training-induced improvement in lipid oxidation in type 2 diabetes mellitus is related to alterations in muscle mitochondrial activity. Effect of endurance training in type 2 diabetes. Diabetes Metab. 2008; 34: 162-168.

43. Friedlander AL, Casazza GA, Horning MA, Buddinger TF, Brooks GA, et al. Effects of exercise intensity and training on lipid metabolism in young women. Am J Physiol. 1998; 275: E853-863.

44. Friedlander AL, Casazza GA, Horning MA, Huie MJ, et al. Training-induced alterations of carbohydrate metabolism in women: women respond differently from men. J Appl Physiol. 1998; 85: 1175-1186.

45. Brandou F, Savy-Pacaux AM, Marie J, Bauloz M, Maret-Fleuret I, et al. Impact of high- and low-intensity targeted exercise training on substrate utilization in obese boys submitted to a hypocaloric diet. Diabetes Metab. 2005; 3: 327-335.

46. Achten J, Venables MC, Jeukendrup AE. Fat oxidation rates are higher during running compared with cycling over a wide range of intensities. Metabolism. 2003; 52: 747-752.

47. Bergman BC, Brooks GA. Respiratory gas-exchange ratios during graded exercise in fed and fasted trained and untrained men. J Appl Physiol (1985). 1999; 86: 479-487.