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High cycling cadence reduces carbohydrate oxidation at given low intensity metabolic rate

AUTHORS: Beneke R[1], Alkhatib A[2]

[1] Abt. Medizin, Training und Gesundheit, Inst. Sportwissenschaft und Motologie, Philipps Universität Marburg, Germany
[2] Academy of Sport and Sport & Physical Activity, Sheffield Hallam University, United Kingdom

ABSTRACT: Cycling cadence (RPM)-related differences in blood lactate concentration (BLC) increase with increasing exercise intensity, whilst corresponding divergences in oxygen uptake ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) decrease. Aim of the present study was to test whether a higher RPM reduces the fraction (%) of the $\dot{V}O_2$ used for carbohydrate oxidation (relCHO) at a given BLC. Eight males (23.9 ± 1.6 yrs; 177 ± 3 cm; 70.3 ± 3.4 kg) performed incremental load tests at 50 and 100 RPM. BLC, $\dot{V}O_2$ and $\dot{V}CO_2$ were measured. At respiratory exchange ratios (RER) < 1, relCHO were calculated and the constant determining 50 % relCHO (k_{CHO}) was approximated as a function of the BLC. At submaximal workload $\dot{V}O_2$, $\dot{V}CO_2$, and relCHO were lower (all $p < 0.002$; $\eta^2 > 0.209$) at 50 than at 100 RPM. No differences were observed in $\dot{V}O_2$peak (3.96 ± 0.22 vs. 4.00 ± 0.25 $l \cdot min^{-1}$) and RER_{peak} (1.18 ± 0.02 vs. 1.15 ± 0.02). BLC was lower ($p < 0.001$; $\eta^2 = 0.680$) at 50 than at 100 RPM irrespective of cycling intensity. At 50 RPM, k_{CHO} (4.2 ± 1.4 $(mmol \cdot l^{-1})^3$) was lower ($p = 0.043$; $\eta^2 = 0.466$) than at 100 RPM (5.9 ± 1.9 $(mmol \cdot l^{-1})^3$). This difference in k_{CHO} reflects a reduced CHO oxidation at a given BLC at 100 than at 50 RPM. At a low exercise intensity, a higher cycling cadence can substantially reduce the reliance on CHO at a given metabolic rate and/or BLC.

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Correspondance author:
Ralph Beneke
Abt. Medizin, Training und Gesundheit, Inst. Sportwissenschaft und Motologie, Philipps Universität Marburg, Jahnstr. 12, 35037 Marburg, Germany, Phone: ++49 6421 2823955, Fax: ++49 6421 2828972, E-mail: ralph.beneke@staff.uni-marburg.de

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INTRODUCTION

Cycling cadence alterations are known to influence oxygen uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), respiratory exchange ratio (RER) and levels of blood lactate concentration (BLC) over a wide range of exercise intensities [1,2,3]. BLC differences between varying cadences increase with exercise intensity. The differences in $\dot{V}O_2$ and $\dot{V}CO_2$ converge as exercise intensity increases with small or no differences at peak power (P_{peak}) [3,4]. At cycling cadences of 90 to 100 revolutions per minute (RPM) this resulted in lower performances at the lactate threshold defined as an identifiable non-linear BLC-increase with increasing workload [4], at the second lactate deflection point [5], or at 2 and 4 $mmol \cdot l^{-1}$ [6] compared to cadences of 40 to 60 RPM. However, top class professional road cyclists prefer cadences above 90 RPM during racing, testing in the laboratory and training [7-9] and at exercise intensities above 85 % of the maximum oxygen uptake they showed lower BLC, $\dot{V}O_2$ and root-mean square EMG values at cadences at 100 RPM than at 60 RPM [8]. Prolonged constant load tests seem to show equivocal BLC, power output and intensity results at the transition from the heavy to the severe intensity domain as indicated by maximal lactate steady state results at different cadences [10-12]. While there is no difference in the critical power-$\dot{V}O_2$ relationship there is a lower critical power at a cycling cadence of 100 RPM compared with 60 RPM [13]. Whether the above partly equivocal cadence-effects on $\dot{V}O_2$, $\dot{V}CO_2$, RER, BLC and sustainable mechanical power at prolonged exercise reflect also changes in the reliance on carbohydrates (CHO) as substrate of aerobic metabolism at a given exercise intensity related to P_{peak} (Int_P) and also as fraction of $\dot{V}O_{2peak}$ (Int_{VO_2}), is not known.

Aerobic CHO oxidation is regulated by the pyruvate dehydrogenase (PDH). Activators of the PDH are pyruvate, CoA and NAD^+. Allosteric cofactors include Mg^{2+}, Ca^{2+} and Mn^{2+} [14,15]. Substrate availability in terms of pyruvate and lactate is possibly one of the most potent effectors of PDH-activation, as food deprivation is known to shift metabolism to increased fat utilization. At a given metabolic rate, this results in a conservation of endogenous carbohydrates [16-21]. The corresponding decrease in muscular PDH-activation counts as a key regulator of this carbohydrate conserving effect. One identified factor of this muscle fibre-specific effect is the muscle fibre-specific PDH phosphatase profile [20].

Cycling cadence-dependent differences in cardio-respiratory and metabolic acute responses have been linked with muscle fibre acti-

vation. Higher fast twitch muscle fibre (FTF) recruitment is associated with higher cadences particularly at relative low exercise intensities [22-26]. Compared to the slow twitch fibre (STF) enzymatic profile, FTF have a higher content of anaerobic glycolytic enzymes combined with lower aerobic mitochondrial protein content [27-29]. These fibre-specific enzymatic properties result in lower PDH-activity combined with higher muscle lactate concentration in exercising muscles with approximately 70 % FTF compared with musculature of approximately 50 % FTF [28]. This higher PDH-activity in the more aerobic muscle has been related to the higher PDH-content rather than to a higher activation of the PDH [28] which in combination with a more marked dephosphorylation of the PDH-E1α site 1 suggests that in FTF the PDH is less sensitive to the availability of pyruvate.

BLC, $\dot{V}O_2$ and $\dot{V}CO_2$ measurements are routine exercise testing measures. RER calculations and estimates of absolute and relative rates of fat and carbohydrate (relCHO) oxidation are often related to $\dot{V}O_2$ and/or exercise intensity [30-34]. Compared to the concept of relating CHO-management to exercise intensity, the interpretation of relCHO as a function of the BLC and thus substrate availability is a change of paradigm. The bi-directional dynamic equilibrium between pyruvate and lactate is very much on the side of lactate. Thus the BLC serves as an indicator of the substrate activation of the whole body relCHO, which functionally reflects the mix of the activation of the PDH-complex of all aerobic tissues [10,35-37]. This approach was already used to address potential maturation-related differences in the reliance on CHO during exercise. It offered an explanation as to why under aerobic exercise conditions the reliance on CHO and the BLC are independent of maturation, whilst BLC but not relCHO is higher at high intensity exercise in more mature subjects [36].

Therefore the present study investigated the within subject effect of cycling cadence on the interrelationship between relCHO and BLC. The experiment was based firstly, on the frequently observed increase in cadence-related BLC-differences and the decreases in corresponding differences of $\dot{V}O_2$ and $\dot{V}CO_2$ at increasing exercise intensity [3,4]; secondly, the concept that the above observations reflect higher FTF-recruitment at higher cadences [24,26]; and thirdly, on data suggesting that in FTF the PDH is less sensitive to the availability of pyruvate as substrate of mitochondrial metabolism [28]. The above background led to the hypothesis that compared with lower cadences a higher cycling cadence causes a higher metabolic rate, a higher BLC and a higher relCHO at a given mechanical power but a lower relCHO at given BLC-levels.

MATERIALS AND METHODS

Subjects. Eight healthy males (Tab. 1) volunteered and gave written informed consent to participate in this study, which was approved by the university's ethics committee. All experiments were performed in accordance with the ethical standards of the Helsinki declaration. The subjects were instructed to avoid any strenuous exercise or alcohol consumption, and consume the same diet for the 24 hours preceding a test, and to arrive in a fully hydrated state without consuming any heavy meals for at least 2 hours prior to the test. Subjects were familiarized with all testing procedures prior to the testing day. All tests were performed at a similar time of day.

Exercise testing

All subjects arrived at the laboratory approximately one hour before testing. Test preparation included a short interview about exercise, sleep, food intake and drinking during the previous 24 hours to exclude issues likely to affect the experimental results. Subjects completed two separate incremental load cycling tests at 50 and 100 RPM on an electro-magnetically braked cycle Ergometer (Lode Excalibur Sport, The Netherlands) in a counter balanced random order. The tests were completed within a period of two weeks and under similar environmental conditions (19.0 ± 0.3 °C, 1022 ± 4 mmHg and 55 ± 3% humidity). Seat and handle bar positions were recorded for the first test and reproduced for the subsequent test. Each test started with a mechanical power of 1.0 $W \cdot kg^{-1}$ body mass, which was increased by 0.5 $W \cdot kg^{-1}$ after every second minute. Subjects cycled until volitional exhaustion, defined as the inability to maintain the required cycling cadence for longer than 15 seconds despite verbal encouragement.

Measurements

$\dot{V}O_2$ and $\dot{V}CO_2$ were continuously measured breath-by-breath throughout the incremental power test (Oxycon Gamma, Mijnhard, The Netherlands). Flow sensor and gas analyzers were calibrated using a 3 l syringe and gases of known concentration prior to each test.

TABLE 1. Anthropometric data, peak performance and corresponding physiological acute response

	50 RPM (mean ± SE)	100 RPM (mean ± SE)	Sig.	η²
Age (years)	23.9 ± 1.6			
Height (cm)	177 ± 3			
Body mass (kg)	68.3 ± 3.5			
P_{peak} (W)	290 ± 18	286 ± 19		
$relP_{peak}$ ($W \cdot kg^{-1}$)	4.3 ± 0.2	4.2 ± 0.2		
$\dot{V}O_{2peak}$ ($l \cdot min^{-1}$)	3.96 ± 0.22	4.00 ± 0.25		
$rel\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	58.2 ± 1.7	58.7 ± 2.1		
BLC_{peak} ($mmol \cdot l^{-1}$)	10.0 ± 0.5	11.8 ± 0.7	p=0.007	0.669
RER_{peak}	1.18 ± 0.02	1.15 ± 0.02		

Note: Peak power (P_{peak}), Peak power related to body mass ($relP_{peak}$), Peak oxygen uptake ($\dot{V}O_{2\,peak}$), Peak oxygen uptake related to body mass ($rel\dot{V}O_{2\,peak}$), highest blood lactate concentration measured during the post-exercise period (BLC_{peak}), respiratory exchange ratio at P_{peak} (RER_{peak})

The breath-by-breath oxygen uptake data were reduced to stationary averages of the final 30 s of each stage.

Capillary blood samples (20 μl) for BLC analysis were collected from the hyperaemic (Finalgon forte®, Thomae) ear lobe during the final 15 s of each stage and every minute after test termination up to the fifth minute. All BLC samples were immediately analyzed utilizing the enzymatic amperometric method (Ebio Plus, Eppendorf).

Data processing and statistics

Peak power (P_{peak}) was equivalent to the power at the final stage if a test was terminated after completion of the full stage. If the test was terminated before 2 min had been completed, P_{peak} was calculated as: P_{peak} (W) = power of previous stage (W) plus power increment (W) times duration of exercise at the final stage (s) divided by 120 s. $\dot{V}O_{2\,peak}$ ($ml \cdot min^{-1}$) was defined as the highest $\dot{V}O_2$ averaged throughout a 30 s time segment of the final minute of the test. BLC_{peak} ($mmol \cdot l^{-1}$) was determined as the highest BLC measured during the post-test period. Int_P and Int_{VO_2} were calculated as percentage of P_{peak} and $\dot{V}O_{2peak}$, respectively. CHO was calculated from $\dot{V}O_2$ ($ml \cdot min^{-1}$) and $\dot{V}CO_2$ ($ml \cdot min^{-1}$) measured at each stage via indirect calorimetry [38]. The relCHO was calculated as the fraction (%) of the $\dot{V}O_2$ used for CHO with 100 % reflecting a respiratory exchange ratio (RER) $\geq$ 1.0. As a first approximation relCHO was individually plotted as a function of the BLC ($relCHO = 100\% / (1 + k_{CHO}/BLC^n)$) [10,35-37]. k_{CHO} is defined as the constant determining relCHO of 50 %, and n is set 3.

Descriptive results are presented as mean ± SE. Respiratory and BLC data at P_{peak} and at all stages below P_{peak}, and relCHO of each stage with an RER < 1.0 were tested for normal distribution using Kolmogorov-Smirnov test. Peak data of 50 RPM and 100 RPM tests were compared using paired t-test. A cadence-by-power ANOVA analysis was used to analyze all sub-peak data measured with 'cadence' as within and 'power' as between factor. Significant interactions and main effects were further analyzed using t-tests and paired samples t-tests as appropriate. Based on cadence and power effects on $\dot{V}O_2$ and BLC observed previously [39], assuming normal distribution an a priori power calculation revealed the necessity of a sample size of n = 8 to achieve a power of 80 % at a significance level of $p \leq 0.05$. Effect sizes in the form of eta^2 (η^2) were calculated.

RESULTS

P_{peak}, peak respiratory data and BLC_{peak}, and all corresponding data at power stages below P_{peak} were normal distributed. P_{peak}, $\dot{V}O_{2peak}$ and RER_{peak} were not different between cycling cadences (Tab.1). There were main effects of cadence and power in $\dot{V}O_2$ ($p < 0.001$; $\eta^2 = 0.528$ and $p < 0.001$; $\eta^2 = 0.874$), $\dot{V}CO_2$ ($p < 0.001$; $\eta^2 = 0.472$ and $p < 0.001$; $\eta^2 = 0.907$), BLC ($p < 0.001$; $\eta^2 = 0.600$ and $p < 0.001$; $\eta^2 = 0.640$) and relCHO ($p = 0.002$; $\eta^2 = 0.209$ and $p < 0.001$; $\eta^2 = 0.785$) with interaction of cadence and power in BLC ($p = 0.042$; $\eta^2 = 0.280$) and relCHO ($p = 0.043$; $\eta^2 = 0.278$).

At both cadences, the $\dot{V}O_2$ and the $\dot{V}CO_2$ increased with each increase in workload ($p < 0.01$). A corresponding increase in the BLC was significant above a workload of 2.5 $W \cdot kg^{-1}$ whilst relCHO increased from 1.5 to 2.5 $W \cdot kg^{-1}$ irrespective of cadence. At both cadences, Int_P and Int_{VO_2} were highly correlated, however, inclination of the regression line was steeper at 50 RPM than at 100 RPM resulting in higher Int_{VO_2} at given Int_P at 100 RPM than at 50 RPM which diminished towards P_{peak} (Fig. 1). Consequently, at a given sub-maximal workload $\dot{V}O_2$ but also $\dot{V}CO_2$ were lower at 50 RPM than at 100 RPM (Fig. 2).

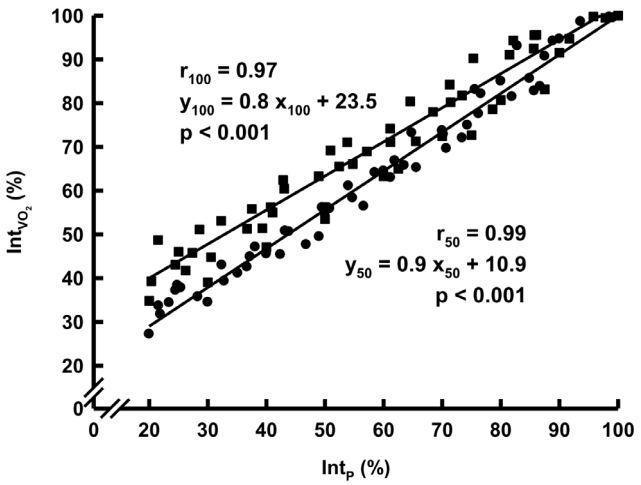

FIG. 1. Exercise intensity related to $\dot{V}O_{2\,peak}$ (Int_{VO_2}) as a function of exercise intensity related to P_{peak} (Int_P) at 50 RPM (●) and at 100 RPM (■).

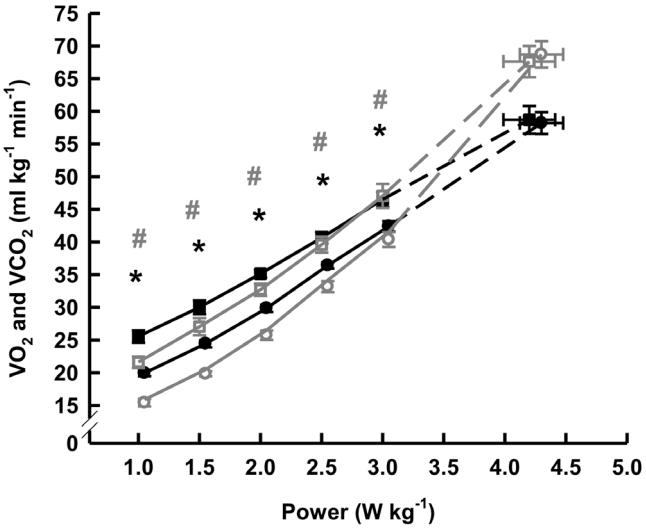

FIG. 2. $\dot{V}O_2$ (black) and $\dot{V}CO_2$ (grey) at 50 RPM (● and ○) and at 100 RPM (■ and □); * = $\dot{V}O_2$ difference between 50 and 100 RPM; mean ± SE; # = $\dot{V}O_2$ difference between 50 and 100 RPM; all $p < 0.05$.

The BLC was lower ($p < 0.001$) at 50 RPM than at 100 RPM irrespective of workload (Fig. 3). A corresponding effect on relCHO was seen up to a workload of 2.0 $W \cdot kg^{-1}$ (Fig. 3), which was equivalent to BLC levels of 1.8 ± 0.2 $mmol \cdot l^{-1}$ vs. 2.7 ± 0.3 $mmol \cdot l^{-1}$ ($p = 0.003$; $\eta^2 = 0.745$), and Int_{VO_2} of 51.6 ± 1.4 % vs. 60.5 ± 2.5 % ($p < 0.01$), whilst Int_P of 47.5 ± 1.8 % vs. 48.4 ± 2.3 % (n.s.) were not different at 50 and 100 RPM. At the individually highest workload (2.9 ± 0.2 $W \cdot kg^{-1}$ vs. 2.8 ± 0.3 $W \cdot kg^{-1}$; n.s.) with an RER < 1.0 (0.97 ± 0.01 vs. 0.99 ± 0.01; n.s.), the BLC was lower (2.9 ± 0.3 $mmol \cdot l^{-1}$ vs. 4.0 ± 0.3 $mmol \cdot l^{-1}$; $p = 0.009$; $\eta^2 = 0.651$) at 50 RPM than at 100 RPM but not Int_P (68.6 ± 2.4 % vs. 64.5 ± 3.4 %; n.s.) and Int_{VO_2} (71.4 ± 3.0 % vs. 74.0 ± 3.0 %; n.s.) or relCHO (89.5 ± 1.9 % vs. 95.1 ± 1.2 %; n.s.).

At 50 RPM k_{CHO} (k_{CHO50}: 4.2 ± 1.4 $(mmol\ l^{-1})^3$) was lower ($p = 0.043$; $\eta^2 = 0.466$) than the k_{CHO} at 100 RPM (k_{CHO100}: 5.9 ± 1.9 $(mmol\ l^{-1})^3$). The approximation model explained 90 ± 3 and 91 ± 4 % of the variance of the relCHO at 50 RPM and 100 RPM, respectively (both $p < 0.001$, Fig. 4). k_{CHO50} and k_{CHO100} were independent of P_{peak}, $\dot{V}O_{2peak}$, RER_{peak} or BLC_{peak} and also the difference between k_{CHO50} and k_{CHO100} was independent of P_{peak} and $\dot{V}O_{2peak}$.

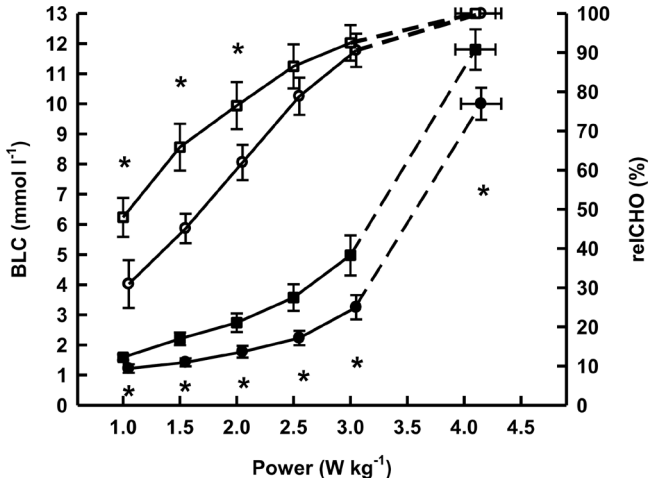

FIG. 3. Blood lactate concentration (BLC) and relative carbohydrate oxidation (relCHO) at 50 RPM (● and ○) and at 100 RPM (■ and □); mean ± SE; * = difference between 50 and 100 RPM (all $p < 0.05$).

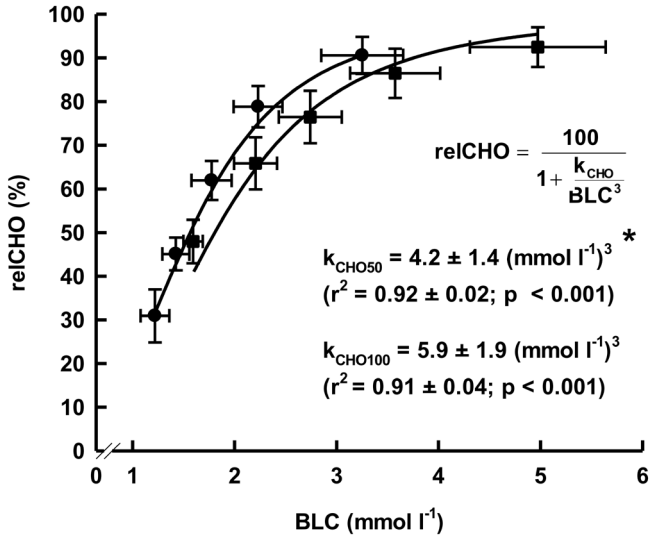

FIG. 4. Relative carbohydrate oxidation (relCHO) related to the blood lactate concentration (BLC) at 50 RPM (●) and 100 RPM (■); mean ± SE; * = difference between 50 und 100 RPM ($p < 0.05$).

DISCUSSION

The main new finding of the present study was that at a higher cycling cadence the relCHO was reduced at given BLC-levels as indicated by a higher k_{CHO} (Fig. 4). This cadence-related CHO-preserving effect was apparent below approximately 50 % Int_P only.

The present findings are consistent with previous observations of higher $\dot{V}O_2$, $\dot{V}CO_2$, RER and BLC values at higher cadences over a wide range of given sub-maximal exercise intensities [1-3]. They confirm that BLC-differences between different cadences increase with exercise intensity, whereas the differences in $\dot{V}O_2$ and $\dot{V}CO_2$ converge as exercise intensity increases with small or no differences at P_{peak} [3,4].

$\dot{V}O_2$, $\dot{V}CO_2$, RER and BLC values do not directly reflect metabolism at muscle fiber level. Respiratory measures represent metabolism of all aerobic tissues and the BLC indicates corresponding substrate availability in its dilution space. In the third decade of life, healthy males have an average relative skeletal muscle mass related to the total body mass of about 40 % [40]. In comparison to the skeletal muscle other tissues with lactate release and oxidation like heart muscle, liver, kidney, brain [41,42] are comparably small or less well perfused during exercise than skeletal muscle. The exercise-induced and cadence-related increase in the metabolic rate at an RER < 1 was approximately up to four to eight fold the resting metabolic rate at 50 RPM and five to nine fold at 100 RPM (Fig. 2). The corresponding BLC-levels were up to approximately three and four times the resting value, respectively. Consequently, skeletal muscle can be seen as the dominant factor of the cycling-induced metabolic response seen in the present study.

The observed within subject differences in the metabolic response at given workload between cycling at 50 vs. 100 RPM below approximately 50 % Int_P are indicative for increased muscle fibre activation at the higher cadence, e.g. a higher fast twitch muscle fibre (FTF) recruitment associated with higher cadences [22-26]. This effect should decrease, disappear or even reverse with increasing exercise intensity [26] as seen in subjects with varied athletic background [24,43] and professional road cyclists [8] cycling at and above 85 % $\dot{V}O_{2peak}$. Other factors which may also affect fibre recruitment during cycling at given workload including muscle strength [44], cycling skills [45], saddle position [46,47], pedal design [48] and test duration [23] have been minimized or excluded through the within subject design and standardization in the present experiment.

BLC and Int_{VO_2} are common measures of exercise intensity of recreational and high performance training [49]. The newly described difference in the BLC-relCHO-relationship indicates, that at a given $\dot{V}O_2$ and/or BLC higher cadences reduce the reliance on CHO as a metabolic substrate of aerobic energy (Fig. 4). Comparable effects on BLC and RER, and a lower use of CHO as metabolic substrate of aerobic energy at given workload have been described at incremental and constant workload exercise in glycogen depletion cycling experiments [50,51]. Such a carbohydrate conserving effect was linked with a decreased muscular PDH-activation [16-21,28]. The localization of glycogen, the pattern of depletion of the muscle cell and carbohydrate availability lead to a fibre type-specific compartmentalization of glycogen metabolism [52,53]. Also muscle fibre-specific PDH phosphatase profiles have been described [20]. The higher PDH-activity in the more aerobic muscle was related to the higher PDH-content [54]. Higher content of anaerobic glycolytic enzymes, lower aerobic mitochondrial protein content of FTF compared with STF [27,29,54], and higher muscle lactate concentration combined with lower PDH-activity in FTF suggest that the PDH of FTF is less sensitive to the availability of pyruvate than STF [54].

The higher BLC and k_{CHO} are therefore consistent with suggestions that cycling cadence-dependent differences in cardio-respiratory and metabolic acute responses reflect higher FTF-recruitment at higher cadences [22-26]. Consequently, at a given metabolic rate the higher BLC, as observed at 100 RPM compared with 50 RPM, does not necessarily reflect a higher glycolytic rate but a reduced sensitivity to the availability of substrate requiring a higher equilibrium concentration of pyruvate and lactate as a substrate of the aerobic rephosphorylation of the whole body aerobic tissue with a cadence-related higher fraction of activated FTF.

Racing road cyclists prefer cadences above 90 RPM during racing, testing in the laboratory and training [7-9]. Depending on seasonal variations, top athletes competing in long distance events train 70 to 90% of their endurance training at intensities corresponding to BLCs below 2 $mmol \cdot l^{-1}$ [49,55]. The present cadence-related CHO-preserving effect of a higher k_{CHO} at a higher cadence is most relevant at BLC-levels up to 2.0 $mmol \cdot l^{-1}$ (Fig. 3 and 4) comparable to approximately 50 % of $\dot{V}O_{2peak}$ (Fig. 2) where relCHO is approximately 65 % (equivalent to 25 $mg \cdot kg^{-1} \cdot min^{-1}$) at 50 RPM and 58 % (equivalent to 22 $mg \cdot kg^{-1} \cdot min^{-1}$) at 100 RPM. This CHO-saving effect of approximately 12 % disappears at an Int_{VO_2} of approximately 70 %, which is roughly comparable to an Int_P of 60 % with corresponding relCHO of 82 and 86 % (equivalent to 46 $mg \cdot kg^{-1} \cdot min^{-1}$ vs. 44 $mg \cdot kg^{-1} \cdot min^{-1}$ CHO oxidation) at 50 and 100 RPM, respectively, above which relCHO approaches saturation. Many endurance athletes monitor their exercise intensity via blood lactate measurements adjusted to exercise intensity domains expressed as a fraction of the peak oxygen uptake rather than mechanical power [49,55]. k_{CHO50} and k_{CHO100} and also the cadence-effect on k_{CHO} were independent of both P_{peak} and $\dot{V}O_{2peak}$ suggesting that in top cyclists athletes with an up to 40 % higher $\dot{V}O_{2peak}$ than the present subjects combined with up to 30 hours of training per week such a cadence related CHO preserving effect may become a protective factor against glycogen depletion.

CONCLUSIONS

Frequently observed higher $\dot{V}O_2$, $\dot{V}CO_2$ and BLC values at higher cycling cadences over a wide range of exercise intensities suitable for high training volumes are combined with a reduced relCHO at given BLC-values as indicated by a higher k_{CHO}. These findings are consistent with suggestions that at such exercise intensities cycling cadence-dependent differences in cardio-respiratory and metabolic acute responses reflect higher FTF recruitment at higher cadences [1-3], and that the PDH of FTF is less sensitive to the availability of pyruvate than that of STF [54]. The present results also suggest that in spite of a higher $\dot{V}O_2$, a higher BLC and a higher CHO oxidation at a given mechanical power compared to a low cycling cadence, a higher cadence can substantially reduce the reliance on CHO at a given low exercise intensity as indicated by a particular $\dot{V}O_2$ and/or BLC.

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