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Inverse Relationship between $\dot{V}O_{2\max}$ and Gross Efficiency

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Key words

- training
- aerobic capacity
- power output
- performance

Abstract

The aim of this study was to identify if an inverse relationship exists between Gross Efficiency (GE) and $\dot{V}O_{2\max}$ in trained cyclists. In Experiment 1, 14 trained cyclist's GE and $\dot{V}O_{2\max}$ were recorded at 5 different phases of a cycling 'self-coached' season using an incremental laboratory test. In Experiment 2, 29 trained cyclists undertook 12 weeks of training in one of 2 randomly allocated groups (A and B). Over the first 6 weeks Group A was prescribed specific high-intensity training sessions, whilst Group B were restricted in the amount of intensive work they could con-

duct. In the second 6-week period, both groups were allowed to conduct high intensity training. Results of both experiments in this study demonstrate training related increases in GE, but not $\dot{V}O_{2\max}$. A significant inverse within-subject correlation was evident in experiment 1 between GE and $\dot{V}O_{2\max}$ across the training season ($r = -0.32$; $P < 0.05$). In experiment 2, a significant inverse within-subject correlation was found between changes in GE and $\dot{V}O_{2\max}$ in Group A over the first 6 weeks of training ($r = -0.78$; $P < 0.01$). Resultantly, a training related inverse relationship between GE and $\dot{V}O_{2\max}$ is evident in these groups of trained cyclists.

Introduction

Gross Efficiency (GE) is defined as the ratio of work accomplished ($\text{kcal}\cdot\text{min}^{-1}$) to the energy expended in performing that work ($\text{kcal}\cdot\text{min}^{-1}$) [6], and has been suggested to be a key determinant of endurance cycling performance [6, 7, 18]. The combination of efficiency, performance $\dot{V}O_2$ and anaerobic capacity has been suggested to determine performance velocity or power output [22].

Historically, the effect of training on GE has been controversial [6, 15, 35, 36]. Whereas other performance determinants, such as lactate threshold and maximal power output, have been shown to change with training [5, 29], GE has been shown to be within a range of 18–23% regardless of individual training status [6, 15, 35–37]. These findings using a cross-sectional approach seemed puzzling given that the main determinants of efficiency have been linked to muscle fibre type [6, 18], which has been shown to be affected by training [40].

Recent longitudinal study designs have found a significant impact of training on efficiency in cycling [8, 16, 17, 39]. Santalla et al. [39] found

that over a 5-year period delta efficiency (DE) (the ratio of change in work accomplished to the change in energy expended) increased from 23.61 to 26.97% in a group of world class cyclists as a result of their normal training. One of the novel findings of their study was an inverse correlation between the training related change in DE and $\dot{V}O_{2\max}$ ($r = -0.63$) over the 5-year study period. They found that cyclists with the lowest $\dot{V}O_{2\max}$ had the largest increase in DE. An inverse relation between GE and $\dot{V}O_{2\max}$ was also reported by Lucia et al. [31] in a group of professional road race cyclists, and between economy and $\dot{V}O_{2\max}$ in highly trained runners [34]. Lucia's study [31] involved single lab tests for GE and $\dot{V}O_{2\max}$ and thus could not determine whether the finding might be due to training influences. Both Lucia et al. [31] and Santalla et al. [39] suggested that a high level of exercise efficiency (either GE or DE) could compensate for a comparatively low $\dot{V}O_{2\max}$. Consequently, this would afford the athlete further potential to achieve an optimal physiological performance velocity or power output. However, it is not clear from either of the studies of Lucia et al. [31] or Santalla et al. [39] if this inverse relationship is due to training

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undertaken by cyclists, i.e., is short-term in nature, or if it is due to an accumulation of a number of years of training. The aim of the present study was to identify if training affected the relationship between GE and $\dot{V}O_{2\max}$ found in the studies of Lucia et al. [31] and Santalla et al. [39]. We were interested to investigate the relationship between GE and $\dot{V}O_{2\max}$ 1) over the course of a competitive road race cycling season, and 2) as a result of a short-term endurance training intervention. Specifically, we aimed to test the hypotheses that there is an inverse relationship between GE and $\dot{V}O_{2\max}$.

Materials and Methods

Experiment 1: Observational data from a road racing season

Participants

14 endurance-trained competitive male cyclists (mean \pm SD: 34 ± 8 years, 74.3 ± 7.4 kg, W_{\max} 406 ± 43 W, $\dot{V}O_{2\max}$ 59.5 ± 3.8 mL \cdot kg $^{-1}$ \cdot min $^{-1}$) were selected from local clubs and professional teams to take part in this investigation. All cyclists had a minimum of 3 years competitive cycling experience (national to international level of competition), and familiarity of laboratory-based testing. Prior to recruitment the study was approved by the University of Kent, Centre for Sports Studies Research Ethics Committee, in accordance with the Declaration of Helsinki and the international standards outlined by Harriss and Atkinson [13]. All cyclists provided written informed consent before participating.

Procedures

All participants visited the laboratory 5 times during the study. The assessment consisted of a combined threshold test and maximal aerobic power test using a continuous incremental protocol. Starting at 150 W, work rate increased by 30 W every 8 min. Blood lactate samples were taken at the end of every 4th and 8th min of each work stage until the measured concentration of lactate in the blood reached 4 mmol \cdot L $^{-1}$. Power output and heart rate were recorded continuously on a Lode Excalibur Sport Ergometer (Lode B.V., Groningen, NL) and Polar heart rate monitor (Polar S725X, Polar, Kempele, Finland), respectively. Expired gases were collected over the final 3 min of each 8-min bout of exercise completed (Quark b2, Cosmed, Italy) for the measurement of $\dot{V}O_2$ and RER. These data were used to calculate GE. Prior to each test the gas analyzer was calibrated according to the manufacturer's recommendations.

Once 4 mmol \cdot L $^{-1}$ was obtained cyclists were immediately switched to a fast incremental protocol to elicit $\dot{V}O_{2\max}$. This protocol used a 30 W per minute increment rate continued until volitional exhaustion. Maximal power output (W_{\max}) was calculated using the W_{\max} formula [23]:

$$W_{\max} = WE + (30W/t \times tE)$$

where W_{\max} = maximal power output (Watts); WE = power output of last stage completed; 30 W = work rate increment; t = work rate duration (seconds); tE = duration of final stage (seconds).

The training period

Initial testing (Test 1) took place in January, Test 2 was conducted in May (pre-competition period), Test 3 (competition period) in

July, Test 4 in September (post-competitive period) and finally Test 5 in December (transition period). All participants were instructed to refrain from strenuous exercise for 24 h immediately prior to each test and were not informed of their performance in any of the assessments until all phases had been completed.

Throughout the study, participants served as their own control maintaining their normal diet and training patterns. Training was not manipulated or altered and the cyclists were asked to train and compete in their normal training macrocycles. Cyclist's training session/race data was monitored and quantified in terms of duration and intensity using heart rate (Polar S725X, Polar, Kempele, Finland) with data sampled every 5 s. From this data total training time and the percentage of time spent training below LT, between LT and OBLA and above OBLA was calculated.

Experiment 2: Training intervention

Participants

Following institutional ethical approval and in accordance with the guidelines of Harriss and Atkinson [13], a group of 29 endurance-trained competitive male cyclists from local clubs and professional teams provided written informed consent to take part in this investigation. The cyclists' physical characteristics at the start of the study were (mean \pm SD): 35 ± 8.5 years, 72.9 ± 4.6 kg, MMP 383 ± 36 W, $\dot{V}O_{2\max}$ 60.8 ± 8.3 mL \cdot kg $^{-1}$ \cdot min $^{-1}$. All cyclists had a minimum of 3 years competitive cycling experience and familiarity of laboratory-based testing.

Procedures

All cyclists visited the laboratory 3 times during the study. Laboratory testing was conducted as outlined in experiment 1 above except that the efficiency test was terminated once a measured blood lactate concentration of 4 mmol \cdot L $^{-1}$ was reached or exceeded. Cyclists then rested for 5 min prior to the commencement of a ramp protocol to elicit $\dot{V}O_{2\max}$. This protocol started at 150 W using a 20 W \cdot min $^{-1}$ ramp rate, which continued until volitional exhaustion.

Training period

Cyclists were then randomly divided into 2 groups. Group A were instructed to supplement their normal training with 2 high intensity training sessions per week (involving both 4-min 'on' and 4-min 'off' intervals at a power output equivalent to the 4 mmol \cdot L $^{-1}$ lactate point, and maximal sprint repetitions). These sessions were individually set for each cyclist based upon their laboratory test results. Group B cyclists were asked to conduct all of their training under the heart rate equating to their 4 mmol \cdot L $^{-1}$ lactate point minus 5 beats.

The study lasted for 12 training weeks, comprising two 6-week training phases over the pre-season period (approximately January to April). For the first 6 weeks of the study cyclists' training was controlled as outlined above. Cyclists were asked not to race and only to engage in training during this period of the study. During the second 6-week phase cyclists' training and racing were not manipulated for either group. The second phase of training was used to see if the introduction of higher intensity training following restricted base training would increase efficiency in Group B. Heart rate was measured at 5 s intervals (Polar S725X, Polar, Kempele, Finland) throughout every training session and race over the 12-week period.



Determination of gross efficiency and $\dot{V}O_{2max}$

Energy expenditure was estimated from the expired gases (steady state $\dot{V}O_2$, and RER) collected during the final 3 min of each submaximal bout of exercise using the formulae:

$$\text{Work rate energy expenditure (kcal}\cdot\text{min}^{-1}) = \text{Power output} \times 0.01433$$

$$\text{Total Energy expenditure (kcal}\cdot\text{min}^{-1}) = \text{kcal per litre } O_2 \text{ consumed} \times \dot{V}O_2$$

Kcal per litre of O_2 consumed was calculated from the thermal equivalents of oxygen for non-protein RQ (as represented by RER). Subsequently, gross efficiency was calculated as the ratio of power output to power input, multiplied by 100%:

$$GE = \frac{\text{Work rate energy expenditure}}{\text{Total energy expenditure}} \times 100\% \quad \text{or}$$

$$\frac{\text{Power output}}{\text{Power input}} \times 100\%$$

Breath-by-breath $\dot{V}O_2$ data was averaged over 1 min periods prior to analysis. Averaged data was then checked for consistency with the criteria for achieving $\dot{V}O_{2max}$ (a last minute rise of ≤ 150 ml/min), as suggested by Midgeley et al. [33]. To ensure a valid data set prior to the above analyses, 3 cyclists were excluded from study 2, as their data were not entirely consistent with the criteria for achieving $\dot{V}O_{2max}$.

Statistical analysis

Prior to all statistical analyses data were checked for violation of the parametric assumptions using a Shapiro-Wilk test. For Experiment 1, changes in GE and $\dot{V}O_{2max}$ were assessed using repeated measures ANOVA, for experiment 2, ANCOVA was used to assess changes in GE and $\dot{V}O_{2max}$ with initial gross efficiency

used as the covariate. For all analysis unadjusted Post Hoc analysis was performed [38]. Within-subject correlation coefficients were calculated [4] to examine the relationship between GE and $\dot{V}O_{2max}$ values. Statistical significance was set at $P \leq 0.05$. All values are expressed as mean and standard deviation (mean \pm SD) unless otherwise stated. For each individual cyclist, submaximal efficiency data were averaged to the highest common power output achieved across the tests.

Results

Experiment 1: Observational data from a season of road race training

Gross efficiency significantly increased across the road-racing season ($P < 0.01$). Specifically, GE was significantly higher at Tests 2–4, compared to 1 and 5 (Table 1).

A significant inverse within-subject correlation was identified between GE and $\dot{V}O_{2max}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) across the training season ($r = -0.32, P < 0.05$; Fig. 1a–d).

Table 1 Mean $\dot{V}O_{2max}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) and gross efficiency (averaged across all power outputs to the highest common power output achieved during all tests). Mean \pm SD.

	Wmax (W)	$\dot{V}O_{2max}$ ($\text{L}\cdot\text{min}^{-1}$)	$\dot{V}O_{2max}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	Gross Efficiency (%)
test 1	406 \pm 43	4.4 \pm 0.4	59.5 \pm 3.8	19.6 \pm 0.7
test 2	421 \pm 46	4.5 \pm 0.4	60.0 \pm 5.6	20.6 \pm 1.0*
test 3	410 \pm 31	4.2 \pm 0.3	59.2 \pm 4.9	20.6 \pm 1.3*
test 4	390 \pm 27 [§]	4.2 \pm 0.3	57.5 \pm 3.4	20.3 \pm 0.8*
test 5	399 \pm 37 [§]	4.4 \pm 0.5	59.8 \pm 4.4	19.4 \pm 0.8

*Significantly higher than Tests 1 and 5 ($P < 0.01$); [§] Significantly lower than Tests 1–3, $P < 0.05$

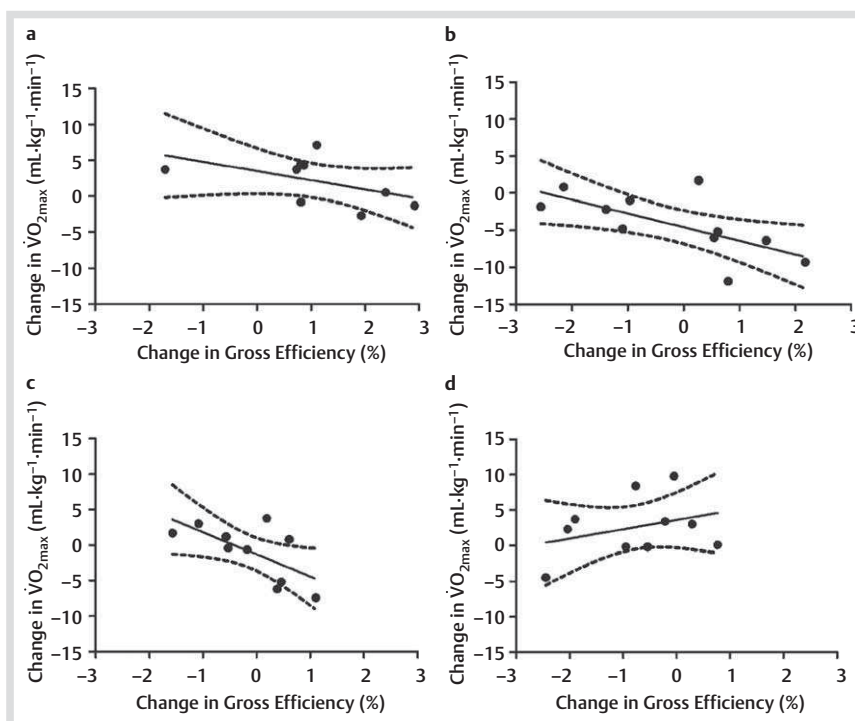


Fig. 1 Inverse relationship between changes in GE and changes in $\dot{V}O_{2max}$ over a training season. **a** Inverse relationship over the pre-season phase. **b** Inverse relationship over the competition phase. **c** Inverse relationship over the post-competition phase. **d** Positive relationship over the transition phase.



Table 2 MMP, $\dot{V}O_{2\max}$ and GE values across the 3 tests in Groups A and B.

	MMP (W)		$\dot{V}O_{2\max}$ (L·min ⁻¹)		$\dot{V}O_{2\max}$ (ml·kg ⁻¹ ·min ⁻¹)		Gross Efficiency (%)	
	Gp. A	Gp. B	Gp. A	Gp. B	Gp. A	Gp. B	Gp. A	Gp. B
test 1	389±34	375±39	4.5±0.4	4.3±0.6	60.0±6.9	58.6±8.8	20.6±1.4	21.2±2.1
test 2	402±37*	383±42	4.3±0.5	4.3±0.5	58.2±7.2	59.0±8.5	21.4±1.6*	21.2±2.5
test 3	400±44	396±31	4.2±0.5	4.2±0.4	58.2±7.2	57.4±5.5	21.8±1.8*	22.6±0.8*

*Significantly higher than baseline ($P < 0.05$)

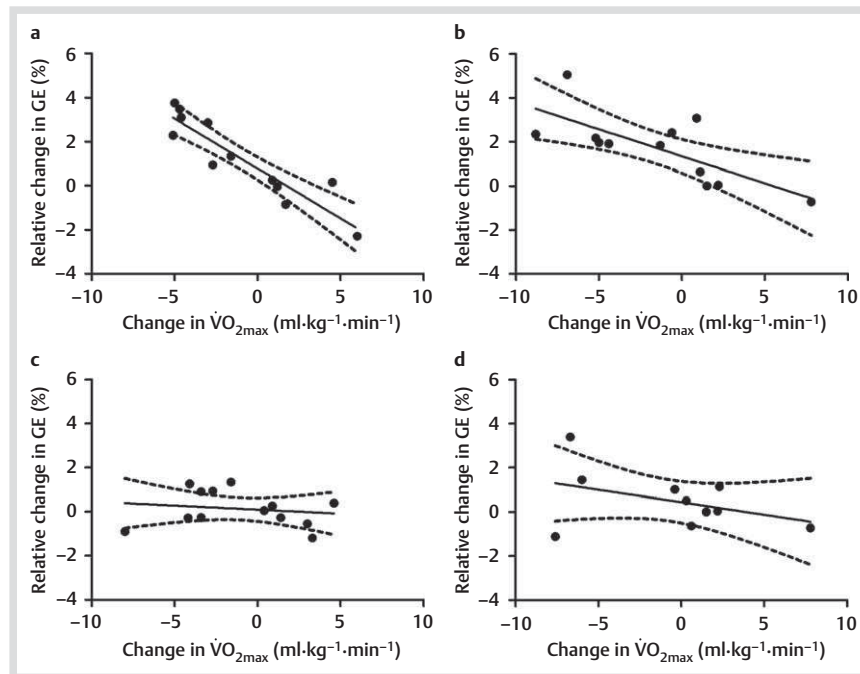


Fig. 2 Inverse correlations between changes in $\dot{V}O_{2\max}$ and changes in GE over 12 weeks of training in Groups A and B. **a** Change in $\dot{V}O_{2\max}$ vs. change in GE in Group A Test 1–2. **b** Change in $\dot{V}O_{2\max}$ vs. change in GE in Group A Test 1–3. **c** Change in $\dot{V}O_{2\max}$ vs. change in GE in Group B Test 1–2. **d** Change in $\dot{V}O_{2\max}$ vs. change in GE in Group B Test 1–3. 3 cyclists were excluded for not achieving a plateau in the $\dot{V}O_2$ response during the last minute of the test.

Experiment 2: Training intervention

GE significantly changed in both groups over the study period (Table 2). The Group A cyclists significantly improved their efficiency over the intervention period between Tests 1 and 2, both in terms of absolute and relative GE ($P < 0.05$). However, there were no significant changes in the GE of Group B over this training period when high intensity training was restricted ($P = 0.81$). Consequently, the change in GE for Group A between Test 1 and 2 was significantly greater than for Group B ($P = 0.027$). Between Tests 2 and 3 Group A demonstrated no significant change in GE, (21.4 vs. 22.0%, $P = 0.42$). Group B significantly improved their absolute GE above baseline (21.2 vs. 22.6%, $P = 0.01$). MMP significantly increased in Group A at Test 2 compared to baseline ($P = 0.01$) and was greater than the change in Group B ($P = 0.05$).

Using data from both Group A and B across the time course of the study, significant within-subject correlations were found between GE and $\dot{V}O_{2\max}$ ($r = -0.59$; $P < 0.01$). Those cyclists with the largest improvement in GE experienced the largest decrease in $\dot{V}O_{2\max}$ (Fig. 2a–d). Data from Group A demonstrated a significant inverse correlation over the first 6-week training period ($r = -0.78$; $P < 0.01$; Fig. 2a). Data are presented over the specific intervention period (2a and c) and then over the whole 12 weeks of the study for Groups A and B, respectively, (2b and d). To further examine the factors that may result in the changes seen in GE, cyclists were ranked based on their observed change in efficiency across Tests 1–3 and divided into equal high- and low-change groups. Using an independent t -test the high-change group was found to have a significantly lower initial GE

at Test 1 compared with the low-change group (GE 19.9 vs. 20.9%, $P < 0.01$). The high-change group also had a significantly higher $\dot{V}O_{2\max}$ at Test 1 ($\dot{V}O_{2\max}$ 4.6 vs. 4.0 L·min⁻¹, $P < 0.01$).

Discussion

The results of the present study suggest that training related changes in GE are inversely related to $\dot{V}O_{2\max}$. These findings are consistent to both longitudinal observational and training intervention study designs. These results are supportive of the findings of Lucia et al. [31] and Santalla et al. [39] and suggest that GE may compensate for a low $\dot{V}O_{2\max}$. Additionally, the current study demonstrates that the inverse within-subject correlation may result from relatively short-term training (~6 weeks), albeit in a group of cyclists with lower training status.

Consistent with the findings of both studies in the present work, Chicharro et al. [5], Lucia et al. [29,30] and Santalla et al. [39] found no significant change in cyclist's $\dot{V}O_{2\max}$ (L·min⁻¹) during a period of training. These authors attribute this finding to the extensive training history of their participants and their already high values. Therefore, it is possible that the inverse relationship may only become evident in cyclists who are highly trained and reach their ceiling for $\dot{V}O_{2\max}$. However, due to the nature of correlational analysis it is difficult to distinguish cause and effect. This speculation is supported by evidence from experiment 1 of the current study where the experienced cyclists' $\dot{V}O_{2\max}$ did not change significantly across a year's training. However, the submaximal parameter of GE did significantly increase with train-

ing, and an inverse relationship between $\dot{V}O_{2\max}$ and GE was evident. This suggests that the training induced increases in GE compensated for static or declining maximal aerobic capacity to facilitate improvements in maximal aerobic power. Consequently, it could be suggested that GE responds more markedly to training than $\dot{V}O_{2\max}$. In agreement with the findings of Hoogeven [14] and Jeukendrup et al. [21] the W_{\max} of the cyclists changed significantly over the training year. Most notably, W_{\max} was highest in the build up to, and during the competitive phases of the season. Thus, it could be suggested that the increase in W_{\max} is to some extent mediated by the improvements in efficiency (due to the inverse correlation between $\dot{V}O_{2\max}$ and GE). Additionally, in experiment 2, when cyclists were regrouped according to their initial Test 1 $\dot{V}O_{2\max}$ cyclists who possessed the highest initial $\dot{V}O_{2\max}$ experienced the smallest change in terms of GE as a result of the training they undertook.

In experiment 2 there was a trend for $\dot{V}O_{2\max}$ to decrease (Group A: 60.0–58.2 ml·kg⁻¹·min⁻¹; Group B: 58.6–57.4 ml·kg⁻¹·min⁻¹) during the 12 weeks of the intervention study, although this was not statistically significant. However, the individual variation in GE and $\dot{V}O_{2\max}$ responses have yielded the significant linear relationship between these variables in this study. The relationship is not evident due to gas measurement error as analyser was carefully calibrated prior to each test according to the manufacturer's recommendations. Similarly there is evidence that equipment drift (causing artificially low O₂ values) has not occurred. If this were the case then cyclists in both groups would have increased GE at all tests. Iaia et al. [19] found decreases in $\dot{V}O_{2\max}$ following a period of intensive endurance training (mean $\dot{V}O_{2\max}$ 54.8 vs. 53.5 ml·kg⁻¹·min⁻¹ before and after 4 weeks of intensive interval training, respectively). These authors used similar protocols with endurance-trained runners. Interestingly, their study also reported significant increases in economy and decreases in the submaximal energy cost of running at a range of running velocities. Unfortunately no relationships between $\dot{V}O_{2\max}$ and running economy were presented.

It could be argued that the results of this study are the artifact of a spurious correlation between $\dot{V}O_{2\max}$ and GE. Atkinson et al. [1] suggested that a spurious correlation could account for the correlation of Lucia et al. [31] as a correlation was performed between cycling economy at 80% $\dot{V}O_{2\max}$ and $\dot{V}O_{2\max}$ itself. However, this is not the case in the current study as a common divisor (i.e., $\dot{V}O_{2\max}$ or body mass) is not used in the calculation of both correlated variables. Additionally, significant inverse correlations were found whether absolute or relative $\dot{V}O_{2\max}$ values were used.

The training prescription used in this study was based upon heart rate equivalents to a measured concentration of 4mmol·L⁻¹ blood lactate. This method of prescribing training intensity is commonplace [12,20,24,28,41], but has distinct limitations. Carbohydrate status, hydration status, fatigue, cardiovascular drift and thermoregulation can all affect the prescribed heart rate (cf. [20]), and so actual training intensities may have varied from what was intended. The use of either laboratory based training or power meters might have enabled a closer scrutiny of the relationship between training intensity and resultant responses, although these methods require significant resources and/or time allocation.

A possible mechanism for the observed inverse within-subject correlation between efficiency and $\dot{V}O_{2\max}$ is suggested by studies that have investigated the effects of nitrate supplementation. Such supplementation has been shown to reduce exercise oxy-

gen cost (i.e., to increase efficiency) [2,3,25–27,32] and to decrease $\dot{V}O_{2\max}$ [25] (cf. [9–11]). Given the similarity of the efficiency/ $\dot{V}O_{2\max}$ response in these studies to those of the current investigation, we might hypothesise that training leads to a natural increase in the body's nitrate levels. Clearly, further research is required to confirm this hypothesis.

Further support for this 'nitrate hypothesis' is provided by studies of high-altitude-living Tibetans. Specifically, these high altitude natives have been shown to have significantly lower $\dot{V}O_2$ at submaximal work rates (i.e., higher efficiency), lower $\dot{V}O_{2\max}$ values and >10-fold higher circulating nitrate levels than inhabitants of lower altitudes [9–11].

Summary

In moderate-highly trained cyclists, training increases GE, but not $\dot{V}O_{2\max}$. Resultantly, an inverse relationship is created between GE and $\dot{V}O_{2\max}$. Those with a high $\dot{V}O_{2\max}$ seem to be more responsive to training related changes in GE compared to those with a lower $\dot{V}O_{2\max}$. This inverse relationship is evident in the results of an observational study where cyclists were free to conduct their own training and in a tightly controlled training intervention study. The impact on performance and the mechanisms responsible for the inverse relationship between GE and $\dot{V}O_{2\max}$ are unclear and require further investigation.

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