



Gross Efficiency and the Relationship with Maximum Oxygen Uptake in Young Elite Cyclists During the Competitive Season

by

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This study assessed gross efficiency (GE) during a single competitive season and determined the relationship between GE and maximum oxygen uptake (VO_{2max}) in young elite cyclists ($n = 15$, 20.1 ± 1.4 yrs, 177.5 ± 5.7 cm, 68.3 ± 6.2 kg, 45.2 ± 7.5 mm of six skinfolds) during a competitive season. Participants completed at two occasions (T1 = April; T2 = July), a progressive bike protocol (initial intensity = 100 W, 35 W increments every 3 min) until volitional exhaustion to assess VO_{2max} and submaximal variables. A single capillary blood sample was drawn from the left earlobe immediately after completion of each exercise load to determine lactate thresholds. Cyclists' GE was calculated as ([work accomplished/energy expended] x 100). No significant differences were obtained in GE at any workload between T1 and T2 or in the mean GE between T1 (19.3%) and T2 (19.4%) testing ($p = 0.93$). No significant association was found between mean GE and VO_{2max} at either T1 ($r = -0.28$, $p = 0.30$), or T2 ($r = -0.27$, $p = 0.32$). GE of young elite cyclists might not vary during the most important phase of the training season and GE was not related to VO_{2max} . A lower accumulated volume and intensity of training of these cyclists may account for their lower GE in comparison to older professional cyclists and might not have been enough to foster higher increases of GE in cyclists with lower VO_{2max} .

Key words: cycling, training, economy, performance, endurance.

Introduction

Successful cycling performance relies on a variety of biomechanical and physiological factors (Castronovo et al., 2013; Michalczyk et al., 2015). Maximum aerobic capacity (measured by maximum oxygen uptake, VO_{2max}) is a physiologic variable strongly related to performance and may be a determinant of potential success (Bell et al., 2017; Lucia et al., 2003; Santalla et al., 2012; Michalczyk et al., 2008; Czuba et al., 2014; Holdys et al., 2013; Gronek et al., 2013). However, training has little to no effect on VO_{2max} in high-level athletes (Hopker et al., 2009; Lucia et al., 2000), possibly due to its high heritability (Bouchard et al., 1999) and association with genetic polymorphisms (Ahmetov and Fedotovskaya, 2015).

Gross efficiency (GE) differs from VO_{2max} in that it is adaptable (Hopker et al., 2009) once a certain level of fitness is reached and it may be a more important determinant of cycling performance (Joyner and Coyle, 2008; Lucia et al., 2003; Santalla et al., 2012). Gross efficiency has good absolute reliability (Moseley and Jeukendrup, 2001) and is defined as the ratio of power output to power input from oxygen uptake (VO_2) and carbon dioxide production (VCO_2) measurements during steady state cycling. Higher GE may account for performance differences in cyclists with matched VO_{2max} (Lucia et al., 2002), as a GE improvement of 0.9% could result in a 25.6 s improvement over a 20 km trial (Hettinga et al., 2007).

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A cyclist's GE is modulated by several external and internal factors including weather conditions, cycling technique, training regimens, and the physiological structure of the cyclist (Hopker et al., 2010). High ambient temperatures and insufficient high-altitude acclimatization negatively affect GE. High ambient temperatures increase the energy cost of the exercise due to greater circulation, sweating, and ventilation (Hettinga et al., 2007), whereas suboptimal-altitude acclimatization increases energy demands during muscle excitation and contraction (Green et al., 2000). These muscle contractions are thought to be aided in those with a higher percentage of type I muscle fiber (Coyle et al., 1992; Horowitz et al., 1994). Regarding technique, an increasing cadence leads to decreased GE (Chavarren and Calbet, 1999; Lucia et al., 2004; Sidossis et al., 1992), but in contrast, lower cadence training is not related to increased GE (Kristoffersen et al., 2014).

Six-weeks of high-intensity training sessions increased GE in endurance-trained competitive cyclists (Hopker et al., 2010) and an accumulated training effect is related to increased GE (Hopker et al., 2010). Therefore, one could expect higher GE in older elite cyclists compared to younger cyclists. Despite GE assessment in older cyclists and the importance of GE as a performance-enhancing variable (Joyner and Coyle, 2008), we are unaware of any study that has assessed the change of GE during a competitive season in youth elite cyclists. Furthermore, a previous study in professional male road cyclists (26 ± 1 yr) showed that GE was inversely correlated to VO_{2max} , arguing that GE could compensate for relatively low VO_{2max} (Lucia et al., 2002). However, there is a lack of data regarding this relationship in young cyclists who aspire to become professionals. Since performance in the "under 23" (U23) category of the Union Cycliste Internationale (UCI) is critical for the transition to the professional level, determining GE and its relationship with VO_{2max} in these cyclists would be useful to further understand and possibly improve cycling performance. It was hypothesized that: 1) GE would not show significant differences in young elite cyclists during a competitive season and, 2) a negative correlation would be found between GE and VO_{2max} in these cyclists.

Therefore, the aims of the present study were: 1) to assess GE of young cyclists during a

competitive season, and 2) to determine the relationship between GE and VO_{2max} in young elite cyclists.

Methods

The present investigation is an observational study in which cyclists ($n = 15$) completed two laboratory-based progressive exercise tests during the competition phase (T1 = April; T2 = July) to assess selected physiological variables.

Participants

Fifteen male road U23 cyclists (20.1 ± 1.4 yrs, 177.5 ± 5.7 cm, 68.3 ± 6.2 kg, 45.2 ± 7.5 mm of six skinfolds), with a mean of two years of competitive experience at the national elite level (1-5 years), were recruited from the same cycling team. All participants competed at national standards or above, covered a total of 20,000 to 25,000 km per year, and had weekly training duration of 18 to 22 hours. The study was approved by the Bioethics Commission of the University of the Basque Country (UPV/EHU), and all participants provided written informed consent prior to any data collection.

Procedures

Participants had previous experience with the experimental protocol, and the laboratory conditions were controlled (*i.e.*, $19-23^{\circ}\text{C}$ and 40-50% humidity). The cyclists refrained from exhaustive exercise within the 48 h of testing, ate standardized diets (no food intake three hours before the test), and were allowed water "ad libitum" before testing. Electric fans were used for cooling during exercise testing.

Anthropometry included stature, body mass, and six skinfold thicknesses (Harpending, Germany) (subscapular, triceps brachii, supraspinale, abdominal, anterior thigh, medial calf). Skinfolds were assessed in accordance with guidelines from the International Society for the Advancement of Kinanthropometry.

The bike ergometer (Lode Excalibur Sport, Lode, Groningen, NL, software LODE v. 5.1.5) was calibrated to daily intensities of 100-1000 W, and again prior to individual tests. Each cyclist's setup (saddle height, reach, handle bar height, clip pedals and set crank lengths) was registered to ensure similar settings in the second trial.

A progressive protocol was performed to assess maximum aerobic capacity and submaximal

variables. The intensity was incrementally increased 35 W every 3 min until volitional exhaustion. The test was not preceded by any warm-up, and participants cycled at their freely chosen cadence at each intensity (initial intensity was 100 W). Participants were asked to keep their cadence constant at their preferred rate based on visual feedback from a display unit. Cyclists received verbal encouragement from the laboratory physiologists and team coach throughout testing.

Maximum intensity (W_{\max}) had to be maintained for at least a 3-min period. When the last intensity could not be completed for 3 min, W_{\max} was computed as $W_{\max} = W_f + [(t/180) \times 35]$ (Kuipers et al., 1985), where W_f was the last completed intensity (in W), t was time in seconds for which the last uncompleted intensity was maintained, and 35 was the power output difference between the last two intensities.

A single capillary blood sample was drawn from the left earlobe immediately after completion of each workload, avoiding any contact with the electrode. Blood lactate concentration [La] was determined with an automatic analyzer (Lactate ProTM; Arkray Factory Inc., Shiga, Japan) that was calibrated per manufacturer's recommendations prior to testing. The first lactate threshold (LT1) was identified on individual [La]-power output curves as the intensity which elicited a 1 mmol·L⁻¹ increase in [La] above mean baseline lactate values when measured at 40–60% of W_{\max} (Hagberg and Coyle, 1983). The exercise intensity corresponding to the onset of blood lactate accumulation or the second lactate threshold (LT2) was identified on the [La]-power output curve by straight-line interpolation between the two closest points as the power output eliciting a [La] of 4 mmol·L⁻¹ (Sjodin and Jacobs, 1981). Intensities at LT1 (WLT1) and LT2 (WLT2) were also determined by straight-line interpolation (Padilla et al., 2008).

Maximum oxygen uptake was determined via a breath-by-breath automated gas analysis system (Jaeger Oxycon Delta System, Hoechberg, Germany) that was calibrated before each testing session in line with the manufacturer's guidelines. Achievement of $VO_{2\max}$, the highest VO_2 value attained toward the end of the test, was assumed on attainment of at least two of the following: a plateau in VO_2 with increasing speeds (<2.0 mL·kg⁻¹·min⁻¹), a respiratory exchange ratio (RER) above

1.10, or a heart rate within ± 10 beats·min⁻¹ of age predicted maximum heart rate (220-age) (Duncan et al., 1997).

Each cyclist's GE was calculated as the ratio of work accomplished·min⁻¹ (*i.e.*, watts converted to kcal·min⁻¹) to energy expended·min⁻¹ (kcal·min⁻¹) *100 (Coyle et al., 1992; Hopker et al., 2009), whereas energy expenditure min⁻¹ (*i.e.*, kcal·min⁻¹) was calculated as the caloric equivalent of steady-state VO_2 and RER using the tables of Lusk (Lusk, 1928). To measure efficiency accurately, only gas collection data under steady-state exercise conditions were considered for GE determination (*i.e.*, below the LT1, intensities between 135 and 275 W in the present study).

Statistical analysis

For the statistical analyses data were assessed for normality using the Shapiro–Wilk test. Group means between the two tests were compared using Paired Samples t-tests. Mean GE across the intensities was assessed using a two-factor repeated-measures ANOVA (*i.e.*, intensity and test session). Bonferroni post hoc analyses were then conducted to identify any significant differences between intensities. Correlations between mean GE and $VO_{2\max}$ at each time were calculated using the Pearson's correlation coefficient. Analyses were conducted with SPSS 15.0 (SPSS Inc., Chicago, USA) software with alpha set at $p < 0.05$. Values are reported as means \pm standard deviations, unless otherwise stated. Practical significance was assessed by calculating Cohen's *d* effect size. Effect sizes (*d*) of above 0.8, between 0.79 and 0.5, between 0.49 and 0.2 and lower than 0.2 were considered as large, moderate, small and trivial, respectively.

Results

No significant differences were found in GE at any workload at either testing session ($p > 0.05$). The Cohen's *d* effect sizes of the difference in the mean values between T1 and T2 ranged from small to trivial across intensities of 135 W ($d = 0.21$), 170 W ($d = 0.01$), 205 W ($d = 0.32$), 240 W ($d = 0.07$) and at 275 W ($d = 0.24$). At T1, GE increased from $18.0 \pm 1.1\%$ at the first workload (135 W) to $21.1 \pm 1.3\%$ at the peak workload (275 W) (17.7% higher). At T2, there was a similar increase from $18.3 \pm 1.4\%$ to $21.5 \pm 1.2\%$ (17.5% higher). The increase in GE from the first workload (135 W) until the peak workload (275 W) was not statistically significant

($p > 0.05$) (Figure 1).

The mean GE value, an average of the GE calculated at 135, 170, 205, 240 and 275 W, was 19.3% during T1 and 19.4% during T2. Differences between mean GE between both tests were not significant ($p = 0.93$, d trivial).

The results of the physiological variables determined at T1 and T2 are shown in Table 1.

Significant differences were observed in maximum lactate ($L_{a_{max}}$, $\text{mmol}\cdot\text{L}^{-1}$) (d large), percentage where W_{LT2} occurred (PW_{LT2} , %) (d large) and heart rate at LT2 (HR_{LT2} , $\text{beats}\cdot\text{min}^{-1}$) (d small).

No significant, small and negative correlations were found between mean GE and VO_{2max} ($\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) at T1 ($r = -0.28$, $p = 0.30$) and at T2 ($r = -0.27$, $p = 0.32$) (Figure 2).

Table 1

Physiological variables obtained during the two measurements (T1 and T2).

Data are presented as mean \pm SD

Variables	T1	T2	p	ES
W_{max} (W)	385.4 \pm 27.2	393.3 \pm 8.7	0.08	0.3
W_{max} (W/kg)	5.7 \pm 0.4	5.8 \pm 0.5	0.13	0.3
VO_{2max} ($\text{L}\cdot\text{min}^{-1}$)	5.1 \pm 0.4	5.2 \pm 0.4	0.6	0.1
VO_{2max} ($\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	75.7 \pm 5.9	76.8 \pm 7.1	0.45	0.2
HR_{max} ($\text{beats}\cdot\text{min}^{-1}$)	179.6 \pm 27.8	186.7 \pm 6.2	0.32	0.3
$L_{a_{max}}$ ($\text{mmol}\cdot\text{L}^{-1}$)	8.4 \pm 1.8	10.4 \pm 1.5*	0.002	1.1
$L_{a_{LT1}}$ ($\text{mmol}\cdot\text{L}^{-1}$)	2.3 \pm 0.2	2.4 \pm 0.2	0.13	0.7
W_{LT1} (W)	290.5 \pm 34.5	295.9 \pm 32.9	0.53	0.2
PW_{LT1} (%)	75.4 \pm 7.1	75.1 \pm 3.1	0.88	0.0
$VO_{2_{LT1}}$ ($\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	57.1 \pm 6.7	58.1 \pm 5.8	0.55	0.1
HR_{LT1} ($\text{beats}\cdot\text{min}^{-1}$)	161.4 \pm 10.0	162.3 \pm 7.8	0.89	0.0
W_{LT2} (W)	334.0 \pm 30.9	329.7 \pm 35.8	0.35	0.1
PW_{LT2} (%)	86.7 \pm 3.4	83.7 \pm 3.0*	0.001	0.8
$VO_{2_{LT2}}$ ($\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	65.5 \pm 5.3	64.3 \pm 6.1	0.31	0.2
HR_{LT2} ($\text{beats}\cdot\text{min}^{-1}$)	174.0 \pm 6.6	171.1 \pm 7.0*	0.04	0.4

T1, first test. T2, second test. p value, difference between T1 and T2. ES, effect size between T1 and T2. W_{max} , maximum intensity. VO_{2max} , maximum oxygen uptake. HR_{max} , maximum heart rate. $L_{a_{max}}$, maximum lactate. LT1, first lactate threshold. LT2, second lactate threshold. $L_{a_{LT1}}$, lactate at LT1. W_{LT1} , intensity at LT1. PW_{LT1} , percentage of intensity at LT1. $VO_{2_{LT1}}$, oxygen uptake at LT1. HR_{LT1} , heart rate at LT1. W_{LT2} , intensity at LT2. PW_{LT2} , percentage of intensity at LT2. $VO_{2_{LT2}}$, oxygen uptake at LT2. HR_{LT2} , heart rate at LT2.

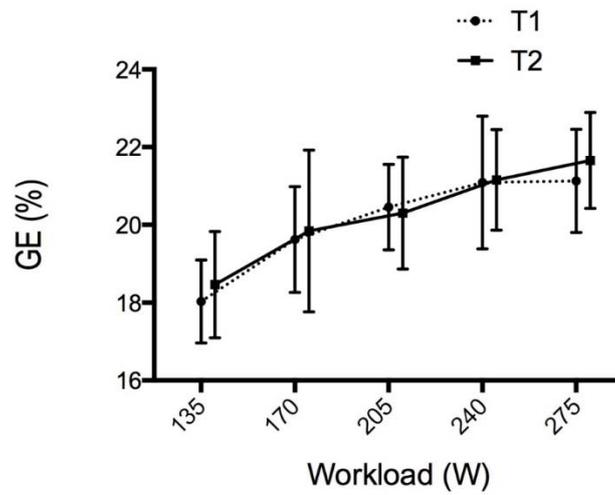


Figure 1
 Mean GE measured across each power output during two phases of the competitive season (April, T1 and July, T2).

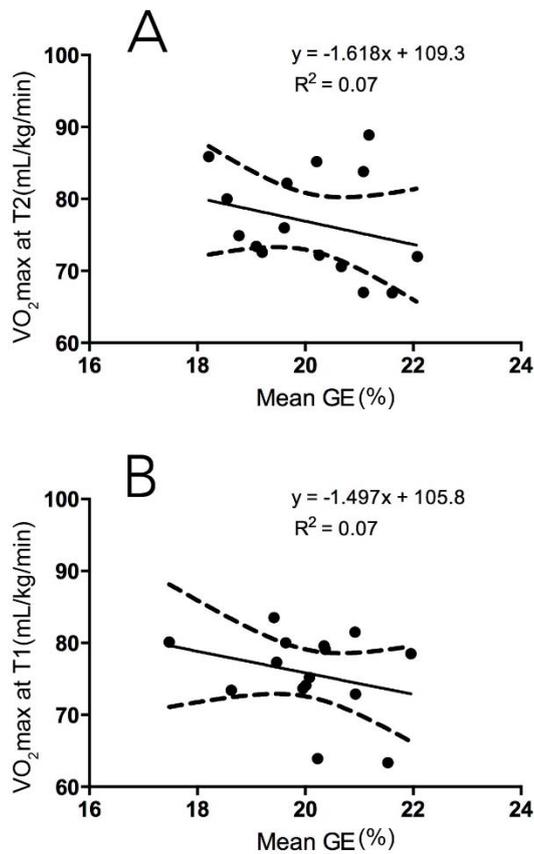


Figure 2
 Relationship between maximum oxygen uptake (VO_{2max}) and cycling gross efficiency (GE) at T1 (B) and T2 (A).

Discussion

Our study aimed to determine GE at two points during a competitive season, and to assess the relationship between GE and $\text{VO}_{2\text{max}}$ in U23 elite cyclists. Our main findings were that GE was lower in U23 cyclists than previously observed in older cyclists and that GE did not vary at the two measurement points within the competitive season. Furthermore, unlike professional riders, GE was not inversely related to $\text{VO}_{2\text{max}}$ in youth elite cyclists.

This is the first study that has determined the evolution of GE during the competitive season in the U23 category of the UCI. GE of trained cyclists was previously reported to be 21.1 to 22.4% (Hopker et al., 2009; Sassi et al., 2008). However, the Hopker et al.'s (2009) sample did not include elite cyclists, and the age ranged considerably (18 to 30 years), whereas the Sassi et al.'s (2008) paper did not isolate younger cyclists either (26 ± 4 yr). Therefore, GE observed in older cyclists was 1.7% and 3% higher than in the U23 cyclists in our study (*i.e.*, 19.3%), respectively.

Considering that appropriate volume and intensity of training are related to increases in GE (Hopker et al., 2010; Jobson et al., 2012) and because the cyclists of the present study were younger than in previous studies (Hopker et al., 2009; Sassi et al., 2008), GE observed in U23 cyclists may be partially due to lower accumulated training volume and intensity. Since lower $\text{VO}_{2\text{max}}$ is related to higher GE as a mechanism for performance compensation (Hopker et al., 2012), lower GE of the cyclists of the present study could be partially due to their higher $\text{VO}_{2\text{max}}$. Hence, it seems that high-intensity training provides the most potent stimulus for changes in GE (Jobson et al., 2012). This could emphasize the necessity for precise quantification of the training load (*i.e.*, external and internal load) undertaken by cyclists to identify the effects of training (Mujika, 2016), including these on GE.

The differences of GE among studies may have been affected by the different work rates for the GE calculation, which may limit comparisons across different studies. GE observed in the present study (Figure 1) was similar to that found in previous works in that it increased with absolute power output (Chavarren and Calbet, 1999), and in a curvilinear fashion (Ettema and Loras, 2009). Our

GE was determined at different intensities (from 135 to 275 W), which may also limit comparisons to other studies that measured GE at different workloads (Sassi et al., 2008).

The evolution of GE remained constant during the competitive phase of the season (*i.e.*, from April to July), which differed from a previous study that observed a 5.6% increase in GE over the course of a competitive season (Hopker et al., 2009). The lack of GE change over a season adds to the literature by highlighting that both endurance-trained cyclists above or within the U23 category might not modify their GE during the competitive period of the season. This lack of change in GE could have been affected by physiological changes through the competitive season (Jobson et al., 2012), as physiological variables improved at each measurement. These improvements found at the end of the season included L_{max} ($\text{mmol}\cdot\text{L}^{-1}$, 18.7%), PW_{LT2} (%), 3.3%), and HR_{LT2} ($\text{beats}\cdot\text{min}^{-1}$, 1.6%), with the rest of the measured variables remaining constant (Table 1).

Previous research findings suggest that short-term strength training can enhance GE (Paton and Hopkins, 2005; Ronnestad et al., 2011; Sunde et al., 2010), even though the mechanisms linking strength training and improvements in GE are unknown (Jobson et al., 2012). Therefore, to eliminate its influence on the GE, no strength training was performed by the cyclists between both tests.

Even though the mechanisms underlying the relationship between $\text{VO}_{2\text{max}}$ ($\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) and GE (%) are unclear (Jobson et al., 2012), a previous study reported a larger negative correlation in elite cyclists ($r = -0.63$) (Lucia et al., 2004) compared to our findings of $r = -0.28$ and -0.27 , at T1 and T2, respectively. Others observed that cyclists with high $\text{VO}_{2\text{max}}$ values (*i.e.*, over $80 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) were unable to reach high efficiency rates (*i.e.*, higher than 23-24%) (Sassi et al., 2008). Along these lines, previous studies reported that cyclists with high $\text{VO}_{2\text{max}}$ seemed less responsive to training related changes in GE, than those with lower $\text{VO}_{2\text{max}}$ (Hopker et al., 2012). Though uncommon amongst elite cyclists, previous studies have reported both high $\text{VO}_{2\text{max}}$ and GE in Tour de France cyclists (Bell et al., 2015; Santalla et al., 2012).

In the present study, contrary to our hypothesis, there was no significant relationship

between GE and $\text{VO}_{2\text{max}}$ in April, nor in July (Figure 2). Since increases in GE are related to accumulated training volume and intensity through the years (Jobson et al., 2012), we could argue that the lack of a significant relationship could be due to the fact that the cyclists from this study had limited intensive training (about two years), while professional cyclists, with more significant relationships had performed high-volume endurance training for several years (e.g. average of $35,000 \text{ km}\cdot\text{yr}^{-1}$) (Lucia et al., 2002).

Acknowledgements

The authors would like to thank the cyclists of the Seguros Bilbao Cycling Team for their participation, effort and support in this investigation. This study was supported by the Government of the Basque Country (SAI10/42, SA-2010/00071)..

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Conclusions

GE of U23 cyclists is lower than GE observed in older cyclists and it does not vary during the most important phase of the training season (i.e., April to July). Additionally, GE was not significantly correlated to $\text{VO}_{2\text{max}}$. The lower accumulated volume and intensity of training in younger cyclists may account for their lower GE in comparison to older professional cyclists and this lack of training did not yield higher increases of GE in cyclists with lower $\text{VO}_{2\text{max}}$.

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