Chapter 6B

Changes in gross efficiency in relation to
time trial length

Noordhof DA, Mulder RCM, Malterer KR, Foster C, de Koning JJ.
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Abstract

The primary purpose of this study was to evaluate if gross efficiency (GE), determined during submaximal cycling, is lower after time trials and if the magnitude of the decrease differs in relation to race distance. Secondary purposes were to study the rate of the decline in GE and the underlying cause. Cyclists completed nine GE tests, consisting of submaximal exercise performed before and after time trials of different length (500 m, 1,000 m, 2,000 m, 4,000 m, 15,000 m, and 40,000 m). Additionally, subjects performed time trials (1,000 m, 4,000 m, and 40,000 m) during which they were stopped at 50% of the final time of the corresponding ‘full’ time trial. Power output, gas exchange, and EMG were measured. A significant interaction between distance and time was found for GE ($p < 0.01$). GE was lower after the time trials and the magnitude of the decrement in GE was larger for shorter trials. The rate of the decline in GE seemed constant for relatively short time trials, but by 50% of the 40,000 m time trial the ultimate decrement in GE was almost attained. A significant effect of time ($p < 0.05$) was found for mean EMG amplitude. However, post-hoc comparisons showed no significant differences between the different time points (before and after the time trial). In conclusion, GE decreases during time trial exercise and it seems that this decline cannot be explained by changes in muscle fiber recruitment. Future modeling studies should consider using a declining instead of a constant GE. In sport situations, the declining GE has to be taken into account when selecting a pacing strategy.
Introduction

Recent literature suggests that the efficiency of the transfer of metabolic energy production (aerobically and anaerobically) to forward propulsion is a performance determining factor. Calculating gross efficiency (GE) requires calculating the ratio of mechanical power output (PO) to metabolic power input (PI) from measures of oxygen uptake (\(\dot{V}_\text{O}_2\)) and carbon dioxide output (\(\dot{V}_\text{CO}_2\)) during steady state submaximal exercise. Therefore, the estimated GE is only representative of efficiency during submaximal steady state exercise. To study athletic performances it would be desirable to know what GE would be during and after maximal and supramaximal exercise, as athletic events are mostly performed at intensities above the ventilatory threshold. For example, Padilla et al. showed that prologue time trials (mean distance 7.1 ± 1.0 km) in professional cycling are performed at a relative intensity of 114 ± 8% of the heart rate (HR) at the lactate threshold and that time trials with a mean distance of 28.2 ± 9.6 km are performed at 108 ± 9% of the HR at the lactate threshold.

There have been attempts to estimate the anaerobic energy contribution to exercise using the method described by di Prampero and Ferretti, which would make it possible to determine GE at intensities above the ventilatory threshold. However, as di Prampero and Ferretti stated in their review, the anaerobic energy contribution can only be estimated from continuous blood lactate measurements, as a continuous rise in blood lactate concentration represents a continuous anaerobic contribution. To circumvent this, de Koning et al. proposed a methodology that makes it possible to estimate GE immediately following a high intensity exercise bout, based on calculations of GE during submaximal exercise performed after a high intensity exercise bout and back-extrapolation using linear regression. During submaximal exercise before the maximal constant PO bout a mean GE of 18.3 ± 1.3% was found. GE was estimated to be 15.8 ± 1.7% immediately at the end of the maximal PO bout, which suggests that GE decreases by 2.5% during 4 min of maximal exercise. Since constant PO trials are unusual in competitive athletic events, the method of de Koning et al. may be the most practical method for estimating GE during and at the end of time trial exercise. Consequently, the first purpose of the present study was to determine GE during submaximal exercise before and after a time trial and to use linear regression to estimate what GE would have been immediately at the end of the time trial.

Athletes will adopt different pacing strategies for time trials of different length. For example, an all-out strategy will be used for 500 m or 1,000 m cycling time trials, but a more even-paced strategy, with a characteristic faster, slower, faster pattern will be
It might be that different pacing strategies and as a result different exercise intensities during time trials will cause dissimilar decrements in GE. Therefore, GE was determined during submaximal exercise before and after time trials of different length (500 m, 1,000 m, 2,000 m, 4,000 m, 15,000 m, and 40,000 m).

Passfield and Doust\(^1\) showed that GE significantly declined during moderate intensity endurance exercise (~60% \(\bar{V}O_{2\text{peak}}\)) from 21.5 ± 1.1% at 25 min to 21.1 ± 1.1% at 50 min. Multiple studies have shown that GE declines during moderate and high intensity exercise,\(^1,12,16,17\) but the rate of the decline in GE during high intensity cycling exercise and the underlying cause, have been less well studied. Therefore, the second purpose of the present study was to investigate the rate of the decline in GE during high intensity exercise by incorporating time trials in the study protocol during which subjects were stopped at 50% of the final time of the corresponding ‘full’ time trial (split trials). In this way, a GE value estimated immediately at the end of the split trial, i.e. at 50% of the final time of the ‘full’ time trial could be obtained. Changes in muscle fiber recruitment have been suggested as underlying cause of the reduction in efficiency.\(^1,17,18\) Therefore, EMG amplitude, an index of muscle fiber recruitment or muscle activation\(^19\) was studied together with changes in GE.

In summary, the goal of the present study was to determine GE during submaximal exercise before and after time trials of different length and gain knowledge about the magnitude and rate of the decline in GE and the underlying cause. It was hypothesized that GE would be lower after time trial exercise and that the biggest decline in GE will be observed for the shortest time trials. The rate of the decline in GE is hypothesized to be larger for the shorter time trials, with a more all-out pacing strategy and presumably more additional motor unit recruitment (most likely type II fibers).

**Methods**

**Subjects**

Nineteen healthy trained male cyclists (mean age 24.2 ± standard deviation (SD) 4.3 year) participated in this study. They trained 12 ± 4.6 h·week\(^{-1}\). Subject characteristics are summarized in Table 6.1. Subjects were instructed to avoid strenuous exercise and alcohol consumption within 24 h before the tests, to consume their last meal at least three hours prior to the experiments, and to avoid caffeinated beverages during the same time frame. Prior to the first test the aim of the study and the experimental protocol were explained, after which subjects provided written informed consent and completed a healthy history form. The local ethics committee approved the study protocol.
Table 6.1 Subject characteristics determined during a maximal incremental exercise test performed before and after the different gross efficiency tests.

<table>
<thead>
<tr>
<th></th>
<th>Before</th>
<th>After</th>
<th>t (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (cm)</td>
<td>183.9 ± 7.4</td>
<td>183.9 ± 7.4</td>
<td>&lt; 0.001 (1.00)</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>76.0 ± 10.1</td>
<td>75.7 ± 9.2</td>
<td>0.55 (0.59)</td>
</tr>
<tr>
<td>(\dot{V}O_{2\text{max}}) (L·min(^{-1}))</td>
<td>4.55 ± 0.45</td>
<td>4.55 ± 0.47</td>
<td>-0.021 (0.98)</td>
</tr>
<tr>
<td>(\dot{V}O_{2\text{max}}) (mL·kg(^{-1})·min(^{-1}))</td>
<td>60.6 ± 5.6</td>
<td>60.7 ± 5.8</td>
<td>-0.14 (0.89)</td>
</tr>
<tr>
<td>P(\dot{V}O_{2\text{max}}) (W)</td>
<td>392 ± 43</td>
<td>413 ± 31</td>
<td>-4.12 (&lt; 0.01)</td>
</tr>
<tr>
<td>P(\dot{V}O_{2\text{max}}) (W·kg(^{-1}))</td>
<td>5.22 ± 0.56</td>
<td>5.52 ± 0.52</td>
<td>-3.77 (&lt; 0.01)</td>
</tr>
<tr>
<td>HR(_{\text{max}}) (beats·min(^{-1}))</td>
<td>193.4 ± 8.4</td>
<td>190.9 ± 7.6</td>
<td>1.92 (0.07)</td>
</tr>
</tbody>
</table>

Values are reported as means ± standard deviations. \(\dot{V}O_{2\text{max}}\), maximal oxygen uptake; P\(\dot{V}O_{2\text{max}}\), power output attained at \(\dot{V}O_{2\text{max}}\); HR\(_{\text{max}}\), maximal heart rate.

**Experimental design**

Each subject completed 13 experimental testing sessions, separated by at least one day of rest, within a period of 79 days. The first and final testing sessions were a maximal incremental exercise test, to monitor possible changes in fitness due to the extensive experimental protocol. The second testing session consisted of familiarization 1,000 m and 4,000 m time trials, separated by 20 min rest. After at least 48 h rest, the subjects completed a 40,000 m familiarization trial. Familiarization trials were performed to select the best individual gear ratio (front gear 48 or 52 and rear 17 or 18) of the bike, to experience the effort that had to be delivered, and to minimize the effect of learning on pacing strategy.\(^{20}\) Subsequently, the following time trials, preceded by and ended with a submaximal exercise bout, were performed in random order during separate visits: 500 m, 1,000 m, 2,000 m, 4,000 m, 15,000 m, 40,000 m, 1,000 m split, 4,000 m split, and 40,000 m split, with the restriction that the split time trial could only be performed when the corresponding ‘full’ time trial had already been completed. Subjects were instructed to complete the time trials in the shortest amount of time possible. Tests were performed on a custom made, electronically braked cycle ergometer (VU-MTO, Amsterdam, The Netherlands). Elapsed time and pedaling frequency were displayed on a screen in front of the cycle ergometer during both the maximal incremental exercise tests and the submaximal exercise bouts. Distance covered was displayed during the time trials. Saddle and handle bar height were adjusted to individual preferences and were recorded on the first visit for replication during subsequent tests. Subjects used their own clipless pedals. Environmental circumstances were controlled in a climate chamber (room temperature 17.1 ± 0.8°C, relative humidity 50.9 ± 6.7%).
Maximal incremental exercise test
The maximal incremental exercise test, necessary for the determination of the maximal oxygen uptake ($\dot{V}O_{2max}$) and PO at $\dot{V}O_{2max}$ ($P\dot{V}O_{2max}$), started with a warm-up of 3 min at 100 W, after which PO was increased by 25 W·min⁻¹. Subjects were instructed to maintain a pedaling frequency of ~90 revolutions per minute (rpm). If subjects were not able to maintain pedaling frequency above 80 rpm, despite verbal encouragement, the test was ended.

Gross efficiency test
The GE tests started with 1 min rest, while the subjects were sitting on the cycle ergometer, followed by 1 min cycling at 25 W, after which exercise intensity was increased to 55% $P\dot{V}O_{2max}$ for 6 min and finally to 65% $P\dot{V}O_{2max}$ for another 6 min, as GE increases with relative exercise intensity. After this submaximal part of the GE test, subjects had 2 min of relative rest at 25 W before they started with one of the time trials (500 m, 1,000 m, 2,000 m, 4,000 m, 15,000 m, 40,000 m, 1,000 m split, 4,000 m split, or 40,000 m split). Following completion of the time trial, subjects rested for 1 min (25 W), which was enough time to allow $\dot{V}O_2$ to drop below the level at 65% $P\dot{V}O_{2max}$ before continuing with the post time trial submaximal part of the GE test (10 min at 65% $P\dot{V}O_{2max}$). After the time trial a submaximal exercise intensity of 65% $P\dot{V}O_{2max}$ was chosen, as each subject was able to cycle at this intensity with a mean RER $\leq$ 1.0 (pre time trial). The protocol of the GE tests is displayed in Figure 6.3.

The three GE tests which included a split time trial followed exactly the same protocol, however, during the split time trials (1,000 m, 4,000 m, or 40,000 m) subjects had to follow the PO profile produced during the corresponding ‘full’ time trial. The PO profile of the ‘full’ time trial and the real-time PO of the split trial were both displayed on the screen in front of the subjects. Subjects were instructed to follow the PO line of the corresponding time trial as closely as possible. When subjects were at 50% of the finish time of the ‘full’ time trial, the split trial was ended and subjects rested for 1 min (25 W), before continuing with the post time trial submaximal part of the GE test. During both submaximal parts of the GE test pedaling frequency had to be maintained at ~90 rpm.
Figure 6.3 Experimental protocol of the gross efficiency test. Mean respiratory values, power output, and EMG amplitude were determined over the shaded areas (pre, post1, post2). ft = finish time.

Data collection
Torque, pedaling frequency, and PO data of the cycle ergometer were sampled using a frequency of 100 Hz. Expired air was analyzed breath-by-breath using open circuit spirometry (Cosmed Quark b², Cosmed S. R. L., Rome, Italy), for the duration of the entire test. Before every test the gas analyzer and volume transducer were calibrated (gas analyzer: room air and a reference gas mixture (16.0% O₂ and 4.02% CO₂), volume transducer: 3 L syringe). HR was measured continuously with radiotelemetry (Polar Electro OY, Kempele, Finland). During all GE tests neuromuscular activity of the vastus medialis, vastus lateralis, biceps femoris (long head), and gluteus maximus muscle of the left leg was recorded using bipolar surface electromyography (EMG; Porti 17, TMS, Enschede, The Netherlands) at a sampling rate of 1000 Hz. A reference electrode was attached to one of the lumbar processus spinosus. The EMG signal was processed with a differential amplifier (22-bits AD conversion after 20 × amplification, input impedance > 1012 Ω, CMRR > 90 dB). Pairs of surface electrodes (Ag/AgCl; Ambu A/S, Ballerup, Denmark; inter-electrode distance 30 mm) were attached on the muscle belly in parallel to the muscle fiber axis, after shaving and cleaning the skin with alcohol. Cable movement artifacts during cycling were minimized by attaching the electrode leads to the skin using Fixomull tape (Fixomull stretch, BSN medical, Hamburg, Germany). After completion of the first test the position of the surface electrodes was marked using henna to enable reproduction of the electrode placement in subsequent tests. Prior to every GE test subjects
performed two repetitions of a static maximal voluntary contraction (MVC) of 5 s, with 20 s rest in between, on the bicycle ergometer by pushing down the pedals maximally, while the pedals were blocked in a horizontal position.

**Data analysis**

Breath-by-breath respiratory data were converted to second-by-second respiratory data using interpolation. $\dot{V}O_2\text{max}$ was determined as the highest $\dot{V}O_2$ of 30 s moving averages. To diminish the breath-by-breath noise, the second-by-second respiratory data of the GE tests was smoothed with a 6 s moving average filter.

Over the final 3 min of the 65% $\dot{P}V\dot{O}_2\text{max}$ step (shaded area in Figure 6.3), of the pre time trial submaximal bout, $\dot{V}O_2$ and RER were averaged in order to calculate GE (GE-pre), as described in Noordhof et al.\textsuperscript{21} Average values of $\dot{V}O_2$ and RER were also determined from minute 3:00 to 6:00 and 6:30 to 9:30 (shaded areas in Figure 6.3), to calculate GE during the post time trial submaximal bout of the GE test (GE-post1 and GE-post2, respectively), with the restriction that mean RER was $\leq 1.0$ and $\dot{V}O_2$ was in steady state. Steady state was accepted if the difference in $\dot{V}O_2$ between min 3:00-4:00 and 5:00-6:00 (or 6:30-7:30 and 8:30-9:30) expressed relatively to the mean $\dot{V}O_2$ over the corresponding 3 min was $\leq 5.0\%$. If RER was above 1.0 or $\dot{V}O_2$ was not in steady state, then the corresponding GE value was excluded from further analysis.

GE was determined from the second-by-second respiratory data (with the restriction that RER $\leq 1.0$) during the final 9 min of the post time trial submaximal bout. A linear regression line was fitted through the second-by-second GE data using the least squares method and back-extrapolation of this line to the end of the time trial or split time trial provided an estimate of GE at the end of maximal exercise (GE-extrap).\textsuperscript{12}

To determine the mean amplitude of the EMG signal, the raw EMG signal of the GE tests and associated MVC trial was high-pass filtered (4\textsuperscript{th} order Butterworth filter, cut-off frequency 20 Hz), band-stop filtered (4\textsuperscript{th} order Butterworth filter, [49 51]), rectified, and then low-pass filtered (4\textsuperscript{th} order Butterworth filter, cut-off frequency 3 Hz) to produce a linear envelope. MVC was determined as the highest moving average over 500 ms. The mean amplitude, expressed as percentage MVC, was determined over the final 3 min of the 65% $\dot{P}V\dot{O}_2\text{max}$ pre time trial submaximal bout and over min 3:00-6:00 (post1) and 6:30-9:30 (post2) of the post time trial submaximal bout.
Statistics

Data are presented as individual values or means ± SD. Visual inspection of Q-Q plots and the Shapiro-Wilks’ statistic were used to check if the differences were normally distributed (SPSS 20.0, IBM Corp., Armonk, NY, USA). A paired samples t-test was used to test the differences in body mass, $\dot{V}O_{2\max}$ (L·min$^{-1}$), $\dot{V}O_{2\max}$ (mL·kg$^{-1}$·min$^{-1}$), $P\dot{V}O_{2\max}$ (W), $P\dot{V}O_{2\max}$ (W·kg$^{-1}$), $HR_{\max}$ (beats·min$^{-1}$) between the two maximal incremental exercise tests. Missing GE-pre values, due to RER values above 1.0 or non steady state conditions of $\dot{V}O_2$, were substituted with the mean of all GE-pre values of the individual, as previous research showed that GE can be determined very reliably.21 The coefficient of variation was 3.5% (standardized typical error 0.57) in the present study, which is good, as the coefficient of variation of most tests is between 1 and 5%.22 Differences in GE were tested using a repeated measures ANOVA, with distance (500 m, 1,000 m, 2,000 m, 4,000 m, 15,000 m, and 40,000 m) and time (pre, post1, and post2) as within factors. A second repeated measures AVOVA, with the extrapolated GE values included, was performed, as the extrapolated values are the best estimate of GE immediately at the end of the time trial. Differences in mean EMG amplitude were tested using a repeated measures ANOVA, with distance (500 m, 1,000 m, 2,000 m, 4,000 m, 15,000 m, and 40,000 m), time (pre, post1, and post2), and muscle (vastus medialis, vastus lateralis, biceps femoris, and gluteus maximus) as within factors. The GE results of the split trials and corresponding ‘full’ time trials were analyzed using a repeated measures ANOVA with distance (1,000 m, 4,000 m, and 40,000 m) and time (pre, extrap-split, and extrap) as within factors. The GE-pre value was determined as the average of the GE-pre value of the split trial and the GE-pre value of the corresponding ‘full’ time trial. The assumption of sphericity was tested using Mauchly’s test. When the assumption of sphericity was met and significant main and/or interaction effects were found, post-hoc comparisons were tested using the Bonferroni method. In case of violation of the assumption of sphericity, the degrees of freedom were adjusted using the Greenhouse Geisser correction if $\varepsilon < 0.75$ or with the Huynh Feldt correction if $\varepsilon \geq 0.75^{23}$ and post-hoc comparisons were tested with the Bonferroni method. Differences were considered to be significant if $p < 0.05$.

Results

Comparing the outcome variables of both maximal incremental exercise tests resulted in a significant difference in $P\dot{V}O_{2\max}$, with a higher $P\dot{V}O_{2\max}$ attained during the maximal incremental exercise test after the extensive experimental period. No significant changes in body mass, $\dot{V}O_{2\max}$, and $HR_{\max}$ were found (Table 6.1).
Due to missing GE-post1 and GE-post2 values, GE data of only 10 subjects were analyzed. No effect of distance (500 m, 1,000 m, 2,000 m, 4,000 m, 15,000 m, 40,000 m) was found on GE ($F = 0.34, p = 0.88$), but a significant main effect of time ($F = 104.4, p < 0.001$) was present. The interaction between distance and time was not significant ($F = 1.48, p = 0.16$). Post-hoc comparisons for time (pre, post1, post2) showed that all differences were significant, GE was lower after the time trials (post1 and post2), compared to before. Including the extrapolated GE values, resulted in no significant main effect of distance on GE ($F = 0.71, p = 0.62$), but in a significant effect of time ($F = 98.6, p < 0.001$) and a significant interaction between distance and time ($F = 5.49, p < 0.01$; Figure 6.4). Post-hoc comparisons for time (pre, extrap, post1, and post2) showed that GE was different between all time points ($p < 0.01$). Further analysis of the interaction effect revealed a significant effect of time for all distances ($p < 0.001$) and a significant effect of distance on the extrapolated GE values ($p < 0.001$; Figure 6.4A and 6.4B, compared to Figure 6.4C).

Mean EMG amplitude was not significantly different between time trial distances ($F = 1.90, p = 0.11$), but a significant main effect of time and muscle was found on mean EMG amplitude ($F = 5.26, p < 0.05$; $F = 18.54, p < 0.001$; Table 6.2). No significant interactions were found. Post-hoc comparisons showed that there was no significant difference in EMG amplitude between the different time points (pre, post1, and post2). Post-hoc comparisons between the different muscles showed that there was a significant difference between the gluteus maximus and the vastus medialis ($p < 0.01$), vastus lateralis ($p < 0.01$), and the biceps femoris muscle ($p < 0.05$). In addition, there was a significant difference between the vastus medialis and the biceps femoris muscle ($p < 0.05$).

Analyzing the GE results of the split trials and corresponding ‘full’ time trials, of 15 subjects, resulted in the finding of a significant main effect of distance ($F = 5.13, p < 0.05$), time ($F = 67.9, p < 0.001$), and a significant interaction between distance and time ($F = 3.33, p < 0.05$). Post-hoc comparisons showed that the GE values of the 1,000 m and 40,000 m were significantly different from each other ($p < 0.05$; Figure 6.5). Post-hoc comparisons between the different time points (pre, extrap-split, extrap) demonstrated that the GE values differed significantly between all time points ($p < 0.01$). Studying the interaction resulted in a significant effect of distance on the extrapolated GE values at the end of the entire time trial ($p < 0.001$) and a significant effect of time for all three distances ($p < 0.01$; Figure 6.5).
Figure 6.4 Gross efficiency values before and after the different time trials (mean values ± standard deviations). A: 500 m (filled circles) and 1,000 m (open circles) B: 2,000 m (filled circles) and 4,000 m (open circles) C: 15,000 m (filled circles) and 40,000 m (open circles).
Table 6.2 Mean EMG amplitude of the vastus medialis (A), vastus lateralis (B), biceps femoris (C), and gluteus maximus muscle (D), expressed as percentage MVC (maximal voluntary contraction), before and after time trials of different length.

<table>
<thead>
<tr>
<th></th>
<th>A – vastus medialis</th>
<th></th>
<th>B – vastus lateralis</th>
<th></th>
<th>C – biceps femoris</th>
<th></th>
<th>D – gluteus maximus</th>
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<tbody>
<tr>
<td></td>
<td>Distance</td>
<td>pre</td>
<td>post1</td>
<td>post2</td>
<td>Distance</td>
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<td>post1</td>
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</tr>
<tr>
<td></td>
<td>500 m</td>
<td>30.54 ± 14.47</td>
<td>31.29 ± 13.77</td>
<td>31.19 ± 12.77</td>
<td>500 m</td>
<td>31.29 ± 13.03</td>
<td>32.01 ± 12.03</td>
</tr>
<tr>
<td></td>
<td>1,000 m</td>
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<td>1,000 m</td>
<td>31.80 ± 11.11</td>
<td>32.58 ± 10.55</td>
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<tr>
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<td>2,000 m</td>
<td>35.69 ± 14.82</td>
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<td>40,000 m</td>
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<td>32.91 ± 12.11</td>
<td>33.28 ± 12.50</td>
<td>40,000 m</td>
<td>32.49 ± 14.23</td>
<td>30.83 ± 11.03</td>
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</table>

Values are reported as means ± standard deviations (n = 10).
Discussion

The main finding of this study is that GE is lower after time trial exercise, compared to before, and that the magnitude of the decrement in GE differs between time trials of different length. The decrement in GE is larger for shorter time trials (i.e. 500-4,000 m) and it seems that GE decreases during these relatively short time trials in a linear fashion (Figure 6.5). In contrast, the decrement in GE during longer time trials (i.e. 15,000 m and 40,000 m) is smaller and at 50% of the final time of the corresponding ‘full’ time trial the ultimate decrement in GE seems to be almost attained (Figure 6.5). It was hypothesized that the decrement in GE could be explained, at least partly, by an increase in muscle fiber recruitment. However no change in mean EMG amplitude was found during submaximal exercise performed before and after the time trials.

An exercise induced decrement in GE has been found by other research groups. Passfield and Doust\(^1\) let subjects perform submaximal warm-up exercise (~60% \(\dot{V}O_{2\text{peak}}\)) and a 5 min performance test before and after 60 min cycling at ~60% \(\dot{V}O_{2\text{peak}}\), to study the effect of endurance exercise on cycling performance. A reduction in GE from 22.6 ± 0.91% to 20.7 ± 1.07% was found when GE was determined during both warm-up bouts. The change in GE between both warm-up bouts was significantly correlated with the change in 5 min performance \(PO\) (\(r = 0.73\)).\(^1\) Besides calculating GE during both warm-up bouts, Passfield and Doust\(^1\) also determined GE during the 60 min submaximal cycling bout and found that GE decreased significantly from 21.5 ± 1.1% at 25 min to 21.1 ± 1.1%
at 50 min. Unfortunately, it is unknown how much GE decreased due to the high intensity 5 min performance test, as the effect of the 5 min performance test and the submaximal bout cannot be separated.

Bangsbo et al.\textsuperscript{16} studied anaerobic and aerobic ATP production in a group of six subjects performing two bouts of 3 min intense knee extensor exercise and found that the amount of work delivered per mmol ATP produced was significantly higher during the first 15 s (39.3 ± 5.1 J/ mmol ATP), compared to the remaining 165 s (25.0 ± 1.3 J/mmol ATP), which implies that muscle efficiency decreases during high intensity exercise. In a follow-up study of Krustrup et al.,\textsuperscript{17} ATP turnover and muscle heat production were studied during 90 s low intensity exercise without and with thigh blood flow occlusion. They showed that mechanical efficiency (power output divided by total energy turnover (rate of heat production + mechanical power output)) was 52% during low intensity exercise with thigh occlusion and was similar during the first 10 s of low intensity exercise without thigh occlusion (56%), but then decreased to 45% after 25 s and to 32% for the remaining time, which was significantly lower than during the low intensity bout with thigh occlusion. A third bout of exercise consisted of 90 s of low intensity exercise followed by 90 s of high intensity exercise (both without thigh occlusion), during which mechanical efficiency was 47% after the first 5 s of high intensity exercise, dropped to 38% after 15 s and then remained constant. So, it seems that mechanical efficiency decreases during 90 s of low and high intensity exercise without thigh occlusion. Besides that, a higher mechanical efficiency was found when ATP is produced mainly anaerobically, as during the low intensity bout with thigh blood flow occlusion. The efficiency values reported by Bangsbo et al.\textsuperscript{16} and Krustrup et al.\textsuperscript{17}, obtained during knee extensor exercise, were much higher than the efficiency values found by Passfield and Doust\textsuperscript{1} and the GE values found in the current study, but still the results are in agreement. In general, a decrease in efficiency is found during both low and high intensity exercise. Based on the results of Bangsbo et al.\textsuperscript{16} it can be argued that the ‘real’ GE at the start of the time trial is most likely higher than GE determined during submaximal exercise before the time trial. Therefore, the estimated decrement in GE during time trial exercise found in the current study (determined during submaximal exercise) may be even larger. Mechanical efficiency and the amount of ATP produced per unit of work performed did not change during 90 s low intensity exercise with thigh occlusion.\textsuperscript{17}

Multiple studies have shown that efficiency decreases during exercise.\textsuperscript{1,12,16,17} However, this is the first study in which the decrement in GE is studied for multiple time trial distances. A possible cause of the larger decrement in GE with relatively short time trials (500-4,000 m) is the higher exercise intensity reached during these time trials.
Exercise at a higher intensity will result in larger homeostatic disturbances, which logically would require large physiologic adjustments, hence a higher PI and a lower GE. The results of the split trails showed that at 50% of the final time of the corresponding ‘full’ time trials (1,000 m, 4,000 m, 40,000 m) the extrapolated GE values were not significantly different from each other, but that at the end of the time trial the extrapolated GE values were significantly different from each other, with a larger decrement in GE for the shorter time trials (Figure 6.5). During the 40,000 m the final decrement in GE is almost attained at 50% of the final time, which suggests that the anaerobic contribution during the start of the time trial seems to cause the decrement in GE, as exercise intensity during the second half of the 40,000 m is around the ventilatory threshold (RER = 0.96). A RER above 1.0 during exhaustive exercise reflects sodium bicarbonate buffering. A high RER thus reflects a considerable contribution of anaerobic energy production. During the shorter time trials a very high RER was found during the final 10% of the trial (500 m, 1.49 ± 0.08; 1,000 m split, 1.28 ± 0.08; 1,000 m, 1.39 ± 0.06; 2,000 m, 1.20 ± 0.05; 4,000 m split, 1.15 ± 0.05; 4,000 m, 1.09 ± 0.05; 15,000 m, 1.01 ± 0.04, 40,000 m split, 0.96 ± 0.03; 40,000 m, 0.97 ± 0.04). Only during the longer time trials (15,000 m and 40,000 m) a RER at or just below 1.0 was found. This suggests that the marked decrement in GE during the shorter time trials is related to the high exercise intensity, but that also the rate of the decline in GE seems to be associated to exercise intensity. Jones et al. 24 showed that during constant PO exercise, performed just below the critical power, muscle phosphocreatine concentration ([PCr]), inorganic phosphate concentration ([Pi]), and pH reached a steady state within 3 min after the start of exercise. During constant PO exercise performed just above the critical power muscle [PCr] and pH decreased progressively and muscle [Pi] increased progressively until subjects reached the point of task failure. It can be hypothesized that a decrease in muscle [PCr] and pH, and an increase in [Pi] correlates with changes in GE and that exercise intensity (i.e. below or above critical power) determines the rate of the decline in GE.

The lower GE after time trial exercise can most likely be explained by a decrement in muscle efficiency (phosphorylative coupling efficiency × contraction coupling efficiency25) and possibly partly by recovery processes in fatigued inactive fibers in combination with an increase in respiratory work and cardiac muscle work that increases metabolic cost. Suggested causes of a reduction in phosphorylative coupling efficiency and contraction coupling efficiency during dynamic exercise are an elevated muscle temperature, Pi accumulation, Ca2+ accumulation, and a change in muscle fiber recruitment. 17,26,27 Willis and Jackman 26 found a ~10% decrease in the phosphorylative coupling efficiency (P/O ratio) of isolated rat skeletal muscle mitochondria when
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temperature increased from 37˚C to 40˚C. In the study of Tonkonogi et al.\textsuperscript{28} no change in P/O ratio was found in isolated mitochondria of human muscle samples immediately after high intensity intermittent exercise. However, mitochondrial function was determined under constant conditions (25˚C, pH 7.35), so without changes in muscle pH, metabolite concentration, oxygen tension, and temperature, which might have influenced the research findings. Because of the difficulties with studying the individual effect of these different physiological variables (i.e. elevated muscle temperature, free phosphate accumulation, \(\text{Ca}^{2+}\) accumulation in the muscle, and a change in muscle fiber recruitment) on phosphorylative coupling efficiency and contraction coupling efficiency under in vivo conditions in humans, it is complicated to estimate the contribution of changes in muscle efficiency on GE. In the current study and in the study of Vanhatalo et al.\textsuperscript{29} the hypothesis that a change in muscle fiber recruitment causes a decrease in efficiency (i.e. an increased \(\text{O}_2\) cost per unit of external work delivered in Vanhatalo et al.\textsuperscript{29}) was investigated. Vanhatalo et al.\textsuperscript{29} showed that a decrement in efficiency during a 3 min all out test, reflected by a ‘mirror-image’ \(\dot{\text{V}}\text{O}_2\) slow component, is not caused by the progressive recruitment of muscle fibers. The ratio between integrated EMG and work rate stayed relatively constant during the second half of the all-out test, while the \(\dot{\text{V}}\text{O}_2\) gain (\(\text{mL} \cdot \text{min}^{-1} \cdot \text{W}^{-1}\)) kept increasing. In contrast, a high intensity constant PO exercise bout, during which \(\dot{\text{V}}\text{O}_2\) showed a slow component, resulted in an increase in integrated EMG.\textsuperscript{29} Thus, it seems that different mechanisms are responsible for the decrement in GE due to constant PO exercise or all-out exercise. Although, we found a significant main effect of time on mean EMG amplitude, the change is really small (no significant post-hoc differences between time points; Table 6.2) and cannot explain the difference in GE decrement between relatively short and the longer time trials. The results of the present study therefore appear to support the findings of Vanhatalo et al.\textsuperscript{29} additional muscle fiber recruitment does not seem to explain the decrease in efficiency during all-out or time trial exercise. Part of the decrement in GE can possibly be explained by recovery processes in fatigued inactive fibers, however thus far we cannot estimate how large this contribution is. The metabolic cost of an increase in ventilation (\(\dot{\text{V}}\text{E}\)) was estimated to be 1.8 ml \(\text{O}_2\)·L\(^{-1}\)·min\(^{-1}\) at 70% \(\dot{\text{V}}\text{O}_2\)\(_{\text{max}}\) and 2.9 ml \(\text{O}_2\)·L\(^{-1}\)·min\(^{-1}\) at 100% \(\dot{\text{V}}\text{O}_2\)\(_{\text{max}}\) in a study of Aaron et al.\textsuperscript{30} Based on the data of Aaron et al.\textsuperscript{30} the relative contribution of an increase in \(\dot{\text{V}}\text{E}\) to the increase in \(\dot{\text{V}}\text{O}_2\) (post1 vs. pre) was estimated to be 12-36% in the current study. The exact contribution is most likely somewhere in between, as the average \(\dot{\text{V}}\text{E}\) was 147.0 L·min\(^{-1}\) at 100% \(\dot{\text{V}}\text{O}_2\)\(_{\text{max}}\) in the study of Aaron et al.\textsuperscript{30} and the average \(\dot{\text{V}}\text{E}\) during the post1 interval was 104.7 L·min\(^{-1}\) (compared to 79.9 L·min\(^{-1}\) during the pre interval) in the current study. The lowest contribution of the increase in \(\dot{\text{V}}\text{E}\) to the increase in \(\dot{\text{V}}\text{O}_2\) was found for the
relatively short time trials, 500-4,000 m (12-15% based on 1.8 ml O₂·L⁻¹·min⁻¹). Passfield and Doust¹ also estimated the contribution of the increased $\dot{V}E$ to the increased $\dot{V}O₂$ and concluded that the contribution was negligible.

**Conclusion**

The current study is the first study that investigated changes in GE during competitive cycling events. We showed that GE is reduced after time trial exercise and that the decrement in GE is larger for shorter time trials (500-4,000 m). The rate of the decline in GE seems to be constant during relatively short time trials, but during longer time trials the final decrement in GE is at 50% of the final time (about halfway) already attained. The exact cause of the reduction in GE during fatiguing exercise remains to be established. Based on the results of the present study the energy flow model for cycling,¹³ which currently assumes a constant GE during races, needs to be adapted. In sport situations, athletes and coaches should be aware of the declining GE when selecting a pacing strategy.
References


Changes in gross efficiency in relation to time trial length