The Effects of Training on Gross Efficiency in Cycling: A Review

Abstract

There has been much debate in the recent scientific literature regarding the possible ability to increase gross efficiency in cycling via training. Using cross-sectional study designs, researchers have demonstrated no significant differences in gross efficiency between trained and untrained cyclists. Reviewing this literature provides evidence to suggest that methodological inadequacies may have played a crucial role in the conclusions drawn from the majority of these studies. We present an overview of these studies and their relative shortcomings and conclude that in well-controlled and rigorously designed studies, training has a positive influence upon gross efficiency. Putative mechanisms for the increase in gross efficiency as a result of training include, muscle fibre type transformation, changes to muscle fibre shortening velocities and changes within the mitochondria. However, the specific mechanisms by which training improves gross efficiency and their impact on cycling performance remain to be determined.

Introduction

Gross efficiency is defined as the ratio of power output to energy expenditure and is a key determinant of endurance cycling performance [14, 17, 18, 36, 49, 53]. Despite this, previous studies investigating gross efficiency during cycling have found no differences between trained and untrained cyclists [6, 45, 50, 52]. The results of these studies supported the recently challenged assumption that training has no effect on a cyclist’s gross efficiency [34]. These observations of non-significant differences between trained and untrained individuals using cross-sectional research designs have limited the extent of investigation into the mechanisms that could be responsible for changes in efficiency with training. Recently, using more rigorous experimental designs researchers have provided evidence that gross efficiency can increase with training and is higher in a trained population [33, 34, 60]. Nonetheless, one recent study documenting improvements in cycling efficiency in a Grand Tour Champion [19] has provoked much debate within the scientific literature [25, 47, 61]. Currently, researchers are beginning to identify the specific circumstances under which training can lead to an increase in gross efficiency during cycling.

Terminology

During steady-state cycle ergometry, efficiency has been extensively used to provide a convenient index of the effectiveness with which an individual can convert chemical energy into mechanical power [4, 23, 28, 42, 52, 66]. The most commonly used measure of efficiency is gross efficiency (\(\text{[work accomplished/energy expended]} \times 100\)). In calculating gross efficiency, the caloric equivalent of steady state VO\(_2\) and the respiratory exchange ratio (RER) are used to calculate energy expenditure. The term ‘gross efficiency’ is normally reported as a percentage of total energy expenditure [15].
Other indices of efficiency used in the literature include work, net and delta efficiency. The calculation of net efficiency requires the energy expenditure at rest to be measured (and assigned to the performance of internal work), which is then subtracted from the total amount of energy expended for the external work accomplished. Thus, net efficiency does not take into account the cost of moving the involved limbs. Gaesser and Brooks [23] suggested that for a valid measurement this cost should be taken into account. For example, the energetic cost of cycling on an ergometer, but with a work rate of OW (i.e. unloaded pedalling) would need to be subtracted from the total energy expenditure for the measured work rate.

The calculation of work efficiency accounts for the additional cost of moving the legs, but this measurement of zero load in cycling can be problematic. Kautz and Neptune [40] have argued that zero load cycling does not provide a valid baseline for reference to a range of work intensities. Gaesser and Brooks [23] proposed a floating baseline measure for the physiological and external energy cost of exercise, i.e. delta efficiency (DE). Here energy expenditure at a lower work rate is subtracted from the energy expenditure at a higher work rate ([delta work accomplished/delta energy expended] × 100). Coyle et al. [16] suggested that this is the most valid calculation of whole-body efficiency as it attempts to partial out the influence of unmeasured work. However, the linear relationship that is produced between energy cost and work rate does not necessarily mean that it is a valid measure (i.e. it is independent of the work rate used). For efficiency to increase with power output [12, 23] energy expenditure must increase non-linearly due to the decreasing relative contribution of non-propulsive factors (e.g. basal metabolism and moving the legs). The use of DE may also be limited as both Moseley and Jeukendrup [49] and Hopker et al. [33] have shown it to have greater day-to-day variability than gross efficiency.

The issues involved in these measures have been extensively discussed by Gasser and Brooks [23], Stainsby et al. [64], Cavanagh and Kram [11] and Kautz and Neptune [40]. Due to the various criticisms of base-line subtraction methods outlined above, it is unrealistic to attempt to attribute a portion of the total body energy cost to muscle work during a whole body exercise such as cycling. Therefore we will focus on gross efficiency for the rest of this review.

The measurement of efficiency and other methodological issues

Steady state testing

In order to measure efficiency accurately all gas collection must take place under steady state exercise conditions, otherwise measured pulmonary VO₂ may not adequately reflect muscle O₂ consumption [55]. At the onset of exercise or in the transition from one work rate to another, during light to moderate intensities, pulmonary VO₂ increases in a mono-exponential manner to reach a steady state within 2–3 min [75]. Heavy exercise intensities, characterised by a sustained metabolic acidosis, have been found to result in a delayed steady state [74]. Consequently, the relationship between VO₂ and power output presents a marked deviation from linearity at higher intensities when examined carefully [26, 78]. The exercise intensities eliciting this additional O₂ consumption, or VO₂ slow component, should not be used to determine efficiency values. The calculated energy equivalent for a given VO₂ depends upon the equivalence of RER and muscle RQ. A decrease of 0.05 in RER reduces calculated energy expenditure by 1.3 % typically increasing GE by 0.4 %. Therefore as VCO₂ affects RER it is important to ensure its stability prior to taking efficiency measurements. During steady state exercise resulting in minimal metabolic acidosis, VCO₂ may take considerably longer (at least 4 min) to reach steady state [13, 72]. For these reasons, long work stages (≥ 5 min) should be used when collecting gas data for the calculation of efficiency. Boning et al. [6] and Moseley et al. [50] used 3 min stage durations for their studies of cycling efficiency. Therefore they may have failed to allow sufficient time to achieve a steady state when investigating differences in efficiency between trained and untrained cyclists.

Exercise intensity

A particular criticism of past research is the low exercise intensities used for assessing efficiency during cycling. Researchers have previously sought to investigate optimal levels of energy expenditure during cycling using untrained participants [23, 62, 66, 77] and Moseley et al. [50] used 3 min stage durations for their studies of cycling efficiency. As a result, it has only been possible to study responses to low work rates. These values are typically very low if inferences from such findings are made to trained cyclists who commonly race at much higher work rates.

Efficiency has been shown to increase with work rate [12, 23]. It is thought that this is largely due to the unmeasured work (i.e. that required to sustain basal metabolism and body position on the bike) forming a smaller percentage of total energy expenditure at higher work rates [64]. However, more recently Moseley and Jeukendrup [49] have demonstrated that a plateau in gross efficiency occurs at the higher work rates (>240W) used by trained cyclists. Ideally, the work rate used for the determination of gross efficiency should encompass the functional range of the population of interest. However, when trained cyclists are considered this is potentially difficult due to the VO₂ slow component associated with higher racing intensities.

Standardisation of other factors

Most authors agree that changes in muscle shortening velocity (i.e. pedal cadence) markedly affect efficiency. For a given power output, increasing cadence has been shown to decrease gross efficiency [12, 28, 45, 63]. Therefore, cadence must be standardised when conducting repeated measurements of efficiency in the same individual. Changes in riding position may also influence the efficiency values obtained. Alterations in seat tube angle and saddle height have been shown to change gross efficiency [30, 56]. Price and Donne [56] found an energetic optimum for combinations of seat tube angle and seat height (70° seat tube angle; 100% trochanter height). Alterations in the muscle length-tension relationships (quadriceps versus hamstrings) and ankle patterns could account for differences in efficiency found with increased seat tube angles and heights. Thus, the dimensions of a cyclist’s bicycle should be replicated when using a cycle ergometer and maintained during any subsequent tests.

High ambient temperatures (35.5 °C) have been shown to cause decreases in gross efficiency [31]. With hyperthermia the energy cost of the exercise may increase due to greater circulation, sweating and ventilation. This in turn may reduce efficiency as the work accomplished would remain unchanged. Therefore ambient temperature should be tightly controlled, especially for competitive cyclists.
when repeating tests during different phases of a season under varied climatic conditions.

Finally, the control of cyclists’ pre-testing regimen presents a further methodological issue. Racing cyclists often train and compete for several hours at intensities of 60% \( \dot{V}O_2 \) peak and above [44]. A gradual \( \dot{V}O_2 \) drift is commonly observed during such prolonged exercise. This \( \dot{V}O_2 \) drift manifests as a reduction in gross efficiency and is apparent even in trained cyclists when exercising for 75 min at 60% \( \dot{V}O_2 \) peak [54]. Passfield and Doust [54] further demonstrated that this acute reduction in gross efficiency was significantly correlated with a lower 5 min time-trial performance. Similarly, there is also some evidence to suggest that muscle damage from high intensity training might decrease efficiency during subsequent exercise performance [43, 71]. Therefore, it is important to monitor training in the days prior to testing athletes for efficiency. The time course for restoration of gross efficiency after exercise has not been established. The ability to detect meaningful changes in cycling performance over time, and to study the effects of selected interventions, requires careful consideration of the validity and reliability of testing methods and equipment [3, 35]. If reliability of repeated measurements is not ensured then there will be increased chances of falsely rejecting the null hypothesis in any research study. Therefore, it is critical that appropriate methods are employed which minimise the variability of within steady state \( \dot{V}O_2 \) and \( \dot{V}CO_2 \) data, exercise intensities utilised, ergometer set-up and participant pre-test preparation.

**Chronic changes in gross efficiency with training**

A question which has been addressed by several researchers is whether cycling efficiency can be increased by training. Previous cross-sectional studies have failed to find any differences in efficiency between trained and untrained cyclists [6, 45, 46, 50, 52]. However a number of these investigations can be criticized on the basis of their methods and their failure to address the risk of committing a type 2 statistical error. Specifically, a lack of statistical power in past research studies has meant an inability to detect significant differences between study populations. Other confounding factors are the usage of short stage protocols and artificially imposed cadences. Table 1 shows the main findings of research studies that have sought to investigate differences in efficiency between participants of varied cycling ability. To illustrate the likely chance of a type 2 statistical error we have calculated the effect size and statistical power using post hoc methods [5, 69]. Effect size was calculated by the division of the mean difference between the two groups (\( \mu_1 - \mu_2 \)) by the pooled standard deviation (SD) [69]:

\[
ES = \frac{\mu_1 - \mu_2}{\text{SD}}
\]

\( \delta \) (the value for non centrality) and achieved statistical power was then calculated using GPower software [22].

**Table 1** Methods and findings of cross sectional research studies that have investigated differences in efficiency between participants of varied cycling ability. The table is restricted to studies which present enough data to estimate effect size and statistical power.

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Sample Size</th>
<th>Stage Duration</th>
<th>Power Outputs used</th>
<th>Efficiency measure</th>
<th>Cadence imposed</th>
<th>Effect Size/ Statistical Power</th>
<th>Efficiency finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boning et al., (1984)</td>
<td>15 (9 trained cyclists; 6 untrained)</td>
<td>3 mins</td>
<td>50, 100, 200W</td>
<td>GE &amp; NE</td>
<td>yes (40, 60, 70, 80, 100rev.min(^{-1}))</td>
<td>0.87/0.33 at 200W</td>
<td>GE (p&lt;0.05) &amp; NE (both 1% mean difference)</td>
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<tr>
<td>Nickleberry &amp; Brooks (1996)</td>
<td>12 (6 competitive; 6 recreational)</td>
<td>4 mins</td>
<td>50–200W</td>
<td>GE &amp; DE</td>
<td>yes (50 and 80rev.min(^{-1}))</td>
<td>0.26/0.07 at 200W</td>
<td>no significant difference (&gt;1% difference between groups)</td>
</tr>
<tr>
<td>Marsh et al., (2000)</td>
<td>31 (11 competitive cyclists; 10 trained runner; 10 non-cyclists)</td>
<td>5 mins</td>
<td>trained cyclists; 100, 150, 200W Untrained 75, 100, 150W</td>
<td>DE</td>
<td>yes (50, 65, 80, 95, 110rev.min(^{-1}))</td>
<td>1.25/0.77 at 80 rev.min(^{-1})</td>
<td>no significant difference (“1% difference between groups at 80 &amp; 95rev. min(^{-1}))</td>
</tr>
<tr>
<td>Moseley et al., (2004)</td>
<td>69 trained cyclists (divided on ( \dot{V}O_2 )peak, low, medium and high)</td>
<td>3 mins</td>
<td>95W increasing by 35W</td>
<td>GE &amp; DE</td>
<td>yes (80–90 rev.min(^{-1}))</td>
<td>2.33/1.00 between low and high ( \dot{V}O_2 )peak groups</td>
<td>GE &amp; DE no significant difference (GE 0.9% difference Med-High groups; DE 1.2% difference Low to High groups)</td>
</tr>
<tr>
<td>Hopker et al., (2007)</td>
<td>30 (14 trained cyclists; 16 recreational)</td>
<td>10 mins</td>
<td>150W, 50 &amp; 60% ( W_{max} )</td>
<td>GE</td>
<td>preferred cadence used</td>
<td>1.51–1.54/0.98 across intensities used</td>
<td>GE significantly higher in trained group (mean + 1.4% p&lt;0.05)</td>
</tr>
</tbody>
</table>

Horowitz et al. [36] have shown that cyclists with a high gross efficiency (21.9%) were able to sustain a significantly higher power output (27W) during a 1-hour cycle time-trial performance than a group with a lower gross efficiency (20.4%). Jeukendrup et al. [39] used a mathematical modeling approach to predict that a 1% increase in a cyclist's gross efficiency would result in a 63-s improvement in 40km time-trial time. It is therefore quite possible that the changes in gross efficiency following training described by Hopker et al. [34] and Sassi et al. [60] will afford a performance advantage.

**Potential mechanisms for chronic changes in gross efficiency**

**Muscle fibre type transformation**

Previous work has demonstrated that there is a positive correlation between the percentage of type I fibres and gross efficiency [16,36]. Horowitz et al. [36] have shown that cyclists with a higher percentage of type I fibres also had a significantly higher efficiency and were able to maintain a 9% higher power output during a one-hour performance trial. Both studies largely provide correlation based evidence, and intervention studies are required to evaluate this relationship more thoroughly. Coyle et al. [15] reported a positive relationship between years of endurance training and percentage of type I fibres (r = 0.75).

Again, due to the cross-sectional study design, it was not possible to determine whether the percentage of type I fibres were a response to years of training, or that those cyclists with more type I fibres continue to train and race longer.

Jansson and Kaijser [38] compared metabolic responses in trained cyclists and untrained individuals exercising at 65% VO2peak. The trained cyclists had a significantly higher percentage of type I fibres (70% vs. 40%) and a greater gross efficiency (22% vs. 19%). Coyle [19] suggested that much of the 8% increase in efficiency observed in a Grand Tour Champion was the result of an increased percentage of type I muscle fibres caused by prolonged intense endurance training and/or prolonged exposure to high altitude conditions. Coyle attributed much of the improvement in efficiency to muscle fibre type adaptations (speculating that the rider's fast twitch fibres took on more of a slow twitch role), even though no muscle biopsies were taken. In contrast, Martin et al. [47] suggested that modifications in diet, training and chemotherapy (with a resultant loss of body and leg mass), were as likely to be responsible.

When training for several hours per day at low force and movement speeds, as in cycling, the possibility of low frequency stimulation-induced transitions from type IIb to type IIa and ultimately type I might be expected [37,59,76]. This has led to the formulation of the 'adaptive range' concept, which describes the adaptive possibilities for each muscle fibre type [27,73,76]. As satellite cells have been shown to be predetermined to end up as a specific muscle fibre type within a certain adaptive range, local genetic factors have been suggested as unimportant [2] in fibre type alteration.

There is a significant difference in tension cost (how much ATP is consumed per unit force generated during an isometric contraction) between human type I and type II fibres [65]. However, during active shortening, thermodynamic efficiency is the same (21–27%), although peak efficiency is reached at different velocities (at around 15% of maximum shortening velocity in both cell types) [29]. Thus, the effect of fibre-type distribution on whole-body efficiency when cycling is probably not due to differences in contractile efficiency between myosin isoforms. Rather, it may be due to the shortening velocities during cycling being closest to those associated with peak efficiency in type I fibres (as has been suggested elsewhere [16]). Interestingly, marathon training increases the peak shortening velocity of type I fibres [70], and therefore, presumably, the shortening velocity at which peak efficiency is attained. This would improve efficiency during running if the new, most efficient, shortening velocity was better matched with the actual shortening velocity during running. Thus, there is a mechanism by which muscle cells could provide improved contractile efficiency in response to training, without any change in metabolic efficiency.

**Factors influencing efficiency in the muscle cell**

Oxidative phosphorylation is the main process by which ATP is produced under aerobic conditions. Changes in the efficiency of oxidative phosphorylation will therefore affect cycling efficiency. Adaptations that might affect efficiency which are detectable early in a training programme may be related to the myosin ATP supply. Mitochondrial volume and aerobic capacity increase greatly within the first 4–6 weeks, especially in type II fibres, whilst anaerobic capacity decreases [32,37,59]. It could also be suggested that decreases in submaximal oxygen uptake may be due to changes in the working muscle's oxidative capacity and metabolic processes, represented by an increase in activity of the mitochondrial enzymes [10].

A precursor for the aerobic adaptations seen as a result of training is the production of adenosine monophosphate-activated protein kinase. This enzyme is released as a result of intensive training [24,41], and may cause the up-regulation of PGC-1α. This in turn is thought to regulate mitochondrial biogenesis in type I, Ila and IIX fibers [58,67,68]. However, these observations are limited to low and moderately trained individuals, who demonstrate marked physiological adaptations and improvements in fitness. Whether the same adaptations are typical of a trained population remains to the determined.

Key questions also remain unanswered regarding the efficiency of energy transfer within the mitochondria and the possible role of the uncoupling of oxidative phosphorylation. Energy liberated from respiratory chain reactions are used to translocate protons across the inner mitochondrial membrane, creating an electrochemical potential [7]. This potential is subsequently used to drive the endergonic re-phosphorylation of ADP to ATP by the ATP-synthase, a reaction which is stoichiometric [8]. However, protons are able to leak back across the inner membrane without driving the ATP-synthase, a phenomenon known as uncoupling. In skeletal muscle, the most important proteins mediating this process appear to be the adenine nucleotide translocase (ANT) and uncoupling protein-3 (UCP3). Thus increases in the content or activity of these proteins might have adverse effects on cycling efficiency.

Uncoupling accounts for around 50% of resting oxygen consumption in rodent muscle [57]. Its contribution to efficiency during exercise is unclear, particularly as proton leak is of diminishing importance as mitochondrial respiratory rate rises [9]. In the only study of UCP3 content and efficiency in cyclists to date, muscle UCP3 content and work efficiency were negatively correlated in a cohort of mixed-ability cyclists [48]. UCP3 content was greater in untrained compared to trained individuals.**

Therefore, the process of training might have reduced the expression of UCP3, increased the coupling of oxidative phosphorylation, and improved gross efficiency when cycling. However, this mechanism is yet to be observed following a period of training. The contribution of ANT activity to whole-body efficiency remains unexplored. Longitudinal or interventional studies are needed to illuminate the contribution of mitochondrial uncoupling to exercise efficiency in humans.

Conclusions

Until recently it has been assumed that training had no impact on cycling efficiency. This was largely based on the results of investigations that did not find a difference between trained and untrained cyclists. It now appears that insufficient methods (e.g. small sample sizes and inappropriate exercise intensities) may have resulted in type II errors in many of these studies. To enable the identification of training related changes and obtain consistency in efficiency measurements pre-test preparation, ergometer set-up and methods used for data collection must all be valid and reliable. Recent evidence suggests that training (especially at high intensities) improves gross efficiency in cycling. Potential mechanisms which might be responsible for training related increases in GE include muscle fibre type transformation, aerobic enzyme capacity within the muscle and the expression of PGC1α, ANT and UCP3. Future studies should seek to fully investigate the mechanisms responsible for determining gross efficiency via the use of intervention methodologies and invasive biopsy techniques. Further research is required to clarify the role of specific training regimes on the development of gross efficiency and the underlying pinching changes.

References

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