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Adaptations to Training in Endurance Cyclists

Implications for Performance

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Abstract

Our present scientific knowledge of the effects of specific training interventions undertaken by professional cyclists on selected adaptive responses in skeletal muscle and their consequences for improving endurance performance is limited: sport scientists have found it difficult to persuade elite cyclists to experiment with their training regimens and access to muscle and blood samples from these athletes is sparse. Owing to the lack of scientific study we present a theoretical model of some of the major training-induced adaptations in skeletal muscle that are likely to determine performance capacity in elite cyclists. The model includes, but is not limited to, skeletal muscle morphology, acid-base status and fuel supply. A working premise is that the training-induced changes in skeletal muscle resulting from the high-volume, high-intensity training undertaken by elite cyclists is at least partially responsible for the observed improvements in performance. Using experimental data we provide evidence to support the model.

The physiological requirements for success in road cycling are unique and have been recently reviewed.[1,2] Riders compete in mass start, multistage races characterised by stochastic shifts in work rate and/or speed that are largely dependent on terrain[3,4] and team tactics.[1,5] In contrast, during individual[5,6] and team time trials[5] a rider/team sustains the highest average power output (W) possible for the duration of a race. As such, training programmes for improving road cycling perfor-

mance should evoke multiple adaptations that enable a rider to increase energy production from both aerobic and oxygen-independent pathways and delay the onset of muscular fatigue.

Unfortunately, our present scientific knowledge of the effects of specific training interventions undertaken by professional cyclists on selected adaptive responses and their consequences for endurance performance is limited. Although much training data have been collected by sport scientists affiliated with National and professional teams (Martin DT, personal communication), we could find only one published study that detailed the power output and heart rate (HR) responses of a professional cyclist to a single interval training session.[7] Therefore, we have undertaken a series of investigations examining the effects of short term (3 to 6 weeks) intensified training on the metabolic adaptations and performance responses of well-trained cyclists.[8-13] Although the cyclists in those studies were all competitive riders [maximal oxygen uptake ($\dot{V}O_{2max}$) >5.0 L/min] training between 300 to 600km per week, they do not meet the physiological or performance criteria of an elite or world-class rider.[1] Unfortunately, it is extremely difficult to conduct studies that incorporate even moderately invasive techniques when using the most competitive cyclists in the world: only recently have sport scientists been able to obtain laboratory and field data from world class cyclists.[3,5,6,14]

The general physiological responses to endurance training in moderately fit individuals have been well documented.[15] Of interest to this review are those physiological adaptations that occur in response to the extremely high-volume, high-intensity training programmes adopted by professional cyclists. Certainly training does have an impact on performance in genetically endowed, nationally competitive riders, but the question is 'what physiological adaptations are responsible for the modest improvements in performance that are observed?' Because of the scarcity of scientific study in this area, we present a theoretical model of the major training-induced adaptations in skeletal muscle that are likely to determine and improve performance capacity in elite cyclists. A working premise is that training-induced changes in skeletal muscle resulting from high-volume, high-intensity training are at least partially responsible for the observed improvement in performance. Using experimental data we provide evidence to support the model.

1. The Training Stimulus

The key components of any training programme aimed to enhance cycling performance are volume, frequency and intensity.[16] The sum of these training inputs can be quantified as the training impulse[17] that can enhance (fitness) or decrease (fatigue) performance. At any given time the 'predicted performance index' is the difference between fitness and fatigue.[18] A primary question of interest for a cyclist is 'how can I attain my best performance on a given day?' Similarly, a primary question of interest for coaches and sport scientists is 'how do we design a training programme to elicit a maximal performance (on a given day)?' Unfortunately, answers to these questions cannot be based on the results from well controlled published studies undertaken on elite cyclists because such data do not exist. Instead, we are left with sporadic, anecdotal reports of the training habits of a few elite performers[1,7] and the empirical field-based observations of a few outstanding coaches.[8,19]

Table I shows an overview of a 'typical schedule' undertaken by a professional world-class endurance cyclist during a 3-week pre-season training camp.[16] Although this information gives an insight into the volume of training undertaken by a successful professional rider during a specific phase of his programme, it does not provide any indication of the training intensity (i.e. power output, speed, HR), nor the length of time this rider has been performing such a regimen. The training block identified in table I reflects only a small portion of this rider's overall training stimulus which includes riding for >30 000km per year and racing ~100 days per year.[1]

Table I. Training programme for World Class endurance cyclists during a 2- to 3-week pre-season training camp. Data are from Hawley & Burke[16]

Day	Description	Intensity	Duration (h)	Distance (km)
Monday	Rest day or easy ride			
Tuesday	Medium long ride	Easy to moderate	3-4	110
Wednesday	Medium long ride	Easy to moderate	4	130
Thursday	Recovery ride	Easy	2	70
	Interval training	Hard	2-3	80-100
Friday	Recovery ride	Easy	2-3	90
Saturday	Endurance ride	Moderate	4-6	170-200
Sunday	Physiological testing, or riding in small groups			

2. The Model

Figure 1presents a theoretical model of 3 major areas of adaptation in skeletal muscle that are likely to contribute to the successful performance of elite cyclists. The model is an extension of one originally developed and validated by Coyle[20] describing the physiological factors determining endurance performance ability in distance runners, cyclists and race-walkers. For a comprehensive description of that model, the reader is referred to the reviews of Coyle.[20,21]

Our model consists of 3 highly interdependent components: the magnitude of any training-induced adaptation within a component is likely to be directly related to the overall training stimulus. The working premise is that the sum of adaptations within the components of the model ultimately exert a major influence on the performance capacity of a rider at any given time.

2.1 Skeletal Muscle Morphology

The morphological component of the model includes, but is not limited to: (i) skeletal muscle fibre type; (ii) oxidative and glycolytic enzyme activity; (iii) capillary density; (iv) lean thigh volume; and (v) neuromuscular recruitment. Several of these morphological factors have been discussed previously.[20] A high proportion of type I fibres in the vastus lateralis muscle is associated with a lower submaximal oxygen cost (i.e. a greater gross efficiency) during cycling,[22] possibly because of a lower ATP turnover during contraction.[23] As type I fibres also possess a higher capillary density and oxidative potential than type II fibres,[24] these factors may influence gross cycling efficiency. However, cross sectional data reveal that the number of type I fibres in the vastus lateralis muscle of a cyclist is highly related to the number of years spent performing endurance training.[25] Thus, either those cyclists with a large percentage of type I fibres are more competitive and advance into the professional ranks, or fibre type conversion from type II to type I is occurring. Accordingly, if fibre transformation does occur, one would expect that gross efficiency would improve over the course of a rider's career. Unfortunately, there are no longitudinal investigations that have reported such a phenomenon. Such studies are warranted because small improvements in cycling efficiency have the potential to result in large enhancements in performance. For example, Jeukendrup et al.[1] have calculated that for a 70kg cyclist who can sustain a power output of 400W for 1 hour, a 1% increase in gross efficiency would result in a 48 second improvement in 40km time-trial time.

In addition to the importance of a high proportion of type I fibres in the working muscles, the role of type IIa fibres for the successful performance of endurance cycling should not be overlooked. The type IIa fibre has a similar oxidative potential to the type I fibre and at competition intensities has a greater force generating capacity.[24]

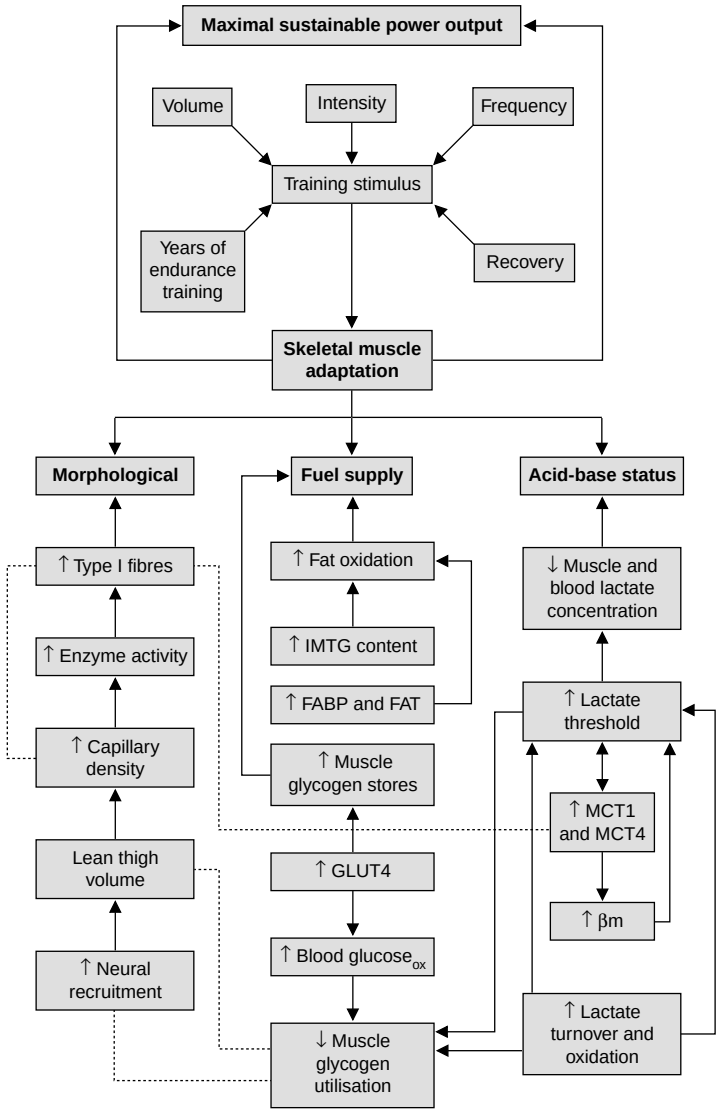

Fig. 1. A model of some of the major adaptations in skeletal muscle that result from the high-volume, high-intensity training undertaken by professional cyclists. **Blood glucose$_{ox}$** = blood glucose oxidation; **FABP** = fatty acid binding protein; **FAT** = fatty acid translocase; **GLUT 4** = glucose transporter protein; **IMTG** = intramuscular triglyceride; **MCT** = monocarboxylate transporter; **βm** = muscle buffering capacity; ↓ indicates a decrease; ↑ indicates an increase.

Although activities of selected skeletal muscle oxidative enzymes (i.e. citrate synthase) have been reported to be higher in a group of 'elite' versus 'good' competitive cyclists,[25] others have found that enzyme activity does not correlate well with endurance cycling capacity.[13] Thus, in some cases factors other than muscle oxidative capacity are important for successful cycling performance. One such factor might be muscle capillary density: a high capillary density would increase muscle perfusion

by reducing diffusion distance and facilitating lactate removal from muscle.[26]

The lean thigh volume of elite cyclists is similar to that of highly trained amateurs,[9,25] so this morphological component cannot account for the large differences in sustainable submaximal and maximal power output between these groups of riders. Instead, Coyle et al.[25] have reported that cyclists with superior performance ability produce a greater power output when riding by adopting a pedalling strategy that allows them to develop greater vertical forces during the 'downstroke.' This superior technique may be related to the number of years of endurance training.[25] It may well be that an important component of sustained, intense riding over a period of many seasons is to directly alter muscle contractility.[27]

Part of this altered muscle contractility may be related to improved patterns of neuromuscular recruitment that would adapt in response to sport-specific overload patterns. Recent data[28] indicate that marked neuromuscular changes occurred [electromyogram (EMG)/force ratio] in both agonist and antagonist leg muscles during a series of 15 × 5-second sprints: as fatigue increased (i.e. power declined) there was 'intermuscular coordination' to transfer force to the pedal. Such an adaptation could be centrally mediated and/or due to reflex changes caused by Golgi tendon organs. In this regard, it is interesting to note that the vast majority of improvements in dynamic muscle strength observed after short term resistance training are attributable to increased voluntary activation of muscle (i.e. better neural recruitment) rather than any measurable increase in the size of muscle fibres.[29] Furthermore, endurance performance can be improved independently of changes in $\dot{V}O_{2max}$ and economy.[30] Taken collectively these observations suggest that prolonged, intense endurance training conducted over many seasons may induce neuromuscular adaptations that enable elite riders to recruit a greater number of muscle fibres and spread the power production over a large active muscle mass while pedalling, compared with less well-trained cyclists.[25]

2.2 Acid-Base Status

A central premise of the model proposed by Coyle[20] is that the individual lactate threshold (LT) is a major 'functional ability' that is highly related to a rider's maximal sustainable power output.[20,31] Indeed, one of the major attributes of professional endurance cyclists is their ability to sustain high absolute workrates for prolonged periods while maintaining steady-state blood lactate levels.[6]

It is now widely accepted that lactate plays an important role as an energy-rich compound during sustained intense exercise.[32,33] Indeed, during progressive cycling, MacRae et al.[34] have reported that up to 80% of lactate is oxidised and accounts for ~45% of overall carbohydrate oxidation. Accordingly, the capacity to transport lactate and H^+ out of the muscle fibres, and the capacity of skeletal muscle to take up lactate are likely to be key determinants of a cyclist's power output at LT.

Transport of lactate across the sarcolemma of skeletal muscle is mediated by the proton-linked monocarboxylate transporters (MCT), the membrane transport system with the highest capacity for H^+ transport during intense exercise.[35-37] Two muscle isoforms of the lactate/H^+ carrier have been cloned: MCT1 and MCT4.[37,38] In humans, MCT1 content is greater in type I fibres, whereas MCT4 content appears to be quite similar in various muscles.[37,39] Of note is the finding that individuals with a high proportion of type I fibres in their active musculature will also have a higher MCT1 content in the type II fibres compared with individuals who possess fewer type I fibres.[37] As a muscle with primarily type II fibres has 50% of the lactate transport capacity of a muscle composed of type I fibres, the importance of these membrane-bound transport proteins for endurance cycling performance is obvious.

Pilegaard et al.[39] examined the capacity to transport lactate across the sarcolemma in individuals of different training status. The relationship between lactate transport capacity and aerobic capacity is shown in figure 2. Lactate transport capacity was significantly higher in the athletes compared with untrained individuals. However, the capacity to

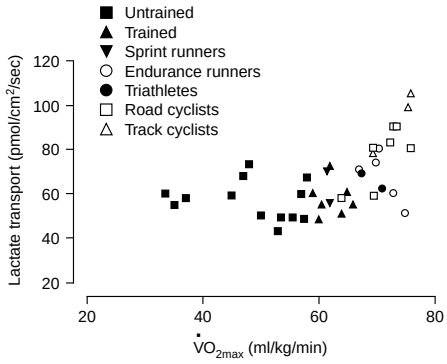

Fig. 2. The relationship between muscle lactate transport capacity and maximal oxygen uptake ($\dot{V}O_{2max}$) in untrained individuals (n = 13), trained individuals (n = 7), sprint runners (n = 2), endurance runners (n = 5), triathletes (n = 3), road cyclists (n = 6) and track cyclists (n = 3) [adapted from Pilegaard et al.[39] with permission from the American Physiological Society].

transport lactate across the sarcolemma was not directly related to an individual's $\dot{V}O_{2max}$. Instead, lactate transport was positively correlated with the number of type I fibres in the vastus lateralis ($r = 0.48$, $p < 0.01$), of which the endurance-trained athletes had a significantly greater proportion.[39] Although these cross-sectional data do not allow inferences to be drawn as to the causality between lactate transport and training status, only those endurance-trained athletes with a $\dot{V}O_{2max}$ >65 ml/kg/min had an elevated lactate transport capacity.[39] In that study,[39] the muscle capillary density was similar for all the endurance-trained athletes and was not related to lactate transport capacity. Thus, it appears that a large volume of endurance training alone is insufficient stimulus to improve the ability to transport lactate.

Indeed, the highest lactate transport values were observed in 2 track cyclists whose specialist event was the 4000m pursuit (one cyclist was a bronze medallist at the 1992 Olympic games). These cyclists were riding an average of ~700km per week including several high-intensity track sessions.[39] This finding highlights the importance of including high-intensity training sessions as preparation for both track and road races. It might also explain why a short term supra-maximal training programme (6 sessions of 12 × 30-second work bouts at ~650W) was just as effective at improving 40km cycle time-trial performance (lasting ~55 minutes) as longer 'aerobic' interval sets (6 sessions of 8 × 4 minutes at ~330W) performed at close to 40km time-trial pace.[10] Indeed, the apparent nadir in 40km performance enhancement after the supra-maximal versus aerobic interval training sets suggest that there may be more than one mechanism by which interval training enhances endurance performance.

It has been reported that the muscle buffering capacity (βm) is enhanced to a similar extent (~16%) after both high-intensity sprint training[40] and when endurance-trained cyclists replace a portion of their base training with sustained (5 minutes) aerobic interval sets.[13] As high-intensity training enhances the sarcolemmal lactate/H^+ transport capacity as well as the content of MCT1 and MCT4 protein in human skeletal muscle,[41] and as such training-induced adaptations are likely to be important for the regulation of muscle lactate and pH, we believe it is reasonable to recommend that supra-maximal repeated sprint workouts be incorporated into the race preparation of all cyclists.

2.3 Fuel Supply

Endurance cycling induces a multitude of profound adaptations in many physiological systems (i.e. muscular, cardiovascular and endocrine). An important effect of these adaptations is to modify the rates at which the major fuel sources (carbohydrate and fat) are utilised during prolonged, intense exercise. Perhaps the single most impressive training adaptation that can enhance endurance cycling performance is that, compared with less well-trained individuals, highly-trained riders oxidise less carbohydrate and more fat during exercise performed at the same absolute power output or oxygen uptake ($\dot{V}O_2$),[42] even though they usually have higher initial glycogen stores.[43] As depletion of the body's endogenous carbohydrate stores is a factor linked to fatigue during prolonged, exhaustive cycling,[44,45] this training-induced shift in substrate selection by the working muscles undoubtedly plays a major

role in the enhanced endurance capacity seen after endurance training.

Part of the training-induced shift in substrate selection (from carbohydrate to fat) at the same absolute work rate has been attributed to the improved respiratory control sensitivity that results from an increased muscle mitochondrial density.[46] However, Coyle et al.[31] have previously reported that in riders with a similar $\dot{V}O_{2max}$ (~67 ml/kg/min) and mitochondrial enzyme activity, muscle glycogen utilisation during strenuous (i.e. 79% of $\dot{V}O_{2max}$) cycling was 2-fold higher in individuals with a low, compared with a high power output at individual LT. Westgarth-Taylor et al.[12] have reported that short term high-intensity training in 8 previously well-trained cyclists ($\dot{V}O_{2max}$ 5.2 L/min) significantly decreased the rates of carbohydrate oxidation (from 3.2, 4.3 and 5.4 g/min to 2.9, 3.6 and 4.8 g/min at 60, 70 and 80% of pre-training $\dot{V}O_{2max}$, respectively), despite no changes in muscle oxidative capacity.[13] Taken collectively these data suggest that the early training-induced shifts in substrate utilisation (from carbohydrate to fat) are caused by increases in muscle respiratory capacity. However, in cyclists with a prolonged history of endurance training who have presumably maximised the oxidative potential of their working musculature, other factors (i.e. neuromuscular recruitment of a greater muscle mass, acid-base status etc.) must be important for subsequent shifts in the patterns of fuel metabolism.

Part of the reduction in carbohydrate utilisation observed after prolonged, intense training could be caused by a decrease in muscle glucose uptake,[47-49] even though endurance training has been shown to increase muscle GLUT-4 content.[49,50] A recent study[51] reported that when muscle glycogen levels were low, glucose uptake was higher in trained versus untrained individuals working at the same relative work rate. This would explain why highly trained endurance cyclists are able to utilise glucose at remarkably high rates during prolonged, submaximal exercise at a time when muscle glycogen stores are low,[45,52,53] and why oral carbohydrate administered to well trained cyclists ($\dot{V}O_{2max}$ 69 ml/kg/min) during prolonged (3-hour) exercise results in a net muscle glycogen synthesis in the nonactive fibres.[54]

The training-induced reduction in carbohydrate oxidation observed in trained athletes may be completely compensated for by the concomitant increase in fat oxidation. The intramuscular triglyceride (IMTG) level has been reported to decrease after several hours of submaximal exercise in well-trained athletes.[55] Furthermore, IMTG content decreased during a standardised bout of submaximal cycling in individuals who completed an intense 12-week training programme,[56] although others have failed to observe a similar phenomenon.[57] As training does not increase net free fatty acid (FA) uptake by working skeletal muscle,[58-60] it seems likely that the additional FAs oxidised during exercise after endurance training are derived mainly from IMTG stores. Of interest is the recent evidence of long-chain fatty acid (LCFA) transporters in skeletal muscle.[61] Bonen and co-workers have reported that LCFA transport is higher in type I than type II muscles,[62] and that when the oxidative capacity of muscle is increased there is a parallel increase in the rate of FA transport at the sarcolemmal membrane.[63] Such adaptations, although reported in moderate- to well-trained individuals, are likely to be highly functional in better trained athletes performing prolonged, intense exercise because they permit the muscle to increase its metabolism of FA at a time when muscle glycogen content is low.

3. Experimental Evidence to Support the Model

We have recently described the muscular and metabolic demands of intense interval training in competitive endurance cyclists.[11] In that study, 7 well-trained cyclists (age 27 ± 5 years, mass 76 ± 3kg, $\dot{V}O_{2max}$ of 5.14 ± 0.23 L/min) who were riding an average of 368 ± 141km per week and had been performing regular endurance for ~8 years performed 8 × 5-minute work bouts at 86 ± 2% of $\dot{V}O_{2max}$ (334 ± 18W) with 60 seconds recovery. Muscle biopsies were taken from the vastus lateralis

muscle immediately before and after the training session, while pulmonary gas exchange and venous blood were sampled at regular intervals throughout exercise. We compared those data[11] with a professional cyclist undertaking the same interval set at the same relative exercise intensity (8×5 minute work bouts at 86% of $\dot{V}O_{2max}$, 358W), under the same laboratory conditions. The characteristics of the professional rider were: age 36 years, mass 77kg, $\dot{V}O_{2max}$ 5.4 L/min. He was riding 600km per week and had been performing prolonged endurance training for 19 years.

Despite the 7% higher absolute work rate (358 *vs* 334W) and the associated elevation in ventilation ($\dot{V}_E$) [111 ± 5 *vs* 107 ± 8 L/min] and $\dot{V}O_2$ [4.7 ± 0.3 *vs* 4.3 ± 0.3 L/min], the professional cyclist had a lower respiratory exchange ratio (0.92 ± 0.02 *vs* 0.93 ± 0.03) and lower rates of total carbohydrate oxidation (300 ± 12 *vs* 336 ± 52 μmol/kg/min) than the well-trained riders. Accordingly, rates of fat oxidation were higher in the professional rider compared with the well-trained group (28 ± 8 *vs* 22 ± 10 μmol/kg/min). The lower rate of carbohydrate oxidation could be accounted for by a lower rate of muscle glycogen oxidation (fig. 3a). Perhaps the most striking observation was the small perturbations in acid-base status in the professional cyclist at high absolute work rates: the blood lactate level was 20% lower (3.0 ± 0.3 *vs* 5.2 ± 1.7 mmol/L) and the muscle lactate level 34% lower than that in the well-trained cyclists (fig. 3b). Interestingly, muscle pH was similar (fig. 3c) indicating that this parameter may not be the most sensitive measure of acid-base status. Subjective ratings of perception of effort on the 20 point Borg scale[64] taken throughout the interval sets at the same relative intensity were 8 ± 1 for the professional rider ('very light') versus 13 ± 1 for the group of well-trained cyclists ('somewhat hard'). In contrast to previous studies,[22,25] cycling economy (79 W/L) was not different between the group of well-trained riders and the professional cyclist who had been performing high-volume, intense endurance training for twice as long (19 *vs* 8 years).

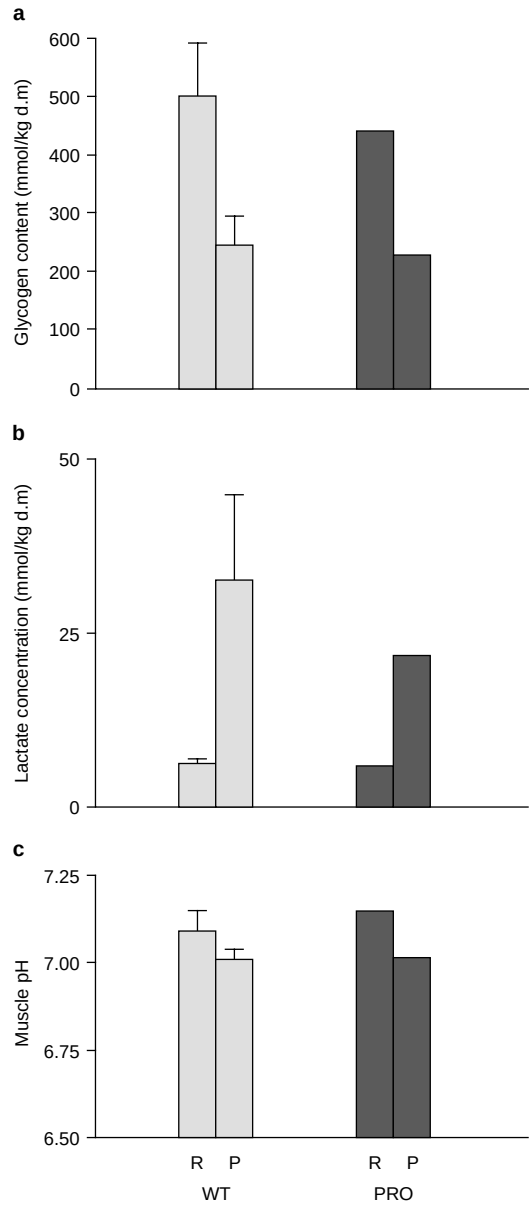

Fig. 3. Skeletal muscle responses to an intense aerobic interval training session in a group of well-trained (WT) competitive cyclists (n = 7) and a professional endurance cyclist (PRO, see section 3 for participant characteristics). After a standardised warm up, all cyclists performed 8 work bouts of 5 minutes duration at 88% of maximal oxygen uptake with a 60-second recovery at ~100W. Muscle biopsies were taken from the vastus lateralis muscle at rest (R) and post-exercise (P). The group data are from Stepto et al.[11]

4. Conclusions

Our current knowledge of the effects of specific training interventions undertaken by professional cyclists on the adaptive responses in skeletal muscle, and their consequences for performance is limited. Owing to the scarcity of scientific study, we presented a theoretical model of the major training-induced adaptations in skeletal muscle that are likely to determine and improve performance capacity in elite cyclists. A working premise was that such perturbations result from the high-volume, high-intensity training undertaken by these cyclists. Using experimental data, we provided evidence to support the model.

This article highlights the fact that our current knowledge of the physiological basis of endurance training techniques employed by professional cyclists is fairly elementary. Certainly there are many unanswered questions that can provide fertile areas of research for sports scientists. These include, but are not limited to:

(i) the effects of intensified training on lactate turnover in trained cyclists;

(ii) the effects of intensified training on lactate transporters in skeletal muscle;

(iii) the effects of different training interventions on muscle recruitment patterns; and

(iv) the effects of different training interventions on intracellular signalling pathways in skeletal muscle and their consequences for control of substrate utilisation.

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