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The Science of Cycling

Physiology and Training – Part 1

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Abstract

The aim of this review is to provide greater insight and understanding regarding the scientific nature of cycling. Research findings are presented in a practical manner for their direct application to cycling. The two parts of this review provide

information that is useful to athletes, coaches and exercise scientists in the prescription of training regimens, adoption of exercise protocols and creation of research designs.

Here for the first time, we present rationale to dispute prevailing myths linked to erroneous concepts and terminology surrounding the sport of cycling. In some studies, a review of the cycling literature revealed incomplete characterisation of athletic performance, lack of appropriate controls and small subject numbers, thereby complicating the understanding of the cycling research. Moreover, a mixture of cycling testing equipment coupled with a multitude of exercise protocols stresses the reliability and validity of the findings.

Our scrutiny of the literature revealed key cycling performance-determining variables and their training-induced metabolic responses. The review of training strategies provides guidelines that will assist in the design of aerobic and anaerobic training protocols. Paradoxically, while maximal oxygen uptake ($\dot{V}O_{2max}$) is generally not considered a valid indicator of cycling performance when it is coupled with other markers of exercise performance (e.g. blood lactate, power output, metabolic thresholds and efficiency/economy), it is found to gain predictive credibility.

The positive facets of lactate metabolism dispel the 'lactic acid myth'. Lactate is shown to lower hydrogen ion concentrations rather than raise them, thereby retarding acidosis. Every aspect of lactate production is shown to be advantageous to cycling performance. To minimise the effects of muscle fatigue, the efficacy of employing a combination of different high cycling cadences is evident. The subconscious fatigue avoidance mechanism 'teleoanticipation' system serves to set the tolerable upper limits of competitive effort in order to assure the athlete completion of the physical challenge.

Physiological markers found to be predictive of cycling performance include: (i) power output at the lactate threshold (LT2); (ii) peak power output (W_{peak}) indicating a power/weight ratio of ≥5.5 W/kg; (iii) the percentage of type I fibres in the vastus lateralis; (iv) maximal lactate steady-state, representing the highest exercise intensity at which blood lactate concentration remains stable; (v) W_{peak} at LT2; and (vi) W_{peak} during a maximal cycling test. Furthermore, the unique breathing pattern, characterised by a lack of tachypnoeic shift, found in professional cyclists may enhance the efficiency and metabolic cost of breathing. The training impulse is useful to characterise exercise intensity and load during training and competition. It serves to enable the cyclist or coach to evaluate the effects of training strategies and may well serve to predict the cyclist's performance.

Findings indicate that peripheral adaptations in working muscles play a more important role for enhanced submaximal cycling capacity than central adaptations. Clearly, relatively brief but intense sprint training can enhance both glycolytic and oxidative enzyme activity, maximum short-term power output and $\dot{V}O_{2max}$. To that end, it is suggested to replace ~15% of normal training with one of the interval exercise protocols. Tapering, through reduction in duration of training sessions or the frequency of sessions per week while maintaining intensity, is extremely effective for improvement of cycling time-trial performance.

Overuse and over-training disabilities common to the competitive cyclist, if untreated, can lead to delayed recovery.

Cycling remains the major means of transportation in many countries of the world and, as a recreational and competitive sport, it continues to grow increasingly popular. Furthermore, during the last decade, off-road cycling has enjoyed an exponential growth in popularity. While cycling has continued to expand as a recreational/fitness activity, so has the competitive nature of the sport. Competitive cycling requires both aerobic and anaerobic power. Road and off-road bicycle racing require the cyclist to possess the ability to generate a relatively high power output of short duration during the mass start, steep climbing and at the race finish.

The typical road race and off-road cross-country cycle events may have durations of approximately 1–5 hours while multi-stage races are characterised by several back-to-back days of racing consisting of mass-start stages and individual and team time trials. Professional cyclists must be able to tolerate high work-loads for long periods (3 weeks) during major tour races such as the Giro d'Italia, Tour de France and Vuelta a España. During these competitive events, the power output of the cyclist is used to overcome the aerodynamic and rolling resistances. Different by the nature of the setting, competitive track cycling demands acute bursts of high power output followed by constant high-intensity sprinting. This article attempts to provide an overview of the physiological and metabolic factors associated with cycling performance. A better understanding of the mechanisms and their interactions that underlie excellence in the dynamics of cycling will enable more educated approaches to testing, training and research.

1. Terminology and Research Design

Recent literature has questioned the application and interpretation of terminology and concepts commonly found in scientific papers that address topics related to exercise science.[1-7] Consequently, prior to this review, some interpretative commentary of concepts and terminology is considered necessary. Furthermore, research design, subject sample, sample size, instrumentation, methodology and data acquisition are addressed.

1.1 Terminology

The following terms are prevalent in cycling literature; however, the definition of these terms is often absent or inconsistent. For clarification and consistency, the terms with their definitions are presented.

'Anaerobic' is considered a non-oxygen metabolic concept that represents enzyme concentrations and activity. In the present article, the term anaerobic relates to those metabolic processes that do not depend on oxygen, irrespective of its availability.[6]

'Maximal oxygen uptake ($\dot{V}O_{2max}$)' represents the maximal oxygen used, and is limited by oxygen delivery and subject to central and peripheral cardiovascular capacity limitations and tissue oxygen demand.

'Efficiency' is a measure of effective work and is most commonly expressed as the percentage of total energy expended that produces external work.[7]

'Lactate threshold 1 (LT1)' is defined as the exercise intensity that elicits 1 mmol/L increase in blood lactate concentration above the average rest value.[8] It is identified on the individual power curve as the intensity eliciting such a blood lactate increase. This threshold has several names, such as lactate threshold, aerobic threshold and anaerobic threshold. However, we will use the term 'threshold 1' to represent the first break-point on the lactate-intensity curve.

'Lactate threshold 2 (LT2)' is the highest exercise intensity at which blood lactate concentration remains stable and is often referred to as maximal lactate steady state (MLSS). MLSS is attained when blood lactate concentration varies <1 mmol/L during the final 20 minutes of constant intensity exercise, reflecting a balance between lactate production

and removal.[9] Another common name for LT2 is the 'onset of blood lactate accumulation'.[10] An additional approach of determining LT2 is lactate D_{max}. This method provides a means of estimating power output corresponding to a rate of glycogen utilisation that can be maintained for 1 hour. The D_{max} method involves calculating the point on the regression curve that yields the maximal perpendicular distance from a curve representing work and lactate variables to the line formed by the two endpoints of the curve.

'Ventilatory threshold 1 (VT_1)' is the point of a non-linear increase in ventilation ($\dot{V}_E$) and carbon dioxide production ($\dot{V}CO_2$), in combination with a decline in expired CO_2 fraction (F_ECO_2) and an elevation of expired O_2 fraction (F_EO_2). In practice, the VT is identified as a point of hyperventilation with respect to oxygen uptake ($\dot{V}O_2$) and is reflected in a systematic increase in ventilatory equivalent for oxygen ($\dot{V}_E/\dot{V}O_2$) without a concomitant increase in the ventilatory equivalent for carbon dioxide ($\dot{V}_E/\dot{V}CO_2$).[11] VT_1 corresponds closely with LT1.

'Ventilatory threshold 2 (VT_2)', the second VT is the respiratory compensation point. The respiratory compensation point is identified as an increase in both $\dot{V}_E/\dot{V}O_2$ and $\dot{V}_E/\dot{V}CO_2$ and a decrease in end-tidal CO_2 tension ($PETCO_2$).[12] VT_2 corresponds very closely with LT2.

'Peak power output (W_{peak})' is the highest work-load sustained for 2–3 minutes during progressive incremental exercise to exhaustion.

'Mechanical efficiency' is the percentage of total chemical energy expended that contributes to external work, the remainder being lost in heat. Within this definition, mechanical efficiency is equal to the actual mechanical work accomplished divided by the input of energy × 100.

'Lactate concentration work-load' is defined as the highest work-load not associated with a rise in lactate concentration above baseline.[13] Blood lactate profiling is widely used for monitoring training-induced changes in the physical fitness status of cyclists. The generation of a blood lactate-velocity curve from incremental exercise testing allows for visual inspection for evidence of improvement, stability, or degradation of fitness condition over time. A rightward shift of the lactate-velocity curve reflects training-induced gains.

'Gross efficiency' is the ratio of work done during cycling to the total energy expended and is expressed as a percentage.[14] It is important to remember that measurement of gross efficiency is limited to intensities that elicit a respiratory exchange ratio of <1.00.

'Economy' is a measure of $\dot{V}O_2$ per unit of power output. It is the amount of oxygen per litre per unit of energy transferred to the cycle ergometer.

1.2 Research Design

A review of cycling literature clearly reveals the importance for a note of caution to the reader. Although published in peer-reviewed journals, several papers have vulnerable internal as well as external validity. Additionally, the over-extension of statistical inference, small sample size and the absence of control groups are often evident. Addressing sample size, Hopkins et al.[15] comments that for the estimate of the within-subject variation, ten athletes and three trials will give a barely acceptable 95% confidence interval. Moreover, additional trials and more athletes allow firmer conclusions about learning effects between trials. Concern is raised regarding the subject sample, which ranges from untrained to professional racers. Furthermore, several studies fail to control for the confounding factors of training, initial training level and the effect of testing.

1.2.1 Graded Exercise Tests

Laboratory and field test methodology and procedures differ among researchers engaged in cycling science. The extent to which dissimilar approaches to cycling exercise research affect the validity of the findings remains unanswered. Ramp protocol on a cycle ergometer is generally adopted for laboratory testing; however, work-load assignment differs. Table I presents a summary of commonly applied aerobic cycling test protocols.

When $\dot{V}O_2$ measuring equipment is not available, $\dot{V}O_2$ may be estimated utilising the following equation:[33]

Table I. Commonly applied aerobic bicycle ergometer test protocols

Study	Initial work-load (W)	Work-load increase (W)	Stage duration (min)	Pedal cadence	Termination
Bentley et al.[16]	100	30	1	Self-select	Exhaustion
Bishop et al.[17]	50	25	3	Self-select	Exhaustion
Fernández-García et al.[18]	100	50	4	Self-select	Exhaustion
Harnish et al.[19]	200	50	2 min for first 6 min, 1 min thereafter	80 rpm	Exhaustion
		25			
Heil et al.[20]	80	30 (men), 25 (women)	1	80 rpm	Exhaustion
Laursen and Jenkins[21]	100	15	30 sec	Self-select	Exhaustion
Lepers et al.[22]	150	25	2	Self-select	Exhaustion
Lindsay et al.[23]	3.3 W/kg	25–50	2.5	80–90 rpm	Exhaustion
Lucía et al.[24]	0	25	1	70–90 rpm	<70 rpm
Lucía et al.[25]	20	25	1	70-90 rpm	Exhaustion
Marsh and Martin[26]	100	50	2	85–110 rpm	Exhaustion
MacRae et al.[27]	100	25	1	Self-select	Exhaustion
Moseley and Jeukendrup[7]	60	35	3	80 rpm	Exhaustion
Padilla et al.[28]	110	35 (with 1 min recovery intervals)	4	75 rpm	<75 rpm
Romer et al.[29]	95	35	3	>60 rpm	<60 rpm
Schabort et al.[30]	100	20	1	Self-select	< power output
Stepto et al.[31]	3.3 W/kg	25–50	2.5	Self-select	Exhaustion
Swensen et al.[32]	200	40	2	80 rpm	<80 rpm

rpm = revolutions per minute.

$\dot{V}O_2$ (mL/min) = kg/min × 1.9 mL/min + ([3.5 mL/kg/min × kg bodyweight] + 260 mL/min)

where: 1.9 mL/min = $\dot{V}O_2$ power relationship; 3.5 mL/kg/min = resting $\dot{V}O_2$; 260 mL/min = O_2 cost of unloaded cycling.

This prediction equation, however, is not recommended for use at power loads above the lactate threshold.[33] Moreover, the accuracy of $\dot{V}O_{2max}$ estimation from regression equations, at least in top-level cyclists, may experience inaccuracy since an attenuation of the $\dot{V}O_2$/W relationship at high work-loads is a distinguished characteristic of the best cyclists.[34]

In the field, energy expenditure in terms of $\dot{V}O_2$ may be estimated for outdoor cycling utilising the equation presented by McCole et al.:[35]

$\dot{V}O_2$ (L/min) = −4.5 + 0.17 * C1 + 0.052 * C2 + 0.022 * C3

where: C1 = rider speed (km/h); C2 = wind speed (km/h); C3 = rider weight (kg).

1.2.2 Anaerobic Tests

The cyclist's ability to generate relatively high power output of brief duration is essential during climbing, sprinting to the finish or to pass other cyclists, starting in mass and individual time trailing. To examine this, ability tests of anaerobic power are often employed. For example, Baron[36] presented a protocol for a 10-second anaerobic power test that began with a 10-minute warm-up period at 1 W/kg bodyweight at 70–80 revolutions per minute (rpm) followed by a 1-minute rest then ten randomised 10-second bouts at maximal cadence ranging from 50 to 140 rpm in 10 rpm increments. Recovery between bouts was 4 minutes, during which time the cyclist continued to cycle with a power output of 1 W/kg. Numerous other protocols designed to test anaerobic power can be found in the literature. Hence, there is a need for consistency.

Specific protocols should be examined for their reliability, validity and appropriateness. Perhaps as

a starting point, a Wingate test might be used with a protocol of an initial load of 0.075kg resistance per kilogram of body mass applied within 3 seconds after the initial inertia and unloaded frictional resistance of the ergometer are overcome. The subsequent work-loads, durations, intervals, recoveries, etc., should then be established according to specific characteristics of the cyclist.

Anaerobic power is generally measured using an isokinetic cycle ergometer equipped with racing handlebars, saddle, toe clips and configured to match closely to the dimensions of the cyclist's bicycle. Throughout the test, lasting generally 10–30 seconds, the cyclist remains seated and generates 50–140 rpm. It appears that to remain seated discounts any confounding effects of standing.

1.2.3 Performance Tests

The laboratory 1-hour time-trial test is frequently performed on a calibrated wind-braked cycle ergometer employing the cyclist's bicycle. For this test, the cyclist is instructed to generate the highest power output possible throughout the 60-minute ride. The initial 8-minute power output is preset for work at 70% of the cyclist's W_{peak} determined from a previous incremental test. After 8 minutes, the cyclist is free to vary both pedal cadence and force. Throughout the ride, the cyclist is continually provided with visual feedback of pedal cadence, power output, heart rate (HR) and elapsed time. A 100km time trial has been developed to mimic the stochastic nature of bicycle road races and includes a series of sprints.[30,37] In this regard, for the research findings to be useful, test protocol should allow pacing that mimics an event and allow the cyclist to reproduce a familiar pacing strategy.

Field research is frequently conducted in an outdoor velodrome or during a road or off-road race. The bicycle is generally equipped with SRM instruments (Schroberer Rad McBtechink, Weldorf, Germany) that measures and stores power output, speed, cadence and subject HR. Credit should be given to those scientists who have examined the effects of treatments on performance in actual competitive events.

Researchers regularly comment that differences between their work and the findings of others cannot be explained or perhaps are due to a different subject sample or test protocol, etc. Currently, there is little information in the literature on the confounding effect of different research methodologies. In this regard, a careful review of the cycling literature reveals the need for consideration of methodology standardisation if research findings are to be useful at the operational level. The relationship between performance in tests and performance in cycling competition has not been explored adequately. The question remains, how a change in performance in a cycling laboratory test translates into a change in performance in the actual competitive setting. Moreover, the selection of the ergometer used in testing as well as the protocol demands serious consideration.

2. Bicycle Ergometer Types

Data generation is accomplished utilising various cycle ergometers including the Ergometrics 900 (Ergo-line, Barcelona, Spain); Superbike IIs or SB-II (USA; Fitrocyle, Fitronic); mechanically braked Monark 818 (Varberg, Sweden); mechanically braked Body Guard 900 (Ste Foy, Quebec, Canada); electromagnetically braked ergometer (Lode, Gronigen, The Netherlands); or the electromagnetically braked ergometer (Orion S.T.E., Toulouse, France). Additionally, the Kingcycle (Kingcycle Ltd, High Wycombe, UK) or the Kreitler windload simulator equipped with a Killer Headwind resistance unit (Kreitler Rollers Inc., Ottawa, KS, USA) is often employed as they allow the cyclist to ride her/his own cycle for various tests. In this instance, the bicycle, after removal of the front wheel, is attached to the ergometry system by the front fork and supported by an adjustable pillar under the bottom bracket. The rear wheel rests on an air-braked flywheel. Conversely, the Computrainer (Seattle, WA, USA) provides resistance to the rear wheel of the bicycle through an electronic load generator. The SRM crank dynamometer of an instrumented chainring that transmits strain-gauge readings by telemetry to a handlebar microprocessor

or to a central computer, along with crank rpm and bicycle speed. An alternative to the SRM is the Power-Tap (Madison, WI, USA), which determines power from strain-gauges placed in the hub of the rear wheel. Undertaking a test on a cyclist's own cycle rather than on a cycle ergometer eliminates the problem of the cyclist not being able to configure to their normal riding posture.

3. Test Termination Criteria

The criteria adopted to terminate a cycling exercise test vary among investigators. Nevertheless, there appears to be nothing to suggest that any one criterion is superior, reliable or valid compared with another. Rather, the investigator's past experience, subject's state of conditioning, concern for the subject's safety and desired data acquisition appear to dictate the adopted test termination conditions. Some of the termination criteria applied include:

- pedal cadence of 70,[24,38] 80[32] or 90 rpm[39,40] can no longer be maintained;
- subject volitional fatigue;[16,17,21]
- a given work-load or power output can no longer be maintained;[30]
- a plateau in $\dot{V}O_2$;[26]
- selected metabolic parameters, such as a rise in respiratory exchange ratio of >1.0,[7] 90% of age predicted peak heart rate (HR_{peak}),[41] HR >95% of age-related maximum (220 − age).[42]

Other test termination variables include various blood lactate and V_E values predetermined by the research design or established guidelines. For example, with regard to blood lactate, the British Association of Sport Sciences[43] guidelines propose that the $\dot{V}O_2$ measurement at exhaustion can be considered a maximal ($\dot{V}O_{2max}$) rather than a peak ($\dot{V}O_{2peak}$) value if the peak blood lactate concentration is >8 mmol/L. The question remains as to which criteria provide the most consistent and valid measure of the cyclist's exercise capacity.

4. Predictors of Cycling Potential

4.1 Characteristics of Road Cyclists

4.1.1 Maximal Oxygen Uptake ($\dot{V}O_{2max}$) and Metabolic Thresholds

The cyclist's potential for road-racing success might be measured from physiological values of elite professional riders. There is substantial evidence demonstrating that successful professional cyclists possess high $\dot{V}O_{2max}$ values (~74 mL/kg/min) and a LT2 that is ~90% of $\dot{V}O_{2max}$.[18,44-47] Padilla et al.[38] have grouped riders into their area of expertise, i.e. uphill riders, flat terrain riders, time-trial specialists and all terrain cyclists, and characterised their physiological attributes. Table II presents absolute and relative power output values at individual lactate thresholds.

A scaling of maximal aerobic power and $\dot{V}O_2$ values was performed using mass exponents of 0.32 to evaluate level of cycling ability and 0.79 to evaluate uphill cycling ability.[48] Frontal area was considered to be 18.5% of body surface area and $\dot{V}O_{2max}$ was estimated utilising the regression equation proposed by Hawley and Noakes[49] where $\dot{V}O_{2max} = 0.01141 \bullet W_{max} + 0.435$. W_{max} is defined at the highest work-load maintained for a complete 4-minute period. The LT1 was identified as the exercise intensity that elicited 1 mmol/L increase in

Table II. Absolute and relative power output values (mean ± SD) at the individual lactate threshold (reproduced from Padilla et al.,[38] with permission)

Variable	Flat terrain	Time trialist	All terrain	Uphill
W_{LT} (W)	356 ± 31	357 ± 41	322 ± 43	308 ± 46
W_{LT} (W/kg)	4.67 ± 0.25	5.0 ± 0.2	4.73 ± 0.48	4.91 ± 0.5
W_{LT} ($W/kg^{-0.32}$)	89.0 ± 6.7	91.0 ± 8.0	83.4 ± 10.0	81.0 ± 10.8
W_{LT} ($W/kg^{-0.79}$)	11.6 ± 0.69	12.25 ± 0.64	11.47 ± 1.23	11.71 ± 1.29
W_{LT} (W/m^{-2} FA)	962.5 ± 59.0	1009.7 ± 65.0	933.7 ± 110.2	940.7 ± 10.3
W_{LT} (%W_{max})	77 ± 2.0	78 ± 3.0	74 ± 7.0	76 ± 3.0

FA = frontal area; W_{LT} = power output at lactate threshold; W_{max} = maximal power output.

Table III. Characteristics of professional road-racing cyclists (reproduced from Padilla et al.,[38] with permission)

Variable	Mean ± SD	Range
Age (y)	26.0 ± 3.0	20–33
Height (cm)	180.0 ± 6.0	160–190
Mass (kg)	68.2 ± 6.6	53–80
W_{max} (W)	431.8 ± 42.6	349–525
W_{max} (W/kg)	6.34 ± 0.30	5.58–6.82
$\dot{V}O_{2max}$ (L/min)[a]	5.63 ± 0.47	4.42–6.42
$\dot{V}O_2$ (mL/kg/min)	78.8 ± 3.7	69.7–64.8
HR_{max} (beats/min)	192 ± 6.0	178–204
La_{max} (mmol/L)	9.8 ± 1.9	6.9–13.7

a $\dot{V}O_2$ was estimated.

HR_{max} = maximal heart rate; **La_{max}** = maximal blood lactate concentration; **$\dot{V}O_2$** = oxygen uptake; **$\dot{V}O_{2max}$** = maximal oxygen uptake; **W_{max}** = maximal power output.

lactate concentration above average lactate values measured when exercising at 40–60% of $\dot{V}O_{2max}$.[8] The LT2 was identified as the exercise intensity eliciting a lactate concentration of 4 mmol/L.[50] When the last work-load was not maintained for 4 full minutes, maximal power output (W_{max}) was calculated as follows.[51]

$$W_{max} = W_f + (t/240) \bullet 35$$

where: W_f = the value of the last complete work-load (W); t = the time the last work-load was maintained (seconds), and 35 = the power output difference between the last two work-loads (W) [tables III, IV and V].

Power output at lactate threshold has been shown to be a valid predictor (r = 0.88) of cycling potential (table VI).[44]

The validity of using the 4 mmol/L intensity determined during an incremental test as a predictor of the maximal steady-state intensity sustainable during prolonged exercise for all individuals has been questioned. Research data indicate that steady-state conditions at exercise intensities eliciting blood lactate values differ from the fixed 4 mmol/L value corresponding to LT2.[52,53] However, LT2 has been reported as the highest possible steady-state work intensity that can be maintained for a prolonged time and, therefore, is an excellent endurance index.[50,53,54] The LT2 of the cyclist who set a new world record in 1999 was 88.2% of his maximal aerobic power. Moreover, power output at blood lactate threshold has been shown to be an important variable for cycling performance.[16,44] As a predictor of performance, Baron[36] used a fixed lactate threshold of 4 mmol/L. In support of this finding, Coyle et al.[44] demonstrated that lactate threshold $\dot{V}O_2$ is a strong predictor (r = 0.96) of endurance performance among trained cyclists with similar maximal aerobic power. The success of tests performed and the related accomplishment of a new 1-hour world record reveal the existence of a close relationship between those measurements for cycling and future performance potential. When standardised environmental and equipment conditions are maintained, adequate models that integrate all major performance-determining variables are used and laboratory-based assumptions are verified in the field, cycling laboratory tests have a high predictive value.

4.1.2 Peak Power Output

There is substantial evidence demonstrating that W_{peak} obtained during a maximal incremental cycling test can be used as predictor of cycling performance.[16,49] Hawley and Noakes[49] report a significant correlation ($r = -0.91$; $p < 0.001$) between W_{max} during a graded exercise test and a 20km

Table IV. Group anthropometric characteristics (mean ± SD) [reproduced from Padilla et al.,[38] with permission]

Variable	Flat terrain	Time trialist	All terrain	Uphill
Age (y)	27 ± 3.0	28 ± 5.0	25 ± 2.0	25 ± 4.0
Height (cm)	186 ± 4.0	181 ± 6.0	180 ± 2.0	175 ± 7.0
Mass (kg)	76.2 ± 3.2	71.2 ± 6.0	68.0 ± 2.8	62.4 ± 4.4
BSA (m^2)	2.0 ± 0.06	1.91 ± 0.11	1.87 ± 0.04	1.75 ± 0.01
FA (m^2)	0.37 ± 0.0011	0.353 ± 0.02	0.345 ± 0.008	0.326 ± 0.019
BSA-BM ($^{-1} \bullet 10^{-3}$)	26.26 ± 0.48	26.82 ± 0.73	27.44 ± 0.53	28.27 ± 0.49
FA-BM ($^{-1} \bullet 10^{-3}$)	4.86 ± 0.09	4.96 ± 0.13	5.07 ± 0.10	5.23 ± 0.09

BM = body mass; **BSA** = body surface area; **FA** = frontal area.

Table V. Absolute and relative power output values at the individual lactate threshold (mean ± SD) [reproduced from Padilla et al.,[38] with permission]

Variable	Flat terrain	Time trialist	All terrain	Uphill
W_{LT} (W)	356 ± 31	357 ± 41	322 ± 43	308 ± 46
W_{LT} (W/kg)	4.67 ± 0.25	5.0 ± 0.2	4.73 ± 0.48	4.91 ± 0.5
W_{LT} (W/kg$^{-0.32}$)	89.0 ± 6.7	91.0 ± 8.0	83.4 ± 10.0	81.0 ± 10.8
W_{LT} (W/kg$^{-0.79}$)	11.6 ± 0.69	12.25 ± 0.64	11.47 ± 1.23	11.71 ± 1.29
W_{LT} (W/m^{-2} FA)	962.5 ± 59.0	1009.7 ± 65.0	933.7 ± 110.2	940.7 ± 10.3
W_{LT} (%W_{max})	77 ± 2.0	78 ± 3.0	74 ± 7.0	76 ± 3.0

FA = frontal area; $\mathbf{W}_{LT}$ = power output at lactate threshold; $\mathbf{W}_{max}$ = maximal power output.

cycle trial. These investigators concluded that W_{max} may be a good parameter for assessment of cycling performance. Test data reveal that W_{peak} measured during a maximal incremental cycle test can be used as a predictor of performance in endurance cyclists.[16,49] A power/weight ratio of >5.5 W/kg is considered a necessary prerequisite for top-level competitive cyclists.[55] However, this criterion must used with caution as the protocol used during testing can affect the outcome of power output, thus further reinforcing the need for a common protocol.

4.1.3 Efficiency

Recent information indicates that in professional cyclists the rate of the $\dot{V}O_2$ rise, elicited by graded exercise, decreases at moderate to high work-loads to the maximal attainable power output.[24] More importantly, these authors observed that mechanical efficiency seemed to increase with rising exercise intensity. These findings reveal that professional road cyclists acquire a high cycling efficiency allowing them to sustain extremely high work-loads for extended periods of time. To that end, professional racers exhibit considerable resistance to fatigue of recruited motor units at high submaximal intensities.[46,56] The years of training and mileage required to achieve this characteristic is unknown. Nonetheless, professional riders generally cycle ~35 000 km/year and compete ~90 days.[24]

This training volume impacts the cyclist's efficiency. The professional cyclist's efficiency during heavy exercise appears to be positively related to the percentage of type I fibres in the vastus lateralis muscle.[57] A higher proportion of type I fibres in the muscle is associated with a lower submaximal oxygen cost, thus a greater gross efficiency.[58] This efficiency is a reflection of the increase in aerobic metabolism and related increases in muscle power output.[34]

4.1.4 Breathing Pattern

The cyclist's breathing pattern appears to have some influence on performance. Professional riders exhibit a unique breathing pattern at high work-loads characterised by a lack of tachypnoeic shift, that is they continue to increase V_E through increasing tidal volume versus breathe frequency.[59] It is suggested that this breathing adaptation may enhance the efficiency and metabolic cost of breathing and partly account for the $\dot{V}O_2$ kinetics exhibited by professional cyclists. Nevertheless, the oxygen cost of breathing in highly fit individuals has been esti-

Table VI. Absolute and relative power output values at the onset of blood lactate accumulation (W_{OBLA}) [mean ± SD; reproduced from Padilla et al.,[38] with permission]

Variable	Flat terrain	Time trialists	All terrain	Uphill
W_{OBLA} (W)	417 ± 45.0	409 ± 46.0	366 ± 38.0	356 ± 41.0
W_{OBLA} (W/kg)	5.46 ± 0.42	5.73 ± 0.21	5.37 ± 0.37	5.70 ± 0.46
W_{OBLA} (W/kg$^{-0.32}$)	104.1 ± 10.3	104 ± 8.9	94.8 ± 8.7	94.8 ± 9.6
W_{OBLA} (W/kg$^{-0.79}$)	13.57 ± 1.10	14.03 ± 0.69	13.01 ± 0.99	13.57 ± 1.14
W_{OBLA} (W/m^{-2} FA)	1125.8 ± 100.3	1156.8 ± 70.0	1061.0 ± 91.0	1090.4 ± 88.0
W_{OBLA} (%W_{max})	90 ± 3.0	89 ± 2.0	84 ± 5.0	88.5 ± 5.0

FA = frontal area; $\mathbf{W}_{max}$ = maximal power output.

Table VII. Lactate threshold responses of National Off-Road Bicycle Association (NORBA) and United States Cycling Federation (USCF) cyclists (mean ± SD) [reproduced from Wilber et al.,[61] with permission]

	Female		Male	
Variable	NORBA	USCF	NORBA	USCF
$\dot{V}O_2$ (mL/kg/min)	48.4 ± 3.0	53.3 ± 3.8	53.9 ± 4.6	56.4 ± 4.4
$\dot{V}O_{2max}$ (%)	83.8 ± 5.6	83.6 ± 2.7	77.1 ± 6.4	80.1 ± 3.2
Lactate (mmol/L)	2.6 ± 0.7	3.0 ± 0.6	2.9 ± 1.1	2.7 ± 0.4
HR (beats/min)	155 ± 8.0	165 ± 12.0	166 ± 13.0	169 ± 13
HR_{max} (%)	87.2 ± 2.7	87.9 ± 2.5	86.4 ± 4.2	84.9 ± 4.3
Power (W)	204 ± 20	224 ± 8.0	271 ± 29.0	321 ± 17
Power (W/kg)	3.6 ± 0.3	3.7 ± 0.3	3.8 ± 0.3	4.4 ± 0.3

HR = heart rate; **HR_{max}** = maximal heart rate; **$\dot{V}O_2$** = oxygen uptake; **$\dot{V}O_{2max}$** = maximal oxygen uptake.

mated to be ~15% of $\dot{V}O_{2max}$.[60] The work of breathing during heavy exercise compromises leg blood flow to working limb muscle.[60] Consequently, a more efficient breathing pattern may not reduce blood flow to the working muscles.

4.2 Characteristics of Off-Road Cyclists

Table VII summarises the physiological responses for male and female riders from the National Off-Road Bicycle Association and the United States Cycling Federation.[61] These data reveal that, in general, elite off-road cyclists possess physiological profiles that are similar to those of elite road cyclists. However, males tend to have higher power outputs. It should be noted that the lactate threshold of elite off-road male cyclists has been determined to correspond to 77% $\dot{V}O_{2max}$ in laboratory conditions.[27]

4.3 Use of Blood Lactate Levels

Blood lactate concentration at various cycling exercise intensities is highly predictive of endurance performance making its measurement valuable for evaluating future performance.[9,44,62,63] Especially meaningful is the MLSS, the highest exercise intensity at which blood lactate concentration remains stable, which reflects a balance between lactate production and removal.[9] Trained cyclists have been observed to reach MLSS at an intensity equivalent to 90% of their average simulated 5km time-trial speed.[32] Harnish et al.[19] demonstrated that MLSS may be estimated non-invasively, within a 2% error, utilising a 5km and 40km cycling time trial. The 40km was found by these authors to be the most convenient method, limiting testing to a single session.

Furthermore, Bishop et al.[17] have shown that lactate parameters and W_{peak} provide better predictors of endurance performance than $\dot{V}O_{2max}$ in trained female cyclists. W_{peak} was found particularly useful for its predictive value of 1-hour cycling performance. The importance of this finding is that W_{peak} discovery does not require the measurement of $\dot{V}O_2$ or lactate. However, if lactate measurement is available, it is a valuable measure for prescription of training intensity.[64,65]

5. $\dot{V}O_{2max}$ and Metabolic Thresholds

$\dot{V}O_{2max}$ is said to be set by metabolic and oxygen transport limits or a combination of both. There is considerable evidence documenting the contention that $\dot{V}O_{2max}$ has limited predictive value for performance in homogeneous groups of high-performance athletes.[54] Nonetheless, $\dot{V}O_{2max}$ measurement remains recommended for the purpose of evaluating and selecting elite cyclists and as a prerequisite to perform at a high level.[66,67] Furthermore, to some extent it appears that depressed $\dot{V}O_{2max}$ values may be indicative of fatigue or overtraining than actual training progress.

There is substantial evidence documenting the high $\dot{V}O_{2max}$ of road-racing cyclists.[18,38,68] Moreover, Pfeiffer et al.[69] have demonstrated $\dot{V}O_{2max}$ is a strong predictor ($r = -0.91$) of cycling performance in a 14-day stage race among trained female cyclists. For males, the mean $\dot{V}O_{2max}$ during the Tour de

France and Vuelta a España was found to be 73.5 mL/kg/min and the LT2 was observed to be 90% of $\dot{V}O_{2max}$.[18] Thus, it is not surprising that Lucía et al.[70] reported leading time realists of the Giro d'Italia, Tour de France, or Vuelta a España stage races were able to tolerate high submaximal constant work-loads close to their VT_2 or ~90% $\dot{V}O_{2max}$ for ~60 minutes. A high VT_2 is an advantage to the cyclists since mountain stage climbs lasting 30–60 minutes demand the cyclist work close to VT_2 or onset of blood lactate.[38,59] Table VIII presents the $\dot{V}O_{2max}$ values recorded for various levels of male and female road and off-road-racing cyclists. These data are useful in the selection and subsequent classification of cyclists.

Table VIII. Maximal oxygen uptake ($\dot{V}O_{2max}$) of road and off-road male and female cyclists

Study	Cycling level	$\dot{V}O_{2max}$ (mL/kg/min)
Male		
Fernández-García et al.[18]	Professional	73.7
Lucía et al.[34]	Professional	72.0
Lucía et al.[34]	Professional	71.3
Lacour et al.[71]	Professional	70.1
Padilla et al.[38]	Professional	78.8
Sjogaard[72]	Professional	71.0
Terrados et al.[73]	Professional	70.0
Gnehm et al.[74]	Elite	69.4
Saltin and Astrand[75]	Elite	74.0
Burke et al.[76]	Elite	67.1
Hermansen[77]	Elite	73.0
Burke[78]	Elite	74.0
Coyle et al.[44]	Elite	69.1
Stromme et al.[79]	Elite	69.1
Wilber et al.[61]	Elite (off-road)	79.3
Wilber et al.[61]	Elite	70.0
Faria[68]	Elite	68.0
Faria et al.[45]	Elite	75.5
Lindsay et al.[23]	Amateur	65.7
Impellizzeri et al.[80]	Amateur	75.9
Lucía et al.[24]	Amateur	69.5
Padilla et al.[81]	Amateur	66.1
Palmer et al.[55]	Amateur	66.7
Palmer et al.[82]	Amateur	73.6
Tanaka et al.[83]	Amateur	69.4
Hopkins and McKenzie[84]	Amateur	68.0
Females		
Wilber et al.[61]	Elite (off-road)	57.9
Wilber et al.[61]	Elite	68.0

Although not to be dismissed as an important quality for endurance cycling performance, $\dot{V}O_{2max}$ alone is not a good predictor of endurance performance when athletes of similar endurance ability are compared.[85] The mechanisms that underlie why lactate parameters provide a better predictor of endurance performance than $\dot{V}O_{2max}$ continue to be examined. The argument has been presented that $\dot{V}O_{2max}$ is limited by the oxygen supply to the muscle mitochondria.[86,87] However, lactate levels are related to the capacity to transport lactate and hydrogen ion or proton (H^+) out of the muscle fibres, and the capacity of skeletal muscle to take up lactate.

Central factors are likely to limit $\dot{V}O_{2max}$, while the lactate response to exercise is primarily related to peripheral factors in the trained musculature. These factors include the percentage of slow-twitch fibres, the activities of key oxidative enzymes and respiratory capacity.[50,88-91]

6. Anaerobic Power

The attainment of very high power output during 30 seconds of cycle sprinting is derived from the anaerobic sources of phosphocreatine (PCr) degradation and glycogenolysis ending in lactate production. The most frequently used test to describe anaerobic power and capacity is the Wingate test.[92] At the onset of sprinting, both the phosphagen and glycolytic systems are fully activated.[93-95] Accelerated glycolysis, PCr degradation and oxidative metabolism provide approximately 50–55%, 23–29% and 16–25%, respectively, of the adenosine triphosphate (ATP) required by the working muscle during a 30-second sprint.[5,96-99] Furthermore, resynthesis of ATP to ~80–100% of resting value requires 2–4 minutes of recovery.[100-102]

For the competitive cyclists, these facts serve as the foundation for sprinting strategy. Initiation of the sprint to the finish too soon will result in a gradual reduction of speed and most certainly a loss of first place. This notion is supported by the fact that PCr degradation begins at the onset of intense

exercise and reaches a maximal rate within 10 seconds, and then ceases to contribute to energy provision as the PCr store depletes sometime between 10 and 30 seconds.[5,93-95,103]

7. Metabolic Acidosis

It is apparent that when cycling at increasing power outputs, lactate accumulates in the vastus lateralis muscle. However, while the mechanisms responsible for its production, accumulation, use and disposal have been well investigated, they are misinterpreted by many athletes, coaches and some scientists. More importantly, there is a need to dispel the myths of 'lactic acid' so often voiced among cycling coaches and athletes. Included among the misnomers are such statements as: "lactic acid is the primary factor in muscle soreness"; "lactic acid is the central cause of oxygen debt"; "lactic acid is the causative agent in muscle fatigue"; "lactic acid is the immediate energy donor for muscle contraction"; and "lactic acid is a dead-end waste product".

Most texts on physiology and biochemistry clearly demonstrate the importance of the lactate reaction in maintaining cytosolic redox (oxidation-reduction reaction) and allowing glycolysis to continue during intensive exercise. Furthermore, Donovan and Brooks[104] were able to demonstrate the significance of lactate as a gluconeogenic substrate during exercise. The most striking finding was the observation that well trained individuals were better able to maintain blood glucose levels through the gluconeogenesis of lactate than were untrained individuals. Recall that gluconeogenesis is the synthesis of glucose from non-carbohydrate precursors such as glycerol, ketoacids or amino acids. While many have come to accept these positive facets of lactate metabolism, some individuals continue to believe that these benefits come at the cost of increasing acidosis whose genesis is 'lactic acid'.

Nevertheless, there is no biochemical evidence to support the belief that lactate production during exercise releases a proton and causes acidosis.[4] A review and comments by Robergs et al.[4] clearly identifies that the intermediates of glycolysis are protonated in a physiological pH (measure of acidity/alkalinity) where 'lactic acid' would exist as lactate and not 'lactic acid'. Moreover, the main form of 'lactic acid' in physiological systems is sodium lactate.[4] Rather, Robergs et al.[4] suggests an increased reliance on non-mitochondrial ATP turnover as the potential source of proton production that is associated with increased lactate creation and development of acidosis. Furthermore, when cell buffering capacity is exceeded, acidosis develops. Clearly, there are plausible mechanisms which explain that 'lactic acid' is not the likely source of protons during exercise. In this regard, Robergs et al.[4] points to the evidence that lactate serves to lower hydrogen ion concentrations rather than raise them. The conversion of pyruvate to lactate not only oxidises the reduced form of nicotinamide-adenine dinucleotide (NADH) but also utilises an H^+ from solution to attach to the middle carbon, thereby serving to lower the H^+ concentration rather than contribute to its increase. In a recent investigation of professional cyclists (exercising at 90% $\dot{V}O_{2max}$, 2 × 6-minute sessions), Santalla et al.[105] demonstrated that despite a pH indicative of acidosis, there was no apparent acidosis-induced impairment in skeletal muscle function in the athletes. These results also suggest, indirectly, that the efficiency of muscle contraction was not altered significantly and adds further support that lactate production retards rather than contributes to acidosis.

It should be evident that the production of lactate should not be viewed as a negative facet of increasing exercise intensity. Every aspect of lactate production is beneficial. Lactate production serves to maintain cytosolic redox, make new glucose, consume H^+ from the cytosol, as well as allow transport of H^+ from the cell. All of these reactions are advantageous to the exercise response. Therefore, it is better to conclude that high levels of lactate are not detrimental to cycling performance.

Moreover, the cause of acidosis is not the result of non-steady exercise intensity.[4] While an increased ability to produce and remove lactate from the cell helps delay the onset of acidosis, lactate production retards, not worsens acidosis.[4] For the competitive cyclist, this means that for a given

$\dot{V}O_2$ during intense cycling, high lactate production is beneficial. Its production is even more beneficial if accompanied by a high capacity for lactate and proton transport from the cell. Both factors are known to increase with endurance and sprint training.[106]

While lactate production does not cause the acidosis it remains an important indirect indicator of an adjustment in metabolism causing acidosis. High lactate production may be due to contributions of anaerobic glycogen and glucose metabolism[107] or due to aerobic overproduction of pyruvate and subsequent conversion to lactate.[108] When oxygen is non-limiting, the higher the exercise intensity the higher the rate of muscle production and release of lactate.[109]

Muscle fibre type recruitment also has implications for the onset of metabolic acidosis. As discussed in the accompanying article,[110] high pedal cadences with reduction of force to the pedal, reduce the force used per pedal stroke. Consequently, muscle fatigue is reduced in type II fibres. However, when pedal cadence is increased without a reduction in force to the pedal or a harder gear is employed, type II muscle fibres become progressively recruited. Type II muscle fibres have a lower mitochondrial density than type I fibres and therefore are more dependent on glycolysis and cytosolic ATP turnover.[4] Combined, these two metabolic characteristics result in an increased rate of proton release from catabolism resulting in substantial proton production when cycling intensity calls upon increased type II muscle fibre recruitment. Consequently, type II fibres will then substantially contribute to acidosis because they have less mitochondrion mass to facilitate ATP regeneration, and the uptake of protons.[4] Accordingly, it is important to recognise their contribution to acidosis is not because type II fibres produce more lactate. For a comprehensive discussion of metabolic acidosis the reader is encouraged to read the paper by Robergs et al.[4]

8. Fatigue

Muscle fatigue may be defined as the failure to maintain a required or expected power output.[111] Evidence has been provided that subjects who possess a high percentage of fast-twitch fibres are more sensitive to fatigue than those with more slow fibres.[112] Repeated sprint cycling results in the fatigue of the gluteus maximus and vastus lateralis muscles. When fatigued, these monoarticular muscles produce less force and power. Two types of fatigue are evident: (i) peripheral fatigue where there is an increase in the EMG/force ratio; and (ii) central fatigue characterised by a constant EMG/force ratio associated with a force decline.[113] There occurs a decrease in the efficiency of the EMG signal of the power producing muscles. However, simultaneously lower activation of antagonist muscles intervenes, thereby allowing the cyclist to efficiently transfer force and power to the pedal.

Prolonged cycling exercise impairs muscle strength capacity associated with changes in contractile and neural properties of leg extensors. There appears to be no relationship between fibre type recruitment pattern and neuromuscular fatigue and subsequent reduction of strength during cycling exercise. Rather, a central component of fatigue exists. Deficits of muscular activation are not significantly different between cadences. Lepers et al.[22] found that at the highest pedalling rate, the central neural input to the vastus medialis and vastus lateralis muscles remained unchanged. Additionally, the findings suggest that the central drive is less altered when a high (69–103 rpm) pedal rate is used. It is interesting to note two important findings of these authors: (i) central drive is less lathered when a high cadence is used; and (ii) freely chosen cadence does not minimise the effects of fatigue on subsequent leg extensors' strength capacities. In practice, this means that a combination of different high cadences may be used effectively.

There is some evidence showing possible mechanisms of CNS fatigue during prolonged exercise.[114] More specifically, increases in serotonin (5-HT) or a depletion in catecholamines during exercise have been examined as possible contributors to early onset of fatigue.[115,116] However, Piacentini et al.[117] demonstrated that a 90-minute cycling time-trial performance was not influenced by a noradrenergic

reuptake inhibitor. Furthermore, the ingestion of 5-hydroxytryptophan (5 mg/kg), a serotonin precursor, had no effect on prolonged cycling performance at 65% $\dot{V}O_{2max}$ followed by a graded effort to fatigue.[118] It appears that the 'central component' of fatigue is not easily linked to increase or decrease of neurotransmitters.

Little has been said concerning fatigue avoidance, especially during stage racing. In this regard, it is hypothesised that there exists a learned subconscious anticipatory/regulation system, known as 'teleoanticipation' originating from the CNS.[119] This subconscious feedback mechanism serves to decrease efferent output from the motor cortex. Prior to the beginning of a given competition it is hypothesised that the cyclist's CNS is aware of the rider's fitness level, endurance capacity and limitations as gained from previous similar competitions. The total exercise load and time the cyclist's body can tolerate the given metabolic level is known. Having this information there occurs, at the subconscious level, the exercise load limit in order to avoid premature fatigue prior to the conclusion of the event. This is accomplished by a 'central programmer' that sets the tolerable upper limits for the total competitive loads.[120] It is postulated that the limitation is accomplished through a decreased efferent output from the motor cortex. This theoretical mechanism may be akin to the 'central governor' hypothesis where it is theorised that a central, neural governor constrains the cardiac output by regulating the mass of skeletal muscle that can be activated as metabolic limits are approached.[121] Operationally, the process might be viewed as 'pacing' in order to complete the near impossible physical challenges encountered during a 3-week stage racing, 161km (100 mile) endurance run or climbing the highest mountain peaks of the world. In this respect, Lucía et al.[122] observed that during the Tour de France and Vuelta a España, cyclists never achieved their maximal exercise limits for two consecutive days. Their subconscious was 'well aware' of thresholds to avoid in order to realise success another day.

9. Pulmonary Limitations

Respiratory muscles may reach the limit of their capacity during exercise of high intensity or long duration. Research data suggest that inspiratory muscles present a potential site of exercise performance limitation.[29] An elevated acid milieu of respiratory muscles or competition for blood flow between locomotor muscles and the respiratory muscles may contribute to inspiratory muscle fatigue following high-intensity exercise.[123] Romer et al.[29] observed a decrease in maximum inspiratory flow rate after both a 20km and a 40km cycling time trial. Furthermore, inspiratory muscle fatigue slows the relaxation rate needed for muscle recovery.[124]

It appears that endurance training fails to provide the necessary training stimulus to strengthen the inspiratory muscles, therefore, a specific inspiratory muscle training protocol may be wise. Attenuation of exercise-induced inspiratory muscle fatigue and recovery time may be accomplished through specific inspiratory muscle training.[29] Inspiratory muscle training incorporating 30 dynamic inspiratory efforts twice daily for 6 weeks against a pressure-threshold load equivalent to ~50% maximum inspiratory mouth pressure (P_O), has been shown to be an effective training stimulus.[29,125] However, improvements in respiratory muscle function do not appear to be transferable to $\dot{V}O_{2max}$ or endurance capacity in competitive athletes.[126]

10. Training

10.1 Monitoring Training

Numerous strategies have been presented for monitoring the training status of competitive cyclists in order to precisely evaluate training methods and their efficacy during a training and competitive season. The magnitude of dependent variable reaction as subsequent alterations in response to training is remarkably varied. No significant change in $\dot{V}O_{2max}$ during a complete training and competitive season is evident.[127-130] However, this variable may increase by about 20% with ranges between 10–40% for athletes having pretraining $\dot{V}O_{2max}$ values be-

tween 40 and 50 mL/kg/min.[67] $\dot{V}O_2$, V_E, mechanical efficiency and HR at VT remain stable relative to pedal speed throughout the training and competitive season. In contrast, submaximal exercise parameters are more sensitive to training and yield valuable information regarding the training status.[131] More importantly, these work-loads correspond to work-loads encountered during racing. Clearly, these findings suggest in experienced cyclists peripheral adaptations in working muscles play a more important role for enhanced submaximal working capacity than central adaptations.

VT appears to be an indicator of endurance performance as $\dot{V}O_2$, mechanical efficiency and HR at the VT remain stable. Training adaptations that have been observed include the interaction between $\dot{V}O_2$, HR, ventilation, and mechanical efficiency at work-loads of 200W and 250W.[67] Interestingly, these work-loads correspond to work-loads encountered during racing.

The training impulse (TRIMP), computed from both exercise HR and duration, is used as an integrative marker of exercise load undertaken by the cyclist during a training or competition bout.[132-136] When used to characterise exercise intensity and load during competition it may subsequently be used effectively to establish appropriate training criteria.

$$TRIMP = A \bullet B \bullet C$$

where: A = competition time (in minutes); $B = [(HR_T - HR_B)/(HR_{max} - HR_B)$; $C = 0.64 . e^{1.92B}$; e = Napiarian logarithm having a value of 2.712; HR_T = average HR during competition; and HR_B = basal HR.

The physiological load of the extremely high exercise demands of 3-week professional cycling competition tours has been characterised by employing the concept of the TRIMP. Its application provides an analysis of the exercise volume.[122] In doing so, Foster et al.'s[137] approach to the concept of the TRIMP,[132,133] which integrates the exercise volume (i.e. total exercise time in minutes) and intensity (i.e. total time spent in each of the three phases) in the three phases was employed. HR data were continuously collected via telemetry during daily stages. Exercise intensity was referenced by HR = VT, respiratory compensation point (RCP), $\%\dot{V}O_{2max}$ and classified accordingly by phases:

- phase I ('light intensity', <VT or approximately <70% $\dot{V}O_{2max}$);
- phase II ('moderate intensity', between VT and RCP, or approximately 70–90% $\dot{V}O_{2max}$);
- phase III ('high intensity', < RCP or approximately <90% $\dot{V}O_{2max}$);[133,138]

(HR = VT, RCP, $\%\dot{V}O_{2max}$ are related to pre-tour metabolic data collected during a ramp protocol cycling test).

The exercise volume or load score in each phase was computed by multiplying the accumulated duration in the phase by multipliers for the phase:

- 1 minute in phase I = 1 TRIMP (1 × 1)
- 1 minute in phase II = 2 TRIMP (1 × 2)
- 1 minute in phase III = 3 TRIMP (1 × 3)

The total TRIMP score was obtained by summing the results of the three phases. Lucía et al.[122] reports that the upper tolerable limits for total loads (volume × intensity) set by the hypothetical 'central programmer' is ~ 6000 TRIMP for mass start stages, 250 TRIMP for individual time trials of 55–70km and ~8300 TRIMP for the entire 3-week period.

Schabort et al.[30] utilising a Kingcycle ergometer system that allows the subjects to use their own cycle, devised a test of cycling performance that simulates the variable power demands of competitive road racing. Beginning with a 5-minute warm-up, the cyclists commence a 100km time trial incorporating 1km sprints after 10, 32, 52 and 72km, and four 4km sprints after 20, 40, 60 and 80km. The object is to complete the total distance in the fastest time possible. Throughout the test the cyclist is free to regulate the power output. Feedback to the subjects includes elapsed distance and HR. This test serves to evaluate the effects of training strategies and may well serve to predict the cyclist's performance. An important finding of this research was that data are more reliable when subjects are allowed to freely choose their effort during testing than when they exercise to exhaustion at a constant work-load.[30]

The laboratory-simulated 40km time trial has shown to be highly reproducible in well trained

cyclists.[82] Hence, such a laboratory test may serve as an indicator of training protocol efficacy. In this regard, Lindsay et al.[23] have shown that 4 weeks of high-intensity interval training significantly improved the cycling performances of highly trained cyclists in laboratory tests ranging from ~60 seconds to ~1 hour. On six occasions during a 28-day period, the cyclists performed 6–8 repetitions of high-intensity interval training. High-intensity training was shown to result in significant improvements in 40km time trial, W_{peak} and muscular resistance to fatigue.

In summary, no significant change in $\dot{V}O_{2max}$ during a complete training and competitive season is evident. Likewise, $\dot{V}O_2$, V_E, mechanical efficiency and HR at VT remain stable relative to pedal speed throughout the training and competitive season. Rather, submaximal exercise parameters are more sensitive to training and peripheral adaptations in working muscles play a more important role for enhanced submaximal working capacity than central adaptations. The TRIMP, computed from both exercise HR and duration, is useful as an integrative marker of exercise load undertaken by the cyclist during a training or competition bout. When evaluating cycling performance, if subjects are allowed to freely choose their effort during testing, data are more reliable than when they exercise to exhaustion at a constant work-load. The laboratory-simulated 40km time-trial test may serve as an indicator of training protocol efficacy.

10.2 Determination of Training Parameters

Lucía et al.[70] provide substantial evidence demonstrating that a single laboratory testing session at the beginning of the cycling season may be sufficient to adequately prescribe training loads based on HR. In this respect, the authors found that mean HR values corresponding to the lactate threshold (LA), first ventilatory (VT_1) [author used at least 0.2 mmol/L] and second ventilatory (VT_2) threshold remained stable throughout the training season despite a significant improvement in performance and shifts in LA, VT_1 and VT_2 toward high work-loads. Use of the SRM training system to measure power output, and a ramp test protocol (employing gradual continuous work-load increases of 5 W/12 seconds or 25 W/min) should provide sufficient data to permit establishment of training protocols corresponding to low-, moderate- and high-intensity training. For example, low intensity would equal $<VT_1$, moderate intensity between VT_1 and VT_2, and high intensity $>VT_2$.

While the most accurate method to determine the MLSS remains the measurement of the athlete's blood lactate response to changes in work intensity, a non-invasive estimation procedure may serve as an alternative.[19,36] To that end, many studies show that training at MLSS is beneficial.[13,50,64,90,139-141] A 5km time trial, on a windload simulator, has been shown to be an effective and valid means to estimate MLSS and HR at MLSS for the purpose of training. The cyclist first performs a maximal 5km time trial on a windload simulator. Subsequently, following complete recovery several days later, a 30-minute MLSS trial is performed on the windload simulator riding at a velocity that is approximately 90% of the average 5km velocity. The mean HR attained during the last 20 minutes of the MLSS trial is used as the MLSS training HR. Whether training at MLSS or using intermittent and steady-state training at a power output corresponding to the anaerobic threshold is more efficient remains unresolved.

10.3 Training Volume

Professional road-racing cyclists train over distances of approximately 30 000–35 000km per year.[18] Distances covered during multi-stage road races range between 5km to almost 300km. Vuelta a España, a 22-day stage race covers ~3725km, of which ~3635km is in-line competition (IL) and ~89km are individual time-trial (ITT) stages. Likewise, the Tour de France multi-stage race covers ~3899km, of which ~3796km are IL competition and ~103 km ITT.

The training protocol employed by the German national 4000m pursuit team during the last 19 days prior to the 2000 Olympic Games is listed in table IX.[142] In this training programme, the 'basic training' is aerobic training. The intensities are determined during an incremental cycling test on ergom-

Table IX. Training protocol employed by the German national 4000m pursuit team during the last 19 days prior to the 2000 Olympic Games[142]

No. of days to competition	Training
15–19	Stage race
14	Rest day
13	115km basic training
12	120km basic training
11	115km basic training
10	120km basic training
9	Rest day
8	Track training 3 × 5000m evolution training
7	Morning 3 × 5000m evolution training
	Afternoon 4 × 5000m evolution training
6	2 × 5000m evolution training
	1 × 2000m, 1 × 1000m 'peak' training
5	75m basic training/recovery (road)
4	Morning 2 × 5000m evolution training
	Afternoon 1h basic training (road)
3	3 × 5000m evolution training
	2 × 2000m peak training
2	75m basic training/recovery (road)
1	2 × 5000m evolution training

eter using the HR at the individual anaerobic threshold (IAS) as the main marker. Basic training ranges between HR (IAS) – 50 beats/min and HR (IAS) – 30 beats/min. For example, an athlete with an HR of 165 beats/min at IAS should perform his basic training at intensities between 115 and 135 beats/min. This training is aimed at pure aerobic adaptation. Training units last from 2 to 7 hours (60–240km) and are usually performed on the road (Schumacher YO, personal communication).

Furthermore, 'evolution training' is calculated in a similar way: HR (IAS) – 5 beats/min and HR at (IAS) + 5 beats/min. (160–170 beats/min in the above-mentioned example). It ranges around the IAS and therefore aims at improving lactate tolerance. Evolution training is usually performed after a period of basic training, closer to the competition. Intervals with or without full recovery (intensive/extensive) are usually used for this type of training. The length is between 3 and 10km or 3 and 15 minutes. Training can be performed on the road or discipline-specific on the track or mountain-bike training. These training zones are used for all cycling disciplines within the national team (road, track, mountain-bike training). For riders using the SRM system, these zones are given in watts (Schumacher YO, personal communication).

10.4 Interval Training

Cyclists traditionally use increases in training volume to induce an overload in the training stimulus. When a rise in volume no longer augments fitness, cyclists employ intervals to intensify their training load. Hawley et al.[143] have suggested that following the base training period, endurance athletes undergo a period of transition training, which entails the use of 'pace' type intervals. Accordingly, when high-intensity intermittent training is adopted, the selection of the most effective work and rest ratios of the exercise bouts must be addressed.

Toward that end, MacDougal et al.,[144] examined the effects of cycling sprint interval training on muscle glycolytic and oxidative enzyme activity and exercise performance. Training consisted of 30-second maximum sprint efforts (Wingate protocol) interspersed by 2–4 minutes of recovery, performed three times per week for 7 weeks. The training programme began with four intervals with 4 minutes of recovery per session in week 1 and progressed to ten intervals with 2.5 minutes of recovery per session by week 7. This training strategy resulted in significant increases in W_{peak}, total work over 30 seconds and $\dot{V}O_{2max}$. Moreover, maximal enzyme activity, sampled from the vastus lateralis, of hexokinase, phosphofructokinase, citrate synthase, succinate dehydrogenase and malate dehydrogenase was also significantly higher following training. These findings clearly demonstrate that relatively brief but intense sprint training can enhance both glycolytic and oxidative enzyme activity, maximum short-term power output and $\dot{V}O_{2max}$. Tabata et al.[145] found that a high-intensity intermittent training programme achieved bigger gains in $\dot{V}O_{2max}$ (+9%) than a programme of 60 minutes of moderate-intensity cycling (70% $\dot{V}O_{2max}$) for a total of 5 hours per week for 6 weeks. The short-term, high-intensity training sessions consisted of eight all-out work bouts, each lasting 20 seconds, with 10

seconds of rest. This group cycled for a total of only 20 minutes per week, yet their $\dot{V}O_{2max}$ improved by 15%.

More specifically and performance applied, the effect of transition training on trained cyclists laboratory-simulated 40km time-trial performance and power output was examined by Lindsay et al.[23] and Stepto et al.,[146] employing similar protocols of 6–8 intervals of 4–5 minutes separated by 1–1.5 minutes at an intensity of 80% to 85% of W_{peak} for 3–4 weeks. The cyclists in these studies replaced approximately 15% of their normal training with one of the interval exercise protocols. Given the relatively short duration of these training protocols, these investigators produced rather impressive increases in W_{peak} of ~3%[31] and ~5%,[23] and improved 40km time-trial velocity by ~3%[23] and 2.8%.[31] A notable variation in the interval training protocol of Stepto et al.[146] allowed an examination of interval training protocols ranging in time from 0.5 to 8.0 minutes. Interestingly, these authors found that 30-second work bouts at 175% of peak power followed by 4.5 minutes of recovery and repeated 12 times was nearly as effective for improving 40km time-trial performance (+2.4% estimated from cubic trend)[146] as the 4-minute interval. It was shown that following the high-intensity training, both absolute and relative intensity during the time trial were improved. The observed increase in absolute intensity is not surprising since W_{peak} was increased. It appears the increase in relative intensity is suggestive of an increase in lactate threshold. The mechanisms that underlie the efficiency of this short-term very high-intensity training may include enhanced ability to sustain, for a longer period of time, a higher fraction of $\dot{V}O_{2max}$ or power output due to improvement in muscle buffering capacity. The data herein demonstrate that very short high-intensity work bouts also enhance endurance performance.

Furthermore, the 4-minute work bouts at 85% peak power with a rest interval of 1.5 minutes repeated eight times, demonstrated the best result for improved W_{peak} of ~3%.[146] However, there was no significant correlation found between the change in peak power and 40km time. This lack of correlation is difficult to explain since the single best predictor of a 40km time-trial performance is the average amount of power output that can be maintained in 1 hour.[44] This predictor represents 89% of the variance in time-trial performance.[48] The authors[146] suggest that the lack of correlation may be the result of interval training producing different responses between individuals in 40km time-trial performance and peak power. These curious findings call for further research.

In support of high-intensity, short-term intervals, Rodas et al.,[147] reported that a high-intensity intermittent training programme increases oxidative enzyme activity in muscle. It was shown that 2 weeks of daily high-intensity intermittent training consisting of two 15-second all-out bouts separated by 45 seconds of rest, followed by two bouts of 30-second all-out sprints separated by 12 minutes of rest resulted in ~10% increase in $\dot{V}O_{2max}$, 38% increase in activity of citrate synthase and a 60% activity increase in 3-hydroxyacyl-coenzyme A (CoA) dehydrogenase. The oxidative enzyme activity modification could enhance the rate of fat oxidation and reduce carbohydrate oxidation. Furthermore, subsequent decline in the accrual of hydrogen irons may augment endurance performance.[143]

Similarly, Hawley et al.[143] investigated the question of intensity and duration of interval training. They found a plateau in both W_{max} and 40km time-trial speed with six sessions, suggesting the transition phase of training only be carried out until these six sessions have been completed. Following the transition period, Hawley et al.[143] suggest a speed/power training phase. However, the training during this phase has not been clearly investigated.

Laursen and Jenkins[21] examined interval optimisation in trained cyclists by engaging cyclists in intervals using W_{max} as the training intensity and 60% of the maximal duration that W_{max} could be sustained for (T_{max}) as the duration of each interval. Interval sessions were performed two times per week for a 4-week period. The results were impressive with increases in $\dot{V}O_{2max}$ (+5.4–8.1%), peak power (+4.7–6.2%) and average velocity during a 40km time trial (+5.1–5.8%). Training of this nature

during the speed/power phase would significantly enhance the improvements already made during transition training suggested by Hawley et al.[143]

Westgarth-Taylor et al.[148] examined the metabolic adaptations to the same training protocol and found that the only changes in metabolism occurred at absolute intensities and not relative intensities, suggesting that the increase in relative intensity during the 40km time trial could not be the consequence of an increase in lactate threshold. Weston et al.[149] examined muscle buffering capacity in response to the same high-intensity interval training and observed an increase in muscle buffering capacity that was related to the increase in time-trial performance. The authors concluded that the increase in relative performance was due to an increase in muscle buffering capacity rather than an increase in threshold.

The limited research on interval training in trained cyclists brings light to an overall important finding that trained athletes can make significant improvements that are measurable in the laboratory. Previously, it has been thought that trained athletes are unable to demonstrate improvements during training studies because of their already high level of fitness. Hence, most of the research on training has been conducted on individuals of low to moderate fitness, suggesting that science knows little about how to best optimise training in already highly trained athletes. Laursen and Jenkins[21] highlight this limitation in their review on interval training, and readers are urged to review this paper for greater discussion on interval training.

In summary, when a rise in training volume no longer augments fitness, cyclists employ intervals to intensify their training load. Relatively brief but intense sprint training can enhance both glycolytic and oxidative enzyme activity, maximum short-term power output and $\dot{V}O_{2max}$. Brief 30-second work bouts at 175% of peak power followed by 4.5 minutes of recovery and repeated 12 times has been found to be nearly as effective for improving 40km time-trial performance as 4-minute interval training bouts. Moreover, high-intensity intermittent training programmes have been effective to increase oxidative enzyme activity in muscle. When examining the efficacy of a training strategy, it should be remembered that the single best predictor of a 40km time-trial performance is the average amount of power output that can be maintained in 1 hour.

11. Tapering

To achieve peak performance, the cyclist must develop a training strategy that blends very hard riding with the correct amount of rest and recovery. A systematic reduction of training is a key part of preparation for an important competition. The extent and rate to which training volume, intensity and frequency should be reduced during a taper can seriously affect the cyclist's race performance. Should there be step reductions in training or exponential decays? Houmard et al.[150] demonstrated that a 7-day exponential decay in training volume each day or 85% reduction in training volume produced dramatic improvements in running economy and 5km race times. Furthermore, MacDougall et al.[66] employed an exponential taper strategy of an overall 87–88% reduction in training and found a 22% increase in endurance performance of runners. More specifically, these athletes gained enhanced leg-muscle enzyme activity, augmented total blood volume, increased red blood-cell density and greater muscle-glycogen storage compared with those using a step-reducing programme. Banister et al.[133] demonstrated that an exponential reduction in training utilising a reduction in the duration of training sessions or the frequency of sessions per week to be extremely effective for improvement of W_{max}. These observations lead to the conclusion that the exponential decay taper is the superior strategy. It appears that the quicker reduction seems to stimulate quicker recovery from and responses to the previous training. To be effective, exponential tapering should include at least 1 day off per week with no exercise.

Trappe et al.[151] showed, in highly trained swimmers, that a 21-day taper resulted in neuromuscular adaptations in type II muscle fibres characterised by increased contractile functional properties (peak isometric force, peak power and unloaded shortening velocity). Additionally, swim performance was im-

proved. It was hypothesised that the increased contraction velocity of type I fibres partly contributed to improved swim performance. Furthermore, in a similar study employing cyclists as subjects, these findings were extended and confirmed by Neary et al.[152] who showed that metabolic (enzymatic) adaptations in single muscle cells improve with specific taper training. Employing 22 male endurance cyclists ($\dot{V}O_{2max}$ 4.42 ± 0.40 L/min, and 60.9 ± 2.4 mL/kg/min), taper training was found to be intensity, volume, frequency and duration specific. Following a high-intensity training (progressive overload) programme, (60 minutes per session × 4 days/week for 7 weeks at 85–90% HR_{max}), the most effective taper protocol was found to be that where the cyclists maintained training at 85–90% HR_{max} for 4 days/week with progressive reduction in exercise duration (45, 35, 25, 20 minutes). During this taper, the volume of training (units of work = exercise duration × number of days × exercise intensity) was 113 units of work. Tapering resulted in a faster simulated 40km cycling time trial by 4.3% and $\dot{V}O_{2max}$ and power output at VT improved. Metabolic adaptations were most evident in oxidative (cytochrome oxidase, β-hydroxyacyl CoA dehydrogenase, succinate dehydrogenase) and contractile (myofibrillar adenosine triphosphatase) enzyme activity and structural (fibre size) changes of type II muscle fibres. Increased activity of β-hydroxyacyl CoA dehydrogenase, a marker of β-oxidation, indicates an improved utilisation of fatty acids. The results of this study suggest that when designing a taper protocol, it is important to maintain high-intensity (85–90% HR_{max}) training while methodically reducing training volume by at least 50%.

Once the taper regime is established, a plan to monitor recovery during and following the taper is required. Indicators that recovery is progressing well include: a noticeable increase in muscle strength and power, fewer sleep disturbances, decline in feelings of stress, reduced fatigue, lower rates of perceived exertion (RPE) during cycling and improved feelings of well-being.[153] In this regard, the best predictor of effective recovery is plasma noradrenaline (norepinephrine) concentration.[154] These researchers observed that changes in concentration of this hormone predicted 82% of the variation between pre- and post-tapering performance. Noradrenaline is secreted by nerve cells of the sympathetic system and the adrenal glands. Its action includes increased HR and contractility and increased rate of breakdown of glycogen and fat.

Increases in noradrenaline suggest overtraining.[155] Consequently, reduced levels of noradrenaline portend that the body is under less stress and adaptation to the taper regime has occurred. It appears that levels of this hormone serve as a reliable marker of training efficacy and work-recovery balance. For those without the means for such blood analysis, use of the Profile of Mood Status (POMS) questionnaire may serve as an alternative measure.[156] A 65- and 30-item questionnaire is available.

In summary, the extent and rate to which training volume, intensity and frequency should be reduced during a taper can seriously affect the cyclist's race performance. To that end, an 85% reduction in training volume has been shown to produce dramatic improvements in running economy and 5km race times. An exponential decay taper is the superior strategy. Employed properly, tapering results in metabolic (enzymatic) adaptations in single muscle cells. A key taper strategy is to maintain high-intensity (85–90% HR_{max}) training while methodically reducing training volume by at least 50%.

12. Overuse

Cycling is associated with overuse and overtraining disabilities, of which knee pain and excessive fatigue are at the forefront. Biomechanical abnormalities of the patellofemoral complex in conjunction with strenuous training appear to be responsible for chronic knee pain referred to as the patellofemoral syndrome.[157] This complex comprises the knee cap, the femur, and their associated muscle and connective tissues. During constant knee flexion and extension associated with cycling, any cartilage breakdown places extra pressure on the bones that come together at the knee. When breakdown occurs, bone pain and inflammation together with cartilage

destruction irritate the synovial membrane of the knee causing synovitis and joint discomfort. The causes of poor biomechanics that lead to patellofemoral pain include:

- poor quadriceps function
- vastus-medialis insufficiency
- subtalar-joint pronation
- poor muscle flexibility
- abnormal lower-limb biomechanics
- incorrect bicycle and equipment settings (saddle height, cleat position, cleat type and shoe type)
- abnormal fore-foot and rear-foot alignment
- leg-length discrepancies
- varus or valgus knee misalignments.

Riding events that are often related with patellofemoral pain comprise:

- hill training
- cycling with high gears at low cadence
- sudden increase in training volume.

Characteristically, patellofemoral pain presents itself as discomfort in the front of the knee area. This diffuse ache around the patella is often described as a vague knee pain 'underneath the kneecap'. At times, the knee may 'lock up', 'catch', 'give way', make 'cracking' noises and display some swelling. In >80% of cyclists presenting with patellofemoral pain, an abnormal mediolateral deviation of the knee during the downstroke of pedalling was demonstrated. The abnormal action was characterised as excessive side-to-side swinging of the knee during the down stroke, as the knee underwent extension. At the first sign of knee pain, the best action to take is active rest, application of ice, and if so recommended local ultrasound. Once symptoms subside, biomechanical corrections are made and quadriceps muscles strengthened, a conservative return to cycling is possible. At first, hill riding must be approached with caution and spinning using low rather than high gears should be considered. If the symptoms return, perhaps surgical procedures deserve consideration. Patellar shaving and patellectomy are common surgical procedures; however, a simple procedure termed 'washing out the knee' or arthroscopic lavage is an alternative to be given consideration.

13. Over-Training

There is fine balance between the training load and recovery. Too much cycling and too little recovery may disturb this balance and the result is a phenomenon referred to as 'over-reaching'.[158] Cyclists who drop out of stage races due to insufficient metabolic recovery caused by a decline in glycogen levels and energy-rich phosphates are examples of over-reaching. When corrected promptly, recovery from over-reaching generally occurs within approximately 14 days.[159]

If training and recovery are not rebalanced, over-reaching leads to the over-training syndrome (OTS). A common symptom of which is reduced cycling performance and subsequent fatigue leading to such symptoms as irritability, sleep problems and a lack of motivation. The mechanisms involved include major disturbances in the hypothalamo-hypophyseal thyroid axis as well as disturbed neuromuscular function.[160] Included among the disturbances are both rest and exercise HR.

HR is a sensitive indicator of the cyclists training state. Resting HR is controlled by sympathetic and parasympathetic divisions of the autonomic nervous system. When sympathetic activity increases, HR increases and short-term HR variability decreases.[161] However, when the parasympathetic activity increases, HR decreases and the short-term HR variability increases.[161] The short-term HR variability has been reported to reflect the activity of the sympathetic and parasympathetic nervous system and balance between these two divisions. Because the balance between and the activity of these divisions of the autonomic nervous system change with training and over-training, HR variability can be used to indicate training effects.[161] For example, HR decreases and HR variability increases with the positive training effect. In over-reaching and in the sympathetic over-training state the HR increases and HR variability decreases.[161] In the parasympathetic over-training state or in exhaustion both HR and HR variability decrease.[161]

If HR during sleep is used to interpret training state or overtraining, consideration must given to the fact that intrinsic day-to-day maximum HR during

sleep varies by ~8 beats/min.[162] Lucía et al.[122] have shown that in professional 3-week cycling races, the best possible marker suggestive of overreaching and fatigue is a decrease in HR_{max}. This phenomenon, in turn, would reflect a certain state of down-regulation of the sympathetic-adrenal system. In fact, a certain state of 'hormonal exhaustion' has been reported by the end of 3-week races.[122,163]

Moreover, the over-trained cyclist generally exhibits performance incompetence, prolonged fatigue, or an inability to train at expected levels. Additionally, muscle soreness and tenderness, persistent muscle soreness that increases with each training session and elevated serum creatine kinase may be observed. The overload may be both psychological as well as physical. Disturbance of mood state, reduction of maximum performance capacity, and competitive incompetence over weeks and months are common.

The OTS has been related to glutamine levels.[163] It has been suggested that the single measure of a lower concentration could be a negative effect of exercise stress. Glutamine is the most abundant amino acid in the body, where skeletal muscle provides the majority of plasma glutamine required by the gastrointestinal tract, cells of the immune system and kidney during acidosis. Moreover, glutamine may be important in the regulation of protein synthesis and degradation. Two factors appear to be responsible for the decline in glutamine concentration over time: (i) increased levels of glucocorticoids may decrease muscular glutamine stores; and (ii) decreased nutritional intake of protein may affect stores because increased protein intake has been suggested for endurance athletes particularly during heavy training.

Glutamate concentration for over-trained athletes presents another perspective to over-training. It appears that high glutamate concentrations are associated with very high-intensity training periods where high blood lactate concentrations and coincidently high hydrogen ion concentrations are observed. Normal rested or low training volume status is represented by a glutamine concentration of 585 ± 54 μmol/L and by a glutamate concentration of 101 ± 16 μmol/L.[164] A glutamine concentration of 585 μmol/L or lower appears to indicate a 'developed work capacity' or tolerance to exercise volume. This reference is associated with athletes who have developed an aerobic base over years of training.

The changes in both glutamine and glutamate concentrations appears to follow a pattern of a decrease in glutamine and an increase in glutamate concentration with increased training load. Consequently, it has been proposed that training status can be represented by the ratio of glutamine to glutamate (Gm/Ga). The Gm/Ga ratio rested or early season training would be >5.88 μmol/L.[164] A maximum value might be 7.66 μmol/L.[164]

A mean glutamine concentration of 522 ± 53 μmol/L, glutamate of 128 ± 19 μmol/L and Gm/Ga ratio of 4.15 ± 0.57 μmol/L have been proposed as the extreme values for athletes who have not met conditions of over-training and are managing the training load imposed.[164] A Gm/Ga ratio of 3.58 μmol/L is a suggestion of over-training.[164] This level may be used as guide to suggest that overreaching, which leads to over-training, may soon manifest itself. In this instance, serial testing is necessary beginning prior to the training season.

Athletes who have a potential for less tolerance to either volume or intensity of training load imposed may be identified by relatively lower Gm/Ga ratios of approximately one standard deviation (5.04 μmol/L) from the mean rested or early season training value.[164] Likewise, a reduced maximal lactate as opposed to the preserved maximal lactate in athletes adapted to a hard training programme suggests over-training. In this regard, Snyder et al.[128] suggests that one of the most sensitive and easy measure criterion for over-reaching/over-training is the plasma blood lactate to RPE ratio (BLa **:** RPE). When the concentration of blood lactate (measured in mmol/L) is reduced and the RPE (using a 10 scale) remains unchanged, the ratio of the two (multiplied by 100) will be reduced to probably <100 (here the blood lactate concentration is less than RPE). Such a ratio suggests over-reaching/over-training.

To avoid the OTS, the long-term training background must be such as to develop an adequate

tolerance for work or that genetic make-up predisposes one to reduced training ability. Development of a sound base of training may prevent both injury and over-training later in the season. Tolerance to training should be reflected in a trend toward higher Gm/Ga ratios. Excessive training load is most likely a major contributor to the likelihood of developing the OTS.

The cyclists who experience 'heavy legs' while riding are voicing a common symptom of over-training. Large volumes of training, systemic inflammation and elevated levels of proinflammatory cytokines, directly and/or indirectly, induce anorexia resulting in reduced caloric intake. Additionally, local muscle membrane injury and reduced availability of GLUT-4 glucose transporters in muscle cell membrane, attenuates movement of glucose into the cell for glycogen resynthesis. Both factors may contribute to reduced muscle glycogen synthesis in the OTS. Moreover, the reduced muscle glycogen could in turn account for the 'heavy legs'. Likewise, reduced blood lactate levels during both submaximal and maximal exercise could be the consequence of reduced muscle glycogen.

Treatment of OTS includes determination and elimination of those factors, in both daily life and training that lead to obvious over-training. Regular sleep is very important. Adequate quantity and quality of nutrition should be ensured regardless of any loss of appetite connected with over-training. Most importantly, rest may be the most potent healing agent.

In summary, if training and recovery are not rebalanced, over-reaching leads to the OTS. A common symptom of which is reduced cycling performance and subsequent fatigue leading to such symptoms as irritability, sleep problems and a lack of motivation. HR variability can be used to indicate whether effects of over-reaching are present. HR decreases and HR variability increases with the positive training effect. In over-reaching and in the sympathetic over-training state, the HR increases and HR variability decreases. It has been shown that in professional 3-week cycling races, the best possible marker suggestive of over-reaching and fatigue is a decrease in HR_{max}.

Furthermore, the over-trained cyclist generally exhibits performance incompetence, prolonged fatigue or an inability to train at expected levels. The overload may be both psychological as well as physical. One of the most sensitive and easiest to measure criterion for over-reaching/over-training is the BLa **:** RPE. Training status may also be represented by Gm/Ga. Rest may be the most potent healing agent.

14. Conclusions

This research review confirms that competitive cycling is a strenuous exercise that requires unique physiological and metabolic demands. To achieve success entails thousands of kilometres of riding while pushing toward extremes of training volume, intensity and duration. The physical and physiological characteristics of high performance cyclists presented in this review serve as morphological and physiological guidelines for optimal performance. To assess road cycling performance, scaling of the physiological characteristics to account for the influence of morphotype on road conditions is recommended. A power output **:** body mass ratio of at least 5.5 W/kg may be used as a prerequisite for top-level cyclists; however, a ratio ≥6.34 W/kg is suggested for professional cyclists.

Research findings clearly demonstrate that relatively brief but intense sprint training serves to enhance both the glycolytic and oxidative enzyme activity, short-term W_{peak}, $\dot{V}O_{2max}$, lactate threshold and 40km cycling TT performance. From a review of the scientific literature, it becomes clear that specific monitoring of training and competition intensity, through the application of the TRIMP and use of SRM power cranks, is essential for performance improvement and competitive success. These data become crucial reference material for the enrichment of training regimes. It is imperative that the cyclist give credence to the importance of tapering and over-training avoidance in order to achieve performance improvement.

In the future, cycling science research will continue to be heavily influenced and fuelled by emerging technologies designed to examine the physiology (molecular and cellular events) of the rider and design mechanics of the bicycle. Cycling issues that should receive further attention include:

- standardisation and validation of bicycle ergometers;
- standardisation and validation of bicycle ergometer protocols for untrained and trained cyclists;
- standardisation and validation measurement of anaerobic power in trained cyclists;
- quality control for internal and external validity of research designs;
- identification of the mechanisms that underlie the training response;
- examination of the effects of different protocols of interval training on sprint and endurance performance;
- identification of mechanisms responsible for enhanced aerobic performance consequent to high-intensity short-duration interval training;
- examination of the performance effect of a combination of speed and power cycling training;
- investigation of how to maximise the benefits derived from taper strategies;
- examination of the relationship between laboratory cycling test results and competition performance.

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References

1. Bassett DR, Howley ET. Maximal oxygen uptake 'classical' versus 'contemporary' viewpoints. Med Sci Sports Exerc 1997; 29: 591-603
2. Brooks GA. Intra- and extra-cellular lactate shuttles. Med Sci Sports Exerc 2000; 32: 790-9
3. Noakes TD. Maximal oxygen uptake: 'classical' versus 'contemporary' viewpoints: a rebuttal. Med Sci Sports Exerc 1998; 30: 1381-98
4. Robergs RA, Ghiasvand F, Parker D. Biochemistry of exercise-induced metabolic acidosis. Am J Physiol Regul Integr Comp Physiol 2004; 287: R502-16
5. Spriet LL. Anaerobic metabolism in human skeletal muscle during short-term, intense activity. Can J Physiol Pharmacol 1990; 70: 157-65
6. Wagner PD. New ideas on limitations to $\dot{V}O_{2max}$. Exerc Sport Sci Rev 2000; 28: 10-4
7. Moseley L, Jeukendrup AE. The reliability of cycling efficiency. Med Sci Sports Exerc 2001; 33: 621-7
8. Hagberg JM, Coyle EF. Physiological determinants of endurance performance as studied in competitive walkers. Med Sci Sports Exerc 1983; 15: 287-9
9. Heck H, Mader A, Hess G, et al. Justification of the 4 mmol/l lactate threshold. Int J Sports Med 1985; 6: 117-30
10. Sjodin B, Jacobs I, Karlsson J. Onset of blood lactate accumulation and enzyme activities in m. vastus lateralis in man. Int J Sports Med 1981; 2: 166-70
11. Wasserman K, Hansen JE, Sue DY, et al. Principles of exercise testing and interpretation. Philadelphia (PA): Lea & Febiger, 1987
12. Davis JA. Anaerobic threshold: a review of the concept and directions for future research. Med Sci Sports Exerc 1985; 17: 6-18
13. Weltman AD, Snead D, Seip R, et al. Reliability and validity of a continuous incremental treadmill protocol for determination of lactate threshold fixed blood lactate concentration, and $\dot{V}O_{2max}$. Int J Sports Med 1990; 11: 26-32
14. Sidossis LS, Horowitz JF, Coyle EF. Load and volecity of contraction influence gross and delta mechanical efficiency. Int J Sports Med 1992; 13: 407-11
15. Hopkins WG, Hawley JA, Burke LM. Design and analysis of research on sport performance enhancement. Med Sci Sports Exerc 2002; 31: 472-85
16. Bentley DJ, Wilson GJ, Davie AJ, et al. Correlation between peak power output, muscular strength and cycle time trial performance in triathletes. J Sports Med Phys Fitness 1998; 38: 201-7
17. Bishop D, Jenkins DG, MacKinnon LT. The relationship between plasma lactate parameters, W_{peak} and 1-h cycling performance in women. Med Sci Sports Exerc 1998; 30: 1270-5
18. Fernández-García B, Perez-Landaluce J, Rodriguez-Alonso M, et al. Intensity of exercise during road race pro-cycling competition. Med Sci Sports Exerc 2000; 32: 1002-6
19. Harnish CR, Swensen TC, Pate RR. Methods for estimating the maximal lactate steady state in trained cyclists. Med Sci Sports Exerc 2001; 33: 1052-5
20. Heil D, Wilcox A, Quinn C. Cardiorespiratory responses to seat tube variation during steady state cycling. Med Sci Sports Exerc 1995; 27: 730-5
21. Laursen PB, Jenkins DG. The scientific basis for high-intensity interval training: optimizing training programs and maximizing performance in highly trained endurance athletes. Sports Med 2002; 32: 53-73
22. Lepers R, Hausswirth C, Maffiuletti NA, et al. Evidence of neuromuscular fatigue following prolonged cycling exercise. Med Sci Sports Exerc 2000; 32: 1880-6

23. Lindsay FH, Hawley JA, Myburgh KH, et al. Improved athletic performance in highly trained cyclists after interval training. Med Sci Sports Exerc 1996; 28: 1427-34
24. Lucía A, Hoyos J, Pérez M, et al. Inverse relationship between $\dot{V}O_{2max}$ and economy/efficiency in world-class cyclists. Med Sci Sports Exerc 2002; 34: 2079-84
25. Lucía A, Hoyos J, Chicharro JL. Preferred pedaling cadence in professional cycling. Med Sci Sports Exerc 2001; 33: 1361-6
26. Marsh AP, Martin PE. Effect of cycling experience, aerobic power, and power output on preferred and most economical cycling cadences. Med Sci Sports Exerc 1997; 29: 1225-32
27. MacRae HS-H, Hise KJ, Allen PJ. Effects of front and dual suspension mountain bike systems on uphill cycling performance. Med Sci Sports Exerc 2000; 32: 1276-80
28. Padilla S, Mujika I, Orbananos J, et al. Exercise intensity and load during mass-start stage races in professional road cycling. Med Sci Sports Exerc 2001; 33: 796-802
29. Romer LE, McConnell AK, Jones DA. Inspiratory muscle fatigue in trained cyclists: effects of inspiratory muscle training. Med Sci Sports Exerc 2002; 34: 785-92
30. Schabort EJ, Hawley JA, Hopkins WG, et al. A new reliable laboratory test of endurance performance for road cyclists. Med Sci Sports Exerc 1998; 30: 1744-50
31. Stepto NK, Martin DT, Fallon KE, et al. Metabolic demands of intense aerobic interval training in competitive cyclists. Med Sci Sports Exerc 2001; 33: 303-10
32. Swensen TC, Harnish CR, Beitman L, et al. Non-invasive estimation of the maximal lactate steady state in trained cyclists. Med Sci Sports Exerc 1999; 31: 742-6
33. Latin RW, Berg KE, Smith P, et al. Validation of a cycle ergometry equation for predicting steady-rate $\dot{V}O_2$. Med Sci Sports Exerc 1993; 25: 970-4
34. Lucía A, Hoyos J, Santalla A, et al. Kinetics of $\dot{V}O_2$ in professional cyclists. Med Sci Sports Exerc 2002; 34: 320-5
35. McCole SD, Claney K, Conte JC, et al. Energy expenditure during bicycling. J Appl Physiol 1990; 68: 748-53
36. Baron R. Aerobic and anaerobic power characteristics of off-road cyclists. Med Sci Sports Exerc 2001; 33: 1387-93
37. Palmer GS, Noakes TD, Hawley JA. Effects of steady-state versus stochastic exercise on subsequent cycling performance. Med Sci Sports Exerc 1997; 29: 684-7
38. Padilla S, Mujika I, Cuesta G, et al. Level ground and uphill cycling ability in professional road cycling. Med Sci Sports Exerc 1999; 31: 878-85
39. Impellizzeri F, Sassi A, Rodriguez-Alonso M, et al. Exercise intensity during off-road cycling competitions. Med Sci Sports Exerc 2002; 34: 1808-13
40. Liedl MA, Swain DP, Branch D. Physiological effects of constant versus variable power during endurance cycling. Med Sci Sports Exerc 1999; 31: 1472-7
41. Laursen PB, Shing CM, Peake JM, et al. Interval training program optimization in highly trained endurance cyclists. Med Sci Sports Exerc 2002; 34: 1801-7
42. Takaishi T, Ishida K, Katayama K, et al. Effect of cycling experience and pedal cadence on the near-infrared spectroscopy parameters. Med Sci Sports Exerc 2002; 34: 2062-71
43. British Association of Sport Sciences (Sports Physiology Section). Position statement on the physiological assessment of the elite competitor. 2nd ed. Leeds: British Association of Sport Sciences (Sports Physiology Section), 1988
44. Coyle EF, Feltner ME, Kautz SA. Physiological and biomechanical factors associated with elite endurance cycling performance. Med Sci Sports Exerc 1991; 23: 93-107
45. Faria IE, Faria EW, Roberts S, et al. Comparison of physical and physiological characteristics in elite young and mature cyclists. Res Q Exerc Sports Sci 1989; 60: 388-95
46. Lucía A, Pardo J, Durantez A, et al. Physiological differences between professional and elite road cyclists. Int J Sports Med 1998; 19: 342-8
47. Saltin B. Anaerobic capacity: past, present and prospective. In: Taylor AW, Gollinck PD, Green HJ, et al. editors. Biochemistry of exercise VII. Champaign (IL): Human Kinetics, 1990: 387-413
48. Swain DP. The influence of body mass in endurance bicycling. Med Sci Sports Exerc 1994; 26: 58-63
49. Hawley JA, Noakes TD. Peak power output predicts maximal oxygen uptake and performance in trained cyclists. Eur J Appl Physiol 1992; 65: 79-83
50. Sjodin B, Jacobs I. Onset of blood lactate accumulation and marathon running performance. Int J Sports Med 1981; 2: 23-6
51. Kuipers H, Verstappen FTJ, Keizer HA, et al. Variability of aerobic performance in the laboratory and its physiological correlates. Int J Sports Med 1985; 6: 197-201
52. Fohrenbach R, Mader A, Hollmann W, et al. Determination of endurance capacity and prediction of exercise intensities for training and competition in marathon runners. Int J Sports Med 1987; 8: 11-8
53. Stegmann H, Kindermann W. Comparison of prolonged exercise tests at the individual anaerobic threshold and the fixed anaerobic threshold of 4 mmol/l-1 lactate. Int J Sports Med 1982; 3: 105-10
54. Sjodin B, Svedenhag J. Applied physiology of marathon running. Sports Med 1985; 2: 83-99
55. Palmer GS, Hawley JA, Dennis SC, et al. Heart rate response during a 4-d cycle stage race. Med Sci Sports Exerc 1994; 26: 1278-83
56. Lucía A, Hoyos J, Chicharro JL. Physiology of professional cycling. Sports Med 2001; 31: 325-37
57. Horowitz JF, Sidossis LS, Coyle EF. High efficiency of type I muscle fibers improves performance. Int J Sports Med 1994; 15: 152-7
58. Coyle EF, Sidossis LS, Horowitz JF, et al. Cycling efficiency is related to the percentage of type I muscle fibers. Med Sci Sports Exerc 1992; 24: 782-8
59. Lucía A, Carvajal A, Calderon FS, et al. Breathing pattern in highly competitive cyclists during incremental exercise. Eur J Appl Physiol 1999; 79: 512-21
60. Harms GA. Effect of skeletal muscle demand on cardiovascular function. Med Sci Sports Exerc 2000; 32: 94-9
61. Wilber RL, Zawadzki KM, Kearney JT, et al. Physiological profiles of elite off-road and road cyclists. Med Sci Sports Exerc 1997; 29: 1090-4
62. Foxdal P, Sjodin B, Sjodin A, et al. The validity and accuracy of blood lactate measurements for prediction of maximal endurance running capacity. Int J Sports Med 1994; 15: 89-95
63. Jacobs I. Blood lactate: implications for training and sports performance. Sports Med 1986; 3: 10-25
64. Kindermann W, Simon G, Keul J. The significance of the aerobic-anaerobic transition for the detection of work load intensities during endurance training. Eur J Appl Physiol 1979; 52: 25-34
65. Keith SP, Jacobs I, McLellan TM. Adaptations to training at the individual anaerobic threshold. Eur J Appl Physiol 1992; 65: 316-23

66. MacDougall JD, Wenger HA, Green HJ, editors. Physiological testing of the high-performance athlete. 2nd ed. Champaign (IL): Human Kinetics Books, 1991: 107-73
67. Barbeau P, Serresse O, Boulay MR. Using maximal and submaximal aerobic variables to monitor elite cyclists during a season. Med Sci Sports Exerc 1993; 25: 1062-9
68. Faria IE. Energy expenditure, aerodynamics and medical problems in cycling: an update. Sports Med 1992; 14: 43-63
69. Pfeiffer RP, Harden BP, Landis D, et al. Correlating indices of aerobic capacity with performance in elite women road cyclists. J Strength Cond Res 1993; 7: 201-5
70. Lucía A, Hoyos J, Perez M, et al. Heart rate and performance parameters in elite cyclists a longitudinal study. Med Sci Sports Exerc 2000; 32: 1777-82
71. Lacour JR, Padilla S, Denis C. The inflection curve frequency of cardiac power is not indicative of the anaerobic threshold [in French]. Sci Motr 1987; 1: 3-6
72. Sjogaard G. Muscle morphology and metabolic potential in elite road cyclists during a season. Int J Sports Med 1984; 5: 250-4
73. Terrados N, Melichna J, Sylven C, et al. Effects of training at simulated altitude on performance and muscle metabolic capacity in competitive road cyclists. Eur J Appl Physiol 1988; 57: 203-9
74. Gnehm P, Reichenbach S, Altpeter E, et al. Influence of different racing positions on metabolic costs in elite cyclists. Med Sci Sports Exerc 1997; 29: 818-23
75. Saltin B, Astrand PO. Maximal oxygen uptake in athletes. J Appl Physiol 1967; 23: 353-8
76. Burke ER, Cerny F, Costill D, et al. Characteristics of skeletal muscle in competitive cyclists. Med Sci Sports Exerc 1977; 9: 109-12
77. Hermansen L. Oxygen transport during exercise in human subjects. Acta Physiol Scand Suppl 1973; 399: 1-104
78. Burke ER. Physiological characteristics of competitive cyclists. Phys Sportsmed 1980; 8: 79-84
79. Stromme SB, Ingjer F, Meen HD. Assessment of maximal aerobic power in specifically trained athletes. J Appl Physiol 1977; 42: 833-7
80. Impellizzeri F, Sassi A, Rodriguez-Alonso M, et al. Exercise intensity during off-road cycling competitions. Med Sci Sports Exerc 2002; 34: 1808-13
81. Padilla S, Mujika I, Cuesta G, et al. Validity of a velodrome test for competitive road cyclists. Eur J Appl Physiol 1996; 73: 446-51
82. Palmer GS, Dennis SC, Noakes TD, et al. Assessment of the reproducibility of performance testing on an air-braded cycle ergometer. Int J Sports Med 1996; 17: 293-8
83. Tanaka H, Bassett Jr DR, Swensen TC, et al. Aerobic and anaerobic power characteristics of competitive cyclists in the United States Federation. Int J Sports Med 1993; 14: 334-8
84. Hopkins SR, McKenzie DC. The laboratory assessment of endurance performance in cyclists. Can J Appl Physiol 1994; 19: 266-74
85. Costill DL, Thompson H, Roberts EL. Fractional utilization of aerobic capacity during distance running. Med Sci Sports Exerc 1973; 5: 248-53
86. Saltin B, Strange S. Maximal oxygen uptake: 'old' and 'new' arguments for a cardiovascular limitation. Med Sci Sports Exerc 1992; 24: 30-7
87. Wagner PD. Muscle $\dot{V}O_2$ transport dependent control of metabolism. Med Sci Sports Exerc 1995; 27: 47-53
88. Farrell PA, Wilmore JH, Coyle EF, et al. Plasma lactate accumulation and distance running performance. Med Sci Sports Exerc 1979; 11: 338-44
89. Ivy JL, Withers RT, van Handel PJ, et al. Muscle respiratory capacity and fiber type as determinants of lactate threshold. J Appl Physiol 1980; 48: 523-7
90. Sjodin B, Jacobs I, Svedenhag J. Changes in the onset of blood lactate accumulation and muscle enzymes after training at OBLA. Eur J Appl Physiol 1982; 49: 45-57
91. Tesch PA, Sharp DS, Daniels WL. Influence of fiber type composition and capillary density on OBLA. Int J Sports Med 1981; 2: 252-5
92. Bar-Or O. The Wingate anaerobic test: an update on methodology, reliability and validity. Sports Med 1987; 4: 381-94
93. Boobis L, Williams C, Wootton SA. Human muscle metabolism during brief maximal exercise [abstract]. J Physiol (Lond) 1982; 338: 21P-2P
94. Hultman E, Sjoholm H. Energy metabolism and contraction force of human skeletal muscle situ during electrical stimulation. J Physiol (Lond) 1983; 345: 525-32
95. Jones NL, McCartney N. Influence of muscle power on aerobic performance and the effects of training. Acta Med Scand Suppl 1986; 711: 115-22
96. Serresse O, Lortie G, Bouchard C, et al. Estimation of the contribution of the various energy systems during maximal work of short duration. Int J Sports Med 1988; 9: 456-60
97. Smith JC, Hill DW. Contribution of energy systems during a Wingagte power test. Br J Sports Med 1991; 25: 196-9
98. Spriet LL, Lindinger ML, McKelvie RS, et al. Muscle glycogenolysis and H^+ concentration during maximal intermittent cycling. J Appl Physiol 1989; 66: 8-13
99. Withers RT, Sherman WM, Clark DG, et al. Muscle metabolism during 30, 60, and 90 s of maximal cycling on an air-braked ergometer. Eur J Appl Physiol Occup Physiol 1991; 63: 354-62
100. Harris R, Hultman E, Nordesjo LO. Glycogen glycolytic intermediates and high energy phosphates determined in biopsy samples of muscles quadriceps femoris of man at rest. Scand J Clin Lab Invest 1974; 33: 109-20
101. McCartney N, Spriet LL, Heigenhauser GJF, et al. Muscle power metabolism in maximal intermittent exercise. J Appl Physiol 1986; 60: 1164-9
102. Sahlin K, Harris RC, Hultman E. Resynthesis of ceratine phosphate in human muscle after exercise in relation to intramuscular pH and availability of oxygen. Scand J Clin Lab Invest 1979; 39: 551-8
103. Gaitanos G, Williams C, Boobis LH, et al. Human muscle metabolism during intermittent maximal exercise. J Appl Physiol 1993; 75: 712-9
104. Donovan CM, Brooks GA. Endurance training affects lactate clearance, not lactate production. Am J Physiol 1983; 244: E83-92
105. Santalla A, Pérez M, Montilla M, et al. Sodium bicarbonate ingestion does not alter the $\dot{V}O_2$ slow component of professional cyclists. J Sports Sci 2003; 1 (1): 39-47
106. Juel C. Muscle pH regulation: role of training. Acta Physiol Scand 1998; 162: 359-66
107. Ward-Smith AJ. Aerobic and anaerobic energy conversion during high-intensity exercise. Med Sci Sports Exerc 1999; 31: 1855-60
108. Conley KE, Kemper WF, Crowther GJ. Limits to sustainable muscle performance: interaction between glycolysis and oxidative phosphorylation. J Exp Biol 2001; 204: 3189-94

109. Hochachka PW, Beatty CL, Burelle Y, et al. The lactate paradox in human high-altitude physiological performance. News Physiol Sci 2002; 17: 122-6
110. Faria EW, Parker DL, Faria IE. The science of cycling: factors affecting performance – part II. Sports Med 2005; 35 (4): 313-37
111. Edwards RHT. Biochemical bases of fatigue in exercise performance: catastrophe theory of muscular fatigue. In: Knuttgen HG, Vogel RD, Poortmans JR. editors. Biochemistry of exercise. Champaign (IL): Human Kinetics, 1983: 3-28
112. Colliander EB, Dudley GA, Tesch PA. Skeletal muscle fiber type composition and performance during repeated bouts of maximal, concentric contraction. Eur J Appl Physiol 1988; 58: 81-6
113. Hautier CA, Arsac LM, Deghdegh K, et al. Influence of fatigue on EMG/force ratio and cocontraction in cycling. Med Sci Sports Exerc 2000; 32: 839-43
114. Meeusen R, De Meirleir K. Exercise and brain neurotransmission. Sports Med 1995; 20: 160-88
115. Davis MJ, Bailey SP. Possible mechanisms of central nervous system fatigue during exercise. Med Sci Sports Exerc 1997; 29: 45-57
116. Davis MJ. Nutrition, neurotransmitters, and central nervous system fatigue. In: Maughan RJ, editor. Oxford: Blackwell Science Ltd, 2000: 171-83
117. Piacentini MF, Meeusen R, Buyse L, et al. No effect of a noradrenergic reuptake inhibitor on performance in trained cyclists. Med Sci Sports Exerc 2002; 34: 1189-93
118. Hansen JJ, Faria EW, Faria IE. Effect of 5-hydroxytryptophan on central fatigue [abstract]. Med Sci Sports Exerc 1999; 31: S118
119. St Clair Gibson A, Schabort EJ, Noakes TD. Reduced neuromuscular activity and force generation during prolonged cycling. Am J Physiol Regul Integr Comp Physiol 2001; 281: R187-96
120. Ulmer HV. Concept of an extracellular regulation of muscular metabolic rate during heavy exercise in humans by psychophysiological feedback. Experientia 1996; 52: 416-20
121. Noakes TD, Peltonen JE, Rusko HK. Evidence that a central governor regulates exercise performance during acute hypoxia and hyperoxia. J Exp Biol 2001; 204: 3225-34
122. Lucía A, Hoyos J, Santalla A, et al. Tour de France vs Vuelta a España: which is harder? Med Sci Sports Exerc 2003; 35: 872-8
123. Johnson DB, Aaron EA, Babcock MA, et al. Respiratory muscle fatigue during exercise: implications for performance. Med Sci Sports Exerc 1996; 28: 1129-37
124. Coirault C, Chemla D, Lecarpentier Y. Relaxation of diaphragm muscles. J Appl Physiol 1999; 87: 1243-52
125. Caine MP, McConnell AK. The inspiratory muscles can be trained differentially to increase strength or endurance using a pressure threshold, inspiratory device. Eur Respir J 1998; 12: 58-9
126. Williams JS, Wongsathikun J, Boon SM, et al. Inspiratory muscle training fails to improve endurance capacity in athletes. Med Sci Sports Exerc 2002; 34: 1194-8
127. Perez HR. The effects of competitive road-racing cycling on the body composition, pulmonary function, and cardiovascular system of sport cyclists. J Sports Med 1981; 21: 165-72
128. Snyder AC, Woulfe TJ, Walsh R, et al. Insensitivity of laboratory measures to training induced improvement in athletes [abstract]. Physiologist 1989; 32: 230
129. Lucía A, Hoyos J, Perez M, et al. Heart rate and performance parameters in elite cyclists: a longitudinal study. Med Sci Sports Exerc 2000; 32: 1777-82
130. Lucía A, Hoyos J, Pardo J, et al. Metabolic and neuromuscular adaptations to endurance training in professional cyclists: a longitudinal study. Jpn J Physiol 2000; 50: 381-8
131. Boulay MR, Barbeau P, Giroux M, et al. Peripheral and central adaptations in cyclists during a training and competitive season [abstract]. Med Sci Sports Exerc 1992; 24: S95
132. Banister EW. Modeling elite athletic performance. In: Green HJ, McDougal JD, Wenger HA, editors. Physiological testing of elite athletes. Champaign (IL): Human Kinetics, 1991: 403-24
133. Banister EW, Carter JB, Zarkadas PC. Training theory and taper: validation in triathlon athletes. Eur J Appl Physiol 1999; 79: 182-91
134. Candau R, Busso T, Lacour JR. Effect of training on iron status in cross-country skiers. Eur J Appl Physiol 1992; 64: 497-502
135. Morton RH, Fitz-Clarke JR, Banister EW. Modeling human performance in running. J Appl Physiol 1990; 69: 1171-7
136. Padilla S, Mujika I, Angulo F, et al. Scientific approach to the 1-h cycling world record. J Appl Physiol 2000; 89: 1522-7
137. Foster C, Florhaug JA, Franklin J, et al. A new approach to monitoring exercise training. J Strength Cond Res 2001; 15: 109-15
138. Skinner JS, Mclellan TH. The transition from aerobic to anaerobic metabolism. Res Q Exerc Sport 1980; 51: 234-48
139. Coen B, Schwarz L, Urhausen A, et al. Control of training in middle- and long-distance running by means of individual anaerobic threshold. Int J Sports Med 1991; 12: 519-24
140. Hollmann W, Rost R, Liesen H, et al. Assessment of different forms of physical activity with respect to preventative and rehabilitative cardiology. Int J Sports Med 1981; 2: 67-80
141. Weltman A, Seip R, Snead D, et al. Exercise training at and above the lactate threshold in previously untrained women. Int J Sports Med 1992; 13: 257-63
142. Schumacher YO, Mueller P. The 4000m team pursuit cycling world record: theoretical and practical aspects. Med Sci Sports Exerc 2002; 34: 1029-36
143. Hawley JA, Myburgh KH, Noakes TD, et al. Training techniques to improve fatigue resistance and enhance endurance performance. J Sports Sci 1997; 15: 325-33
144. MacDougall JD, Hicks AL, MacDonald JR, et al. Muscle performance and enzymatic adaptations to sprint interval training. J Appl Physiol 1998; 84: 2138-42
145. Tabata I, Nishimura K, Kouzaki M, et al. Effects of moderate-intensity endurance and high-intensity intermittent training on anaerobic and $\dot{V}O_{2max}$. Med Sci Sports Exerc 1997; 28: 1327-30
146. Stepto NK, Hawley JA, Dennis SC, et al. Effects of different interval-training programs on cycling time-trial performance. Med Sci Sports Exerc 1999; 31: 736-41
147. Rodas G, Ventura JL, Cadefau JA, et al. A short training programme for the improvement of both aerobic and anaerobic metabolism. Eur J Appl Physiol 2000; 82: 480-6
148. Westgarth-Taylor C, Hawley JA, Rickard S, et al. Metabolic and performance adaptations to interval training in endurance-trained cyclists. Eur J Appl Physiol 1997; 75: 298-304
149. Weston AR, Myburgh KH, Lindsay FH, et al. Skeletal muscle buffering capacity and endurance performance after high-intensity training by well-trained cyclists. Eur J Appl Physiol 1997; 75: 7-13

150. Houmard J, Scott BK, Justice CL, et al. The effects of a taper on performance in distance runners. Med Sci Exerc Sports 1994; 26: 624-31

151. Trappe S, Costill DL, Thomas R. Effects of swim taper on whole muscle and single muscle fiber contractile properties. Med Sci Sports Exerc 2001; 33: 48-56

152. Neary JP, Martin TP, Quinne HA. Effects of taper on endurance cycling capacity and single muscle fiber properties. Med Sci Sports Exerc 2003; 35: 1875-81

153. Hooper SL, MacKinnon LT, Howard A, et al. Markers for monitoring overtraining and recovery. Med Sci Sports Exerc 1995; 27: 106-12

154. Hooper SL, MacKinnon LT, Howard A. Physiological and psychometric variables for monitoring recovery during tapering for major competition. Med Sci Sports Exerc 1999; 31: 1205-10

155. Hooper SL, MacKinnon LT, Gordon RD, et al. Hormonal responses of elite swimmers to overtraining. Med Sci Sports Exerc 1993; 25: 741-7

156. McNair DM, Lorr M, Droppleman LF. EDITS manual for the profile of mood status. San Diego (CA): Educational and Industrial Testing Services, 1971: 1-29

157. Gregor RJ, Broker JP, Ryan MM. The biomechanics of cycling. In: Holloszy JO, editor. Exercise and science review. Baltimore (MD): Williams & Wilkins, 1991: 127-9

158. Kuipers H. Training and overtraining: an introduction. Med Sci Sports Exerc 1998; 30: 1137-9

159. Snyder AC. Overtraining and glycogen depletion hypothesis. Med Sci Sports Exerc 1998; 30: 1146-50

160. Kuipers H, Keizer HA. Overtraining in elite athletes. Sports Med 1988; 6: 79-92

161. Uusitalo A, Tahvanainen K, Uusitalo A, et al. Noninvasive evaluation of sympathovagal balance in athletes by time and frequency domain analyses of heart and blood pressure variability. Clin Physiol 1996; 16: 575-88

162. Wiriam RW, Lambert MI. Heart rate during sleep: implications for monitoring training status. J Sports Sci Med 2003; 2: 133-8

163. Fernández-Garcĭa B, Lucĭa A, Hoyos J, et al. The response of sexual and stress hormones of male pro-cyclists during continuous intense competition. Int J Sports Med 2002; 23: 555-60

164. Smith DJ, Norris SR. Changes in glutamine and glutamate concentrations for tracking training tolerance. Med Sci Sports Exerc 2000; 32: 684-9

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