Effects of long-term training cessation in young top-level road cyclists

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ABSTRACT

In cycling it is common practice to have a break in the off-season longer than four weeks whilst adopting an almost sedentary lifestyle, and such a break is considered to be long-term detraining. No previous studies have assessed the effect of training cessation with highly-trained young cyclists. The purpose of the present investigation was to examine effects of five weeks of training cessation in 10 young (20.1±1.4yr) male road cyclists for body composition, haematological, and physiological parameters. After training cessation body mass of cyclists increased (*P*=0.014; ES=0.9). $\dot{V}O_{2\,max}$ (L·min^{-1}=-8.8±5.0%, mL·kg^{-1}·min^{-1}=-10.8±4.2%,), W_{max} (W=-6.5±3.1%, W·kg^{-1}=-8.5±3.3%,), W_{LT1} (W=-12.9±7.0%, W·kg^{-1}=-14.8±7.4%,), W_{LT2} (W=-11.5±7.0%, W·kg^{-1}=-13.4±7.6%,) and haematological (red blood cells count, -6.6±4.8%; Haemoglobin, -5.4±4.3% and Haematocrit, -2.9±3.0%) values decreased (*P* ≤0.028; ES ≥0.9). Five weeks of training cessation resulted in large decreases in physiological and haematological values in young top-level road cyclists suggesting the need for a shorter training stoppage. This long-term detraining is more pronounced when expressed relative to body mass emphasizing the influence of such body mass on power output. A maintenance program based on reduced training strategies should be implemented to avoid large declines in physiological values in young cyclists who aspire to become professionals.

Introduction

Road cycling is predominantly an endurance sport, where performance is highly correlated with maximum oxygen uptake ($\dot{V}O_{2\,max}$), muscle fibre type, economy and lactate threshold (LT1) (Atkinson, Davison, Jeukendrup, & Passfield, 2003). There is also substantial evidence that maximum external power output (W_{max}) and power at LT1 and onset of blood lactate accumulation (OBLA or LT2) obtained during a maximum incremental cycling test predict cycling performance (Atkinson et al., 2003; Faria, Parker, & Faria, 2005; Padilla, Mujika, Cuesta, & Goiriena, 1999). The $\dot{V}O_{2\,max}$ is considered one of the gold standards for the purpose of evaluating and selecting elite-standard cyclists and as a prerequisite to perform at high level. The upper limit for this $\dot{V}O_{2\,max}$ is usually achieved during relatively large muscle mass exercise and represents the integrative ability of the heart to generate a high cardiac output, total body haemoglobin, high muscle blood flow and muscle oxygen extraction, and in some cases the ability of the lungs to oxygenate the blood (Joyner & Coyle, 2008) Furthermore, cyclists who are able to tolerate high submaximal constant intensities, *i.e.* close to LT2, have a further advantage, since most of the racing time during professional road cycling completion is not spent at W_{max}. Accordingly, different road specialists have high power output at both LT1 and LT2 and possess the ability to generate those high powers of short duration during the mass start, steep climbing and at the race finish (Faria et al., 2005; Mujika & Padilla, 2001c). These physiological variables have been used for monitoring the training status of competitive cyclists in order to evaluate training methods and their efficacy, both during the competitive season and in the post-season break (Faria et al., 2005; Mujika & Padilla, 2003).

Cycling periodization in young top-level cyclists, who are not professionals, typically incorporates a transition period of reduced stress to allow physical and mental

recovery after the end of the competition season, *i.e.* complete training cessation in the off-season. However this period is usually longer than four weeks with no tradition of reduced training strategies, but instead the adoption of an almost sedentary lifestyle. Long term detraining has been defined as the partial or complete loss of training-induced anatomical, physiological and performance adaptations, as a consequence of more than four weeks training reduction or cessation and in response to an insufficient training stimulus (Mujika & Padilla, 2000a; Mujika & Padilla, 2000b). Large decreases in cardiorespiratory metabolic and muscular characteristics have been presented as a result of detraining in highly trained individuals (Mujika & Padilla, 2001a; Mujika & Padilla, 2001b). Consequently, specific athletic performance could decline quickly in high level athletes (Mujika & Padilla, 2000b).

The effects of training cessation have been investigated in athletes such as soccer players (Koundourakis et al., 2014), swimmers (Ormsbee & Arciero, 2012), kayakers (Garcia-Pallares, Sanchez-Medina, Perez, Izquierdo-Gabarren, & Izquierdo, 2010), handball players (Marques & Gonzalez-Badillo, 2006), rowers (Godfrey, Ingham, Pedlar, & Whyte, 2005) and runners (Houmard et al., 1992), but data are scarce in cycling after a period of long-term detraining. Thus, studies are needed, first to demonstrate the effects of cessation of training in a sport where physiological markers are determinants of performance, and second to challenge the traditional training concepts in young cyclists. A previous study evaluated the influence of aging on cardiovascular effects after two months of detraining in male cyclists showing nearly similar left ventricular morphological modifications in the two age groups (*i.e.,* young, range 19-25yr, and older, range 50-65yr) (Giada et al., 1998). However, it has not been investigated yet the effect of training cessation in highly trained young riders who aspire to become professional cyclers.

Therefore, the purpose of the present investigation was to examine the effects of five weeks of training cessation in young top-level road cyclists on body composition, haematological, and physiological parameters related to performance.

Methods

Study design

The present investigation is an observational study without a control group, where the cyclists completed two laboratory-based progressive exercise tests to assess selected physiological variables. One test was at the end of the competition phase of the cycling season (September = T1) and the second after five weeks of training cessation coinciding with the start of the season (November = T2). During the cessation period, cyclists discontinued any kind of physical training with no control over the cyclists' diet.

Participants

Ten young male road cyclists were recruited from the same cycling team. Characteristics of participants were: age 20.1±1.4 yr, body mass 68.4±6.3 kg, stature 177.9±5.8 cm (mean±SD) with a mean of two years of competitive experience at national level (range of 1-5 yr). Their $\dot{V}O_{2\,max}$ was 5.3±0.4 $L \cdot min^{-1}$, 78.5±5.5 $mL \cdot kg^{-1} \cdot min^{-1}$ and 1386±87$mL \cdot kg^{-0.32} \cdot min^{-1}$ with a W_{max} of 396±31W, 5.8±0.4 $W \cdot kg^{-1}$ and 103±7 $W \cdot kg^{-0.32}$. All participants competed at national standard or above covering a total of 20,000 to 25,000 km per year, with a mean weekly training duration of 18 to 22h.

The study was approved by the Bioethics Commission of the first author's University.

Procedures

Participants were accustomed to the experimental protocol. Laboratory conditions under which the cyclists performed the tests were controlled (*i.e.,* 19-23ºC and 40-50% humidity), including no exhaustive exercise during the 48 h before testing and a standardised diet, with no food intake three hours before the test, allowing water "ad libitum". Athletes were cooled using an electric fan during testing.

Anthropometry included stature, body mass, and six skinfold thicknesses (Harpenden, Germany) (subscapular, triceps brachii, supraspinale, abdominal, anterior thigh, medial calf). Skinfolds were assessed on the right side of the body by the same experienced investigator in accordance with guidelines from International Society for the Advancement of Kinanthropometry (Norton et al., 1996).

The assessment consisted of a progressive incremental protocol to volitional exhaustion on an electrically braked ergometer (Lode Excalibur Sport, Lode, Groningen, NL, software LODE v. 5.1.5) with increments of 35 W every 3 min. The ergometer was calibrated every day before starting the tests for intensities of 100-1000 W, and after that, prior to every single test. Each cyclist's bike setup (saddle height, reach, and handle bar height) was recorded and registered for both tests. Handle bar height and reach were also adjusted to allow a comfortable position. Clip pedals and set crank lengths of 170 mm were also used. These settings were replicated in the second trial. The tests were not preceded by any type of warming-up, and participants cycled at their freely chosen cadence at each intensity. Initial intensity was 100 W. Participants were asked to keep their cadence constant at their preferred rate based on visual feedback from a display unit. During the test, athletes were encouraged verbally by the laboratory technicians as well as by their team coach. The highest intensity

(W_{max}) was taken to be the highest a cyclist could maintain for a complete 3-min period. When the last intensity was not completed for 3 min, W_{max} was computed as: W_{max} = Wf + [(*t*/180) x 35], (Kuipers, Verstappen, Keizer, Geurten, & Van, 1985) where Wf is the value of the last completed intensity (in W), *t* is the time the last uncompleted intensity was maintained (in s), and 35 is the power output difference between the last two intensities. A single capillary blood sample was withdrawn from the left ear lobe immediately after completion of each intensity avoiding any contact with the electrode. Blood lactate concentration was determined with an automatic analyzer (Lactate Pro™). The analyzer was calibrated before each test as recommended by the manufacturer. The exercise intensity corresponding to LT2 was identified on the blood lactate concentration-power output curve by straight line interpolation between the two closest points as the power output eliciting a blood lactate concentration of 4 mmol·l^{-1} (Sjödin & Jacobs, 1981). The lactate threshold was identified on individual blood lactate concentration-power output curves as the exercise intensity eliciting a 1 mmol·l^{-1} increase in blood lactate concentration above mean base-line lactate values measured when exercising at 40–60% of W_{max} (Hagberg & Coyle, 1983). Intensities at LT2 (W_{LT2}) and LT1 (W_{LT1}) were also determined by straight line interpolation (Padilla, Mujika, Santisteban, Impellizzeri, & Goiriena, 2008). Maximum oxygen uptake was determined via a breath-by-breath automated gas analysis system (Jaeger Oxycon Delta System, Hoechberg,Germany) calibrated before each testing session in line with the manufacturer's guidelines. Maximum oxygen uptake was defined as the highest $\dot{V}O_2$ value attained toward the end of the test. Achievement of $\dot{V}O_{2\,max}$ was assumed on attainment of at least two of the following three criteria: a plateau in $\dot{V}O_2$ with increasing speeds (<2.0 mL·kg^{-1}·min^{-1}); a respiratory exchange ratio above 1.10; a heart rate within ±10 beats·min^{-1} of age predicted maximum heart rate (220-age) (Duncan, Howley, & Johnson, 1997). Previous studies have shown that mass exponents of 0.32 and 1 can evaluate level and uphill cycling ability, respectively

(Padilla et al., 1999). Therefore, because road cycling occurs in a variety of terrains (including uphill roads) both values adjusted to body mass raised to the power 0.32 and simple ratio standards were used to compare the participants (Winter, E.M. & Nevill, A.M., 2009).

For the determination of complete and differential blood counts (haematological variables), all venous blood samples were processed by impedancemetric method in a blood analyser habitually used in Medikosta laboratory (Sysmex XE 2100 Roche). All the samples were analysed within four hours of collection in BD Vacutainer Plastic SSTII Advance Tube 8.5 ml. The analysers (Sysmex XE 2100 Roche) were regularly calibrated and underwent quality controls, as described by the manufacturers. Blood was drawn between 8 am and 10 am in fasting condition, in the same specialized clinical chemistry centre for the two tests, using standardized venepuncture techniques in the antecubital vein in the bend of the elbow. Samples were collected by a phlebotomist.

Statistical analyses

Before all statistical analyses, data were checked for violations of normality using a Shapiro–Wilk test. To evaluate the effects of training cessation on each outcome, two-sample paired *t*-tests were performed using data at baseline and five weeks later. Change between T2 and T1 data were also presented as a percentage of the baseline values and were arcsine-transformed before being compared to reduce skewness. To distinguish the effects of detraining expressed in relative (normalized by dividing by body mass and by allometric scaling, *i.e*. body mass to the power of 0.32) and absolute terms, changes (expressed as percentages) were compared using two-

sample paired *t*-tests. Additionally, ninety-five per cent confidence interval (95% CI) constructed around means.

Effect size was calculated by dividing the difference between means for the outcome variable by the pooled standard deviation and interpreted as 0.0 to 0.19 trivial, 0.20 to 0.49 small, 0.50 to 0.79 moderate and 0.80 and above large (Cohen, 1988). Analyses were carried out with SPSS 15.0 (SPSS Inc, Chicago, USSA) software with alpha set at $P<0.05$. Values are reported as mean±standard deviation, unless otherwise stated.

Results

Body mass and composition. Changes in body composition are reported in Table 1. Compared with baseline (T1), body mass (68.4±6.3*vs* 70.1±7.2 kg; 95% CI=-2.8 to 0.4; ES=0.9; *P*=0.014, +2.3±2.4%) and sum of six skinfolds (45.2±9.5 *vs.* 50.6±7.4 mm; 95% CI=-13.5 to 2.7; ES=0.5; *P*=0.16; +15.1±23.7%) increased after five weeks of detraining period (at T2).

Haematological variables

There were decreases at T2 for red blood cell count (4.9±0.2 *vs.* 4.6±0.2 $10^6/\mu L$; 95% CI=0.1 to 0.5; ES=1.3; *P*=0.007; -6.6±4.8%), haemoglobin (14.9±0.7 *vs.* 14.0±0.5 g/dL; CI=0.3 to 1.4; ES=1.2; *P*=0.010, -5.4±4.3%) and haematocrit (43.0±2.0*vs.* 41.7±1.9%; CI=0.2 to 2.4; ES=0.9; *P*=0.028, -2.9±3.0%) after the training cessation period (Table 1).*Physiological variables*

There were decreases (*P*<0.001) at T2 in physiological responses for W_{max} (-6.5±3.1%, CI=0.3 to 0.6), $\dot{V}O_{2\,max}$ (-8.8±5.0%, CI=6.1 to 11.1), W_{LT1} (-12.9±7.0%,

CI=0.4 to 0.9), W_{LT2} (-11.5±7.0%, CI=0.4 to 0.9) (Table 1). There were larger decreases (*P*<0.05) when changes were expressed in relative terms (*i.e.* accounting for changes in body mass) when compared with absolute terms (Table 1 and Fig. 1). Results did not differ when measures allometrically scaled to $kg^{-0.32}$ compared with ratio standard (Table 1). Effect sizes for all the physiological variables between T1 and T2 were large (>1) (Table 1).

Table 1. Mean values±standard deviation for anthropometric, haematological and physiological values in the two tests.

Variables	T1	T2	*P* value1	ES1	%Change
ANTHROPOMETRICS					
Mass (kg)	68.4±6.3	70.1±7.2	0.014	0.9	2.3±2.4
Skinfolds (mm)	45.2±9.5	50.6±7.4	0.16	0.5	15.1±23.7
HAEMATOLOGICAL					
RBC (10^6/µL)	4.9±0.2	4.6±0.2	0.007	1.3	-6.6±4.8
Haemoglobin (g/dL)	14.9±0.7	14.0±0.5	0.010	1.2	-5.4±4.3
Haematocrit (%)	43.0±2.0	41.7±1.9	0.028	0.9	-2.9±3.0
PHYSIOLOGICAL					
VO_{2max} ($L \cdot min^{-1}$)	5.3±0.4	4.8±0.4	<0.001	1.7	-8.8±5.0
VO_{2max} ($mL \cdot kg^{-1} \cdot min^{-1}$)	78.5±5.5	69.9±4.6	<0.001	2.4	-10.8±4.2
VO_{2max} ($mL \cdot kg^{-0.32} \cdot min^{-1}$)	1386±87	1253±79	<0.001	2.0	-9.4±5.0
W_{max} (W)	396±31	371±28	<0.001	1.9	-6.5±3.1
W_{max} ($W \cdot kg^{-1}$)	5.8±0.4	5.3±0.3	<0.001	2.4	-8.5±3.3
W_{max} ($W \cdot kg^{-0.32}$)	103±7	95.8±5.1	<0.001	2.2	-7.1±3.0
W_{LT1} (W)	303±31	264±36	<0.001	2.1	-12.9±7.0
W_{LT1} ($W \cdot kg^{-1}$)	4.4±0.3	3.8±0.4	<0.001	2.0	-14.8±7.4
W_{LT1} ($W \cdot kg^{-0.32}$)	78.4±6.6	67.8±8.1	<0.001	2.1	-13.5±6.8
W_{LT2} (W)	336±36	298±40	<0.001	1.8	-11.5±7.0
W_{LT2} ($W \cdot kg^{-1}$)	4.9±0.4	4.3±0.5	<0.001	1.7	-13.4±7.6
W_{LT2} ($W \cdot kg^{-0.32}$)	87.0±7.6	76.4±9.1	<0.001	2.0	-12.1±7.0

T1, first test at the end of the competition phase of the cycling season. T2, second test after five weeks of training cessation. *P* value1, difference between T1 and T2. ES1, effect size between T1 and T2. % of change between tests. *P* value2, difference between absolute and relative values for the same variable. ES2, effect size between absolute and relative values for the same variable. RBC, red blood cells; $\dot{V}O_{2max}$, maximum oxygen uptake; W_{max}, maximum intensity; W_{LT1}, intensity at lactate threshold; W_{LT2}, intensity at onset of blood lactate accumulation.

Discussion

To our knowledge, this is the first study to report changes in body composition, haematological and physiological variables related to performance after five weeks of training cessation in young top-level cyclists. The results of the study indicate that all

the investigated outcomes were adversely affected by the abrupt cessation of training stimulus, with increases in body mass and decline in haematological and physiological variables (Table 1). The larger decreases for physiological variables expressed relative to body mass, emphasize the importance of the body mass of a cyclist and its influence on the outcomes associated with performance (Atkinson et al., 2003).

The present study has shown a decline in variables (absolute and relatives, *i.e*. normalized by dividing by body mass and by body mass to the power of 0.32, respectively) associated with endurance performance for the young top-level cyclists after five weeks of insufficient training stimulus (Table 1). Detraining has been shown to decrease the $\dot{V}O_{2\,max}$ by 6 to 20% due to reduced total blood volume (*i.e.* red cell volume plus plasma volume), and to increase blood lactate levels during submaximal exercise at the same absolute and relative intensities (Mujika & Padilla, 2000b). In the present study the mean decline (*i.e.* difference between T2 and T1 expressed as % of T1) in $\dot{V}O_{2\,max}$ was by 8 to 11%. These decreases were similar to those for swimmers after five weeks of swim detraining (-7.7%) (Ormsbee & Arciero, 2012) and kayakers after four weeks of detraining (-11.3%) (Garcia-Pallares et al., 2010). Longer periods of detraining (12 weeks) have shown mean decreases in $\dot{V}O_{2\,max}$ of 16% (Coyle et al., 1984). There is substantial evidence demonstrating that successful professional cyclists possess high $\dot{V}O_{2\,max}$ values, and those high values are required for cycling performance (Faria et al., 2005; Lucia, Pardo, Durantez, Hoyos, & Chicharro, 1998). It has also been shown that long term inactivity may promote a decline in cardiac dimensions and ventilatory efficiency, affecting both $\dot{V}O_{2\,max}$ and endurance performance of athletes (Giada et al., 1998; Mujika & Padilla, 2000b). The decline in $\dot{V}O_{2\,max}$ is mainly a consequence of a decrease in oxygen delivery to the muscle (Bosquet & Mujika, 2012). The rapid decrease in blood volume after the first days of

training cessation probably plays an important role in the cascade of events that reduces maximum cardiac output, and consequently $\dot{V}O_{2\,max}$ (Bosquet & Mujika, 2012; Coyle et al., 1984; Coyle, Hemmert, & Coggan, 1986). In the present study, the analysis of the haematological variables suggests a decrease in blood volume because of declines in red blood cell count (-6.6±4.8%) and haemoglobin (-5.4±4.3%). Rather than complete cessation of training, published studies have presented reduced training valuable strategies leading to, instead of detraining, maintenance of the physiological adaptations achieved during previous training periods (Mujika, 1998). Indeed, unchanged $\dot{V}O_{2\,max}$ has been reported during periods of reduced training (Hickson, Kanakis, Davis, Moore, & Rich, 1982; Madsen, Pedersen, Djurhuus, & Klitgaard, 1993). Accordingly, taking into account that $\dot{V}O_{2\,max}$ measurement remains recommended for the purpose of evaluating and selecting elite cyclists and as a prerequisite to perform at a high level (Barbeau, Serresse, & Boulay, 1993), it would be interesting to adopt these strategies (*i.e.,* reduced training programs) in athletes who have to continually undertake assessment for professional team selection. A superior performance in endurance sports, such as cycling, clearly requires high LT1 and LT2 values, since these have been shown to be better predictors of endurance performance than $\dot{V}O_{2\,max}$ (Atkinson et al., 2003). The findings of the present study for maximum data (*i.e.,* $\dot{V}O_{2\,max}$, W_{max}) showed smaller decreases (P<0.001) than the mean declines in submaximal values (*i.e.,* values at LT1 and LT2) also in absolute terms (-12.9±7.0% for W_{LT1} and -11.5±7.0% for W_{LT2}). Similarly, in an elite rower after eight weeks of training cessation greater decreases occurred in LT1 (27%) than at peak intensity (20%) (Godfrey et al., 2005). Thus, the excessive loss of performance at these metabolic zones (*i.e.* LT1 and LT2) during the off-season in the present study could have undesired detrimental consequences for the cyclists' performance in subsequent competitive season. Previous studies that investigated intensity of exercise during

mass-start stage races in professional road cycling, concluded that aerobic capability dominated and that most of competition time during the mass start stages was spent at intensities near LT1 (Padilla et al., 2001; Vogt et al., 2006). Participants of the present study cover around 25,000 km in a year with in excess of 50 days of competition and participation in races on more than three consecutive days. Nevertheless, since they were younger than professional riders, and consequently had fewer years of training volume, the decline at these submaximal metabolic zones (*i.e.* LT1 and LT2) after a detraining period could have for a larger impact on performance compared with professional riders who have experienced more regular and structured training. In this regard, recently, it has been shown that older trained individuals appear to have smaller age-related declines in both maximum and submaximal exercise responses than younger trained ones (Hopker et al., 2013). Additionally, the decrease in maximum power output (396 *vs*. 370 $W_{max,}$ -6.5±3.1%; 5.8 *vs*. 5.3 $W \cdot kg^{-1}$, -8.5±3.3% and 102.5 *vs*. 95.1 $W \cdot kg^{-0.32}$, -7.1±3.0% from T1 to T2) after five weeks of training cessation is estimated to correspond to a reduction in cycling speed of 1.3 $km \cdot h^{-1}$ in a 1-h race (Bassett, Jr., Kyle, Passfield, Broker, & Burke, 1999). Thus, there is little doubt about the consequences of training cessation on performance.

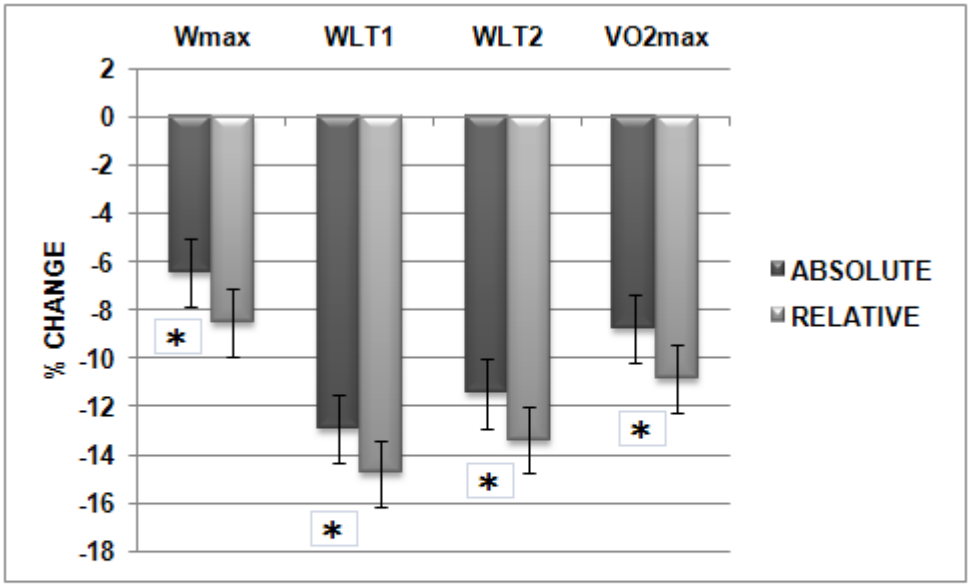

Figure 1. Percentages of change for physiological and performance variables.

$\dot{V}O_{2max}$, maximum oxygen uptake; W_{max}, maximum intensity; W_{LT1}, intensity at lactate threshold; W_{LT2}, intensity at onset of blood lactate accumulation

Difference between all absolute and relative values (*$p < .05$)

For the physiological outcomes, there were larger decreases (P<0.05) in relative (to body mass) changes than for absolute changes (Fig. 1). This difference is related to the increase in body mass (2.3±2.4%, P=0.01) and also the increase in sum of six skinfolds (15.1±23.7%, P=0.16) at T2. It is well known that body mass, and specifically body fat, is a key factor that limits endurance performance because it determines gravity-dependent resistance, having a major influence on uphill cycling performance (Padilla et al., 1999). A key limitation of the present study is the lack of a control group who continued with a reduced training programme. A previous study has shown that a reduced training intensity and volume for 21 days could maintain physiological adaptations, as measured during submaximal and maximal exercise (Rietjens, Keizer, Kuipers, & Saris, 2001).

Given that improvements from retraining after training cessation take considerably longer to achieve than losses from detraining (Godfrey et al., 2005), there is a need to programme some endurance stimuli during the off-season period to minimize losses in physiological and performance measures in top-level cyclists. It is clear that during the break after the competition season, an alternative training stimulus including exercises that involve the same muscle groups as the competitive activity are necessary to maintain the metabolic adaptations to training (Bosquet & Mujika, 2012).

Conclusion

In young top-level cyclists, five weeks of training cessation results in large decreases in haematological and both submaximal and maximal physiological variables. This long-term detraining is more pronounced when expressed relative to body mass via allometric scaling, emphasizing the relevance of the body mass of cyclist and its influence on performance. This suggests that the training break should be shorter and that a maintenance programme should be implemented to avoid such a large decline in physiological values in young cyclists who aspire to become professionals. We interpret these findings as being consistent with previous investigations in other sports, emphasizing the importance of establishing the optimal training load in each phase of the training plan to avoid excessive declines in performance.

Acknowledgments

The authors would like to thank the cyclists of Seguros Bilbao Cycling Team for their participation, effort and support in this investigation.

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