

Improved muscular efficiency displayed as Tour de France champion matures

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Coyle, Edward F. Improved muscular efficiency displayed as Tour de France champion matures. *J Appl Physiol* 98: 2191–2196, 2005. First published March 17, 2005; doi:10.1152/jappphysiol.00216.2005.— This case describes the physiological maturation from ages 21 to 28 yr of the bicyclist who has now become the six-time consecutive Grand Champion of the Tour de France, at ages 27–32 yr. Maximal oxygen uptake ($\dot{V}O_{2\max}$) in the trained state remained at ~ 6 l/min, lean body weight remained at ~ 70 kg, and maximal heart rate declined from 207 to 200 beats/min. Blood lactate threshold was typical of competitive cyclists in that it occurred at 76–85% $\dot{V}O_{2\max}$, yet maximal blood lactate concentration was remarkably low in the trained state. It appears that an 8% improvement in muscular efficiency and thus power production when cycling at a given oxygen uptake ($\dot{V}O_2$) is the characteristic that improved most as this athlete matured from ages 21 to 28 yr. It is noteworthy that at age 25 yr, this champion developed advanced cancer, requiring surgeries and chemotherapy. During the months leading up to each of his Tour de France victories, he reduced body weight and body fat by 4–7 kg (i.e., $\sim 7\%$). Therefore, over the 7-yr period, an improvement in muscular efficiency and reduced body fat contributed equally to a remarkable 18% improvement in his steady-state power per kilogram body weight when cycling at a given $\dot{V}O_2$ (e.g., 5 l/min). It is hypothesized that the improved muscular efficiency probably reflects changes in muscle myosin type stimulated from years of training intensely for 3–6 h on most days.

maximum oxygen uptake; blood lactate concentration

MUCH HAS BEEN LEARNED about the physiological factors that contribute to endurance performance ability by simply describing the characteristics of elite endurance athletes in sports such as distance running, bicycle racing, and cross-country skiing. The numerous physiological determinants of endurance have been organized into a model that integrates such factors as maximal oxygen uptake ($\dot{V}O_{2\max}$), the blood lactate threshold, and muscular efficiency, as these have been found to be the most important variables (7, 8, 15, 21). A common approach has been to measure these physiological factors in a given athlete at one point in time during their competitive career and to compare this individual's profile with that of a population of peers (4, 6, 15, 16, 21). Although this approach describes the variations that exist within a population, it does not provide information about the extent to which a given athlete can improve their specific physiological determinants of endurance with years of continued training as the athlete matures and reaches his/her physiological potential. There are remarkably few longitudinal reports documenting the changes in physiological factors that accompany years of continued endurance training at the level performed by elite endurance athletes.

This case study reports the physiological changes that occur in an individual bicycle racer during a 7-yr period spanning

ages 21 to 28 y. Description of this person is noteworthy for two reasons. First, he rose to become a six-time and present Grand Champion of the Tour de France, and thus adaptations relevant to this feat were identified. Remarkably, he accomplished this after developing and receiving treatment for advanced cancer. Therefore, this report is also important because it provides insight, although limited, regarding the recovery of "performance physiology" after successful treatment for advanced cancer. The approach of this study will be to report results from standardized laboratory testing on this individual at five time points corresponding to ages 21.1, 21.5, 22.0, 25.9, and 28.2 yr.

METHODS

General testing sequence. On reporting to the laboratory, training, racing, and medical histories were obtained, body weight was measured (± 0.1 kg), and the following tests were performed after informed consent was obtained, with procedures approved by the Internal Review Board of The University of Texas at Austin. Mechanical efficiency and the blood lactate threshold (LT) were determined as the subject bicycled a stationary ergometer for 25 min, with work rate increasing progressively every 5 min over a range of 50, 60, 70, 80, and 90% $\dot{V}O_{2\max}$. After a 10- to 20-min period of active recovery, $\dot{V}O_{2\max}$ when cycling was measured. Thereafter, body composition was determined by hydrostatic weighing and/or analysis of skin-fold thickness (34, 35).

Measurement of $\dot{V}O_{2\max}$. The same Monark ergometer (model 819) equipped with a racing seat and drop handlebars and pedals for cycling shoes was used for all cycle testing, and seat height and saddle position were held constant. The pedal's crank length was 170 mm. $\dot{V}O_{2\max}$ was measured during continuous cycling lasting between 8 and 12 min, with work rate increasing every 2 min. A leveling off of oxygen uptake ($\dot{V}O_2$) always occurred, and this individual cycled until exhaustion at a final power output that was 10–20% higher than the minimal power output needed to elicit $\dot{V}O_{2\max}$. A venous blood sample was obtained 3–4 min after exhaustion for determination of blood lactate concentration after maximal exercise, as described below. The subject breathed through a Daniels valve; expired gases were continuously sampled from a mixing chamber and analyzed for O_2 (Applied Electrochemistry S3A) and CO_2 (Beckman LB-2). Inspired air volumes were measured using a dry-gas meter (Parkinson-Cowan CD4). These instruments were interfaced with a computer that calculated $\dot{V}O_2$ every 30 s. The same equipment for indirect calorimetry was used over the 7-yr period, with gas analyzers calibrated against the same known gasses and the dry-gas meter calibrated periodically to a 350-liter Tissot spirometer.

Blood LT. The subject pedaled the Monark ergometer (model 819) continuously for 25 min at work rates eliciting $\sim 50, 60, 70, 80,$ and 90% $\dot{V}O_{2\max}$ for each successive 5-min stage. The calibrated ergometer was set in the constant power mode, and the subject maintained a pedaling cadence of 85 rpm. Blood samples were obtained either from

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a catheter in an antecubital vein or from piercing a fingertip during the 5th min of exercise at each stage or 4 min after maximal exercise. Whole blood was deproteinized in perchloric acid and later analyzed for lactate using an enzymatic spectrophotometric method (20). The blood LT was determined, as previously described (14), by graphing the lactate vs. $\dot{V}O_2$ relationship and determining the $\dot{V}O_2$ at which blood lactate increased 1 mM above baseline. Maximal blood lactate concentration was determined from a blood sample obtained during the 4th min after exhaustion during the $\dot{V}O_{2\max}$ determination.

Mechanical efficiency. Gross efficiency was calculated as the ratio of work accomplished per minute (i.e., watts converted to kcal/min) to energy expended per minute (kcal/min). Energy expenditure per minute (i.e., kcal/min) was calculated from $\dot{V}O_2$ and respiratory exchange ratio using the tables of Lusk (31). On a given date of testing, gross efficiency was generally similar at all work rates evaluated when cycling at 50–90% $\dot{V}O_{2\max}$ and 80–90 rpm, as previously described in trained cyclists (10, 31). Therefore, gross efficiency was reported as the average of the values obtained at the five work rates (10).

Delta efficiency is defined as the ratio of the change in work accomplished per minute and the change in energy expended per minute (10, 31). Delta efficiency was identified from linear regression ($y = mx + b$) of the relationship (i.e., 5 data points at ~50, 60, 70, 80, and 90% $\dot{V}O_{2\max}$) between energy expended per minute (i.e., y ; kcal/min) vs. work accomplished per minute (i.e., x ; kcal/min). Delta efficiency was calculated from the slope of the relationship and was equal to the reciprocal of m (i.e., $1/m$) (31).

Body composition. Body density was determined from hydrostatic weighing, with direct measurement of residual lung volume using the nitrogen dilution technique (34, 35). Furthermore, skinfold thickness at five sites was determined, and the sum of these measures was related to body density. Percent body fat and lean body weight were calculated from body density and body weight (35).

RESULTS

Training and medical history of the subject. This individual was born on September 18, 1971. He engaged in competitive swimming at ages 12–15 yr and competitive running and triathlon racing at ages 14–18 y. Thereafter, he competed in and trained primarily for bicycle road racing. Table 1 contains

Table 1. Highlights of the bicycling racing history and medical history of the subject

Year	Age, yr	Event
1991	19	U.S.A. National Amateur Champion
1992	20	14th place in Olympic Road Race; Barcelona
1993	21	1st place in World Championships, Road Racing; Oslo. Winner, one stage in Tour de France
1995	23	Winner of one stage in Tour de France
1996	24–25	12th place in Olympic Road Race; Barcelona. 6th place in Olympic Individual Time trial; Barcelona. Diagnosed with testicular cancer; chemotherapy; brain surgery in October 1996. Last chemotherapy treatment December 1996.
1998	26	4th place in World Championships, Road Racing 4th place in World Championships, Time trial
1999	27	1st place—Tour de France Grand Champion
2000	28	1st place—Tour de France Grand Champion 13th place in Olympic Road Race; Sydney 3rd place in Olympic Individual Time trial; Sydney
2001	29	1st place—Tour de France Grand Champion
2002	30	1st place—Tour de France Grand Champion
2003	31	1st place—Tour de France Grand Champion
2004	32	1st place—Tour de France Grand Champion

the highlights of his racing career from 1991 to 2004, with focus on his placing in the Tour de France, the World Bicycling Championships, and the Olympic Games. Before turning 22 yr old in 1993, he became the youngest winner of the World Championships in Bicycle Road Racing, a 1-day road race. At age 25 yr, this individual was diagnosed with testicular cancer. Thereafter and during the period of October through December of 1996, he underwent surgeries to remove the involved testicle and then to remove cancerous brain tumors and he received chemotherapy as described by Armstrong (1). He resumed international bicycle racing in 1998 and remarkably placed 4th in the World Championships that year. He went on to become the now six-time Grand Champion of the Tour de France over years 1999, 2000, 2001, 2002, 2003, and 2004. The Tour de France is arguably the world's premier bicycle road race. It covers ~3,800 km, competed in 21–22 stages (day of racing) over a period of 3 wk during the month of July.

Anthropometry. Total body weight during laboratory testing ranged from ~76 to 80 kg from 1992 through 1997 as well as during the preseason in 1999. However, when competing in the Tour de France in 1999–2004, body weight was reported by the subject to be ~72–74 kg. Lean body weight was ~70 kg during the period of 1992–1997 (Table 2). His height was ~178 cm.

$\dot{V}O_{2\max}$, maximal heart rate, and the blood LT. $\dot{V}O_{2\max}$ during the preseason months of November through January generally ranged from 5.56 to 5.82 l/min during the period of 1992–1999. $\dot{V}O_{2\max}$ during the competitive season of 1993, soon after winning the World Road Racing Championships (September 1993), was 6.1 l/min and 81.2 ml·kg⁻¹·min⁻¹, results that were corroborated by the United States Olympic Committee (Colorado Springs, CO). Eight months after chemotherapy for cancer and during a period of inconsistent and reduced training (i.e., August 1997), $\dot{V}O_{2\max}$ was 5.29 l/min and 66.6 ml·kg⁻¹·min⁻¹. Furthermore, at this time of reduced training, maximal blood lactate concentration measured 4 min after exhaustion was 9.2 mM compared with previously recorded values in the range of 6.3–7.5 mM. Maximal heart rate declined from 207 to 200 beats/min from 1992 through 1999. The $\dot{V}O_2$ corresponding to the blood lactate threshold was 4.5–4.7 l/min when measured in 1992–1993 and, as expected, it was reduced to 4.02 l/min during the period of reduced training in August 1997.

Mechanical efficiency. Gross efficiency and delta efficiency during the period from 1992 to 1999 are displayed in Fig. 1. These progressive increases in efficiency amount to an 8–9% improvement over the period. This improvement is also displayed in the measure of mechanical power generated when cycling at a given $\dot{V}O_2$ of 5.0 l/min, in that it increased from 374 to 403 W (i.e., 8%; Table 2). Given that success in the Tour de France is typically determined when cycling uphill on mountains, it is best to normalize power to body weight (i.e., W/kg). Given this individual's reduction in body weight from 78.9 kg (in 1992) to ~72 kg during his victories in the Tour de France and given his increased muscular efficiency, his power-to-body weight ratio (i.e., power/kg) when cycling at 5.0 l/min is calculated to have increased by a remarkable 18% from 1992 to 1999 (i.e., 4.74 vs. 5.60 W/kg when $\dot{V}O_2$ is 5.0 l/min). In that his $\dot{V}O_{2\max}$ remained at ~6 l/min, this given $\dot{V}O_2$ of 5.0 l/min represents ~83% $\dot{V}O_{2\max}$. Therefore, his "power per kilo-

Table 2. *Physiological characteristics of this individual from the ages of 21 to 28 yr*

	Age, yr				
	21.1	21.4	22.0	25.9	28.2
Date: Month-Year	Nov 1992	Jan 1993	Sept 1993	Aug 1997	Nov 1999
Training stage	Preseason	Preseason	Racing	Reduced	Preseason
<i>Anthropometry</i>					
Body weight, kg	78.9	76.5	75.1	79.5	79.7
Lean body weight, kg	70.5	69.8		70.2	71.6
Body fat, %	10.7	8.8		11.7	
<i>Maximal aerobic ability</i>					
Maximal O ₂ uptake, l/min	5.56	5.82	6.10	5.29	5.7
Maximal O ₂ uptake, ml·kg ⁻¹ ·min ⁻¹	70.5	76.1	81.2	66.6	71.5
Maximal heart rate, beats/min	207	206	202	200	200
Maximal blood lactic acid, mM	7.5	6.3	6.5	9.2	
<i>Lactate threshold</i>					
Lactate threshold O ₂ uptake, l/min	4.70	4.52	4.63	4.02	
Lactate threshold, % maximal O ₂ uptake	85	78	76	76	
<i>Mechanical efficiency</i>					
Gross efficiency, %	21.18	21.61		22.66	23.05
Delta efficiency, %	21.37	21.75		22.69	23.12
Power at O ₂ uptake of 5.0 l/min, W	374	382		399	404

gram” at a given percentage of $\dot{V}O_{2\max}$ (e.g., 83%) increased by 18%.

DISCUSSION

This case study has described the physiological characteristics of a renowned world champion road racing bicyclist who is currently the six-time Grand Champion of the Tour de France. It reports that the physiological factor most relevant to performance improvement as he matured over the 7-yr period from ages 21 to 28 yr was an 8% improvement in muscular efficiency when cycling. This adaptation combined with relatively large reductions in body fat and thus body weight (e.g., 78–72 kg) during the months before the Tour de France contributed to an impressive 18% improvement in his power-to-body weight ratio (i.e., W/kg) when cycling at a given $\dot{V}O_2$ (e.g., 5.0 l/min or $\sim 83\%$ $\dot{V}O_{2\max}$). Remarkably, this individual was able to display these achievements despite the fact that he developed advanced cancer at age 25 yr and required surgeries and chemotherapy.

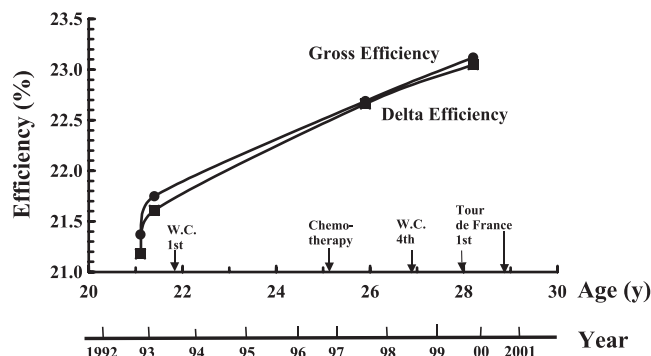


Fig. 1. Mechanical efficiency when bicycling expressed as “gross efficiency” and “delta efficiency” over the 7-yr period in this individual. WC, World Bicycle Road Racing Championships, 1st and 4th place, respectively. Tour de France 1st, Grand Champion of the Tour de France in 1999–2004.

In the trained state, this individual possessed a remarkably high $\dot{V}O_{2\max}$ of ~ 6 l/min, and his blood LT occurred at a $\dot{V}O_2$ of ~ 4.6 l/min (i.e., 76–85% $\dot{V}O_{2\max}$). These physiological factors remained relatively stable from age 21 to 28 yr. These absolute values are higher than what we have measured in bicyclists competing at the US national level (9), several of whom subsequently raced professionally in Europe during the period of 1989–1995. The five-time Grand Champion of the Tour de France during the years 1991–1995 has been reported to possess a $\dot{V}O_{2\max}$ of 6.4 l/min and 79 ml·kg⁻¹·min⁻¹ with a body weight of 81 kg (28). Laboratory measures of the subject in our study were not made soon after the Tour de France; however, with the conservative assumption that $\dot{V}O_{2\max}$ was at least 6.1 l/min and given his reported body weight of 72 kg, we estimate his $\dot{V}O_{2\max}$ to have been at least 85 ml·kg⁻¹·min⁻¹ during the period of his victories in the Tour de France. Therefore, his $\dot{V}O_{2\max}$ per kilogram of body weight during his victories of 1999–2004 appears to be somewhat higher than what was reported for the champion during 1991–1995 and to be among the highest values reported in world class runners and bicyclists (e.g., 80–85 ml·kg⁻¹·min⁻¹) (6, 15, 16, 28, 29).

It is generally appreciated that in addition to a high $\dot{V}O_{2\max}$, success in endurance sports also requires an ability to exercise for prolonged periods at a high percentage of $\dot{V}O_{2\max}$ as well as the ability to efficiently convert that energy (i.e., ATP) into muscular power and velocity (5, 7, 8, 29). Identification of the blood LT (e.g., 1 mM increase in blood lactate above baseline) in absolute terms or as a percentage of $\dot{V}O_{2\max}$ is, by itself, a reasonably good predictor of aerobic performance (i.e., time that a given rate of ATP turnover can be maintained) (7, 8, 14, 21), and prediction is strengthened even more when measurement of muscle capillary density is combined with LT (11). Capillary density is thought to be an index of the working muscle’s ability to clear fatiguing metabolites (e.g., acid) from muscle fibers into the circulation, whereas the LT is thought

to reflect production of fatiguing metabolites in muscle fibers (7, 8).

As expected, this individual possessed a high LT in the range of 76–85% $\dot{V}_{O_2 \max}$. However, the most unique aspect of this individual's blood lactate profile was the extremely low lactate concentration measured 4 min after exhaustion during measurement of $\dot{V}_{O_2 \max}$. Maximal blood lactate in the trained state was only 6.5–7.5 mM in the present subject. By comparison, all the competitive cyclists we have tested, including team mates training with this subject, possessed maximal blood lactate postexercise in the range of 9–14 mM (9, 11). The mechanism for this extremely low maximal blood lactate concentration in this individual is not clear, although it probably reflects reduced lactate production when exercising to exhaustion at intensities above $\dot{V}_{O_2 \max}$. One possibility is that activity of the muscle enzymes largely responsible for lactate production [i.e., lactate dehydrogenase (LDH) and phosphofruktase] are greatly attenuated in this individual when he is trained (12, 24). It should be noted that this individual indeed became exhausted during $\dot{V}_{O_2 \max}$ testing, displaying the typical pattern for competitive cyclist, including a "plateau" of \dot{V}_{O_2} and heart rate at maximal values for 1–3 min, moderate hyperventilation, respiratory exchange ratio >1.05, and a progressive loss of pedal cadence at constant power during the 30–60 s before exhaustion.

Interestingly, when $\dot{V}_{O_2 \max}$ and maximal blood lactate concentration were measured during the period of reduced training 8 mo after chemotherapy (age 25.9 yr), maximal blood lactate concentration was increased to 9.2 mM. This agrees with our previous observation that detraining in well-trained endurance athletes increases maximal blood lactate from 10 to 12.5 mM in association with a 21% increase in total LDH activity (i.e., 21%) (12, 13, 24). It should be noted that blood lactate concentration was measured, in this study and in our previous studies, exclusively with spectrophotometric analysis of NADH produced from the LDH reaction after completely lysing red blood cells (20). It has been our experience that maximal lactate concentrations measured using commercially available automated analyzers are lower, possibly due to incomplete lysing of red blood cells despite addition of detergent into the reagents (3).

Physiological evaluation was performed 8 mo after chemotherapy during a period of reduced training. Regarding his prior training, during the 3rd and 4th mo after chemotherapy, he cycled ~5 day/wk for 2–5 h/day at moderate intensity. During the 5th and 6th mo, training intensity was increased. During the 6- to 7.5-mo period after chemotherapy, he did not perform endurance training. However, during the 8 days before our physiological laboratory evaluation (i.e., 8 mo after chemotherapy), he bicycled 1–2 h/day at moderate intensity, eliciting heart rates of 120–150 beats/min. During this laboratory evaluation 8 mo after completing chemotherapy, this individual displayed no ill effects from his previous surgeries and chemotherapy. In particular, ventilatory volume during maximal exercise appeared typical, and his cardiovascular responses were normal at heart rates of 120–150 beats/min. Furthermore, maximal heart rate achieved the healthy level for this individual (i.e., 200 beats/min). However, as expected from his reduced training, $\dot{V}_{O_2 \max}$ was lowered by 6–12% to 5.3 l/min and 67 ml·kg⁻¹·min⁻¹. If this individual performed

no training for 3 mo, we predicted his $\dot{V}_{O_2 \max}$ would stabilize at 5 l/min (e.g., 61–63 ml·kg⁻¹·min⁻¹ for a body weight of 80 kg) based on our previous measurements in well-trained endurance athletes during detraining (13; see Fig. 1). A $\dot{V}_{O_2 \max}$ in the range of 56–62 ml·kg⁻¹·min⁻¹ is generally believed to be the highest value that the average man who is not genetically endowed for endurance can achieve with prolonged and very intense endurance training (13, 23). As such, it appears that in the detrained state, this individual's $\dot{V}_{O_2 \max}$ is in the range of the highest values than normal men can achieve with training.

The physiological mechanisms responsible for the 8% improvements in both gross and delta efficiency when cycling, as well as the stimuli that provoked this adaptation, are unclear. The observation that both gross and delta efficiency improved to the same extent and also with the same time course (Fig. 1) suggests an improved efficiency of ATP turnover within muscle fibers during contraction (10, 31). This is because the measure of delta efficiency, defined as the increase in power output relative to the rate of increase in energy expenditure (calculated from \dot{V}_{O_2}) throughout a wide range of work rates provides the best reflection of power production from actin-myosin cross-bridge turnover in the active muscles (26) as it eliminates or minimizes the influence of the energy cost of unloaded cycling, ventilatory work, and other metabolic processes not directly linked to muscle power production (31). We previously reported from cross-sectional observation of competitive bicyclists that the percentage of type I muscle fibers of the vastus lateralis is directly and positively related to both delta and gross mechanical efficiency measured either during bicycling or with the simple task of knee extension (10, 25). Therefore, one possible mechanism for increased efficiency is that this individual increased his percentage of type I muscle fibers during this 7-yr period of study.

Using our previously reported prediction of the percentage type I muscle fibers from our direct measurements of gross and mechanical efficiency in this individual, we predict that he might have increased his percentage of type I muscle fibers from 60 to 80%. Interestingly, this magnitude of increase in percentage of type I fibers with 7 yr of continued endurance training in this individual is remarkably similar to our prediction made in 1991 based on cross-sectional observations of competitive cyclists (9; see Fig. 8). To our knowledge, there have been no longitudinal studies performed over years on humans directly testing the hypothesis that type II fibers can be converted to type I muscle fibers with continued intense endurance training. However, during periods of extreme endurance training of rats, skeletal muscle appears to display conversion of type II to type I fibers (18). Other factors that have been reported to increase cycling efficiency and running economy are intermittent exposure to hypoxia for several weeks as encountered by athletes who spend periods living at high altitude or in hypoxic environments (19, 30). Like many endurance athletes, this individual has incorporated hypoxic exposure into his annual plan, which may be another factor contributing to improved cycling efficiency.

It has been recognized for decades that endurance training of rats increases the myosin ATPase activity of type I fibers while decreasing it in type II fibers (2). More recent studies on humans by Fitts, Costill, and colleagues (17, 32, 33) directly

measured maximal velocity of shortening of isolated single muscle fibers (i.e., using the slack test) obtained from biopsy samples. Ten weeks of intense swimming (e.g., 4–5 km/day) increased the maximal velocity of type I fibers, whereas in type II fibers it was decreased (17). Furthermore, Widrick et al. (32, 33) found that men who performed high levels of physical activity for 20–25 yr and who were elite master runners also displayed increased maximal velocity of type I fibers that was associated with altered myosin type (i.e., 28% greater myosin light chain 3 vs. 2). Therefore, intense endurance training performed for prolonged periods results in alterations in myosin ATPase activity whereby type II become more like type I fibers and type I fibers increase ATPase activity and alter myosin type and increase maximal velocity of shortening. These observations support the possibility that in the subject of the present study, 7 yr of extremely intense endurance training and improved muscular efficiency when cycling was related to altered myosin type that allowed more of the energy released from ATP hydrolysis during contraction to be converted to power production.

Muscle samples were not surgically obtained from this athlete to directly test the hypothesis that muscle fiber-type conversion contributed to the large increases in mechanical or muscular efficiency when cycling. Therefore, this hypothesis that the percentage of type I muscle fibers increased in this individual requires identification of other performance characteristics that clearly changed in this individual over that 7-yr period with discussion as to whether they are consistent with the hypothesis of increased percentage of type I muscle fibers. Although during all laboratory measures of mechanical efficiency, cycling cadence was held constant at 85 rpm, this individual's freely chosen cycling cadence during time trial racing of 30- to 60-min duration increased progressively during this 7-yr period from ~85–95 rpm to ~105–110 rpm. This increase in freely chosen revolutions per minute when cycling at high intensity is indeed consistent with increases in type I muscle fibers because cyclists with a higher percentage of type I fibers choose a higher pedaling cadence when exercising at high power outputs (22). Although this may initially seem paradoxical, higher cycling cadence serves to both bring muscle fiber contraction velocity closer to that of maximum power and reduce the muscle and pedaling force required for each cycling stroke. Keep in mind that when exercising at a given rate of oxidative metabolism, an 8% increase in mechanical efficiency will result in 8% more muscle power and force development on the pedals when cycling cadence is held constant. As cycling efficiency increases due to increased percentage of type I muscle fibers, it is possible that increased power is manifested by increasing cycling cadence (i.e., velocity) rather than increasing the muscle forces directed to the pedals. This approach appears to produce less sensation of effort relative to muscular strength (27). Therefore, it is likely that the increases in freely chosen cycling cadence displayed over the years by this Tour de France champion reflect his increased mechanical efficiency, agreeing with the pattern expected to result from muscle fiber conversion from type II to type I.

This report has identified the physiological factor that improved the most from ages 21 to 28 yr in the bicyclist who has now become the six-time consecutive Grand Champion of the

Tour de France as muscular efficiency. As a result, power production when cycling at an absolute $\dot{V}O_2$ of 5.0 l/min increased by 8%. Another factor that allowed this individual to become Grand Champion of the Tour de France was his large reductions in body weight and body fat during the months before the race. Therefore, over the 7-yr period, he displayed a remarkable 18% improvement in steady-state power per kilogram body weight when cycling at a given $\dot{V}O_2$ (e.g., 5 l/min). We hypothesize that the improved muscular efficiency might reflect alterations in muscle myosin type stimulated from years of training intensely for 3–6 h on most days. It is remarkable that at age 25 yr this individual developed advanced cancer, requiring surgeries and chemotherapy, yet these events did not appear to impede his physiological maturation and athletic achievements. Clearly, this champion embodies a phenomenon of both genetic natural selection and the extreme to which the human can adapt to endurance training performed for a decade or more in a person who is truly inspired.

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Very intense exercise-training is extremely potent and time efficient: a reminder

THIS ISSUE of the *Journal of Applied Physiology* contains a report by Burgomaster et al. (2) that reminds us of the “potency” of very intense exercise, performed as 30-s sprints, for stimulating metabolic adaptations within skeletal muscle. This group employed “sprint interval training” on a bicycle ergometer, involving 30-s sprints performed “all out,” with 4 min of recovery. Recreationally active college students performed only 2–4 min of exercise per session and just six sessions over 2 wk. The remarkable finding of this study was that this small total amount of very intense exercise training was sufficient to “double” the length of time that intense aerobic exercise could be maintained (i.e., from 26 to 51 min). Although peak oxygen uptake was not increased, aerobic adaptations did occur within active skeletal muscle as reflected by a 38% increase in activity of the mitochondrial enzyme citrate synthase.

This study is significant because it contains a “documented” first, and more importantly it serves as a reminder to the scientific community and society. It appears that this is the first scientific documentation that very intense sprint training in untrained people can markedly increase aerobic endurance and that the total “dose” of exercise over the 2-wk period, performed in six sessions, amounted to only 15 min. This serves as a dramatic reminder of the potency of exercise intensity for stimulating adaptations in skeletal muscle that improve performance and have implications for improving health. In other words, we are reminded that intense sprint interval training is very time efficient with much “bang for the buck.”

The findings of Burgomaster et al. (2) challenge the concept that aerobic endurance performance is only enhanced by aerobic endurance training. On the surface, this concept seems logical, but it has been long ago proven wrong both in the realm of athletics as well as muscle biochemistry. In athletics, this concept is not generally held by elite athletes competing in middle-distance running because they incorporate sprint interval training to improve aerobic endurance. Sprint interval training was introduced by Woldemar Gerschler 70 yr ago and was followed by world-record performances. Indeed, it is likely that if an experienced runner or bicyclist had only 2 wk and very limited time to prepare for a race of ~30-min duration, that sprint interval training would become a mainstay of their preparation. Therefore, this scientific report of the potency of sprint interval training appears to have practical support in athletics. Furthermore, it seems to have gained some recent popularity in the fitness and health club industry in the form of “spinning” cycling classes. From the perspective of muscle biochemistry, it has long been recognized that 6–8 wk of sprint interval training increases aerobic enzyme activity in muscle (5, 6, 9, 10).

The authors Burgomaster et al. (2) “hope that the present observations will stimulate additional research to clarify the precise nature, time course, and significance of the physiological adaptations induced by short sprint interval training.” The large increase in citrate synthase activity in skeletal muscle, a marker of mitochondrial activity, implies that a host of adaptations typical of aerobic endurance training have been initiated. The exercise stimulus that promotes mitochondrial biogenesis also appears to stimulate other healthy metabolic adaptations in skeletal muscle, such as improved insulin action, improved lipoprotein lipase activity, and greater clearance of

plasma triglycerides (1, 3, 8). Future research might focus on the ability of short-term sprint interval training for promoting these healthy adaptations. Given the growing epidemic of physical inactivity in today’s modern society, the effectiveness of all types of exercise and methods of delivery needs to be considered. As discussed by Chakravarthy and Booth (3), “a continuous sedentary lifestyle has resulted in a stalling of glycogen and triglyceride stores at high levels in skeletal muscle and of those proteins producing their cycling with physical activity-rest cycles. Conceivably, such metabolic stalling may cause the organism to cross a biological threshold, beyond which chronic health conditions develop.” Sprint interval training has promise as an efficient method to reverse metabolic stalling in skeletal muscle in a segment of the sedentary population.

Obviously, sprint interval training performed all-out and repeatedly requires a high level of motivation, and it causes a feeling of severe fatigue lasting for at least 10–20 min. That is the “price” for its effectiveness and remarkable time efficiency. It remains to be determined which populations, depending on age, health status, and psychology, are mostly likely to adhere and benefit from sprint interval training. As mentioned, competitive endurance athletes have long realized its importance. They have also come to appreciate the need for a coach and training partners to provide accountability and motivation during such training. Exercise physiologists, working with other health care professionals, need to better develop a clinical application of sprint interval training. Additionally, the incidence of injury needs to be studied. Chance for impact injury during sprint stationary cycling or swimming seems low and might be compared with sprint running, for example.

The literature contains a number of reports that 6–8 wk of either sprint interval training or “prolonged submaximal aerobic exercise training” promotes increased mitochondrial enzyme activity in skeletal muscle (5, 6). It has also been demonstrated that significant increases in skeletal muscle mitochondria activity can be displayed after only 7–10 days of aerobic training when it is performed daily for 2 h at 65% peak oxygen uptake (11). It is likely that the potency of all-out sprint interval training is derived in large part from the high level of motor unit activation. All-out sprint training especially stresses recruitment and adaptation of type II (i.e., fast twitch) muscle fibers that are remarkably and equally responsive as type I (i.e., slow twitch) muscle fibers in their ability to increase mitochondrial enzyme activity to high absolute levels (4, 5, 7). In fact, the low-intensity aerobic exercise that is typically prescribed for endurance training or health is not very effective at increasing aerobic enzyme activity in type II muscle fibers, which comprise approximately one-half of the fibers within the thigh (vastus) and calf (gastrocnemius) muscle in most people (6). Thus low-intensity aerobic training is not a very effective or efficient method for maximizing aerobic adaptations in skeletal muscle because it generally does not recruit type II muscle fibers. The present report by Burgomaster et al. (2) provides a reminder of the effectiveness of sprint interval training, performed three times per week, and it demonstrates that large increases in aerobic enzyme activity and aerobic performance capacity previously measured after 7–8 wk (6) can occur after as little as 2 wk and only six sessions.

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