國立體育學院九十五學年度研究所博士班入學考試試題

（本試題共15頁）

體育運動論文評論（運動生理組）

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Improved muscular efficiency displayed as Tour de France champion matures

Edward F. Coyle

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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/japplphysiol.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., ~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 ~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., l/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 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.

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

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 $ml \cdot kg^{-1} \cdot min^{-1}$, 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 $ml \cdot kg^{-1} \cdot min^{-1}$. 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
Anthropometry					
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	
Maximal aerobic ability					
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	
Lactate threshold					
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	
Mechanical efficiency					
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 ~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.

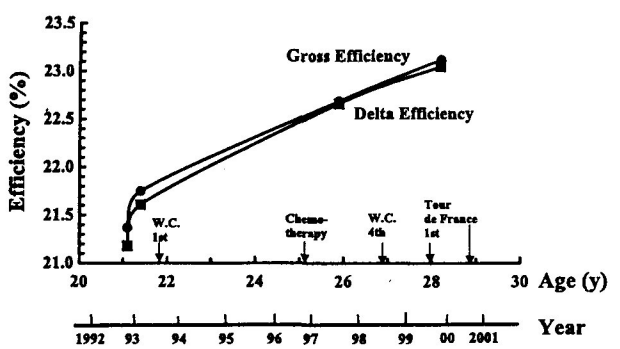

Fig. 1. Mechanical efficiency when bicycling expressed as "gross efficiency" and "delta efficiency" over the 7-yr period in this individual. WC, World Bicycle Road Racing Championships, 1st and 4th place, respectively. Tour de France 1st, Grand Champion of the Tour de France in 1999–2004.

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 phosphorylase] 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 $ml \cdot kg^{-1} \cdot min^{-1}$. 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 $ml \cdot kg^{-1} \cdot min^{-1}$ 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 $ml \cdot kg^{-1} \cdot min^{-1}$ 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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Scientific considerations for physiological evaluations of elite athletes

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The following is the abstract of the article discussed in the subsequent letter:

Coyle EF. Improved muscular efficiency displayed as Tour de France champion matures. *J Appl Physiol* 99: 2191–2196, 2005. First published March 17, 2005; doi:10.1152/japplphysiol.00563.2005.—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., ~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.

Scientific considerations for physiological evaluations of elite athletes

To the Editor: Elite athletes are valuable study objects for exercise physiology: successful sportsmen offer unique insight into the extreme adaptation of the human organism to certain types of exercise and illustrate the amazing adaptation capacity of human physiology (9). Because of the unique characteristics of the study subjects, sample sizes in these investigations are usually low. Even case reports, such as in the article written by Dr. Coyle (1), can therefore be a valuable contribution to the scientific knowledge in this field.

Nevertheless, such studies should respect the basic principles of scientific investigations. We feel that the investigation presented by Dr. Coyle has serious limitations in this context.

Experimental design. The aim of the study was, according to the author, to report "the physiological changes that occur in an individual bicycle racer during a 7-yr period" and thereby illustrate "the extreme to which the human can adapt to endurance training." Unfortunately, the data presented in the manuscript do not contain enough physiological information of the athlete in question (Lance Armstrong) to draw a picture sufficient to illustrate his physiological profile and the associated adaptations over 7 yr: in fact, no testing was performed in immediate connection with his Tour de France wins. It can be assumed that his physiological performance at that moment was much higher than the ones measured and described by the manuscript. The performance data reported in the manuscript are common to many elite cyclists (4, 5), none of whom matches the wins of Armstrong. Furthermore, the exercise tests outside the cancer period date from the months of January, November, and September; these are periods where professional cyclists, who target peak form for races in July, have barely the same condition as during their peak season. Therefore, all speculations in the manuscript on potential data during his Tour de France wins are not supported by any of the presented test results. To display a complete physiological profile of the athlete and to draw the present conclusions, at least some data from peak season testing should have been included. Interestingly, no data from the years of best performance of the athlete are presented: during the period from 2000–2005, Armstrong won five consecutive Tours de France; unfortunately, no exercise test seems to have been conducted during that time, which is rather surprising for an athlete of Armstrong's caliber.

Methods. To evaluate exercise performance and draw valid conclusions, it is essential to report data on the reliability and accuracy of the testing equipment, especially when only small changes are expected or the accuracy of the testing equipment is poor. In exercise physiology, especially the assessment of respiratory data is prone to errors linked to the testing procedure. This error, together with biological variation of maximal oxygen uptake, has been demonstrated to reach up to 5% (3, 8), thereby almost equaling the changes described in the manuscript. The same applies to the ergometry equipment: it has been demonstrated that many ergometers yield a high inaccuracy in their measurements, especially mechanically braked models, such as the one used for the present investigation (6, 10). In a comparable case report which uses the same type of mechanically braked ergometer (9), the authors included a 9% correction for their power measurements.

Unfortunately, the author does not report any data on the accuracy and reliability (such as calibration data) of his testing equipment. Especially when evaluating the calculations and conclusions drawn from the data, this would be of great help.

Furthermore, we are not aware of a reliable constant power mode in mechanically braked ergometers, such as the Monark model used for several tests in the present study.

Results and discussion. The author highlights the importance of improved muscular efficiency as being the main reason for Armstrong's outstanding gain in performance. We feel that this assumption cannot be made on the basis of the presented information, because no records are available from periods where the athlete actually had peak form. In this context, Fig. 1 is not correct, because it implies that Armstrong's gross and delta efficiency have been constantly rising since the age of 20 yr, despite a period of more than reduced physical condition during cancer treatment. On the basis of the presented data, the author cannot judge the efficiency of any other moment than the ones studied (November 1992, January 1993, August 1997, November 1999). Furthermore, the conclusion of the manuscript is even more surprising, because it has been shown that efficiency is not a key factor to differentiate between successful and unsuccessful cyclists (2, 7). Unpublished data from our laboratory support these assumptions: elite cyclists do not show higher efficiency than recreational cyclists. Furthermore, a high interindividual variability can be noted. In a longitudinal follow-up (intraindividually), however, efficiency remains remarkably stable, even when overall physiological exercise performance highly varies.

It is therefore more likely that, in addition to very favorable genetic assets of the athlete, common physiological adaptations associated with endurance training, such as an improved aerobic and possibly anaerobic energy metabolism, increased power-to-weight ratio, or enhanced recovery functions, might have added to the truly outstanding sporting achievements of Lance Armstrong.

It has to be considered that, aside from being determined by purely physiological factors, performance in sporting competitions is highly influenced by many other variables, such as tactical race understanding and motivational and psychological issues. Although speculative, the latter two might play a prominent role in Armstrong's sporting achievements, especially when considering the athlete's unique medical history and human experience as a cancer survivor. Armstrong might have gained the edge over his physiologically equally strong competitors by these means.

We feel obliged to raise these issues to the scientific community on behalf of all scientists working with elite athletes. Even when the popularity of an athlete might strongly influence the interest of publishing data, both from the author working with the athlete and the editor's side, the basic principles for scientific investigations should be respected. Published data (especially if published in a highly regarded scientific journal like the *Journal of Applied Physiology*) represent the base of knowledge and interpretation for future investigations and should therefore fulfill these scientific principles to allow upcoming studies to rely on the validity of their outcomes.

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REPLY

To the Editor: I thank Dr. Schumacher et al. for the opportunity to discuss the reliability and validity of our methods. Regarding "scientific considerations," this study focused on physiology and not the science of bicycle racing. Our main purpose was not to make measurements around the Tour de France or to compare this subject (Lance Armstrong) with other champions. The fact that our subject happened to eventually win the Tour de France was interesting but not the main "scientific consideration." Changes in muscle efficiency with 7 yr of training was the focus.

Reliability was most important, both in terms of the subject as well as the measurements of indirect calorimetry and power. This subject's level of training and accessibility were most reliable from year to year in the early part of the competitive season when most of our measures were made. Besides, our study of Armstrong began before he ever competed in the Tour de France. The fact that we did not report data after this subject won his first Tour de France emphasizes, again, that our purpose was to observe the maturation and not report the characteristics of the existing champion.

Schumacher et al. have requested data regarding the reliability of our respiratory testing equipment for measuring oxygen consumption. During submaximal exercise at 60–70% maximal oxygen consumption in a group of competitive cyclists (circa 1994), we have observed that oxygen consumption when measured on 8 separate days in a given individual displayed an average range of 0.08 l/min and a coefficient of variation of ±0.87% (2). See Martin et al. (5) for additional insight. The notion that a set 9% correction should be applied to all Monark ergometers is not supported by Maxwell et al. (6). The model 819 Monark ergometer used by Armstrong was calibrated statically and dynamically using pedal dynamometers and found valid to within ±3% (1, 4), and power can be held constant [as detailed in Martin et al. (5)].

Schumacher et al. state that "Fig. 1 is not correct" and then say that "on the basis of presented data, the author cannot judge the efficiency of any other moment than the ones studied (November 1992, January 1993, August 1997, November 1999)." The manuscript never "judged" or speculated about efficiency as it only reported actual data. Removing data from 1997 does not alter the line between 1992 and 1999. These data over years, to our knowledge, are the only published addressing long-term efficiency and training. These data seem to conflict with notions of Schumacher et al., because they state "efficiency is not a key factor to differentiate between successful and unsuccessful cyclists" on the basis of their own unpublished data as well as the work of others (7). We have presented a model of how numerous physiological factors interact to determine endurance performance and have discussed that efficiency by itself does not account for most of the interindividual variations in performance. In fact, in our 1991 manuscript (1), we also report that efficiency in a group of elite cyclists does not differ significantly from a group of good cyclists because of the high degree of individual variation in efficiency and fiber type. However, in a following study during which maximal oxygen consumption and lactate threshold were matched in a pairs of competitive cyclists, it was clear that performance power was significantly higher in subjects with greater gross efficiency and greater percentage of type I fibers (3). In fact, Armstrong makes this point in that his efficiency was only average when he was 21–22 yr despite the fact that he was already elite and world champion. However, his efficiency improved and he was able to generate 8% more power when cycling at a constant $\dot{V}O_2$ of 5.0 l/min.

We appreciate that winning the Tour de France requires tactical race understanding and motivational and psychological issues, among other things. However, nonphysiological factors and the winning of the Tour de France, although interesting, are not the focus of this investigation.

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Has Armstrong's cycle efficiency improved?

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Has Armstrong's cycle efficiency improved?

To the Editor: The concept that extensive endurance training improves cycling efficiency is intuitively appealing but not well supported by the literature. Recently, Coyle (1) has published efficiency data from Tour de France Champion, Lance Armstrong. In this case study Coyle concluded 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" (1). Case studies documenting adaptations in truly elite endurance athletes are important (3); however, we believe Coyle's case study is insufficient to support his conclusions because of limitations in study design and methodology.

Timing of testing sessions. Armstrong was tested five times over a period of 7 yr. Only the first and last test occurred during the same month (November), making it difficult to distinguish seasonal effects from maturation effects. Unfortunately, Armstrong's fitness data within 3 mo of racing a Tour de France tour is not reported. The majority of the improvement in gross cycling efficiency (GE) occurred after January 1993 (21.6%) and before August 1997 (22.7%), 8 mo after cancer treatment. Consequently, if there were real changes in GE it becomes difficult to distinguish whether the improvements in GE are due to cancer treatment or important aspects of training (e.g., training load, altitude training, high-cadence training, time-trial training, or resistance training).

Accuracy and reliability of efficiency. Coyle does not present data documenting the accuracy and reliability of the techniques used to calculate cycling efficiency (oxygen uptake, carbon dioxide production, and power output). Friction-braked bicycle ergometers have been shown to be inaccurate when dynamically calibrated (4). Previous research has reported that Monark ergometers tend to underestimate power output by ~2–8% (4). If Coyle's Monark ergometer was inaccurate, then Armstrong's actual GE before winning his first Tour de France may have been ~19–21%, values similar to those reported for recreational cyclists (5). Also of concern is the observation that the accuracy of Monark ergometers can change with age (4). Without routine assessment of accuracy with a dynamic calibration rig, it is difficult to know whether accuracy of the Monark used in Coyle's study changed over the 7-yr period of data collection.

Were all tests performed on same ergometer? The terminology used by Coyle to describe the "same Monark ergometer (model 819) used for all cycle testing" is confusing. In the METHODS section, Coyle states that "the calibrated ergometer was set in the constant power mode" and in the DISCUSSION section that there was "a progressive loss of pedal cadence at constant power during the 30–60 s before exhaustion." Although we are unaware of a constant power mode for Monark (model 819) ergometers, this mode of operation is commonly used with a Lode electromagnetic ergometer. A Lode ergometer has been used in Coyle's laboratory (2). It is possible that either inappropriate terminology was used in the METHODS section or Armstrong was tested on two different types of ergometers.

Is efficiency responsible for success? Without the appropriate data, Coyle is left to speculate that, during the Tour de France tours (1999–2004), Lance possessed a maximal oxygen uptake ($\dot{V}O_{2\,max}$) of ~6.1 l/min (based on the September 1993 testing session) and a body mass of ~72 kg (based on "his reported body weight") and therefore a relative $\dot{V}O_{2\,max}$ of 85 $ml \cdot kg^{-1} \cdot min^{-1}$. These estimations suggest that efficiency improved (21.2–23.1%; ~9%), while $\dot{V}O_{2\,max}$ rose (70–85 $ml \cdot kg^{-1} min^{-1}$; ~21% increase) and body mass fell (from 78.9 to 72.0 kg; ~9% decrease). In contrast to Coyle's conclusions, it appears that conventional physiological adaptations to modifications in diet (loss in body mass) and training (gains in aerobic power) may be equally, if not more, important to Armstrong's performance than the 9% improvements in cycling efficiency.

In summary, although great insight into human physiology can be gained from carefully controlled examinations of elite athletes, poor experimental design and methodology can lead to inappropriate conclusions, which in the case of a sporting hero can quickly become more hype than fact. Coyle's data supporting the assumption that training can improve cycling efficiency in an elite cyclist are not compelling. It appears that other more conventional explanations describing why Armstrong is such a successful cyclist may be equally tenable.

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REPLY

To the Editor: I appreciate this opportunity to answer the four points and address the terminology that Martin et al. find "confusing" (*point 3*).

1) Point 1: Timing of testing sessions. I agree that it is not possible to distinguish what aspects of Armstrong's training over the 7-yr period were related to his improved gross efficiency. Thus it was not discussed (4). Again, it can only be pointed out that he continued to train and his efficiency improved. Because the first measure in 1992 and the last measure in 1997 were both made in November when Armstrong's training was similar, the most appropriate design was indeed used to control for the possibility of seasonal variations in efficiency. The idea that cancer or chemotherapy might have improved Armstrong's efficiency cannot be determined from these data.

2) Point 2: Accuracy and reliability of efficiency. Oxygen uptake ($\dot{V}o_2$) and carbon dioxide production displayed a coefficient of variation of 0.87 and 0.92%, respectively, when measured on eight separate weekly occasions in a group of competitive cyclists in 1994 (6). Furthermore, the range (high minus low) of $\dot{V}o_2$ during these eight separate bouts averaged ±0.08 l/min (6). The point that bicycle ergometers can be inaccurate is well taken and appreciated. The Monark ergometer was chosen because it can be and was statically calibrated for each test. Martin et al. raise the possibility that the calculation of efficiency changed because of Monark ergometer aging instead of Armstrong aging (i.e., maturation). First of all, the mechanical components of Monark ergometer were kept in good condition with the regular cleaning and maintenance of the friction belt, flywheel, drive chain, and bearings, and thus, according to Maxwell et al. (8), it should not have "aged" significantly. Second, an "aging ergometer" according to Maxwell et al. will raise the oxygen cost and thus lower efficiency, which is the exact opposite of what was observed in Armstrong, who increased efficiency with age. The best dynamic calibration of the Monark 819 ergometer in my experience is derived when a pedal dynamometer is compared with simultaneous integration of forces and velocity of the flywheel. This dynamic calibration was performed on this exact "same" Monark ergometer using elite cyclists as subjects (3, 7). It was observed that ergometer power outputs between 20 and 400 W agreed with the right pedal dynamometer with a range of ±3%. It should be noted that our references to "a specially designed ergometer" (3, 7) include continuous and integrated measurement of the Monark pendulum displacement force using a potentiometer with a reliable measurement accuracy of ±0.4 N. Furthermore, cycling cadence was measured (±0.18 rpm) continuously throughout each pedal revolution (3, 7).

3) Point 3: Were all test performed on the same ergometer? All the data presented on Armstrong in this manuscript (4) were indeed collected from the "same" ergometer (i.e., only one unit used). Monark did indeed manufacture an ergometer (819) in the 1980s that possessed electronics that integrated cadence and force in order to hold power constant. I hope this addresses the suspicions. For what it is worth, the electronic circuitry of our 819 ergometer became nonrepairable as did our system for measuring indirect calorimetry. However, Armstrong is still going strong, albeit with a few repairs.

4) Point 4: Is efficiency responsible for success? Improved mechanical efficiency and power (watts) accounted for approximately one-half of Armstrong's improvement (i.e., 8–9%), and an 8–9% reduction of body weight (kilograms) accounted for the other one-half (4). Thus watts per kilogram increased by 18%. Speculation about maximal $\dot{V}o_2$ ($\dot{V}o_{2\,max}$) during the Tour de France is not needed to calculate watts per kilogram. The notion that endurance performance is related only to $\dot{V}o_{2\,max}$ was conventional long ago (5), and Martin et al. might find enlightenment by considering models that also integrate submaximal muscle stress (e.g., lactate threshold) and performance power or velocity (1, 2).

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