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Journal of Applied Physiology 98:2191-2196, 2005; First published May 17, 2005;
doi:10.1152/japplphysiol.00216.2005

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Improved muscular efficiency displayed as Tour de France champion matures

Edward F. Coyle
Human Performance Laboratory, Department of Kinesiology and Health Education, The University of Texas at Austin, Austin, Texas
Submitted 22 February 2005; accepted in final form 10 March 2005

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.00162.2005.—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 (\(V_{\text{O}_2 \text{max}}\)) 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 76–83% \(V_{\text{O}_2 \text{max}}\), 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 (\(V_{\text{O}_2}\)) is the characteristic that improved most as the athlete matured from ages 21 to 28 yr. It is noteworthy that at age 35 yr, this champion developed advanced cancer, requiring surgery 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 \(V_{\text{O}_2}\) (e.g., 5 l/min). It is hypothesized that the improved muscular efficiency probably reflects changes in muscle myosin type stimulated by years of training intensely for 3–6 h on most days.

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, CYCLE 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 (\(V_{\text{O}_2 \text{max}}\)), 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 THEIR 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 yr. 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 (~70 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% \(V_{\text{O}_2 \text{max}}\). After a 10- to 20-min period of active recovery, \(V_{\text{O}_2 \text{max}}\) when cycling was measured. Thereafter, body composition was determined by hydrostatic weighing and/or analysis of skin-fold thicknesses (34, 35).

Measurement of \(V_{\text{O}_2 \text{max}}\). 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, \(V_{\text{O}_2 \text{max}}\) was measured during continuous cycling lasting between 8 and 12 min, with work rate increasing every 2 min. A leveling off of oxygen uptake (\(V_{\text{O}_2}\)) 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 \(V_{\text{O}_2 \text{max}}\). A venous blood sample was obtained ~3 min after exhaustion for determination of blood lactate concentration after maximal exercise, as described below. The subject breathed through a Daniells valve; expired gases were continuously sampled from a mixing chamber and analyzed for \(O_2\) (Applied Electrochemistry S3A) and \(CO_2\) (Beckman LB-2). Inpired air volumes were measured using a dry-gas meter (Parkinson-Cowan CD4). These instruments were interfaced with a computer that calculated \(V_{\text{O}_2}\) every 30 s. The same equipment for indirect calorimetry was used over the 7-yr period, with gas analyzers calibrated against the same known gases and the dry-gas meter calibrated periodically to a 350-liters 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% \(V_{\text{O}_2 \text{max}}\) 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
a catheter in an antecubital vein or from placing a finger during the 3rd min of exercise at each stage or 4 min after maximal exercise. Whole blood was deproteinated 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. $V_O_2$ relationship and determining the $V_O_2$ 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 $V_O_2_{max}$ 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 $V_O_2$ 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% $V_O_2_{max}$ 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. Delta efficiency was 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 of the relationship (i.e., 5 data points at —50, 60, 70, 80, and 90% $V_O_2_{max}$) 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 measurements 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 yr. 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 Cycling 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 1995, 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 new 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.

$V_O_2_{max}$ maximal heart rate, and the blood LT. $V_O_2_{max}$ during the pre-season months of November through January generally ranged from 5.56 to 5.82 l/min during the period of 1992—1999. $V_O_2_{max}$ 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), $V_O_2_{max}$ 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 $V_O_2$ 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 $V_O_2$ 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., Whg). 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 $V_O_2$ is 5.0 l/min). In that his $V_O_2_{max}$ remained at ~6 l/min, this given $V_O_2$ of 5.0 l/min represents ~83% $V_O_2_{max}$. Therefore, his "power per kilo-

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

<table>
<thead>
<tr>
<th>Year</th>
<th>Age, yr</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>1991</td>
<td>19</td>
<td>115-A, National Amateur Champion</td>
</tr>
<tr>
<td>1992</td>
<td>20</td>
<td>14th place in Olympic Road Race; Barcelona</td>
</tr>
<tr>
<td>1993</td>
<td>21</td>
<td>1st place in World Championships, Road Racing; Oslo. Winner, one stage in Tour de France</td>
</tr>
<tr>
<td>1995</td>
<td>23</td>
<td>Winner of one stage in Tour de France</td>
</tr>
<tr>
<td>1996</td>
<td>24-25</td>
<td>12th place in Olympic Road Race; Barcelona. 6th place in Olympic Individual Time trial</td>
</tr>
<tr>
<td>1998</td>
<td>26</td>
<td>4th place in World Championships, Road Racing</td>
</tr>
<tr>
<td>1999</td>
<td>27</td>
<td>1st place—Tour de France Grand Champion</td>
</tr>
<tr>
<td>2000</td>
<td>28</td>
<td>1st place—Tour de France Grand Champion</td>
</tr>
<tr>
<td>2001</td>
<td>29</td>
<td>1st place—Tour de France Grand Champion</td>
</tr>
<tr>
<td>2002</td>
<td>30</td>
<td>1st place—Tour de France Grand Champion</td>
</tr>
<tr>
<td>2003</td>
<td>31</td>
<td>1st place—Tour de France Grand Champion</td>
</tr>
<tr>
<td>2004</td>
<td>32</td>
<td>1st place—Tour de France Grand Champion</td>
</tr>
</tbody>
</table>
Table 2. Physiological characteristics of this individual from the ages of 21 to 28 yr

<table>
<thead>
<tr>
<th></th>
<th>21.1</th>
<th>21.4</th>
<th>22.0</th>
<th>Avg 1997</th>
<th>28.2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Date: Month-Year</td>
<td>Nov 1992</td>
<td>Jan 1993</td>
<td>Sept 1993</td>
<td>Racing</td>
<td>Nov 1999</td>
</tr>
<tr>
<td>Training stage</td>
<td>Preseason</td>
<td>Preseason</td>
<td>Racing</td>
<td>Reduced</td>
<td>Preseason</td>
</tr>
<tr>
<td><strong>Anthropometry</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body weight, kg</td>
<td>78.9</td>
<td>76.5</td>
<td>75.1</td>
<td>79.5</td>
<td>79.7</td>
</tr>
<tr>
<td>Lean body weight, kg</td>
<td>70.5</td>
<td>69.8</td>
<td>70.2</td>
<td>71.6</td>
<td></td>
</tr>
<tr>
<td>Body fat, %</td>
<td>10.7</td>
<td>8.8</td>
<td>11.7</td>
<td>11.7</td>
<td>11.7</td>
</tr>
<tr>
<td><strong>Maximal aerobic ability</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maximal O2 uptake, l/min</td>
<td>4.56</td>
<td>5.22</td>
<td>6.10</td>
<td>5.59</td>
<td>5.7</td>
</tr>
<tr>
<td>Maximal Heart rate, beats/min</td>
<td>207</td>
<td>206</td>
<td>203</td>
<td>200</td>
<td>200</td>
</tr>
<tr>
<td>Maximal blood lactate, mM</td>
<td>7.3</td>
<td>6.3</td>
<td>6.5</td>
<td>9.2</td>
<td></td>
</tr>
<tr>
<td><strong>Lactate threshold</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactate threshold O2 uptake, l/min</td>
<td>4.70</td>
<td>4.52</td>
<td>4.63</td>
<td>4.02</td>
<td></td>
</tr>
<tr>
<td>Lactate threshold, % maximal O2 uptake</td>
<td>85</td>
<td>78</td>
<td>76</td>
<td>76</td>
<td></td>
</tr>
<tr>
<td><strong>Mechanical efficiency</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gross efficiency, %</td>
<td>21.18</td>
<td>21.61</td>
<td>22.66</td>
<td>23.05</td>
<td></td>
</tr>
<tr>
<td>Delta efficiency, %</td>
<td>21.37</td>
<td>21.75</td>
<td>22.69</td>
<td>23.12</td>
<td></td>
</tr>
<tr>
<td>Power at O2 uptake of 5.0 l/min, W</td>
<td>374</td>
<td>382</td>
<td>399</td>
<td>404</td>
<td></td>
</tr>
</tbody>
</table>

In the trained state, this individual possessed a remarkably high V\textsubscript{O2 max} of -6 l/min, and his blood LT occurred at a V\textsubscript{O2} of ~4.6 l/min (i.e., 76-85% V\textsubscript{O2 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 V\textsubscript{O2 max} of 6.4 l/min and 79 ml kg\textsuperscript{-1} min\textsuperscript{-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 V\textsubscript{O2 max} was at least 6.1 l/min and given his reported body weight of 72 kg, we estimate his V\textsubscript{O2 max} to have been at least 85 ml kg\textsuperscript{-1} min\textsuperscript{-1} during the period of his victories in the Tour de France. Therefore, his V\textsubscript{O2 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\textsuperscript{-1} min\textsuperscript{-1}) (6, 15, 16, 28, 29).

It is generally appreciated that in addition to a high V\textsubscript{O2 max} success in endurance sports also requires an ability to exercise for prolonged periods at a high percentage of V\textsubscript{O2 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 V\textsubscript{O2 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 fatigue metabolites (e.g., acid) from muscle fibers into the circulation, whereas the LT is thought...
Furthermore, maxima heart rale achieved the healthy level for 5.3 l/min and 67 ml·kg\(^{-1}\)·min\(^{-1}\) from his reduced training, \(\text{V}O_2\) this individual (i.e., 200 beals/min). However, as expected responses were normal at heart rates of 120-150 beats/min. Maximal exercise appeared typical, and his cardiovascular history evaluation 8 mo after completing chemotherapy, this motherapy), he bicycled 1-2 h/day at moderate intensity, and chemotherapy. In particular, ventilatory volume during performance training. However, during the 8 days before

During the 6- to 7.5-mo period after chemotherapy, he did not encounter by athletes who spend periods living at high altitude or in hypoxic environments (19, 30). Like many competitive cyclists 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 (13, 24). As such, it appears the measure of delta efficiency, defined as the increase in power output relative to the rate of increase in energy expenditure (calculated from \(\text{V}O_2\)) throughout a wide range of work rates provides the best reflection of 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 during bicycling or with the simple task of knee extension (10, 25).

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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, Willier 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 20- 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 speed (e.g., 51/min), increases in freely chosen revolutions per minute when cycling at high intensity are 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 speed (e.g., 51/min), increases in freely chosen revolutions per minute when cycling at high intensity are 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).

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 V̇O₂ 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 V̇O₂ (e.g., 5.0 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 surgery and chemotherapy, yet these events did not appear to impede his physiological maturation and athletic achievements. Clearly, this champion embodies a phenomenon of both 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.

ACKNOWLEDGMENTS

The author very much appreciates the respectful cooperation and positive attitude of Lance Armstrong over the years and through it all.

REFERENCES


