

The Physiology of the World Record Holder
for the Women's Marathon

by

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The Physiology of the World Record Holder for the Women's Marathon

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ABSTRACT

The purpose of this paper is to review the physiological determinants of endurance exercise performance by using the data of the World Record holder for the women's marathon (PR), to illustrate the link between an athlete's physiology and success in distance running. The maximal oxygen (O_2) uptake, O_2 cost of running at sub-maximal speeds (running economy), and blood lactate response to exercise can all be determined using standard physiology laboratory exercise tests and the results used to track changes in 'fitness' and to make recommendations for future training. PR's data demonstrate a 15% improvement in running economy between 1992 and 2003 suggesting that improvements in this parameter are very important in allowing a distance runner to continue to improve their performance over the longer-term. PR's data demonstrate how 15 years of directed training have created the 'complete' female distance runner and enabled the setting of an extraordinary World record of 2:15:25 for the Marathon.

Key words: Endurance Running, Lactate Threshold, Lactate Turnpoint, Oxygen Uptake, Running Economy, Women's Marathon

INTRODUCTION: PHYSIOLOGICAL LIMITATIONS TO ENDURANCE RUNNING PERFORMANCE

"Endurance" might be usefully defined as the capacity to sustain a given speed or work rate for the longest possible time. The majority of the energy supply during exercise of greater than ~60–120 s duration is derived through oxidative metabolism and therefore performance in endurance sports is heavily dependent upon the aerobic resynthesis of adenosine triphosphate (ATP; the energy 'currency' of the cell). This requires an adequate delivery of O_2 from the atmosphere to cytochrome oxidase in the mitochondrial electron transport chain and the availability of fuels in the form of carbohydrates and lipids.

In distance running, the competitive events can be conveniently divided into the short endurance events (800 m and 1,500 m), the long endurance events (5,000 m, 10,000 m, and the Marathon) and the ultra-long endurance events (ultra-marathons). The limitations to performance in events spanning such a large range of exercise durations (from 101 s to

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several days) and exercise intensities (from $\sim 50\%$ to $\sim 115\%$ of maximal $\dot{V}O_2$) are likely to vary considerably [1–3]. For example, in the middle-distance events, the power and capacity of the anaerobic energy pathways and the athlete's ability to tolerate the consequent metabolic acidosis will impact on performance. In the longer events, the availability of metabolic substrate (principally muscle glycogen and blood glucose) and the ability to regulate core body temperature become progressively more important determinants of success [2–4]. Nevertheless, because all these events rely predominantly on energy supply through oxidative metabolism, there are a number of parameters of 'aerobic fitness' (reviewed below) that are important across the entire spectrum of endurance events [1].

Some of the physiological factors that are known to be related to endurance running performance include: maximal O_2 uptake ($\dot{V}O_2$ max); running economy; and the fractional utilisation of the $\dot{V}O_2$ max (which is itself related to markers of blood lactate accumulation during exercise, including the lactate threshold and maximal lactate steady state; [2, 4]). The rate at which $\dot{V}O_2$ rises following the onset of exercise (i.e. the $\dot{V}O_2$ kinetics) is also important in minimising the magnitude of the ' O_2 deficit' that is incurred, although this will be much more important in the middle-distance events [1]. The manner in which these factors interact to determine the highest average speed that can be sustained during a distance running event is summarised in Figure 1, which is adapted from that presented by Coyle [2].

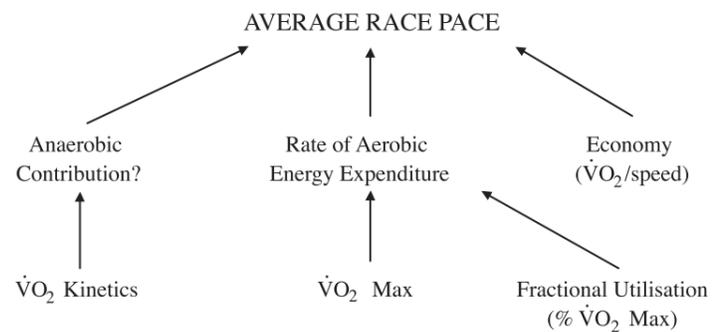


Figure 1. Some of the Physiological Determinants of Endurance Running Performance.

The rate of oxidative metabolism that can be maintained for long periods is a crucial determinant of success and this will be linked to the maximal O_2 uptake ($\dot{V}O_2$ max; this represents the 'ceiling' for aerobic respiration) and the fractional utilisation of the $\dot{V}O_2$ max (linked, in turn, either directly or indirectly, to the accumulation of lactate in the muscles and blood). The running speed that this metabolic rate will correspond to will be dictated by the running economy characteristics of the athlete.

This paper will briefly review these physiological factors and also provide information on the methods used by the author to measure these factors as part of his physiological support work with PR, one of the best female distance runners in the World and the present World Record holder for the Women's marathon. Some of PR's test data will be presented to illustrate the relationships between these physiological variables and distance running performance.

MAXIMAL OXYGEN UPTAKE

The maximal rate at which ATP can be re-synthesised through aerobic pathways is an important determinant of endurance running performance [4, 5]. Indeed, international standard male distance runners typically have $\dot{V}O_2$ max values of 70–85 mL · kg⁻¹ · min⁻¹ while their female counterparts have $\dot{V}O_2$ max values of 60–75 mL · kg⁻¹ · min⁻¹ [4, 6, 7]; these values are ~45% higher than age- and sex-matched sedentary individuals [8]. In the author's experience, the highest $\dot{V}O_2$ max values are often found in 5,000 m specialists, presumably because these athletes are required to run at 94–98% $\dot{V}O_2$ max to achieve best performances [9]. Middle-distance athletes (i.e. 800 m and 1500 m specialists) tend to have slightly lower $\dot{V}O_2$ max values (unpublished observations), reflecting the importance of the capacity to generate energy through oxygen-independent metabolism in these events, which are run at supra-maximal intensities. Marathon and ultra-marathon runners tend to have slightly lower $\dot{V}O_2$ max values compared to 5,000 m specialists probably because their events are run at lower intensities and other physiological factors become relatively more important to success [4].

Given the relationship between $\dot{V}O_2$ max and endurance exercise performance, the assessment of $\dot{V}O_2$ max is of interest in applied work with distance runners. $\dot{V}O_2$ max is perhaps best assessed using an incremental treadmill test that brings the athlete to exhaustion in 7–10 minutes. An acceptable protocol both for the athlete and the physiologist is a constant speed test in which the treadmill gradient is increased by 1% every minute. However, the highest $\dot{V}O_2$ measured during a 'multi-stage' treadmill test, in which running speed is increased by a certain amount every few minutes (for example, by 1 km · h⁻¹ every 3 minutes) until the athlete becomes exhausted, is typically only slightly (1–4%) lower than the $\dot{V}O_2$ max achieved in the incremental gradient test.

RUNNING ECONOMY

Running economy (RE) can be defined as the O₂ cost (in mL · kg⁻¹ · min⁻¹) of running at a certain speed, or the O₂ cost of running a certain distance (i.e. mL · kg⁻¹ · km⁻¹). There is considerable inter-individual variability in RE, even in elite distance runners. For example, at a running speed of 16 km · h⁻¹ (6:00 min/mile pace), the $\dot{V}O_2$ can range between approximately 45 mL · kg⁻¹ · min⁻¹ and approximately 60 mL · kg⁻¹ · min⁻¹ [4, 6]. A running speed of 16 km · h⁻¹ is often used in the assessment of RE because, in trained runners, it is a sub-maximal speed that will be frequently encountered in training. When RE is expressed in units of mL · kg⁻¹ · km⁻¹, a value of around 200 is considered average, with values above and below this value representing poor and good economy, respectively. Good RE, i.e. a low $\dot{V}O_2$ for a given running speed, results in the utilisation of a lower percentage of the athlete's $\dot{V}O_2$ max while running at that speed (and consequently a reduction in muscle glycogen utilisation, and potentially less reliance on O₂-independent metabolism resulting in a reduction in metabolic acidosis). Long distance runners tend to be more economical than middle-distance runners at sub-maximal running speeds [4, 10], but it is not clear whether this difference has a genetic origin or is related to the larger training volumes that are typically completed by these athletes [11]. It is known that both physiological and anthropometric factors influence economy [12–14]. However, biomechanical factors connected to the running action are also of great importance in the determination of RE [14, 15].

Although it might be considered ideal to measure RE at the athlete's "race pace", in practice this is complicated by the existence of a 'slow component' of $\dot{V}O_2$ at all running speeds exceeding the lactate threshold [16]. The $\dot{V}O_2$ slow component elevates the O₂ cost of exercise and can even prevent the attainment of a steady state at higher exercise intensities, complicating the measurement of RE. Therefore, RE is typically measured over the range of

sub-maximal running speeds at which the athlete habitually performs his or her continuous endurance training. It is important that $\dot{V}O_2$ is measured in the 'steady state,' so stage durations in treadmill tests must be at least 3 minutes long. Setting the treadmill gradient to 1% is useful in compensating for the lack of air resistance in the laboratory such that the energy cost of running is equivalent to that when running outdoors on the road [17].

INTERACTION BETWEEN RUNNING ECONOMY AND $\dot{V}O_2$ MAX

It has been known for many years that although a high $\dot{V}O_2$ max is important for high level performances in distance running events, $\dot{V}O_2$ max in itself does not discriminate performance capability in groups of athletes with similarly high $\dot{V}O_2$ max values. In such situations, other parameters of aerobic fitness (such as RE) are important in explaining inter-individual differences in performance [18] (Figure 2). Good RE can therefore compensate to some extent for relatively low $\dot{V}O_2$ max values in elite athletes.

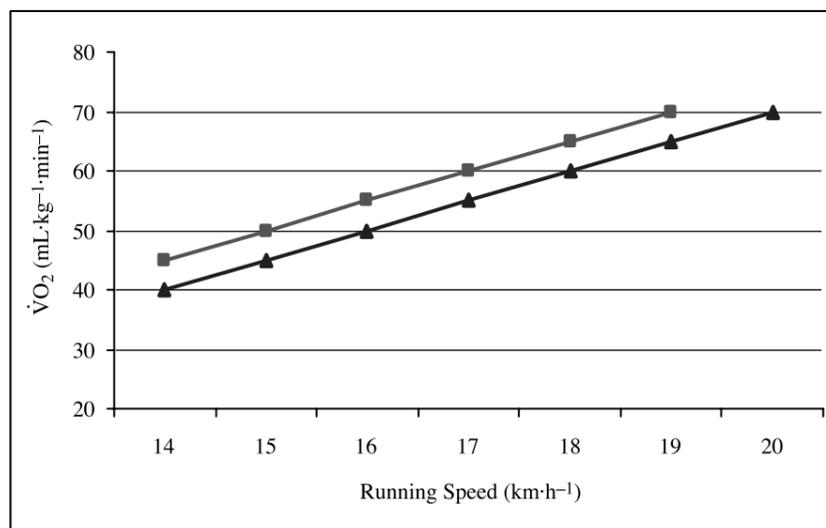


Figure 2. $\dot{V}O_2$ max and Running Economy

The interaction of $\dot{V}O_2$ max and running economy is illustrated here in two athletes who have the same $\dot{V}O_2$ max of 70 mL · kg⁻¹ · min⁻¹ but different running economy characteristics. The athlete represented by the triangle symbols uses less oxygen to run at sub-maximal speeds (i.e., has better running economy) than the athlete represented by the square symbols. This means that at any given $\dot{V}O_2$ (in mL · kg⁻¹ · min⁻¹), the athlete with the better running economy will be running faster. In this example, despite having the same $\dot{V}O_2$ max value, the athlete with the better running economy will be running at 20 km/h compared to 19 km/h for the athlete with the inferior running economy.

The expression of $\dot{V}O_2$ max in units of mL · kg⁻¹ · min⁻¹ is of limited value to runners because it does not, in itself, allow prediction of race performance or the prescription of potentially 'optimal' training. However, with direct measurements of both RE and $\dot{V}O_2$ max, it is possible to calculate the running speed associated with $\dot{V}O_2$ max ($S\text{-}\dot{V}O_2$ max; [19]). Essentially, the regression equation describing the relationship between the measured steady

state $\dot{V}O_2$ and running speed for sub-maximal exercise can be 'solved' for $\dot{V}O_2$ max, such that the latter is expressed as a running speed. For example, an athlete with a running economy of $200 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{km}^{-1}$ and a $\dot{V}O_2$ max for $65 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ would have an $S\dot{V}O_2$ max of $19.5 \text{ km} \cdot \text{h}^{-1}$ ($65 \times 60 / 200$). With certain correction factors [9], this value can be used to provide predictions of best performances over 1,500 m, 3,000 m, and 5,000 m. Furthermore, it has been suggested that training to improve $\dot{V}O_2$ max can be optimised by training at $S\dot{V}O_2$ max [8, 20, 21], and therefore the 'functional expression' of $\dot{V}O_2$ max in this way is useful in applied work with runners. The highest speed attained during a fast incremental treadmill test (involving $1 \text{ km} \cdot \text{h}^{-1}$ increments every minute) has also been positively correlated with distance running performance [22]. However, the highest speed attained at exhaustion in such a test will be higher than the $S\dot{V}O_2$ max because it will incorporate a (variable) contribution from O_2 -independent metabolism following a possible plateau in $\dot{V}O_2$ at its maximum.

LACTATE THRESHOLD AND LACTATE TURNPOINT

The ability to exercise for long periods at high fractions of the $\dot{V}O_2$ max is a characteristic of elite endurance athletes and is an important determinant of performance [4, 11, 23, 24]. The fractional utilisation of the $\dot{V}O_2$ max during endurance competition appears to be intimately linked to markers of blood lactate accumulation during exercise, such as the lactate or ventilatory threshold, lactate turnpoint, or 'onset of blood lactate accumulation' [1, 2, 4, 7, 23]. For this reason, the measurement of blood lactate concentration ($[La]_b$) during exercise can provide useful information on endurance performance potential and the extent of the physiological adaptations to a period of training [1, 6].

During an incremental treadmill test (involving ~ 7–8 stages at progressively increasing speeds), $[La]_b$ initially remains close to the resting value (i.e. ~ 1.0 mM). However, at a particular running speed (or, more properly, a particular metabolic rate), $[La]_b$ begins to increase above the resting value [16]. The running speed at which this occurs is known as the lactate threshold (LT). The LT typically occurs at 50–70% $\dot{V}O_2$ max, although it can be as high as 80–85% $\dot{V}O_2$ max in highly-trained marathon and ultra-marathon runners [4]. The elevation of $[La]_b$ at the LT represents alterations in cellular phosphorylation and redox potentials to drive oxidative metabolism, and does not necessarily indicate that the exercise has become partially 'anaerobic' [25]. It should also be remembered that the $[La]_b$ reflects a balance between the rate of lactate production in the muscles, the rate of efflux of lactate from the muscles to the blood, and the clearance of lactate from the blood [26, 27]. Exercise near the LT can be sustained for > 2 hours with $[La]_b$ being perhaps slightly elevated but not accumulating over time [4]. The LT is therefore useful in the assessment of 'long endurance' and in estimating performance times in events such as the marathon.

As the incremental treadmill test progresses to running speeds exceeding the LT, a second 'sudden and sustained' increase in $[La]_b$ (at around 2–4 mM) can often be discerned. This second threshold has become known as the lactate turnpoint (LTP) [28, 29]. During continuous longer-term (20–60 minutes) exercise at running speeds between the LT and the LTP, $[La]_b$ will be elevated above baseline values but will remain relatively stable over time. In contrast, during continuous exercise at running speeds above the LTP, $[La]_b$ will continue to increase with time until the exercise is terminated. The LTP therefore provides a reasonable approximation of the so-called 'maximal lactate steady state' intensity [28, 29].

Fatigue in endurance events has traditionally been linked to a reduction in muscle cell pH (assumed to be reflected, albeit indirectly, by an accumulation of blood lactate) [4, 30]. Therefore, the ability to delay and/or tolerate an increase in metabolic acidosis has been

considered to be an important adaptation to endurance training [1]. Although this concept is now recognised as being an over-simplification of a complex process [9, 31–33], improved endurance fitness is characterised, in part, by a rightward shift in the $[La]_b$ – running speed relationship, such that there is less lactate accumulation for a given running speed post-training. Therefore, although understanding of the underlying physiology has changed, a right-shifted $[La]_b$ profile still reflects positive metabolic adaptations to endurance training, and the measurement of $[La]_b$ during exercise remains useful in the longitudinal assessment of endurance athletes.

An appropriate incremental treadmill test protocol for the assessment of LT and LTP involves increasing running speed by $1 \text{ km} \cdot \text{h}^{-1}$ every 3 minutes and determining $[La]_b$ from fingertip blood samples taken during short breaks between stages. If heart rate (HR) is determined during the last 30 s of each stage, this enables a number of HR training ‘zones’ to be determined, based upon the metabolic and gas exchange responses to exercise. In the author’s experience with elite distance runners, continuous running below the LT is appropriate for ‘easy’ recovery sessions (20–40 minutes) or long relaxed runs (60–120 minutes), while continuous running between the LT and LTP is suitable for general, good-quality ‘steady’ aerobic training sessions (30–60 minutes) [34, 35]. Continuous ‘tempo’ or ‘brisk’ running at and just above the LTP for (20–40 minutes in total) is appropriate for race preparation, for elevating LTP and for developing ‘lactate tolerance’ [34, 35]. Higher intensity training to stimulate development of $\dot{V}O_2 \text{ max}$ and the capacity to generate ATP through O_2 -independent metabolism generally requires interval-type training at and above the $S\dot{V}O_2 \text{ max}$ [21, 34–36].

PHYSIOLOGICAL SUPPORT TO PR

PR is one of the World’s greatest ever female distance runners and, at the time of writing, is the World Champion and World Record holder (2 hours, 15 minutes, and 25 seconds) for the marathon. The author has had the privilege of working as PR’s physiologist since 1991, when she was a promising junior, through to the present day. The purpose of this section is to briefly describe the physiological tests that have been conducted over this 15 year period and to relate how the physiological changes that have been measured (i.e. improvements in the parameters of aerobic fitness alluded to above) might have facilitated improvements in athletic performance.

METHODS

The methods used in PR’s physiological assessment have remained essentially unchanged over 15 years. Following measurements of height, body mass, body composition (through skinfold thicknesses), haemoglobin concentration ($[Hb]$), pulmonary function, vertical jump height, sit-and-reach test, and a warm-up, PR completes a multi-stage incremental treadmill protocol, typically involving 7–9 exercise stages, each of 3 minutes duration. The starting speed for the test has increased appreciably over the years (from $12 \text{ km} \cdot \text{h}^{-1}$ in 1991 to $15 \text{ km} \cdot \text{h}^{-1}$ in 2003). Running speed has typically been increased in $1.0 \text{ km} \cdot \text{h}^{-1}$ increments, although, more recently, $0.5 \text{ km} \cdot \text{h}^{-1}$ increments have been introduced to more clearly define the LT and LTP. Throughout the test, the treadmill gradient is set at 1% [17]. Exercise is interrupted for 20–30 s at the completion of each stage to facilitate the collection of blood from a punctured fingertip into a capillary tube for subsequent determination of $[La]_b$. Throughout the test, HR is recorded using a telemetric system and pulmonary gas exchange is determined on a breath-by-breath basis. Originally, the multi-stage test was terminated at a sub-maximal intensity ($\sim 95\% \dot{V}O_2 \text{ max}$) and a separate test for the assessment of $\dot{V}O_2 \text{ max}$ was administered following a suitable recovery period [37]. However, in recent years, the

protocol has been modified so that a single multi-stage treadmill test is continued until exhaustion. This protocol is time- and labour-efficient in that it enables the responses of $\dot{V}O_2$, HR, and $[La]_B$ to be measured simultaneously over a wide range of running speeds; RE, LT and LTP, and $\dot{V}O_2$ max and $S\dot{V}O_2$ max can therefore all be assessed within a 25–30 minute test.

Physiological testing has been, and continues to be, used by PR and her coaches for the objective assessment of changes in the parameters of aerobic fitness brought about by the preceding period of endurance training. These changes inform the nature of the training to be conducted in the next training period. As alluded to earlier, the test data (and particularly the HR and running speeds at the LT, LTP and $\dot{V}O_2$ max) are used to regulate the intensity of the training performed. The test data have also been used to indicate the best achievable race time over a variety of distances (from 3,000 m to the marathon) and these predictions have typically been within 0.2–0.4% of the actual race finishing time. The prediction of potential best race times from the physiological test data has been of particular value when PR has gone into major competitions with limited race preparation. The physiological test sessions also serve as a source of motivation and re-assurance/confidence-boosting. Finally, many of the ‘adjunct’ measurements made during the test session (e.g. body mass and body composition; [Hb]; lung function; flexibility (sit-and-reach test); and leg power (vertical jump test)) have proven useful in providing a comprehensive picture of PR’s health and general fitness status and have also influenced her nutritional and training practices.

RESULTS

Although physiological test data exist for most, if not all, of the years between 1992 and 2003, for the sake of clarity of presentation, only representative data are shown in the following section. Where two or more tests were completed in any given year, the data have been averaged to more closely represent the changes in physiology that occurred over this period of time. Some of these data have been presented previously [37].

Maximal Oxygen Uptake ($\dot{V}O_2$ max)

PR’s $\dot{V}O_2$ max has varied according to the time of year when the test was conducted and the stage in her career, with a “low” of approximately $65 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and a “high” of

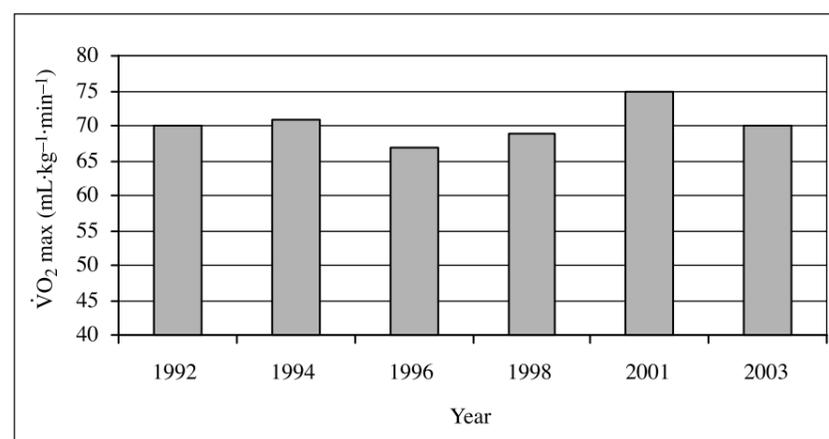


Figure 3. PR’s $\dot{V}O_2$ max Values, 1992-2003

For clarity, only representative data are shown.

approximately $80 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. However, when two or more tests from the same year are averaged together, it becomes clear that $\dot{V}\text{O}_2$ max has remained relatively stable at approximately $70 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ between 1992 (when PR was 18 years of age) until 2003 (when she was 29 years of age), (Figure 3). It should be noted that a $\dot{V}\text{O}_2$ max of this order is extremely high, even in elite female athletes, supporting the view that a high $\dot{V}\text{O}_2$ max is a prerequisite for successful performance at the international level. Clearly, however, physiological factors other than $\dot{V}\text{O}_2$ max must have been enhanced to enable the dramatic improvement in PR's distance running performances over this same time period.

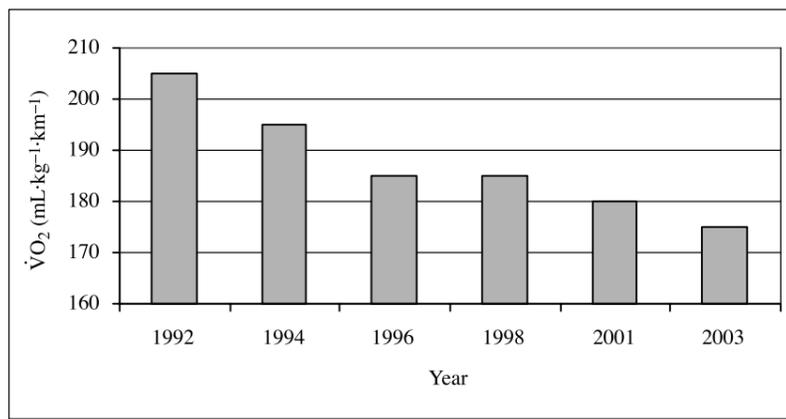


Figure 4. PR's $\dot{V}\text{O}_2$ While Running at $16.0 \text{ km} \cdot \text{h}^{-1}$, 1992-2003

For clarity, only representative data are shown.

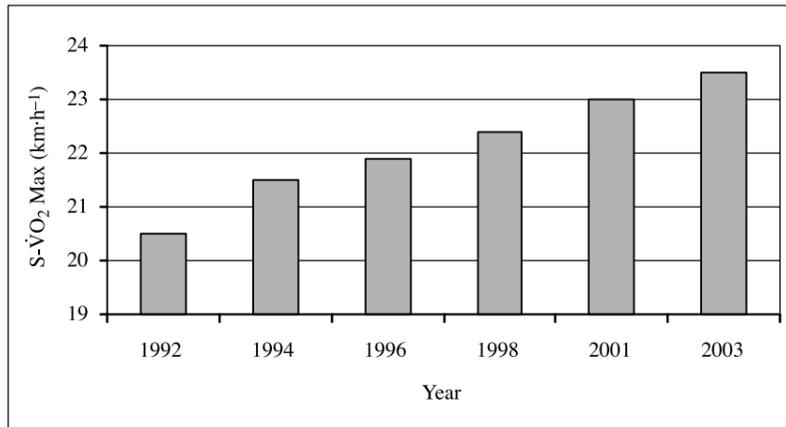


Figure 5. PR's Running Speed at $\dot{V}\text{O}_2$ max, 1992-2003

For clarity, only representative data are shown. Despite the similar $\dot{V}\text{O}_2$ max value, the improved running economy allows the calculated running speed at $\dot{V}\text{O}_2$ max to increase appreciably.

Running Economy (RE)

Figure 4 shows that the O_2 cost of running at $16 \text{ km} \cdot \text{h}^{-1}$, expressed in units of $\text{mL} \cdot \text{kg}^{-1} \cdot \text{km}^{-1}$, has decreased markedly between 1992 (when it was approximately $205 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{km}^{-1}$) and 2003 (when it was approximately $175 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{km}^{-1}$), with this representing a 15% improvement in RE. This improvement in RE shows no sign of abating; most recently, RE was measured at $165 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{km}^{-1}$. These results share similarities with those recently reported by Coyle [38] for Lance Armstrong, the multiple Tour de France cycle champion. It appears, therefore, that the physiological adaptations which permit a reduction in the O_2 cost of sub-maximal exercise are the key to continued improvements in endurance exercise performance over the longer term.

Running Speed Associated With Maximal Oxygen Uptake ($S-\dot{V}O_2 \text{ Max}$)

Although $\dot{V}O_2 \text{ max}$, *per se*, did not change appreciably between 1992 and 2003, the improvement in RE that occurred over this same time period meant that the running speed corresponding to $\dot{V}O_2 \text{ max}$ (and at any given fraction thereof) was greatly increased in the latter years (Figure 5). In other words, the improved RE enabled exercise at a given absolute or relative intensity to be performed at higher running speeds.

Lactate Threshold (LT) and Lactate Turnpoint (LTP)

Representative blood lactate response profiles for several tests undertaken between 1992 and 2003 are shown in Figure 6. Notice the classical rightward shift of the $[\text{La}]_b$ -running speed relationship. To exemplify this shift, the running speed at an absolute $[\text{La}]_b$ of 3 mM was $16 \text{ km} \cdot \text{h}^{-1}$ in 1992 and $21 \text{ km} \cdot \text{h}^{-1}$ in 2003. Notice also that the point at which $[\text{La}]_b$ first rises above baseline values occurs at progressively higher speeds in later years. Indeed, the LT increased from $14\text{--}15 \text{ km} \cdot \text{h}^{-1}$ in 1992–1994 to $17.5\text{--}18.5 \text{ km} \cdot \text{h}^{-1}$ in 2000–2003; similarly, the LTP increased in a step-wise fashion from $16 \text{ km} \cdot \text{h}^{-1}$ in 1992 to $20 \text{ km} \cdot \text{h}^{-1}$ in 2003 (data not shown).

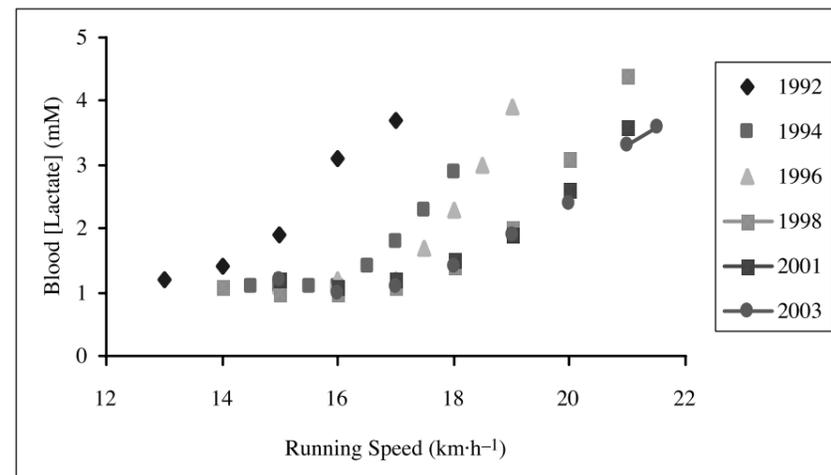


Figure 6. PR's Blood Lactate – Running Speed Relationship, 1992-2003

For clarity, only representative data are shown. Note the 'classic' rightward shift of the curve.

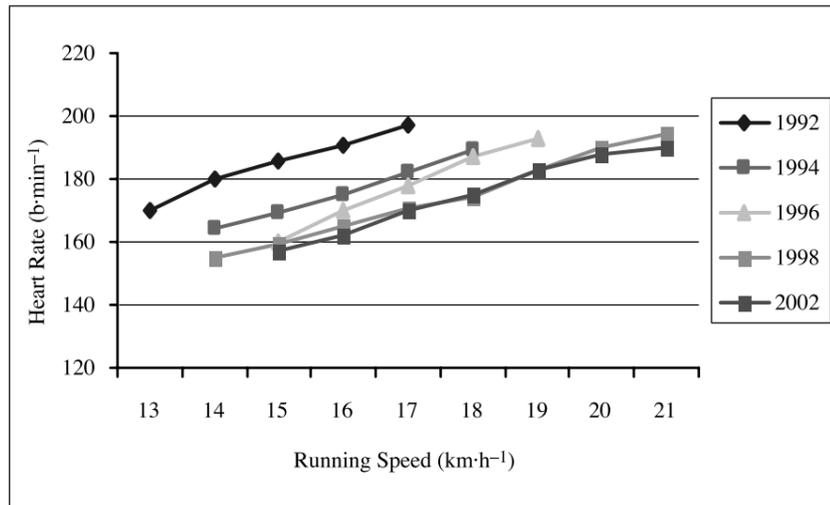
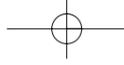


Figure 7. PR's Heart Rate - Running Speed Relationship, 1992-2003

For clarity, only representative data are shown. Note the 'classic' rightward shift of the curve.

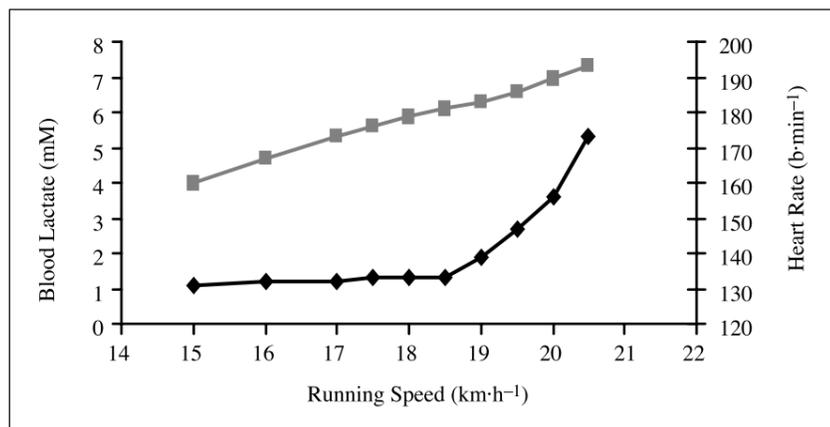


Figure 8. PR's Blood Lactate and Heart Rate Responses to Multi-Stage Incremental Treadmill Test in 2003

Blood lactate is shown in diamonds and heart rate in squares. Note that blood lactate does not rise above resting values until a speed close to 19 km·h⁻¹ is reached. The running speed at the lactate threshold (18.5 km·h⁻¹) and the running speed at the lactate turnpoint (20.0 km·h⁻¹) were very similar to the average running speeds sustained during recent best performances in the marathon and the 10,000 m, respectively.



Heart Rate (HR)

The HR response to the multi-stage treadmill test for a number of years between 1992 and 2002 is shown in Figure 7. Again, note the rightward shift in the HR-running speed relationship which is considered to be characteristic of enhanced cardio-vascular fitness. To illustrate the scale of the change in HR during sub-maximal exercise, a HR of $180 \text{ b} \cdot \text{min}^{-1}$ corresponded to a running speed of $14 \text{ km} \cdot \text{h}^{-1}$ in 1992 and to a running speed of $18.5 \text{ km} \cdot \text{h}^{-1}$ in 2002. The maximal HR declined from $203 \text{ b} \cdot \text{min}^{-1}$ to $197 \text{ b} \cdot \text{min}^{-1}$ over this same time period. Interestingly, the HR values corresponding to LT and LTP have remained essentially unchanged since 1992.

The actual $[\text{La}]_{\text{B}}$ and HR responses to a multi-stage treadmill test completed in 2003 (close to the time when PR set the World Record of 2 hours, 15 minutes, and 25 seconds for the marathon) is shown in Figure 8. Note that $[\text{La}]_{\text{B}}$ did not rise above baseline until a running speed of $18.5 \text{ km} \cdot \text{h}^{-1}$ (corresponding to a HR of approximately $182 \text{ b} \cdot \text{min}^{-1}$) was exceeded. It can be calculated that if the running speed at LT (i.e. $18.5 \text{ km} \cdot \text{h}^{-1}$) was sustained for the full marathon distance, the finishing time would be around 2 hours and 16 minutes – remarkably close to the time that was actually set. Moreover, the running speed at the LTP in this same test ($20 \text{ km} \cdot \text{h}^{-1}$) was identical to the average speed that PR sustained over 10,000 m some months earlier when setting the European record of 30 minutes and 1 second.

DISCUSSION

The physiological mechanisms responsible for the remarkable improvements in the parameters of aerobic fitness over a 12-year period in this exceptional athlete can only be speculated upon. Naturally, invasive procedures for the assessment of, for example, cardiac output, muscle blood flow, muscle fibre type, and mitochondrial and capillary density are not possible in longitudinal scientific support work in athletes of this calibre.

Before discussing the physiological adaptations which might have been responsible for the improvement in the aerobic fitness parameters presented earlier, it is perhaps worth noting that PR has unexceptional lung function values; her forced vital capacity, forced expiratory volume in 1 s, peak expiratory flow, and estimated maximal voluntary ventilation values all being very close to those predicted for someone of her age and height. It is also worth noting that, despite an increased stature between 1992 and 2003 (from 1.68 to 1.73 m), her body mass has been essentially unchanged (approximately 54 kg); her values for the sum of 4 skinfolds also indicate that she has become increasingly lean as her career has progressed. Having the appropriate body size and body composition for distance running has obviously played an important role in PR's successes to date.

Success in all sporting activities results from the combination of genetic predisposition, commitment, to many years of hard training. With regard to genetic predisposition, there are examples of champion athletes in PR's family. It is also noteworthy that PR's $\dot{V}\text{O}_2$ max at 18 years of age (when her training was minimal: i.e. less than 25–30 miles per week) was $72 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$; clearly, she was exceptionally talented. However, this athletic potential was only achieved following 10 further years of increasingly arduous training, underpinned throughout by scientific principles. PR's weekly training volume has increased considerably over her career, and today she will perhaps cover between 120 and 160 miles a week when in full marathon training. However, one cornerstone of PR's training philosophy is that she has never compromised training quality for quantity. Indeed, nowadays PR's "steady" continuous running, which makes up a large fraction of her total weekly mileage, will typically be performed at a pace of between 5:15 and 5:45 min:sec per mile (3:20–3:40 min:sec per km).

When she feels too tired to undertake a session at the appropriate intensity, she will rest rather than complete it 'sub-optimally'. Another important element in PR's training programme has been the inclusion of regular "tempo" running during which she will run at speeds close to her LTP (i.e. around 5:00 min:sec per mile or 3:08 min:sec per km) for extended periods. PR's weekly training programme will also typically include 1–2 higher-intensity (i.e. requiring 95–100% $\dot{V}O_2$ max) interval or repetition sessions performed on the track, road or cross-country, along with perhaps two weight training sessions. Much of her training is completed at altitude in Font Romeau in the French Pyrenees or in Albuquerque, New Mexico. Each and every one of these various features of her training programme might be considered to have played an important role in the dramatic improvement in endurance fitness that has been recorded (as reflected in measures of RE, LT and LTP).

As was emphasised earlier, it appears that enhanced exercise economy is central to the continued improvements in endurance exercise performance noted in elite athletes [37, 38]. However, because exercise economy is itself influenced by a wide variety of factors [12, 14, 15], it is very difficult to pinpoint the mechanism responsible for the improved RE reported herein for PR. There is some evidence that type I (slow-twitch) muscle fibres are more efficient than type II (fast-twitch) muscle fibres; that is, compared to type II fibres, type I fibres consume less O_2 for a given amount of muscle work [13, 39]. Endurance athletes tend to have a high percentage of type I fibres in the muscles that are predominantly used in their sports [40], although whether this results from genetic or training-related factors is unclear. It has been suggested that chronic endurance training might result in a transformation of type II fibres into type I fibres [38]. Certainly, with exposure to endurance training, type II fibres take on many of the properties of type I fibres, with reduced myosin ATPase activity, increased mitochondrial density and oxidative enzyme activities, and a greater capillary density [40, 41]. Given the above, a reduced recruitment of type II muscle fibres during sub-maximal exercise would be expected to reduce the O_2 cost of exercise and this, therefore, could be one explanation for the improved RE with continued endurance training noted in PR. The low peak $[La]_b$ recorded following maximal exercise is consistent with PR having a high oxidative capacity (and commensurately low glycolytic capacity) and/or a high proportion of type I fibres in the working muscles. It should also be remembered that subtle changes either in running technique or in anthropometric variables (body mass, composition, and dimensions) over the period of study might also have been responsible for the enhanced RE [12, 14, 15].

Other, perhaps more controversial, explanations for PR's improved RE with continued training include changes in muscle strength and/or flexibility, and her frequent training sojourns to altitude. Over the period of study, PR's weight training programme became more sophisticated and she improved her leg strength and power. For example, her vertical jump test performance improved from 29 cm in 1996 to 38 cm in 2003. There is some evidence that explosive strength training can improve both RE and running performance [14, 42, 43], although the mechanism for this effect remains to be resolved. Also over the period of study, PR's lower body "flexibility" as assessed, albeit rather crudely, with the sit-and-reach test, declined slightly (from 8 cm past her toes in 1996 to 4 cm past her toes in 2003), possibly as a consequence of her increased training mileage. There is a suggestion that "stiffer" muscle-tendon structures might improve RE by allowing a greater storage and subsequent return of elastic energy during the stretch-shortening cycle [14, 44]. It has also been suggested that exposure to hypoxia through altitude training might improve RE [14, 45] although this is controversial and the putative mechanisms responsible for any effect remain obscure.

The reduced HR measured during sub-maximal exercise (Figure 7) could be the consequence of a continued enlargement of the volume of the left ventricle with chronic

training [46]. This would increase the heart's stroke volume and, for the same cardiac output, allow a reduction in HR. An increased stroke volume would also offset the small decline in maximal HR with age and thus preserve the maximal cardiac output and the $\dot{V}O_2$ max. Another explanation for the reduced HR at absolute running speeds is that the improved RE with continued training reduced the leg blood flow requirement and therefore the cardiac output.

The dramatic reduction in blood lactate accumulation during sub-maximal exercise and the commensurate improvements in LT and LTP (of the order of 20–30%; Figure 6) would be expected to be advantageous to distance running performance. Metabolic acidosis has been suggested to be an important cause of fatigue during many types of exercise [although see refs. 31 and 33], and a reduced hydrogen ion accumulation (as reflected indirectly by measurements of $[La]_b$) following training might therefore be associated with enhanced exercise performance. Also, since lactic acid can only be produced when muscle glycogen (or glucose) is used as the metabolic substrate, a reduced lactic acid production might also signify a lower rate of muscle glycogen utilisation, an effect that might also be expected to improve endurance [2]. A greater oxidation of lactate as a fuel, or greater gluconeogenesis from lactate during exercise, are also likely to be important in the association between lower $[La]_b$ and enhanced endurance performance [47]. Finally, a reduction in metabolic acidosis should be associated with a reduced rate of pulmonary ventilation [48], which might, in turn, reduce the perception of effort [49].

An interesting feature of the $[La]_b$ response curves shown in Figure 6 is the rather low peak $[La]_b$ measured shortly after the cessation of the final stage of the exercise test. In PR, a maximal effort during a treadmill test typically results in a peak $[La]_b$ of just 4–6 mM, much lower than is normally observed in moderately-trained or even sub-elite athletes (8–12 mM). Coyle [38] has recently reported that Lance Armstrong has this same characteristic: this champion cyclist had a peak $[La]_b$ of ~ 6.5–7.5 mM compared to the 9–14 mM normally measured in competitive cyclists tested in the same laboratory.

Like RE, the dramatic improvements in LT and LTP observed with continued training are also difficult to attribute to any single physiological adaptation. Greater capillarisation would facilitate an increased oxygenation of muscle tissue that would tend to result in reduced muscle lactic acid production [25]. In addition, increased mitochondrial volume and density, and increased oxidative metabolic enzyme activity with training [40, 50], would increase the muscle's ability to extract and utilise the available O_2 . A greater ability to utilise free fatty acids as a metabolic substrate following training [49] would reduce muscle lactic acid production, and also potentially enhance endurance running performance by 'sparing' muscle glycogen. The ability to mobilise and oxidise carbohydrate through the 'shuttling' of lactate might also be important [26, 27, 47, 52]. Again, it should be remembered here that the reduced metabolic rate required when running at sub-maximal speeds post-training (i.e. improved RE) might itself have contributed to a lower $[La]_b$.

CONCLUSION

This paper has reviewed the physiological parameters of aerobic fitness and illustrated, using the data of PR, how they contribute independently and in combination to the determination of distance running performance. While a high $\dot{V}O_2$ max is a prerequisite for success at the highest level, factors such as a low O_2 (and thus energy) cost of running at sub-maximal speeds and a delayed accumulation of lactate in the blood, are also of great importance and appear to be responsive to training both in the short term and over the much longer term. In particular, the profound improvement in RE might be considered to be key to the continued

improvement in performance observed since 1992. Study of the great human athletes therefore continues to provide insights into the ultimate limits to exercise performance. Through determination, commitment, and consistently hard training, PR has achieved her athletic potential and become one of the greatest endurance athletes of all time. I have been greatly honoured to have been associated with her.

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