4
Physiological Limiting Factors and Distance Running: Influence of Gender and Age on Record Performances

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INTRODUCTION

Results from competitive distance running events can provide valuable insight into several key areas of integrative physiology. First, world records and other marks by elite athletes offer a framework for the discussion of how various physiological factors interact as determinants of performance [13, 50, 52, 54, 58, 62, 84]. Such analysis can also serve as fuel for the never-ending speculation about just how fast the "ultimate" time for a given distance might be [50, 52, 73, 74, 86]. Second, the decline in performance in elite older athletes can be used to gain insight into the overall reduction in physiological function with advancing years. This approach may be especially useful since it can isolate the effects of aging from the confounding influences of decreased physical activity and degenerative diseases that are seen in some older individuals [11, 32, 38, 41, 78].

With this information as background, the overall purpose of this review is to examine the current state of human performance in a physiological context. Key questions addressed include:

1. Can the continued improvements in performance seen in younger men and women be explained on a physiological basis?
2. Is the rate of improvement leveling off?
3. When does the age-related decline in endurance performance begin?
4. What is the rate of decline in performance with aging?
5. What physiological factors might be responsible for the decline in performance seen in older elite athletes.

In discussing each of these issues, attempts will be made to identify areas were experimental data are lacking.

PERFORMANCES CONSIDERED

Most of the discussion of performances will focus on the 10,000-m and marathon (42,195 m) runs since a large database exists for comparison in

younger and older athletes of both sexes [82, 92]. Occasionally, other performances will be considered when they illustrate an interesting point. The International Amateur Athletic Federation (IAAF) stipulates that official world records be set on a track, and refers to marks set on the road as "notable performances." The source for worldwide marks in open competition is the "Progression of World Best Performances and Official IAAF World Records" by zur Megede and Hymans [92]. Only records achieved prior to 1992 are used.

"Master" athletes will be defined as those over 40 yr old. Most of the age group records considered will come from TACSTATS/USA "Road Running Rankings" for 1990 [82]. This source provides a large and reliable database (primarily on U.S. citizens) that can be used to make valid comparisons across age groups. Other performances of interest by master athletes (and open competitors in their late 30s) have been culled from *Track and Field News* and *Runners World* magazines. The physiological data considered come from the scientific literature and books authored by international experts [4, 13, 59, 90].

PROGRESS OF HUMAN PERFORMANCE

Men have participated in organized competitions, over standard distances, with accurate time- and record-keeping for a little more than 100 years [86, 92]. In the past 20–30 years, similar competitions have become open to women. In both sexes, times continue to fall (Fig. 4.1, Table 4.1). Examples that highlight this ongoing improvement include:

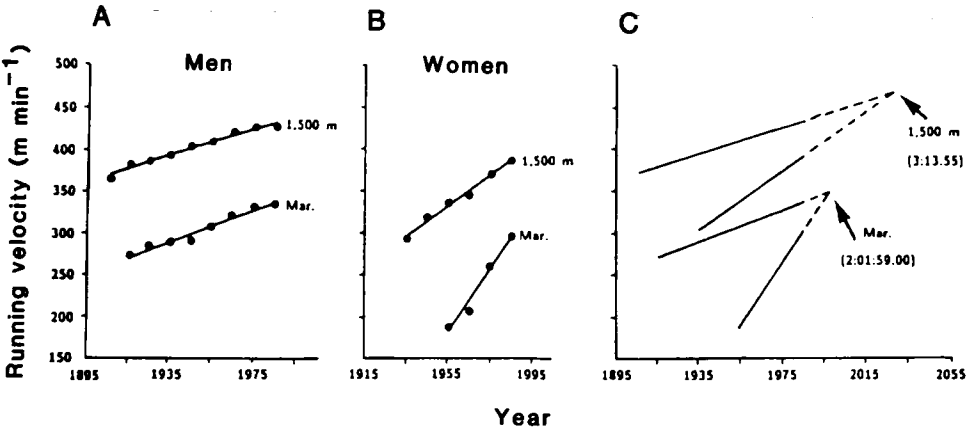
1. In open men's competition, the pace for the current world best in the marathon (approximately 333 $m \cdot min^{-1}$) is faster than the pace for the world-record 10,000-m run up to the late 1930s [92].
2. The current world best marathon for women (2:21.06) is faster than the men's mark was until 1952 [92].

A number of authors have analyzed the rates of decline in human performance over the years. Some have concluded that the decline is linear with time, while others have suggested that it is curvilinear and leveling off [59, 74, 86]. Additionally, the rate of decline in women is faster than that for men and has led to speculation about performances by women either equalling or "surpassing" those by men at some point in the future (Fig. 4.1 [86]).

There have also been remarkable performances by master athletes including a marathon best of 2:11.04 by a 41-yr-old man (Table 4.2). Such performances confirm laboratory investigations that suggest

FIGURE 4.1

Panel A *plots the change in average running velocity for the world record marathon* **(Mar.)** *and 1500 m distances against time for men.* **Panel B** *is a similar plot for times achieved by women.* **Panel C** *is a linear extrapolation of the lines from* **Panels A** *and* **B** *suggesting that if times for both men and women continue to improve at the same rate and in a linear manner, then the times by women will eventually equal and possibly surpass those by men. (Modified from Whipp, B. J., and S. A. Ward. Will women soon outrun men?* Nature *355:25, 1992.)*



intense exercise training can reduce dramatically the decline in physiological function with aging [1–4, 23, 38, 41, 64, 71].

The possible determinants of these remarkable performances by younger or older athletes of both sexes will be discussed in the context of the physiological limiting factors in endurance exercise [50]. By focusing on the achievements of elite athletes, issues related to maximum human adaptability will be highlighted.

PHYSIOLOGICAL FACTORS THOUGHT TO LIMIT DISTANCE-RUNNING PERFORMANCE IN YOUNG MALES

Three physiological factors: maximal oxygen uptake ($\dot{V}O_2max$), the so-called "lactate threshold," and running economy appear to interact as determinants of distance running performance [1, 4, 11–14, 16, 22, 30, 40, 50, 53, 62, 75, 79, 90]. Many of the studies on these factors have been conducted in highly trained male runners successful in local but not national or international competitions. However, the data available in elite athletes of both sexes and all age groups also suggest that these

TABLE 4.1
Some Notable Distance Running Performances

Men						
10,000 m (Track)	31:40.0 George 1884	30:58.8 Bouin 1911	29:52.6 Mäki 1939	28:54.2 Zàtopek 1954	27:39.4 Clarke 1965	27:08.23 Barrios 1989
Marathon	2:37:23 Siret 1908	2:29:39.2 Kolohmainen, A.W., 1912	2:18:40.2 Peters 1953	2:09:36.4 Clayton 1967	2:06:50 Densimo 1988	
Women						
10,000 m (Track)	35:30.5 Pigni 1970	34:01.4 Vahlensieck 1975	32:52.5* Shea 1979	31:35.3 Decker 1982	30:59.42 Kristiansen 1985	30:13.74 Kristiansen 1986
Marathon	2:55:22 Bonner 1971	2:49:40 Bridges 1971	2:38:19 Hanson 1975	2:27:32.6 Waitz 1979	2:21:06 Kristiansen 1985	

*Faster times had been run prior to this in "mixed" gender race.

TABLE 4.2
Some Notable Marks by Men and Women 35 Years of Age or Older

Men				*Women*			
1500 m	3:33.91	Boit	36 yr	1500 m	3:57.73	Puica	35 yr
Mile	3:55.19	Walker	38 yr	Mile	4:17.33	Puica	35 yr
	4:05.39	Waigwa	40 yr				
	4:30.6	Roberts	53 yr	5000 m	15:15.2	Larrieu	35 yr
10,000 m	27:17.48	Lopes	37 yr	10,000 m	31:35.52	Larrieu	35 yr
	36:04	Jensen	65 yr		32:41.98	Palm	45 yr
Marathon	2:07:12	Lopes	38 yr	Marathon	2:27:35	Larrieu	38 yr
	2:11:04	Campbell	41 yr		2:38:00	Palm	48 yr
	2:27:42	Green	55 yr		2:52:02	Irvine	54 yr
	2:38:46	Turnbill	60 yr				
	2:42:49	Davies	66 yr				

factors act as physiological determinants of performance in elite athletes [1, 12, 13, 19, 40, 60].

Maximal Oxygen Uptake

$\dot{V}O_2max$ is thought to represent the maximum integrative ability of the body systems to transport oxygen from air to the active muscles where adenosine triphosphate (ATP) is generated via oxidative processes for muscle contractions [4, 13, 72, 75, 88, 90]. Values in healthy but sedentary 20- to 30-year-old males are usually in the range of 40–50 $ml \cdot kg^{-1} \cdot min^{-1}$ [2–4, 13, 88]. In elite endurance runners tested on the treadmill, values range from 68–70 $ml \cdot kg^{-1} \cdot min^{-1}$ on the low side to approximately 85 $ml \cdot kg^{-1} \cdot min^{-1}$ for an upper limit [4, 13, 14, 16, 59, 62, 63, 67, 69, 88]. The high values seen in elite endurance athletes probably represent the combination of prolonged intense training and genetic factors that result in more marked adaptations than normal to training. Bouchard and colleagues have reviewed the complex issues related to the genetics of $\dot{V}O_2max$ in humans and concluded that "trainability" (i.e., the increase in $\dot{V}O_2max$ in response to a standardized training program) appears to be, in large part, genetically determined [8].

There has been continuing controversy about whether $\dot{V}O_2max$ in humans is limited by "central" (i.e., O_2 delivery) or peripheral factors in the active muscles [24, 72, 76]. If one takes the simple position that the muscle cannot extract what has not been delivered, then the conclusion is that the absolute highest $\dot{V}O_2max$ achievable for any individual is set by the maximum O_2 flux (cardiac output × arterial oxygen content) at the aorta. This means that maximum cardiac output, the hemoglobin concentration of the blood, and the ability of the lung to oxygenate adequately the blood returning from the active muscles and other tissues are of paramount importance as determinants of $\dot{V}O_2max$ [13, 28, 29, 51, 66, 85]. In this context, it appears that a large maximum stroke volume is the key adaptation that explains the high $\dot{V}O_2max$ values in elite endurance athletes [72, 76, 85].

The concept of a central, O_2 delivery-dependent, limitation of $\dot{V}O_2max$ is supported by experimental evidence that demonstrates maneuvers to either increase or decrease maximum O_2 flux cause directionally similar shifts in $\dot{V}O_2max$ [28, 29, 66, 72, 85]. In contrast, it is possible to show that peripheral factors such as muscle mitochondrial content (or function) can be altered without having much effect on $\dot{V}O_2max$ [21, 46, 47].

Of particular interest in the discussion of a central limitation of $\dot{V}O_2max$ are the recent observations demonstrating that, during very heavy exercise (>80% $\dot{V}O_2max$) in some elite distance runners, the lung is unable to oxygenate fully the returning venous blood which results in arterial desaturation (SaO_2 = 86–92%) in a large percentage of such subjects. Such desaturation might decrease $\dot{V}O_2max$ as much as 12–14% [24, 49]. This desaturation may limit $\dot{V}O_2max$ in the affected individuals.

The reasons for the desaturation are complex, but center on several observations: *(a)* breathing occurs at a rate and depth that is limited by intrinsic mechanical factors in the lung; *(b)* ventilation/perfusion mismatch is increased, and *(c)* the high ($>30\ l \cdot min^{-1}$) cardiac outputs that speed the transit of red cells through the pulmonary capillaries limit the time for equilibration of alveolar and arterial O_2 tensions. Taken together, these arguments favor the concept that the heart and lungs limit $\dot{V}O_2$max in elite human endurance athletes [24, 49, 72, 76].

While $\dot{V}O_2$max represents the upper limit of "aerobic" performance, in most athletes the pace used in a 10,000-m run or marathon requires less than 100% of $\dot{V}O_2$max [1, 12–14, 16, 30, 62, 75, 79]. Studies conducted in good (but not world class) runners suggest that humans can run at speeds that require between 79 and 98% of $\dot{V}O_2$max during 10,000-m races and between 68% and 88% of $\dot{V}O_2$max during the marathon [30]. Similar conclusions are drawn from other studies at various distances [12–14, 16, 53, 79]. There have also been examples of champion runners who could sustain approximately 90% or more of their $\dot{V}O_2$max values for the marathon [13].

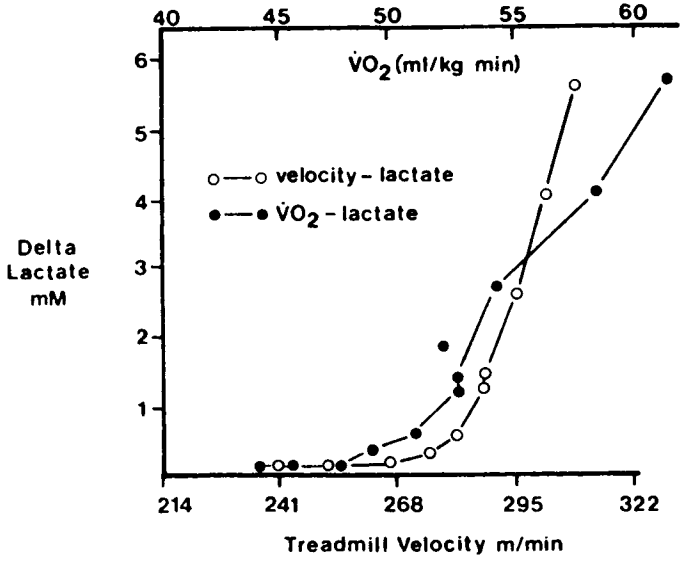
Lactate Threshold

Since $\dot{V}O_2$max is probably not sustained by most individuals over the entire course of a 10,000-m or marathon race, a key question is what factor(s) determines the fraction of $\dot{V}O_2$max that an individual can sustain for efforts between roughly 30 minutes and several hours? A number of studies suggest (based on blood samples) that the fraction of $\dot{V}O_2$max used in endurance races is somehow related to the accumulation of lactic acid in the active muscles [4, 16, 30, 53, 79]. In trained subjects, there is little or no increase in blood (or presumably muscle) lactate until a running speed that uses 60–85% of $\dot{V}O_2$max is reached [13, 14, 30]. This initial increase in blood lactate (Fig. 4.2) is followed by an exponential rise at faster running speeds [14, 30].

The phenomenon associated with and cause(s) of the changes in blood lactate have been described with a variety of terms, and the physiological events in active muscle that result in an increase in the blood lactate concentration during heavy exercise remain immersed in controversy [22]. An extensive discussion of this issue exceeds the scope of this review. However, the view that the rise in blood lactate is representative of inadequate oxygen delivery to the mitochondria in the active muscles is probably simplistic [22]. A more attractive explanation is that the increase in blood lactate is somehow reflective of a mismatch between the rate of pyruvate delivery to, and utilization by mitochondria in the otherwise "oxygenated" active muscles [46–48]. For the purposes of this review, the point at which there is a detectable (1–2 mMol) increase in the blood lactate concentration will be referred to as the "lactate threshold."

The essential concern from an applied point of view is that well-

FIGURE 4.2
An individual record of how blood lactate values change above resting as running speed and oxygen consumption increase. Note that there was little increase in blood lactate above resting values until the treadmill velocity exceeded 268 m·min^{-1} and the oxygen consumption was above 50 ml·kg^{-1}·min^{-1}. (Reproduced with permission from Farrell, P. A., J. H. Wilmore, E. F. Coyle, J. H. Billing, and D. L. Costill. Plasma lactate accumulation and distance running performance. Med. Sci. Sports *11:338–344, 1979.)*



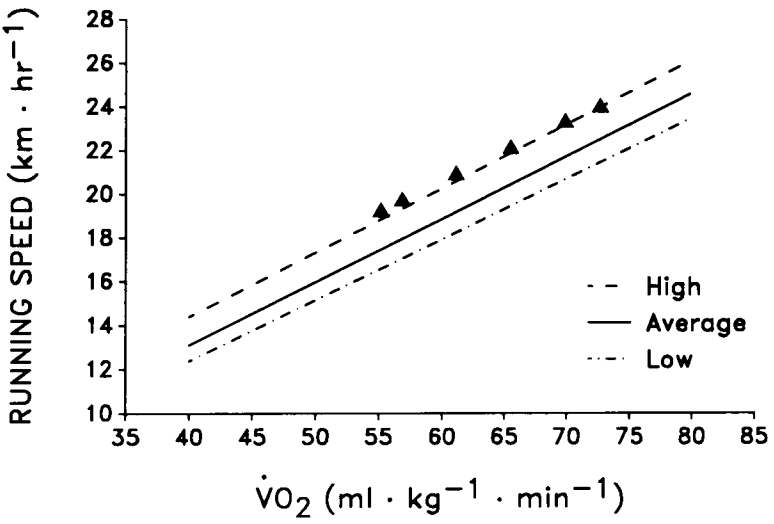
trained subjects can sustain a fraction of their $\dot{V}O_2$max that is slightly above their lactate threshold for several hours, and that the lactate threshold is highly predictive of the fraction of $\dot{V}O_2$max that can be sustained during shorter events as well [14, 30, 50, 53, 79]. The extremely high percentages of $\dot{V}O_2$max that can be sustained by some elite athletes in marathon races is probably explained by their very high lactate threshold values [13].

Running Economy

The relationship between running speed and oxygen consumption (in ml · kg^{-1} · min^{-1}) has been termed running economy. It can differ among individuals so that two hypothetical runners with identical $\dot{V}O_2$max values may require different amounts of O_2 to run the same submaximal speed [12, 13, 16, 19, 20, 60, 63] (Fig. 4.3).

The oxygen cost to run progressively faster speeds appears to increase linearly when this issue is studied using relatively slow speeds in both average subjects and competitive runners on the treadmill at speeds up

FIGURE 4.3
The relationship between running speed and oxygen consumption for individuals with "low," "average," and "high values" for running economy is demonstrated. Individuals with low running economy curves achieve substantially less running speed than individuals with high values for a given $\dot{V}O_2$. The high and low curves were generated using linear regression equations for the two most economical and two least economical individuals studied by Conley and Krahenbuhl [12, and Krahenbuhl, personal communication]. The "average" running economy curve was generated using the mean values from these data [12]. These curves were then extrapolated to higher running speeds, and individual examples **(triangles)** *of excellent running economy values were plotted at high speed in elite athletes [Daniels, unpublished observations] to confirm that linear extrapolation of the data collected at lower speeds by Conley was justified. (For more details, see Joyner [50].)*



to approximately 320 m·min^{-1} [12, 13, 37, 59, 63]. While a few studies and anecdotal data suggest that the increase remains linear at faster speeds, there has been little systematically collected data at running speeds ≥20 km·hr^{-1} that would have relevance to competitive efforts by elite athletes. There is also little information on how the O_2 cost of actual overground running differs from treadmill running at higher speeds because of factors like wind resistance [18, 50]. Finally, most measurements of running economy have been made during brief treadmill runs, so there is little information on how any upward drift in $\dot{V}O_2$max over time might alter running economy during several hours of exercise [50].

Integration of Factors

In groups of subjects with widely differing $\dot{V}O_2max$ values, a strong correlation between performance and $\dot{V}O_2max$ exists. The relationship between $\dot{V}O_2max$ and performance is much weaker in subjects with more homogeneous $\dot{V}O_2max$ values [12, 13, 30, 59, 63]. The current concept is that the lactate threshold determines (or is related to) the fraction of $\dot{V}O_2max$ that can be sustained by an individual in events lasting beyond 10–15 minutes, and that this value interacts with running economy to determine the actual running speed in competition [13, 30, 40, 50]. The so-called running speed at lactate threshold appears to be highly predictive of distance running performance at events including the 10,000-m run and marathon [1, 11–13, 30, 50, 53, 79].

This interaction explains how a subject with relatively "low" $\dot{V}O_2max$ values for elite runners can remain competitive with those whose $\dot{V}O_2max$ values are 80–85 $ml \cdot kg^{-1} \cdot min^{-1}$ or more. The individuals with "low" $\dot{V}O_2max$ values usually do not reach their lactate threshold until 85–90% of $\dot{V}O_2max$. They also generally have excellent running economy values. These concepts are illustrated by the fact that at 5000-m and 10,000-m, the performances of Frank Shorter ($\dot{V}O_2max$ of ~70 $ml \cdot kg^{-1} \cdot min^{-1}$) are virtually identical to those of Steve Prefontaine ($\dot{V}O_2max$ ~85 $ml \cdot kg^{-1} \cdot min^{-1}$) [13, 59, 63]. Shorter was known to have excellent running economy values and probably also had a very high lactate threshold [13, 63].

If $\dot{V}O_2max$, lactate threshold, and running economy interact as discussed, then it should be possible to predict an "optimal" time for the marathon if the same individual possessed exceptional values for all three variables, and race conditions were otherwise ideal. This means that for an individual with a very high $\dot{V}O_2max$ value (84 $ml \cdot kg^{-1} \cdot min^{-1}$), a lactate threshold at 85% of $\dot{V}O_2max$, and an excellent running economy curve, the predicted optimal marathon time would be about 1:57, an improvement of roughly 9 min in comparison to the current world best [50]. This calculation leads to several conclusions: *(a)* there are additional poorly understood limiting factors in truly elite athletes; *(b)* exceptional values for one of the limiting factors might preclude exceptional values in another (i.e., $\dot{V}O_2max$ and running economy?); and *(c)* there is a relative lack of systematically collected lactate threshold and running economy data in truly elite athletes running at fast (>20 $km \cdot hr^{-1}$) but submaximal speeds during both treadmill and overground running.

PHYSIOLOGICAL IMPROVEMENT OVER THE LAST 50–100 YEARS?

Can improvement in any one or all three of these factors explain the improvements in performance in men over the last 100 years? Robinson,

Edwards, and Dill, working at the Harvard fatigue laboratory, observed very high $\dot{V}O_2$max values in elite runners in the 1930s [69]. A value of 82 $ml \cdot kg^{-1} \cdot min^{-1}$ was observed in Donald Lash who was one of the first men to run 2 miles in less than 9 min and held the world record for that distance at the time he was studied. They also noted values in the 75-$ml \cdot kg^{-1} \cdot min^{-1}$ range in several individuals [67, 69]. Based on recent studies demonstrating that brief but very intense training can elicit and maintain large changes in $\dot{V}O_2$max, it would appear reasonable to assert that the training programs used by elite athletes of the 1930s probably allowed the runners studied by Robinson to achieve their individual genetically limited $\dot{V}O_2$max values [33, 42–45, 67, 69, 91]. These data also make it seem unlikely that large improvements in $\dot{V}O_2$max are responsible for the 10% (or more) improvements in the world records for 10,000 m and the marathon since the 1930s.

In the 1960s, Saltin and Åstrand observed similar $\dot{V}O_2$max values to those reported by Robinson in elite runners [75]. They speculated that the factor(s) responsible for the improvement in performance by elite world-class runners from the 1930s to the 1960s was probably related to an improved ability to sustain a high fraction of $\dot{V}O_2$max in competition and to better technique [75]. This view is consistent with how training has evolved in this century. Initially, there was little year-round training, and not even daily training during the competitive season [91]. From 1900 to about 1960, there was a steady increase in the frequency, intensity, and duration of training so that, by the late 1950s or early 1960s, it was not uncommon for athletes to average 2 hr of training (some of it very intense) per day on a year-round basis. Since that time, the training programs used by top athletes have not changed dramatically [91].

These changes in training methodology are consistent with the observation that the mitochondrial adaptations in skeletal muscle that probably cause the lactate threshold to increase to the high values seen in elite athletes are maximized with 1.5–2.0 hr of daily training [27, 46, 47]. Therefore, it would appear reasonable to speculate that training-induced mitochondrial adaptations appear to have allowed athletes to sustain higher percentages of their $\dot{V}O_2$max values in competition as a result of improved lactate threshold values. These adaptations would seem to explain much of the improvement in performance from the 1930s to the 1960s [75].

This view is supported by the lactate threshold and running economy values first observed in elite competitors from the 1960s [13, 14, 16]. These data suggest that there have been no dramatic improvements in these factors over the last 20–30 yr since the currently used approaches to training emerged. However, the improvements in performance for both the 10,000 m and marathon have both been only about 2% since this time.

If champion human athletes have been training "as hard as possible"

and, hence, have maximized their training-induced physiological adaptations (i.e., $\dot{V}O_2max$, lactate threshold) since the early 1960s, and if running economy is only minimally altered by training, why have world records continued to fall? One explanation is that synthetic tracks and better footwear have contributed to the improved performances. Although there are few hard data to support this contention, there is evidence to suggest that the design and composition of running tracks can improve competitive performance by about 2–3% [55, 56]. If the current synthetic surfaces and shoes offer an advantage of this magnitude, the last world records set on natural tracks (i.e., cinder or clay) in the middle 1960s came remarkably close to matching the current world marks if they are "corrected" by about 2% for the different surfaces (Table 4.3). This interpretation supports the contention that physiological improvement by record holders in the last 25 yr has probably been minimal.

Competitive Opportunities

A variety of sociological factors have probably contributed to the improvements in world record performance in men over the last 30 yr. First, beginning with Alain Mimoun (a native of Algeria who competed for France) at Melbourne in 1956, and Abeke Bikila (Etheopia) in 1960 at Rome, there has been the emergence of competitive opportunities for individuals from the so-called "developing countries." Their contribution is highlighted by the success of East Africans in the Olympic games. Second, the financial rewards to those successful in international competition have increased dramatically.

These factors mean that more potential record setters have the opportunity, motivation, and means to train and to compete (full time) beyond their early 20s into their 30s. This increases the number of top-class athletes that can be drawn to selected races and enhance the competitive atmosphere, contribute to the early pace, share the burden of overcoming wind resistance, and generally improve the chances for a record in current competitions.

An example of how these sociological factors might improve performance can be seen by comparing results of the 1969 Antwerp marathon in which Derek Clayton set a world best of 2:08:33, with the results of the 1988 Rotterdam marathon that produced a world best of 2:06:50 for Belayneh Densimo. The second place finisher in the 1969 race was almost 3 minutes behind in comparison to a gap of only 17 seconds between first and second in 1988. The tenth place time in 1969 was 2:22:13 vs. 2:12:42 in 1988. There were no finishers from the developing nations in the top 12 in 1969. Seven of the 12 finishers, including the first four who all broke 2:10, were from developing countries in the 1988 Rotterdam race [92].

TABLE 4.3
Comparison of World Records on "Natural" and Synthetic Track Surfaces*

Surface	*1500 m*	*1 Mile*	*3000 m*	*5000 m*	*10,000 m*
Natural	3:33.1 Ryun, 1967	3:51.1 Ryun, 1967	7:39.6 Keino, 1966	13:16.6 Clarke, 1966	27:39.4 Clarke, 1965
Synthetic	3:29.46 Aouita, 1985	3:46.32 Cram, 1985	7:29.45 Aouita, 1989	12:58.39 Aouita, 1987	27:08.23 Barrios, 1989
Improvement	1.7%	2.1%	2.2%	2.3%	1.9%

*Records set before 1992.

Doping

It is possible that the use of banned substances may have contributed to improved performances by some distance runners. In endurance competition, much attention has been focused on autologous transfusions of red blood cells to athletes or "blood-doping," and the more recent related use of synthetic erythropoietin [28, 29, 66, 87]. Both approaches are effective in increasing $\dot{V}O_2$max and, possibly, performance in fit subjects [87]. However, neither technique has been extensively studied in truly elite athletes.

In any case, it is impossible to say what effect (if any) these procedures have had on world records because of a lack of reliable information. The success of athletes from developing countries and the formidable logistical challenge that blood-doping might present to these individuals makes it seem less likely that blood-doping has had a major impact on international competitions. Additionally, the previously stated argument that the record holders from the middle 1960s are as physiologically gifted as those today suggests a minimal impact. It can only be hoped that this is the case.

Performances by Women

The IAAF did not sanction races for women that were longer than 1000 m until 1967 when the 1500 m and mile were recognized. The 10,000-m event was not added until 1981. It is unclear when the IAAF first began to acknowledge the women's marathon, but the event was not contested in the Olympics until 1984 [92]. When "pre-IAAF" times are considered, the time span available to analyze women's records should probably be limited to the last 20–25 yr because of insufficient opportunities for women to compete at almost every level. In the last 20 yr, the record for 10,000 m has dropped from 35:00.4 in 1975 to the current mark of 30:13.74 set by Ingird Kristiansen in 1986. The marathon best has fallen from 2:46:36 (set by Gorman in 1973) to 2:21:06 (also by Kristiansen in 1985). Based on these improvements, it is clear that, in the recent past, world records for women have fallen at a much faster rate than for men (Fig. 4.1, Table 4.1). An interesting issue is whether this improvement is due primarily to physiological or sociological factors (or both), and whether the rate will continue.

When historical performances for women and men are plotted against time (in years), several authors have concluded that performances are advancing at a linear rate (see Fig. 4.1 [74, 86]). Others can view similar data and conclude that the rate of improvement is subtly curvilinear and may be leveling off [59]. If care is not taken, it is possible to imply that the reason for the continued "linear" improvement is due to "physiological" improvement while ignoring the previously discussed sociological and other factors that might be contributing to improved performance. This is especially possible when evaluating trends in running performance by

women who, unlike men, experienced a dramatic increase in the severity of their training regimens and opportunities to compete *at about the same time*. This experience contrasts with the more gradual changes in training and competitive opportunities previously discussed for men. These issues will be discussed below.

Physiologically Limiting Factors in Women

There was little information on the physiological determinants of running performance in women until the 1970s. The currently available data, while limited in comparison to those available on men, suggests that $\dot{V}O_2$max, lactate threshold, and running economy interact in women as determinants of performance in a manner similar to that in men [13, 15, 19, 60, 88]. The areas where information is particularly lacking for women in comparison with men are on the relationship among the lactate threshold, running economy, and performance.

By the middle 1970s, at least a few women runners who were studied had $\dot{V}O_2$max values approaching 70 $ml \cdot kg^{-1} \cdot min^{-1}$, with values up to 77 $ml \cdot kg^{-1} \cdot min^{-1}$ seen in a cross-country skier [4, 25, 88]. These values are near the upper limit of those currently observed. However, the best early studies (1970s) on groups of elite female runners suggested that the average $\dot{V}O_2$max value in these groups was 58–60 $ml \cdot kg^{-1} \cdot min^{-1}$ [19, 60, 89]. Subsequent studies indicated that $\dot{V}O_2$max values in elite female athletes are approximately 67 $ml \cdot kg^{-1} \cdot min^{-1}$ [60]. Individual values of 73–77 $ml \cdot kg^{-1} \cdot min^{-1}$ seem to be near the upper limit reported for women runners [13, 59, 60]. These values are about 10–15% lower than the average value of 77 $ml \cdot kg^{-1} \cdot min^{-1}$ and highest value of 84–85 $ml \cdot kg^{-1} \cdot min^{-1}$ seen in comparable groups of men [59, 88].

When absolute ($l \cdot min^{-1}$) values for $\dot{V}O_2$max are considered in elite runners, they are much lower (3.15 vs. 5.19 $l \cdot min^{-1}$) in women than in men [60, 63]. This should not be surprising since the women are much smaller than the men and, therefore, probably have smaller stroke volumes and lower cardiac outputs during peak exercise. Additionally, the hemoglobin concentrations in elite female runners are lower [26, 57].

The current explanation of the gender differences in $\dot{V}O_2$max among elite runners when expressed relative to body weight is 2-fold. First, elite females have more (14% vs. 5%) body fat than men [35, 65, 89]. Much of the difference in $\dot{V}O_2$max disappears when it is expressed relative to lean body weight [60, 80]. Second, the hemoglobin concentration of elite female runners in 5–10% lower in women than in men [26, 57, 85].

Lactate Threshold

The mitochondrial adaptations in the skeletal muscles of highly trained male and female runners appear similar [15, 31]. These adaptations are thought to play a key role in explaining the very high lactate threshold values observed in elite athletes and other trained runners [46, 47].

While comprehensive studies relating lactate values to performance similar to those by Costill [14, 16] and Farrell et al. [30] have not been conducted on female athletes, there is no reason to believe that values for the lactate threshold will be lower in women than in men. It is interesting to wonder if some of the extremely high anecdotal values seen in men for the lactate threshold (90% of $\dot{V}O_2max$) will also be seen in women.

Running Economy

The average oxygen cost to run a given speed (241 $m \cdot min^{-1}$) by groups of elite male and female runners appears to be similar [12, 13, 60, 63]. This comparison requires some extrapolation because the running speeds used in the best study to date on women [60] differ from those used in the studies of men (230 and 248 $m \cdot min^{-1}$ for women vs. 241, 268, 295, and 322 $m \cdot min^{-1}$ in men) [12, 63]. Additionally, the range of values observed within a group of female athletes also appears to be similar and probably explains much of the difference in performance observed among women with high $\dot{V}O_2max$ values [12, 60].

Of note are the fairly complete running economy data available on Grete Waitz, the dominant female marathon runner of the late 1970s and early 1980s. Her running economy values at speeds from 215–295 $m \cdot min^{-1}$ are almost identical to those recorded by Derek Clayton and other male athletes noted for their excellent running economy values [12, 13, 50, 63]. This is further evidence that running economy values for elite male and female runners are similar and probably play the same role in determining success in distance running performance. As is the case for men, there are few reported data for elite women at very fast speeds during both treadmill and overground running.

Integration of Factors

When reviewing the current information about how the three key factors thought to limit human performance interact in women, it appears that although comprehensive data is lacking, they probably operate in the same manner in women and in men. The major physiological reason that would appear to "explain" the slower records by women than men is probably the lower $\dot{V}O_2max$ values observed in women. However, this conclusion relies on average data and ignores the fact that a number of female athletes have $\dot{V}O_2max$ values that are as *high or higher* than champion male runners! If such a value occurred in a women with excellent running economy and a high lactate threshold, it is easily conceivable that the world best marathon for women could fall to at least several minutes under 2:20, and that the world record for 10,000 m might drop below 30 minutes. Such times would certainly seem feasible based on currently available data and concepts [13, 50, 60].

However, the same caution required in concluding that there is substantial "physiological" room for improvement in record perform-

ance by elite male athletes should also be applied to women [50]. If, for example, an exceptional value for $\dot{V}O_2$max somehow precludes outstanding values for either running economy or the lactate threshold, then the marked improvements speculated about would be less likely to occur [50]. It should be remembered that while a number elite male and female runners have similar $\dot{V}O_2$max values, these values are at the low end of the elite range for men versus the upper end of the elite range for women. If an excellent value for one of the limiting factors is mutually exclusive with another, this means that it might be more likely for some of the males with $\dot{V}O_2$max values in the low range for men to have outstanding running economy and lactate threshold values and, therefore, faster times in competition than a female competitor with the same $\dot{V}O_2$max [50].

Competitive Opportunities

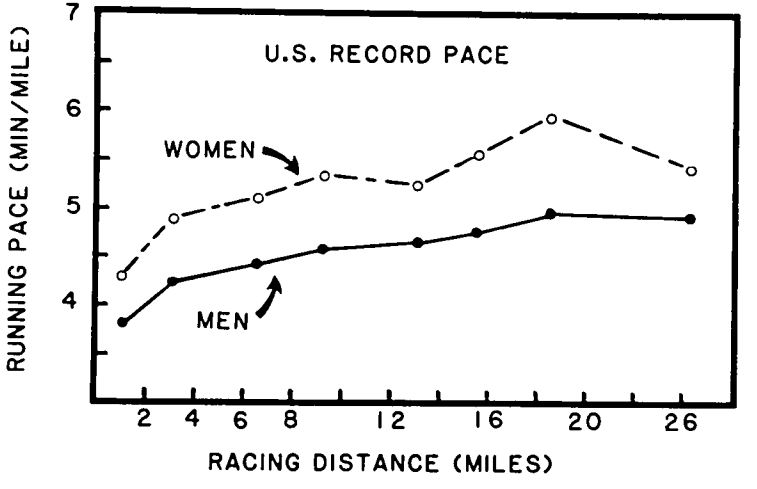
The rapid improvement in distance running performance by women over the last 20 yr would appear to represent the synergistic effects of both physiological improvement and enhanced competitive opportunities occurring at the same time for women in contrast to the more gradual experience of men. The rapid physiological improvement probably resulted from the fact that women were quick to adopt training programs similar to those developed by men through trial and error over many years. These improvements were magnified by improved competitive opportunities and a larger talent pool.

Additionally, most of the improvements in distance running times by women have been achieved by competitors from the developed countries. This means that, in some respects, the situation for women is similar to what it was for men before the emergence of runners from the developing countries in the 1960s. Recently though, women from East Africa have started to make their presence known in international competition. It is likely that these athletes will contribute to the continued improvement in performance by women.

It is also tempting to speculate that women from the developing countries will continue to improve since it is now more socially acceptable for young girls to participate in sports. Such participation could enhance any long-term adaptations that might occur during growth and development and contribute to improved performances in the future. It is unclear, however, if such changes will overcome the negative societal trends toward inactivity and obesity by children in the developed countries, particularly in the U.S. [90].

Will distance running records set by women ever match or exceed those by men (Fig. 4.4 and 4.5) [86]? The previously discussed data indicate that, at least for men, and possibly for women, there has been little physiological improvement among champions since the adoption of prolonged intense daily training regimens. If this is the case, then it is unlikely that records will continue to fall at a linear rate [59, 74, 86]. A

FIGURE 4.4
The record running pace for women and men in races ranging from 1500 m to the marathon in the middle 1980s is plotted. The differences in record performance by men and women are fairly constant across a wide range of endurance running events. (Reproduced with permission from Costill, D. L. Inside Running: Basics of Sports Physiology. *Indianapolis, IN: Benchmark, 1986, pp. 1–189.)*



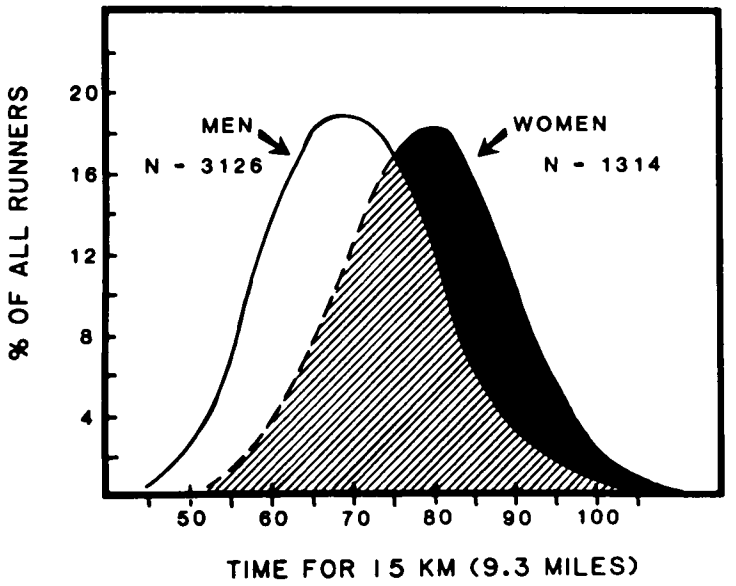
closer look at the data shows that all of the world records for distance races (more than 1500 m) by women were set before 1986, whereas a number of men's records have improved since then. These observations would seem to make it less likely that men's and women's records will converge in the near future. However, sociological factors may be more likely to lead to further marked improvements in women.

Performances by Older Elite Athletes

A number of notable marks by athletes in their late 30s or older are listed in Table 4.2. When the best times by citizens (of both sexes) of the United States for the 10,000 m (road courses) and marathon are plotted against age, it appears that the rate of decline in performance with aging is slight until the late 30s (Figs. 4.6 and 4.7). Thereafter performance appears to fall at the rate of between 6–9% per decade until the late 50s when it accelerates further. The decline in records by women appears to be greater than that for men. It is likely that this reflects the fact that there are fewer women engaged in master's competition than men, and that many more of the men have probably been active and engaged in competitive sports throughout life.

A similar rate of decline in performance also appears to occur when all

FIGURE 4.5
The distribution of performances for men and women in a 15-km race is shown. While a large number of women achieve faster times than men, the sex-based differences seen when record performances are considered (Fig. 4.4) are also evident when larger groups of nonelite performers are considered. (Reproduced with permission from Costill, D. L. Inside Running: Basics of Sports Physiology. *Indianapolis, IN: Benchmark, 1986, pp. 1–189.*



finishers of endurance competitions are considered, indicating that it applies to highly trained (but non-elite) competitors in the older age groups ([6, 7] and Fig. 4.8). These and other performances (many by athletes in their 50s and 60s), along with physiological data, clearly suggest that the rate of decline in performance in well-trained aging endurance athletes is probably less than the frequently cited 9–10% per decade drop in $\dot{V}O_2$max seen in sedentary subjects [2–4, 9, 10, 23, 25, 32, 36, 38, 39, 63–65, 78, 81].

Physiological Factors and Aging

$\dot{V}O_2$MAX. The study of how the physiological factors that limit endurance running performance decline with age has focused primarily on measurements of $\dot{V}O_2$max [2–4, 23, 32, 36, 41, 67–69, 81]. Large population-based studies are difficult to apply to highly motivated athletes because most study "normal" older subjects, and the decline in $\dot{V}O_2$max observed reflects both the effects of aging per se and any changes in habitual physical activity and body composition that are

FIGURE 4.6
Year-by-year age record performances in 10,000-m road races by U.S. citizens. The **left panel** *demonstrates that, in men, the decline in record time with age appears to be about 6% per decade until the middle or late 50s. Thereafter, the rate of decline appears to accelerate. In women, the average rate of decline with aging appears to be closer to 9% per decade into the late 50s. However, some of the individual values suggest a rate of decline similar to that seen in men. After age 60 yr, the rate of decline in women appears to be substantially greater than that for men. It is unclear if this apparent faster decline in women has a biological basis, or is merely the result of sociological factors that have limited the participation of women (in comparison with men) in distance running competitions. (Data from* TACSTATS/USA Road Running Rankings, *1990.)*

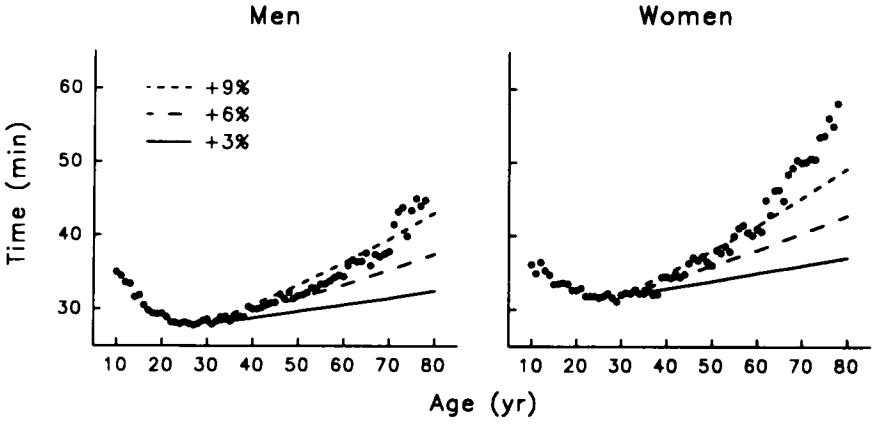


FIGURE 4.7.
Year-by-year U.S. age record performances in the marathon for men and women. These data confirm the observation made in Figure 4.6. (Data from TACSTATS/USA Road Running Rankings, *1990.)*

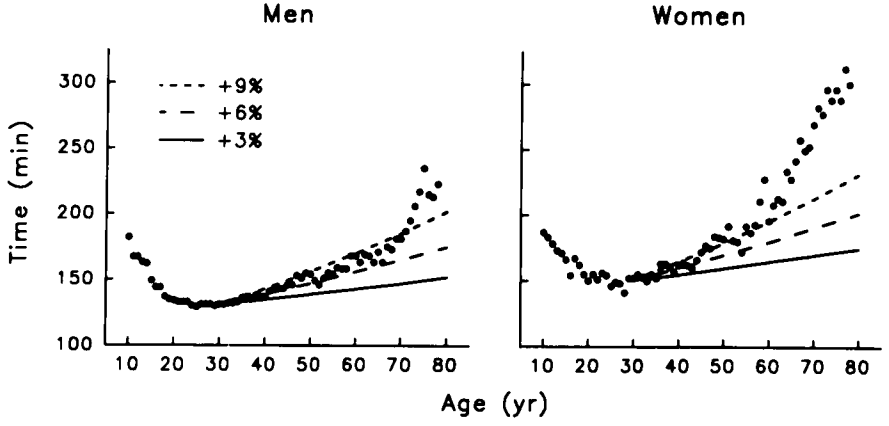
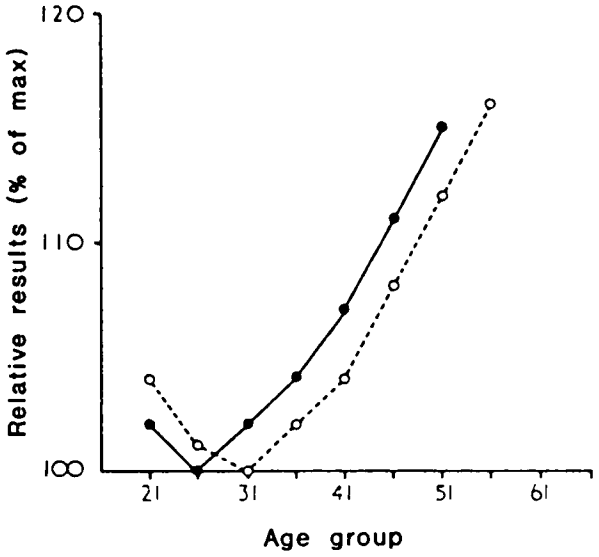


FIGURE 4.8

The increase in either running **(closed circles)** *or cross-country skiing* **(open circles)** *times during two long distance races is shown. The results are the relative time it took competitors in various age groups to complete the courses in comparison with the average time for the fastest age group. These data suggest that the decline in performance among well-trained, but nonelite competitors is also 6–7% per decade. This value is similar to the decline in performance seen when only record performers are considered. (Reproduced with permission from Böttiger, L. E. Regular decline in physical working capacity with age.* Br. Med. J. *3:270–271, 1973.)*



frequently associated with aging [9, 10, 32, 41, 81]. In general, these studies suggest that $\dot{V}O_2$max falls at the rate of about 9–10% per decade beginning at age 30 yr in healthy but sedentary older subjects of both sexes. For the general population, cross-sectional studies may underestimate the rate of decline because it is likely that only more vigorous and disease-free subjects volunteer to be tested [81]. In longitudinal studies of athletes who stop training, a similar decline is seen [67].

The factors responsible for the age-related decline in $\dot{V}O_2$max in inactive subjects are complex and controversial. It is likely that a reduction in maximum cardiac output because of a decline in maximum heart rate and stroke volume plays a major role [34, 38, 41, 81]. While there is some evidence to suggest that maximum cardiac output is maintained in older subjects [70], this observation has not been confirmed by other studies [34, 81]. Additionally, there is no reason to expect that the well-established relationship between maximum cardiac

output and $\dot{V}O_2max$ seen among other populations does not also occur with aging. There is also probably a modest decline in peripheral oxygen extraction [34, 81]. Age-related increases in body weight and body fat also contribute to the decline in $\dot{V}O_2max$ when it is expressed relative to body weight [81]. Some have also argued that the loss of lean body tissue plays a role [5, 32]. It is unclear how changes in total body hemoglobin that can alter $\dot{V}O_2max$ in younger subjects might affect $\dot{V}O_2max$ in older individuals [4, 85].

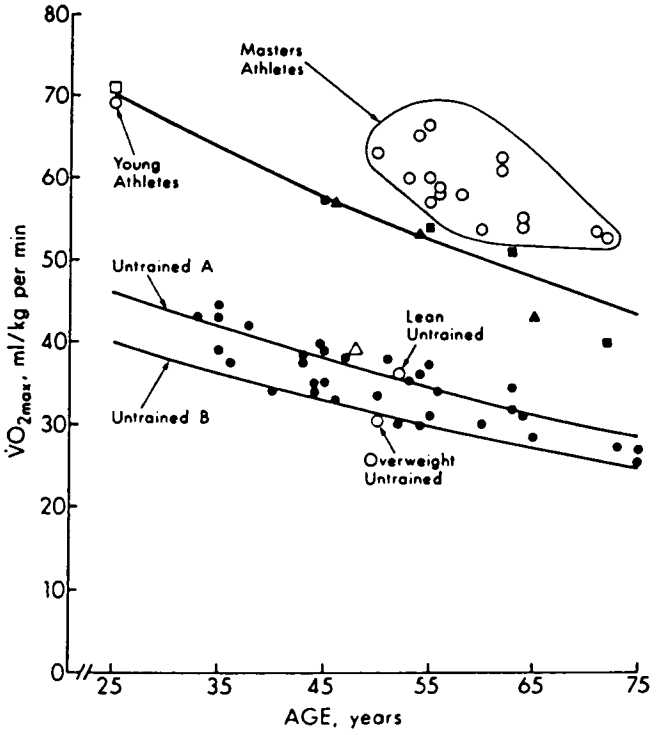
The rate of decline in $\dot{V}O_2max$ in sedentary subjects first noted many years ago appears to be substantially greater than the rate of decline in performance seen in older athletes [6, 7]. These observations suggested that training might limit some of the age-related decline in $\dot{V}O_2max$. This possibility was supported by observations of high $\dot{V}O_2max$ values in at least some older athletes, and observations that the $\dot{V}O_2max$ fell less in more active subjects followed in longitudinal studies [9, 23, 36]. Additionally, one of the champion runners followed by Robinson continued moderately hard training into his 50s [67, 69, Wilt, personal communication]. Fred Wilt, who ran 4:08 for the mile, and is a noted authority on the history of distance running training, did not "retire" from competition until age 36. He then continued to train vigorously 5 days/week, frequently running repeat 200-m intervals (Wilt, personal communication). His $\dot{V}O_2max$ fell from 4.7 $l \cdot min^{-1}$ in his early 20s to 4.4 $l \cdot min$ in his middle 50s. This type of limited duration but intense training is thought to be capable of maintaining $\dot{V}O_2max$ at or near its upper limit [33, 42–45].

Subsequent well controlled cross-sectional and longitudinal studies of older elite distance runners have been conducted demonstrating that the fall in $\dot{V}O_2max$ with aging can be blunted by about one-half (i.e., to 5% per decade [see Fig. 4.9]) with continued hard training [41, 64, 71]. The primary reason for the decline appears to be an age-related reduction in maximum heart rate that causes a decline maximum cardiac output [36, 39, 41, 61, 68]. It appears that highly trained aging subjects are able to maintain their stroke volume, peripheral oxygen extraction, and body composition at or near the levels they possessed in their 20s and 30s [41, 64, 71]. Continued training may also limit the age-related decline in maximum heart rate [71].

Some have speculated that the pulmonary factors which cause arterial desaturation during heavy exercise that might limit $\dot{V}O_2max$ in some younger athletes might become more common in older athletes due to age-related deterioration of pulmonary function [49]. However, the preliminary evidence to date suggests that these events are no more common in older than younger subjects primarily because of a decreased metabolic demand and, therefore, of pulmonary ventilation in the older athletes [49]. Additionally, it is possible that the pulmonary system might impair performance (not $\dot{V}O_2max$) in some older subjects because of

FIGURE 4.9

The decline in $\dot{V}O_2max$ vs. age for a variety of groups is plotted. The **top line** *shows the decline in $\dot{V}O_2max$ among older participants in orienteering [36]. The* **two bottom lines** *are examples of the rate of decline among individuals who were either sedentary as young adults or champion runners who stopped training. The cluster of* **open circles** *labeled* **"Masters Athletes"** *in the upper right-hand corner were obtained in highly trained older champions and demonstrate that the $\dot{V}O_2max$ values in these individuals are substantially higher than any other group of older subjects. These data form the basis for the observation that prolonged intense endurance exercise training by older individuals can reduce dramatically the decline in $\dot{V}O_2max$ with aging. (A variety of data sources were used in the construction of this figure, see Heath et al. [41] for details.)*



age-related increases in the oxygen cost of breathing during exercise that result from an increase in physiological dead space. This means that a larger percentage of O_2 consumption during exercise would be used by the ventilatory muscles and unavailable to the other exercising muscles [49, 61].

In summary, it appears that continued prolonged intense endurance

exercise training by older male subjects limits the age-related decline in $\dot{V}O_2$max by roughly one-half to 5% per decade beginning at age 30 yr (or perhaps not until the late 30s in some individuals). There are almost no data on this issue in older female athletes.

Lactate Threshold

As is the case with younger competitors, master athletes probably do not run 10,000-m and marathon races at speeds that require $\dot{V}O_2$max. In older subjects, there is some evidence that the absolute $\dot{V}O_2$max at which the lactate threshold occurs remains constant or only declines minimally with aging. This means that the lactate threshold as a percentage of $\dot{V}O_2$max may increase somewhat with aging [1, 11]. If the lactate threshold changes in this way, then such alterations would serve to maintain performance in the face of declines in $\dot{V}O_2$max. Such a mechanism could explain the continued world class performances of some individuals in open competition into their late 30s and early 40s.

This possibility is consistent with observations that suggest that training preserves the mitochondrial adaptations in skeletal muscles that are thought to play a key role in regulating lactate production in contracting skeletal muscles [11]. It also appears that the normal mitochondrial adaptations to training can be made in older subjects [79]. These data indicate that either continued training or the initiation of training by older humans can prevent or eliminate the age-related decline in skeletal muscle oxidative capacity [83]. They also indicate that the decline in muscle oxidative capacity with aging results from inactivity and not from advancing years.

Running Economy

There are few data on the extent of running economy changes with aging in well-trained older athletes. The currently available information culled from various sources and reports of oxygen uptake at various stages of treadmill exercise protocols suggests that aging per se does not alter the oxygen cost to perform a given treadmill workload [1, 11, 78]. This issue needs further study to determine what effect (if any) the increased oxygen cost of breathing during exercise might have on running economy [61].

Integration of Factors

Elite older (male) endurance athletes can remain competitive in open competition at the highest international levels until their middle or late 30s, with a few individuals remaining competitive until their early 40s. The current concept is that the normal age for peak performance for distance running occurs during the late 20s or early 30s [77]. That some individuals are able to achieve very high levels of performance later in life probably reflects some combination of genetic variability (i.e., they are "slow" agers), natural ability, and continued hard training.

In the early stages of aging, any declines in $\dot{V}O_2max$ may have little impact on performance because of the maintenance of the lactate threshold at the same absolute workload (i.e., running speed). Later on it appears $\dot{V}O_2max$ declines at rate (5%) per decade beginning in the 30s and that this rate is slightly less than the decline in performance (6–7%). It is unclear if these rates of decline will be lower in individuals who were champions in their 20s who continue to train intensely and compete at a high level throughout life.

It is not clear why the decline in performance appears to be slightly greater than the fall in $\dot{V}O_2max$ with aging. It is possible that, even among the most competitive runners, there might be an age-related decline in the frequency, intensity, and duration of training due to a combination of the inevitable reduction in physiological factors along with orthopedic or motivational considerations. This means that although older competitors may be far more active than the average individuals of the same age, they might be marginally less active than they once were. In follow-up studies of master athletes it appears that there might be at least some reduction in absolute training intensity [64, 71]. This might explain the slightly faster decline in performance and the further slowing seen beginning in the seventh decade of life. Another possibility is that the oxygen cost of achieving a given alveolar ventilation increases with aging due to age-related increases in dead space. If this occurred, it could result in a respiratory muscle steal syndrome, and limit performance since a higher fraction of total body $\dot{V}O_2$ would be used by the respiratory as opposed to locomotor muscles [49, 61]. These issues will only be resolved by continued studies on elite older runners including longitudinal evaluations of aging champion athletes who remain highly competitive.

Based on the simple evaluation of the decline in record performance (Figs. 4.6 and 4.7), it appears that similar events might occur in older women. However, as is the case for younger elite females, there are much fewer (almost no) data on elite older female athletes. Studies on these individuals are especially needed to determine if lifelong exercise training is as effective in reducing the age-related decline in physiological function in women as men.

CONCLUSIONS

Based on the previously discussed information, the following conclusions appears to be justified:

1. The improvement in performance by men in open competition probably resulted from progressive increases in $\dot{V}O_2max$ as a result of changes in training from the late 1800s to the 1930s. From the 1930s

to the 1960s, performance improved because training programs changed in a manner that allowed the top athletes to sustain a greater fraction of their $\dot{V}O_2max$ in competition. There is little evidence to suggest that current record holders have physiologically improved over the last 25–30 yrs. It is likely that improvement since the 1960s represents the combination of better tracks and equipment along with enhanced competitive opportunities for a larger fraction of the world's population.

2. The rapid improvement in performance by women over the last 20 yr seems to have resulted from the rapid emergence of improved competitive opportunities and harder training regimens at roughly the same time. If lifelong competitive opportunities for women (throughout the world) continue to increase, records by women may continue to improve at a faster rate than those by men for some time. Some elite female runners clearly have $\dot{V}O_2max$ values and running economy curves comparable to those of world class male runners. If these women are able to train in a manner that allows them to develop very high lactate threshold values, it is likely that women will run (at a minimum) 2:20 for the marathon and well under 30:00 minutes for 10,000 m.
3. The concept that there is physiological "room" for dramatic improvements in distance running performance in both men and women should be viewed with caution. This approach is useful primarily because it highlights the relative lack of information on how $\dot{V}O_2max$, running economy, and lactate threshold (or some other poorly understood factor) interact in truly elite runners of both sexes (particularly women) as determinants of distance running performance.
4. In older humans, endurance training can blunt or eliminate the changes in $\dot{V}O_2max$, body composition, and muscle oxidative capacity seen in sedentary subjects as they age. In older competitors, the decline in performance with aging appears to be (at best) 6–7% per decade for men up to their late 50s. This rate of decline is probably slightly greater than the decline in $\dot{V}O_2max$ in these individuals. These reductions are substantially less than those observed in the normal sedentary population. When considered in the context of the higher baseline values at the "onset" of aging in trained subjects, they indicate that prolonged intense endurance training throughout life allows the performance of older humans to surpass that of a majority of the younger population. Similar trends in performance are seen in older women, but there are much fewer (almost no) data on such women and few if any have had lifetime opportunities to compete in sports. Cross-sectional and longitudinal studies of elite female athletes as they age are needed to fill this gap. A key question is whether the decline in physiological function and competitive

performance with aging will be even less as larger numbers of athletes who were highly trained in their 20s continue truly vigorous exercise programs throughout life?

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