Lactate-related Factors as a Critical Determinant of Endurance

Kiyoji TANAKA

Institute of Health and Sport Sciences, University of Tsukuba, Tsukuba, Ibaraki 305, Japan

Many interrelated physiological and/or morphological factors have been demonstrated to influence endurance exercise performance. Some of these factors include skeletal musculature, running economy, maximal oxygen uptake ($\dot{V}O_{2\max}$), maximal steady state (MSS), onset of blood lactate accumulation (OBLA), onset of plasma lactate accumulation (OPLA), and anaerobic (or lactate) threshold (AT or LT). The present paper focuses mainly on $\dot{V}O_{2\max}$, MSS, OBLA, OPLA and LT, all of which have been postulated as a prerequisite in endurance exercise success. This paper consists of: (1) significance of La-related variables, (2) longitudinal studies, (3) comments, and (4) conclusion. Briefly, it is suggested that estimation of endurance exercise potential could be obtained with relatively high precision using laboratoriously measured La-related variables. The most critical determinant of endurance exercise performance such as marathon time is considered running velocity (V) at which LT is detected (V@LT), $\dot{V}O_{2\text{@LT}}$, or V@MSS, while V@OBLA appears to be the best predictor of performance in endurance events of 16 km or shorter distances.

Key words: Anaerobic threshold, Lactate threshold, Maximal steady state, Endurance training-induced changes, Estimation of marathon time

Accumulated lines of evidence suggest that success in any sport including endurance events, depends upon many interrelated physiological, anthropometric, and/or morphological factors including cardiorespiratory fitness and skeletal musculature. More specifically, maximal oxygen uptake ($\dot{V}O_{2\max}$), lactate (La) related variables, running economy at a standardized velocity, muscle fiber type, substrate availability, and possibly body composition and anthropometric factors are known to contribute to successful performance in distance running.

Above all, a high $\dot{V}O_{2\max}$ has traditionally been proposed as a critical determinant of success in endurance events. Studies using a relatively homogeneous sample of highly trained runners in terms of both running performance and $\dot{V}O_{2\max}$ have found low-to-moderate correlations ($r = -0.12$ and -0.58) between $\dot{V}O_{2\max}$ and race times (Conely and Krahenbuhl, 1980; Pollock, 1977). Our observation agrees

with these findings (Fig.1). Allen et al. (1985) have reported, despite having 9% lower $\dot{V}O_{2\max}$ values, master runners had the same 10-km running performances as matched young runners in terms of training mileage, training pace, and type of training.

In recent years, however, the work intensity or oxygen uptake ($\dot{V}O_2$) corresponding to the onset of lactate accumulation in blood has gained great

Fig. 1. Relationship between $\dot{V}O_{2\max}$ expressed as ml/kg/min and 10,000-m race time.

attractiveness in exercise and sports science research, particularly in endurance research, as reflecting a metabolic shift from aerobic to predominantly anaerobic energy contribution. This interest is simply because La-related variables measured during submaximal exercise are more sensitive to endurance training-induced adaptations than other variables including $\dot{V}O_{2\max}$. Indeed, many recent studies (Williams et al., 1967; Davis et al., 1979; Sjodin et al., 1982; Denis et al., 1982; Denis et al., 1984; Yoshida et al., 1982; Hurley et al., 1984; Tanaka et al., 1987) with the exception of the studies conducted by Tanaka et al. (1984, 1986a), have reported greater relative changes in La-related variables as compared with changes in $\dot{V}O_{2\max}$ as a result of endurance training. Furthermore, decreases in muscle and blood La accumulation at given absolute or relative exercise intensities have been demonstrated to occur as a result of endurance training (Ekblom et al., 1968; Karlsson et al., 1972).

Blood La-related terms such as anaerobic (AT) or La threshold (LT), onset of plasma La accumulation (OPLA), onset of blood La accumulation (OBLA), maximal steady state heart rate (HR@MSS, where MSS=2.2 mM of La), maximal steady state oxygen uptake ($\dot{V}O_2$ @MSS), maximal steady state treadmill running velocity (Vt@MSS), maximal steady state La (MSSLa), La turning point, excess La, and aerobic-anaerobic threshold are all variables which have been used to describe the kinetics of La accumulation during submaximal exercise (Davis et al., 1983; Tanaka and Matsuura, 1984; Jacobs, 1986). Thus, some of these variables are expressed as $\dot{V}O_2$ at which a given La accumulation occurs; while others are referred to as the exercise intensity (i.e., treadmill running velocity or cycle ergometer power outputs) which elicits a La concentration just above resting and/or warm-up values. These variables can also be expressed as the percentage of $\dot{V}O_{2\max}$ at which they are detected.

The concept of the AT was originally proposed by Wasserman and McIlroy (1964) and Wasserman

et al. (1973). Later, Wasseman et al. (1981) redefined the AT as the work rate or $\dot{V}O_2$ just below the point where La begins to systematically increase from its resting or low-level exercise value during an incremental exercise test (Fig.2). Thus, the AT concept implies that at a certain exercise intensity, anaerobic metabolism systematically increases to supplement deficiencies in aerobic metabolism. More recently, the LT has been used interchangeably with the AT. OPLA is defined as the treadmill velocity at which plasma La starts to accumulate, which is also roughly synonymous with the LT. The OBLA or Vt@OBLA is defined as the running velocity at which a La accumulation of 4 mM occurs (Fig.3), while the MSS is the Vt, $\dot{V}O_2$, or HR at which a La accumulation of 2.2 mM is observed (Fig.3).

It is logically appropriate to hypothesize that a combination of a number of factors (e.g., La-related variable, $\dot{V}O_{2\max}$, muscle fiber type, substrate availability, the degree of fatness, hematological and iron status, or probably lipoprotein pattern) would be highly predictive of endurance exercise performance or aerobic work capacity. However, the present review focuses on which

Fig. 2. Relationship between treadmill velocity (●) and La (or delta La) and between $\dot{V}O_2$ (▲) and La (or delta La) for two representative subjects. Vertical arrows indicate LT and OPLA.

La-related variable would best account for endurance exercise performance.

I. SIGNIFICANCE OF LA-RELATED VARIABLES

Maximal steady state (MSS).

Maximal steady state was first defined by Londeree and Ames (1975) as the $\dot{V}O_2$, HR and/or treadmill running velocity (V_t) at which plasma La concentration corresponded to a fixed value of 2.2 mM (Fig.3). The plasma La of 2.2 mM seems to be a level which clearly exceeds resting values but can be tolerated for substantial periods of time. These authors noted that although highly fit subjects worked close to their $\dot{V}O_{2max}$ without significant La accumulation, they produced La very rapidly once they exceeded their MSS. Thus, the MSS is similar to, but probably slightly higher than AT, LT, and OPLA as used by other investigators. The only difference is that the MSS is detected as a fixed La value, as in the case of the OBLA, while the LT and OPLA reflect the metabolic rate or work rate just below the point where La abruptly increases above the resting value. It should be noted that MSS and OPLA are determined from a series of discontinuous 10-min steady state runs.

In relation to the endurance exercise performance, Londeree and Ames (1975) reported correlations of 0.90 and 0.82 between relative HR@MSS and relative $\dot{V}O_2$ @MSS, respectively, and level of conditioning. LaFontaine et al. (1981) also found that the MSS, expressed as the V_t where venous La concentration was 2.2 mM, correlated highly with the race pace for 402.3-m ($r=0.843$), 3.22-km ($r=0.993$), 8.05-km ($r=0.995$), 16.09-km ($r=0.981$ for overall pace and $r=0.984$ for 6.44 km flat portion), and 20-km ($r=0.917$ for overall pace and $r=0.917$ for 6.44 km flat portion) distances. Conclusions drawn by LaFontaine et al. (1981) were that the MSS represented the approximate upper limit where the necessary energy can be derived almost entirely via oxidative pathways and that the objectively

selected race pace would aid in the prevention of premature fatigue caused by excessive early utilization of glycogen which would severely limit performance.

Onset of plasma lactate accumulation (OPLA).

The data used for the detection of the OPLA are obtained from a series of 10-min treadmill steady state runs (8 different speeds in the Farrell et al.'s study). No more than one run is performed on any day. When plotting delta La (=exercise La-resting La) versus V_t , visual inspection indicates that the portion of the graph above the OPLA exhibits an exponential rise in La (Fig.2).

Farrell et al. (1979) found correlation coefficients of 0.91-0.98 and 0.85-0.91 between the V_t and $\dot{V}O_2$, respectively, at the OPLA and performance at 3.2-km, 9.7-km, 15-km, 19.3-km, and 42.2-km distances. Significant relationships were also obtained for the V_t @OPLA and/or $\dot{V}O_2$ @OPLA versus the running economy ($\dot{V}O_2$ @268m/min), gastrocnemius muscle fiber composition expressed as a percent of slow twitch (%ST) fibers, and $\dot{V}O_{2max}$. In their study, the highest correlation was 0.98 between the V_t @OPLA and the actually measured marathon race velocity (VM). It is interesting to note that the mean difference between V_t @OPLA and the VM was only 0.48 ± 0.32 km/h with a range of -0.3 to 11.4 km/h. The authors suggested that distance runners would set a pace which allows the utilization of the largest possible $\dot{V}O_2$ that just avoids the exponential rise in La.

Lactate threshold (LT) or anaerobic threshold (AT).

According to Wasserman et al. (1981), the LT (or AT) is now a term that refers to the $\dot{V}O_2$ or work rate during exercise above which the rate of La production exceeds the rate at which it can be catabolized, thus causing La to increase in blood (Fig.2). The increase in blood La has been hypothesized to reflect intramuscular La production, which

may in turn be due to the development of anaerobiosis within the active muscle tissue. It should be noted, however, that the increase in La production seems to occur under conditions of increased glycolytic flux and/or increased concentrations of pyruvate during fully aerobic conditions (Brooks, 1986). Some contradictory postulations about the La production mechanisms during submaximal exercise need to be clarified.

$\dot{V}O_2$ @LT and/or V_t @LT have been postulated as the single best determinant of endurance exercise performance in many studies (Tanaka et al., 1981; Kumagai et al. 1982; Tanaka et al., 1983; Hagberg et al., 1983; Powers et al., 1983; Tanaka and Matsuura, 1984; Miller et al., 1987; Tanaka et al., 1989a); while in some other studies (Weltman et al., 1987; Tanaka et al., 1988a; Tanaka et al., 1989b) similar or even higher relationships have been found between $\dot{V}O_{2max}$ and endurance running performance. It can be generally stated that $\dot{V}O_2$ @LT and/or V_t @LT correlate equally or to a greater extent to endurance exercise performance than other physiological variables including $\dot{V}O_{2max}$, V_t @OBLA, and/or $\dot{V}O_2$ @OBLA.

Muscle biopsy studies have suggested that endurance training is associated with an increase in the

Fig. 3. Relationship between treadmill velocity and La for two representative subjects. Vertical arrows indicate MSS and OBLA.

oxidative capacity of skeletal muscle fibers (Costill et al., 1976; Eriksson et al., 1972; Gollnick and King, 1969a; Gollnick and King, 1969b; Gollnick et al., 1973; Kiessling et al., 1971; Morgan et al., 1971). These findings were amplified by the study of Ivy et al. (1980), who found a significantly high relationship between the $\dot{V}O_2$ @LT and the *in vitro* oxygen uptake (termed "the muscle respiratory capacity") by incubated muscle samples, and between the $\dot{V}O_2$ @LT and %ST fibers.

Onset of blood lactate accumulation (OBLA).

Sjodin and Jacobs (1981) reported a very high correlation coefficient of 0.96 between the V_t at which the OBLA occurred and VM. The authors suggested that success in the marathon race was tightly associated with V_t @OBLA and to the ability to run at a pace close to that velocity during the race. Fohrenbach et al. (1987) have found an even higher relationship ($r=0.98$) between V_t @OBLA and VM.

However, in the study of Tanaka and Matsuura (1984), V_t @OBLA (19.1 km/h) was found to differ significantly from the VM (16.2 km/h); whereas, V_t @LT (16.4 km/h) closely approximated the VM. Furthermore, the V_t @LT correlated ($r=0.781$) with VM to a greater extent than did the V_t @OBLA ($r=0.682$). The intensity of OBLA seems to correspond to approximately 85-90% of $\dot{V}O_{2max}$, while that of LT ranges from 70 to 75% in young, trained endurance athletes when measured on the treadmill. The differences reported between V_t representing LT-MSS and OBLA seems to range from 20 to 50 m/min (Davis et al. 1983; Fay et al. 1989; Lehmann et al. 1983; Londeree and Ames 1975; Londeree 1986; Tanaka and Matsuura 1984), which correspond to approximately 10-20% differences in terms of % $\dot{V}O_{2max}$ (Fig.4).

Very close relationships ($r=0.88-0.99$) were reported by Fohrenbach et al. (1987) between the running velocities determined at 2.5, 3, and 4 mM of La in an incremental field test and the VM. The minimum

Fig. 4. % $\dot{V}O_{2max}$ corresponding to LT, OPLA, MSS, and OBLA in marathon runners.

*Significantly different from the other three.

difference was obtained between the test running velocity (16.5 km/h) at a concentration of 2.5 mM and the VM (16.6 km/h). Since the subjects were top national and/or international marathoners, elite runners may be expected to run at a La corresponding to La@LT or La@MSS. Vt@OBLA appears to be identical to the average 10-km or 16-km race pace in male runners (Tanaka and Matsuura, 1984) and to the average 5-km or 10-km race pace in female runners (Fay et al., 1989).

II. LONGITUDINAL STUDIES

Longitudinal Assessment of LT.

Table 1. A comparison of the change in $\dot{V}O_{2max}$ with the change in submaximal La-related variable reported in endurance training studies.

	Subjects	Change ($\Delta\%$) in $\dot{V}O_{2max}$	Change ($\Delta\%$) in La-related Variable	Training Duration (wk)
Williams et al. (1967)	Labourers	7	16	4-16
Davis et al. (1979)	Middle-aged men	25	44	9
Sjodin et al. (1982)	D* runners	NS	5	14
Denis et al. (1982)	Students	NS	18	40
Denis et al. (1984)	Students	19	42	20
	Middle-aged men	8	38	20
Hurley et al. (1984)	Young men	26	39	12
Yoshida et al. (1982)	Students	14	37	8
Tanaka et al. (1986)	Obese middle-aged women	13	18	18
Tanaka et al. (1984)	D runners	6	5	42
Tanaka et al. (1986)	D runners	5	6	18

*D: distance

Not only cross-sectional but also longitudinal studies have demonstrated that endurance training results in decreased muscle and blood La accumulation at given submaximal work intensities (Ekblom et al., 1968; Hermansen and Stensvold, 1972; Karlsson et al., 1972; Davis et al., 1979; Tanaka et al., 1984; Tanaka et al., 1986a). Davis et al. (1979) have shown that, in previously sedentary middle-aged males, after 9 weeks of endurance training the LT increased 44% in terms of absolute $\dot{V}O_2$ and 15% relative to $\dot{V}O_{2max}$. Many other studies have observed greater changes in La-related variables than in $\dot{V}O_{2max}$ as a result of endurance training (Table 1). In the study of Tanaka et al. (1984), both $\dot{V}O_2$ @LT and $\dot{V}O_{2max}$ for trained distance runners increased significantly but only slightly (4-5%) during a 9-month period. The more interesting finding was that significant relationships existed at almost all assessment points between the change in $\dot{V}O_2$ @LT (or Vt@LT) and the change in 10-km running performance.

The metabolic consequences of increase in the LT after an extended period of endurance training (Davis et al., 1979; Tanaka et al., 1984; Tanaka et al., 1986a) may be that individuals can perform sustained exercise with less obligatory glycogen depletion and nonrespiratory acidosis (Wasserman

et al. 1981). A possible physiological mechanism that might account for the observed reduction in La at a given submaximal exercise intensity, together with the increase in $\dot{V}O_2$ @LT after endurance training, can be partly attributed to an increased activation of relatively more slow-twitch (ST) muscle fibers, which are predominantly recruited below and at the LT. Other concomitant contributing factors may be increased oxidative capacity at the cellular level, as represented by mitochondrial hypertrophy and/or hyperplasia, as well as an increase in oxidative cellular enzymes in muscle (Tanaka et al., 1984).

Holloszy and Coyle (1984) have interpreted the major metabolic consequences of the adaptations of skeletal muscle to endurance training as a slower utilization of muscle glycogen and blood glucose, a greater reliance on fat oxidation, and less La production during exercise of a given exercise intensity (i.e., an increase in LT). The authors suggest that these metabolic consequences may contribute to the increase in endurance and the ability to exercise at a higher percentage of $\dot{V}O_{2max}$ in the trained state.

Longitudinal Assessment of OBLA.

Some German researchers have suggested that the optimal training intensity which induces a qualitative stimulus for adaptation to endurance training corresponds to a steady state La concentration of approximately 4 mM (i.e., OBLA). Changes in the OBLA after relatively high-intensity training at V_t @OBLA were studied by Sjodin et al. (1982). In their study, well-trained distance runners added a 20-min treadmill run at a velocity calculated to elicit a La of 4 mM once a week to their regular training program. Interestingly, analyses of the data indicated that a steady state training intensity (85-87% of $\dot{V}O_{2max}$) which approximated V_t @OBLA increased V_t @OBLA (4.69 ± 0.25 to 4.89 ± 0.27 m/s) and $\dot{V}O_2$ @OBLA (58.6 ± 2.3 to 60.6 ± 3.2 ml/kg/min), and resulted in measurable local metabolic adaptations (i.e., increases in the activity of citrate synthase (CS) and the relative activity of the heart-specific lactate

dehydrogenase (H-LDL) isozyme, and decreases in the activity of phosphofructokinase (PFK) and the ratio of PFK to CS) in the skeletal muscles without a significant change in $\dot{V}O_{2max}$. H-LDH bound to the inner membrane of the mitochondrion is known to facilitate an oxidative translocation reaction of La in the presence of nicotinamide adenine dinucleotide (NAD^+), while PFK is an important glycolytic enzyme and a key link in the anaerobic yield of ATP. It was postulated, therefore, that the rate of glycolysis (or glycogenolysis) during the 20-min exercise was slower and the potential to oxidize pyruvate and/or La increased. Consequently, the authors suggested that for the purpose of laboratory tests V_t @OBLA could be a more sensitive indicator for the monitoring of training-induced adaptations (i.e., lower muscle and/or blood La accumulation at a given absolute and relative exercise intensity) than is the determination of $\dot{V}O_{2max}$.

In contrast, the relationship found by Sjodin et al. (1982) between the change in OBLA and the ST fiber distribution was not observed in the study of Denis et al. (1984). Rather, it was reported that, while % ST fibers for male students remained unchanged or tended to decrease (i.e., $54.0 \pm 18.4\%$ to $47.2 \pm 8.8\%$) following a 20-wk training program, $\dot{V}O_{2max}$, maximal work rate, and work rate corresponding to OBLA of this group all increased significantly. This is partly supported by other studies (Jacobs and Kaiser, 1982; Tesch et al., 1982).

COMMENTS

La kinetics during exercise has been a big focus of academic inquiry in the area of exercise science. The major role of La production is to release some of the energy contained in the glucose molecule and to transfer it to adenosine diphosphate (ADP) for the production of adenosine triphosphate (ATP). The relative importance of the anaerobic and aerobic components of metabolism to ATP production is largely related to both the intensity and duration of the exercise. Although the intensity of exercise that

elicits an increase in muscle and blood La is highly variable among individuals, and increase in blood La does not occur in highly trained endurance athletes until the exercise intensity of 70-80% of $\dot{V}O_{2max}$ is attained. In fact, the data from Londeree and Ames (1975) showed that in poorly conditioned subjects La increased in a nearly linear fashion as intensity increased, while the relationship became more curvilinear as conditioning increased. As the exercise intensity increases above LT, therefore, there is an exponential rise in the La particularly in trained athletes. All La-related variables discussed in this paper occur just prior to or around the onset of the middle-part of the exponential La-curve obtained during moderate to very heavy intensity exercise.

A series of correlational studies have indicated that blood La responses to submaximal exercise as represented by the LT, MSS, OPLA, and OBLA are all quite sensitive discriminators between elite and good runners or between good and poor runners. The possible reason why these La-related variables have been shown to have equal or even greater associations with endurance exercise performance than other physiological variables including $\dot{V}O_{2max}$ is the following. A substantial period of endurance training causes a decrease in the accumulation of La during a given submaximal exercise (Holloszy et al., 1973; Hurley et al., 1984; Saltin et al., 1969; Seals et al., 1984) due possibly to the increased rate of La removal, together with the increased activation of more ST fibers. The increased recruitment of more ST fibers may favor oxidative combustion instead of anaerobic glycolysis due to the enzymatic nature of those fibers. Consequently, lower muscle and blood La levels and the slower muscle glycogen utilization during submaximal exercise in the trained as compared to the untrained state (Cobb and Johnson 1963; Ekblom et al., 1968; Saltin and Karlsson 1971; Karlsson et al., 1972; Saltin et al., 1977) may be explained on the basis of the inhibition of anaerobic glycolysis brought about by enhance-

ment of fat oxidation (Fredholm 1971; Rennie et al., 1976; Essen 1977; Hickson et al., 1977).

La is produced predominantly in the active muscle cell, diffused into neighboring cells, and released to the blood. Once produced, a large portion of La is transported to other tissues such as the liver and heart, and again to active musculature. La can, then, be metabolized within the muscle cell and many other tissues. The measured concentration of La is therefore the net result of all of these processes.

Sjodin et al. (1982) reported the significantly reduced ratio of key glycolytic to oxidative enzyme activities after running training at a velocity calculated to elicit 4 mM La. Jacobs and Kaiser (1982) found that, when blood and muscle tissue samples were taken simultaneously, the blood La concentrations up to 4 mM (y) were indeed reflective ($y = 2.52 + 0.12x$, $r = 0.89$) of La accumulated within the active muscle (x). The close association was not observed at higher ($La > 4$ mM) blood La concentrations. These authors speculate that a specific muscle La concentration and the fatiguing effects of the accompanying acidosis may act as a threshold for a number of cellular processes at approximately the blood La values of 3 to 4 mM (Jacobs, 1986). Blood and muscle La may be related closely up to about 4 mM if time is sufficient for diffusion. Above 4 mM muscle La may be higher due to limited diffusion and/or saturation of a carrier system. However, physiological mechanisms for why blood La of about 4 mM is indicative of muscle La remain obscured. Since La-related variables in general have high associations with the %ST fibers (Farrell et al., 1979; Ivy et al., 1980; Tesch et al., 1981; Sjodin and Jacobs, 1981) and La is likely to diffuse from muscle fibers of low to those of high oxidative capacity, the mechanisms of La production and diffusion between fiber types during exercise should be a topic of additional research (Chwalbinska-Moneta et al., 1989).

There is no clear, physiological rationale of why the La "break point" should occur at some specific

blood La concentration (Davis et al., 1983). A fixed, absolute La value is simpler and easier to identify than a La "break point". Nevertheless, LT may be a more physiologically supportable measure.

Very little data are available, however, which compared responses at the LT (or OPLA) and those at a La of 2.2 mM and 4 mM in relation to marathon performance. Our observations (Tanaka et al., 1988b; Tanaka and Matsuura, 1984) and others (Costill and Fox, 1969; Maron et al., 1976; Scrimgeour et al., 1986) indicate that during competition elite marathoners utilize approximately 75-85% of their $\dot{V}O_{2\max}$ with a velocity (V) that elicits a La of approximately 2 mM (i.e., at the LT to MSS) up to approximately 20 m/min faster than the pace. Less conditioned, non-elite runners may run a marathon at a V corresponding to the LT or only slightly below or above that pace (Table 2, Tanaka et al., 1988b). During a 2-hr field run at a velocity corresponding to $V_{t@LT}$, La slightly increased from its resting level (1.17 ± 0.19 mM) by 1.48 ± 0.38 mM at the 20th min and 1.46 ± 0.53 mM at the 70th min, but

tended to decrease thereafter and returned to the resting level at the end of the 2-hr run (1.17 ± 0.20 mM, Table 3). Additional research should attempt to determine why elite marathoners are able to compete with higher La and/or % $\dot{V}O_{2\max}$ values. Perhaps some of these differences are related to time of running rather than distance (Fay et al. 1989).

Jog/run training-induced adaptations in the metabolic and physiologic systems could be observed even when the exercise testing modality is cycling. However, jog/run training elicits significantly higher $\dot{V}O_{2@LT}$ on the treadmill compared with the cycle ergometer (Withers et al., 1981; Tanaka et al., 1986b). These results support the concept of training specificity. Careful attention should be paid even to the LT test which may be selected to assess physiologic changes resulting from physical training.

Finally, it is suggested that estimation of endurance exercise potential could be obtained with relatively high precision using laboratoriously measured La-related variables. The close association of endurance performance with La-related variables may be attributable not only to the functional capacity of the central circulatory apparatus to transport oxygen to the working muscles, but also to the peripheral capacity of the musculature to utilize the oxygen, as indicated by some metabolic and histochemical consequences of the endurance training-induced adaptations. Although any physiological or morphological variable could not stand alone as a very strong, decisive factor of endurance exercise performance in untrained individuals as well as in endurance-type athletes, most of the variance in the endurance exercise performance could be accounted for by a single La-related variable such as $V@LT$, $\dot{V}O_{2@LT}$, or $V@MSS$. As to the marathon runner, it appears likely that, regardless of the competitive level, he or she can maintain an average velocity during the race which is around the $V@LT$ or $V@MSS$.

Table 2. Blood La values and % $\dot{V}O_{2\max}$ observed during 2-hr run at a velocity corresponding to the average marathon race pace (Tanaka et al., 1988b)

	La (mM)	% $\dot{V}O_{2\max}$
Rest	1.02 ± 0.28	
20min	1.68 ± 0.57	71.4 ± 4.8
65min	1.34 ± 0.33	71.9 ± 5.9
120min	1.26 ± 0.40	74.1 ± 5.6

n=10; less-conditioned runners

Note: n=7 was reported in the paper

Table 3. Blood La values and % $\dot{V}O_{2\max}$ observed during 2-hr run at a velocity corresponding to $V_{t@LT}$ (Tanaka et al., unpublished)

	La (mM)	% $\dot{V}O_{2\max}$
Rest	1.17 ± 0.19	
20 min	1.48 ± 0.38	74.0 ± 3.4
70 min	1.46 ± 0.53	73.6 ± 2.5
120 min	1.17 ± 0.20	74.0 ± 2.7

n=7; well-trained runners

CONCLUSION

Potential endurance performance probably is limited by $\dot{V}O_{2\max}$. La-related variables permit accurate prediction of current endurance performance. In this review the most critical determinant of endurance exercise performance such as marathon time is considered $V@LT$, $\dot{V}O_2@LT$, or $V@MSS$. $V@OBLA$ appears to be the best predictor of performance in endurance events of 16 km or shorter distances. Additional research is needed to determine why elite runners are able to compete with higher La values than non-elite runners.

Acknowledgment: The author wish to thank Dr. Yoshiyuki Matsuura, Head of the Institute of Health and Sport Sciences, University of Tsukuba, and Dr. Ben R. Londeree, Department of Health and Physical Education, University of Missouri for their helpful review of this manuscript.

REFERENCES

- Allen, W.K., D.R. Seals, B.F. Hurley, A.A. Ehsani, and J.M. Hagberg, 1985: Lactate threshold and distance-running performance in young and older endurance athletes. *J. Appl. Physiol.*, 58: 1281-1284.
- Brooks, G.A., 1986: The lactate shuttle during exercise and recovery. *Med. Sci. Sports Exerc.*, 18: 360-368.
- Chwalbinska-Moneta, J., R.A. Robergs, D.A. Costill, and W.L. Fink, 1989: Threshold for muscle lactate accumulation during progressive exercise. *J. Appl. Physiol.*, 66: 2710-2716.
- Cobb, L.A., and W.P. Johnson, 1963: Hemodynamic relationships of anaerobic metabolism and plasma free fatty acids during prolonged, strenuous exercise in trained and untrained subjects. *J. Clin. Invest.*, 42: 800-810.
- Conley, D.L., and G.S. Krahenbuhl, 1980: Running economy and distance running performance of highly trained athletes. *Med. Sci. Sports Exerc.*, 12: 357-360.
- Costill, D.L., and E.L. Fox, 1969: Energetics of marathon running. *Med. Sci. Sports*, 1: 81-86.
- Costill, D.L., W.J. Fink, and M.L. Pollock, 1976: Muscle fiber composition and enzyme activities of elite distance runners. *Med. Sci. Sports*, 8: 96-100.
- Davis, J.A., M.H. Frank, B.J. Whipp, and K. Wasserman, 1979: Anaerobic threshold alterations caused by endurance training in middle-aged men. *J. Appl. Physiol.*, 46: 1039-1046.
- Davis, J.A., V.J. Caiozzo, N. Lamarra, J.F. Ellis, R. Vandagriff, C.F. Prietto, and W.C. McMaster, 1983: Does the gas exchange anaerobic threshold occur at a fixed blood lactate concentration of 2 or 4 mM? *Int. J. Sports Med.*, 4: 89-93.
- Denis, C., R. Fouquet, R. Poty, A. Geyssant, and J.R. Lacour, 1982: Effect of 40 weeks of endurance training on the anaerobic threshold. *Int. J. Sports Med.*, 3: 208-214.
- Denis, C., D. Dormis, and J.R. Lacour, 1984: Endurance training, $\dot{V}O_{2\max}$, and OBLA: A longitudinal study of two different age groups. *Int. J. Sports Med.*, 5: 167-173.
- Eklblom, B., P.O. Astrand, B. Saltin, J. Stenberg, and B. Wallstrom, 1968: Effect of training on circulatory response to exercise. *J. Appl. Physiol.*, 25: 518-528.
- Eriksson, B.O., P.D. Gollnick, and B. Saltin, 1972: Muscle metabolism and enzyme activities after training in boys 11-13 years old. *Acta Physiol. Scand.*, 87:231-239.
- Essen, B., 1977: Intramuscular substrate utilization during prolonged exercise. *Ann. N.Y. Acad. Sci.*, 301: 30-44.
- Farrell, P.A., J.H. Wilmore, E.F. Coyle, J.E. Billing, and D.L. Costill, 1979: Plasma lactate accumulation and distance running performance. *Med. Sci. Sports*, 11: 338-344.
- Fay, L., B.R. Londeree, T.P. LaFontaine, and M.R. Volek, 1989: Physiological parameters related to distance running performance in female athletes. *Med. Sci. Sports Exerc.*, 21: 319-324.
- Forenbach, R., A. Mader, and W. Hollman, 1987:

- Determination of endurance capacity and prediction of exercise intensities for training and competition in marathon runners. *Int. J. Sports Med.*, 8: 11-18.
- Fredholm, B.B., 1971: Fat mobilization and blood lactate concentration. (In) *Muscle Metabolism During Exercise*. (Eds.) Pernow, B., and B. Saltin, Plenum Press, New York, pp.249-255.
- Gollnick, P.D., R.B. Armstrong, B. Saltin, C.W. Saubert IV, W.L. Sembrowich, and R.E. Shephard, 1973: Effect of training on enzyme activities and fiber composition of human skeletal muscle. *J. Appl. Physiol.*, 34: 107-111, 1973.
- Gollnick, P.D., and D.W. King, 1969a: Energy release in the muscle cell. *Med. Sci. Sports*, 1: 23-31.
- Gollnick, P.D., and D.W. King, 1969b: Effect of exercise and training on mitochondria of skeletal muscle. *Am. J. Physiol.*, 216: 1502-1509.
- Hagberg, J.M., and E.F. Coyle, 1983: Physiological determinants of endurance performance as studied in competitive racewalkers. *Med. Sci. Sports Exerc.*, 15: 287-289.
- Hermansen, L., and I. Stensvold, 1972: Production and removal of lactate during exercise in man. *Acta Physiol. Scand.*, 86: 191-201.
- Hickson, R.C., M.J. Rennie, R.K. Conlee, W.W. Winder, and J.O. Holloszy, 1977: Effects of increased plasma free fatty acids on glycogen utilization and endurance. *J. Appl. Physiol.*, 43: 829-833.
- Holloszy, J.O., 1973: Biochemical adaptations to exercise: aerobic metabolism. (In) *Exercise and Sports Sciences Reviews*, (Ed.) Wilmore, J.H., Academic Press, New York, pp.45-71.
- Holloszy, J.O., and E.F. Coyle, 1984: Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *J. Appl. Physiol.*, 56: 831-838.
- Hurley, B.F., J.M. Hagberg, W.K. Allen, D.R. Seals, J.C. Young, R.W. Cuddihee, J.O. Holloszy, 1984: Effect of training on blood lactate levels during submaximal exercise. *J. Appl. Physiol.*, 56: 1260-1264.
- Ivy, J.L., R.T. Withers, P.J. Handel, D.H. Elger, and D.L. Costill, 1980: Muscle respiratory capacity and fiber type as determinants of the lactate threshold. *J. Appl. Physiol.*, 48: 523-527.
- Jacobs, I., and P. Kaiser, 1982: Lactate in blood, mixed skeletal muscle, and FT or ST fibres during cycle exercise in man. *Acta Physiol. Scand.*, 114: 461-466.
- Jacobs, I., R. Schele, and B. Sjodin, 1985: Blood lactate vs exhaustive exercise to evaluate aerobic fitness. *Eur. J. Appl. Physiol.*, 54: 151-155.
- Jacobs, I., 1986: Blood lactate. Implications for training and sports performance. *Sports Med.*, 3: 10-25.
- Karlsson, J., L.O. Nordesjo, L. Jorfeldt, and B. Saltin, 1972: Muscle lactate, ATP, and CP levels during exercise after physical training in man. *J. Appl. Physiol.*, 33: 199-203.
- Kiessling, K.H., K. Piehl, and C.G. Lundquist, 1971: Effect of physical training on ultrastructural features in human muscle mitochondria. (In) *Muscle Metabolism During Exercise*, (Eds.) Pernow, B., and B. Saltin, Plenum Press, New York, pp.97-101.
- Kumagai, S., K. Tanaka, Y. Matsuura, A. Matsuzaka, K. Hirakoba, and K. Asano, 1982: Relationships of the anaerobic threshold with the 5 km, 10 km, and 10 mile races. *Eur. J. Appl. Physiol.*, 49: 13-23.
- LaFontaine, T.P., B.R. Londeree, and W.K. Spath, 1981: The maximal steady state versus selected running events. *Med. Sci. Sports Exerc.*, 13: 190-193.
- Lehmann, M., A. Berg, R. Kapp, T. Wessinghage, and J. Kuel, 1983: Correlations between laboratory testing and distance running performance in marathoners of similar performance ability. *Int. J. Sports Med.*, 4: 226-230.
- Londeree, B.R., and A. Ames, 1975: Maximal steady state versus state of conditioning. *Eur. J. Appl. Physiol.*, 34: 269-278.

- Londeree, B.R., 1986: The use of laboratory test results with long distance runners. *Sports Med.*, 3: 201-213.
- Maron, M.B., S.M. Horvath, J.E. Wilkerson and J.A. Gliner, 1976: Oxygen uptake measurements during competitive marathon running. *J. Appl. Physiol.* 40: 836-838.
- Miller, F.R., and T.G. Manfredi, 1987: Physiological and anthropometrical predictors of 15-kilometer time trial cycling performance time. *Res. Q. Exerc. Sport*, 58: 250-254.
- Morgan, T.E., L.A. Cobb, F.A. Short, R. Ross, and D. R. Gunn, 1971: Effect of long-term exercise on human muscle mitochondria. (In) *Muscle Metabolism During Exercise*, (Eds.) Pernow, B., and B. Saltin, Plenum Press, New York, pp.87-95.
- Pollock, M.L., 1977: Submaximal and maximal working capacity of elite distance runners, Part I: cardiorespiratory aspects. *Ann. N.Y. Acad. Sci.*, 301: 301-322.
- Powers, S.K., S. Dodd, S. Deason, R. Byrd, and T. McKnight, 1983: Ventilatory threshold, running economy, and distance running performance of trained athletes. *Res. Q. Exerc. Sport*, 54: 179-182.
- Rennie, M.J., W.W. Winder, and J.O. Holloszy, 1976: A sparing effect of increased plasma free fatty acids on muscle and liver glycogen content in the exercising rat. *Biochem. J.*, 156: 647-655.
- Saltin, B., J. Henriksson, E. Nygaard, P. Andersen, and E. Jansson, 1977: Fiber types and metabolic potentials of skeletal muscles in sedentary men and endurance runners. *Ann. N.Y. Acad. Sci.*, 301: 3-29.
- Saltin, B., L.H. Hartley, A. Kilbom, and I. Astrand, 1969: Physical training in sedentary middle-aged and older men. II. Oxygen uptake, heart rate, and blood lactate concentration at submaximal and maximal exercise. *Scand. J. Clin. Lab. Invest.*, 24: 323-334.
- Saltin, B., and J. Karlsson, 1971: Muscle glycogen utilization during work of different intensities. (In) *Muscle Metabolism During Exercise*, (Eds.) Pernow, B., and B. Saltin, Plenum Press, New York, pp.289-299.
- Scrimgeour, A.G., T.D. Noakes, B. Adams, and K. Myburgh, 1986: The influence of weekly training distance on fractional utilization of maximum aerobic capacity in marathon and ultramarathon runners. *Eur. J. Appl. Physiol.* 55: 202-209.
- Seals, D.R., B.F. Hurley, J. Schultz, and J.M. Hagberg, 1984: Endurance training in older men and women. II. Blood lactate response to submaximal exercise. *J. Appl. Physiol.*, 57: 1030-1033.
- Sjodin, B., and I. Jacobs, 1981: Onset of blood lactate accumulation and marathon running performance. *Int. J. Sports Med.*, 2: 23-26.
- Sjodin, B., I. Jacobs, and J. Svedenhag, 1982: Changes in onset of blood lactate accumulation (OBLA) and muscle enzyme after training at OBLA. *Eur. J. Appl. Physiol.* 49: 45-57.
- Tanaka, K., and Y. Matsuura, 1984: Marathon performance, anaerobic threshold, and onset of blood lactate accumulation, *J. Appl. Physiol.*, 57: 640-643.
- Tanaka, K., Y. Matsuura, and T. Moritani, 1981: A correlational analysis of maximal oxygen uptake and anaerobic threshold as compared with middle and long distance performance. *Jpn. J. Sports Med. Phys. Fitness*, 30: 94-102.
- Tanaka, K., Y. Matsuura, A. Matsuzaka, K. Hirakoba, S. Kumagai, S. Sun-O, and K. Asano, 1984: A longitudinal assessment of anaerobic threshold and distance-running performance. *Med. Sci. Sports Exerc.*, 16: 278-282.
- Tanaka, K., H. Watanabe, Y. Konishi, R. Mitsuono, S. Sumida, S. Tanaka, T. Fukuda, and F. Nakadomo, 1986a: Longitudinal associations between anaerobic threshold and distance running performance. *Eur. J. Appl. Physiol.*, 55: 248-252.
- Tanaka, K., F. Nakadomo, T. Yoshimura, M. Wakita, T. Fukuda, S. Sumida, H. Watanabe, T. Yamada, and K. Maeda, 1986b: Specificity of training on the anaerobic threshold in middle-aged obese women. *Jpn. J. Appl. Physiol.* 16: 293-298.

- Tanaka, K., F. Nakadomo, S. Kumagai, and M. Nishizumi, 1987: Clinical application of lactate threshold to the treatment of obesity. *J. Human Ergol.*, 16: 145-150.
- Tanaka, K., F. Nakadomo, and K. Maeda, 1988: Effects of Jogging/running training on cardiorespiratory fitness, serum lipids, and body composition in healthy males. *Ann. Physiol. Anthropol.*, 7: 31-37.
- Tanaka, K., K. Mimura, H.S. Kim, T. Kawabata, M. Tajima, F. Nakadomo, and K. Maeda, 1989a: Prerequisites in distance running performance of female runners. *Ann. Physiol. Anthropol.*, 8: 79-87.
- Tanaka, K., F. Nakadomo, T. Fukuda, and H. Watanabe, 1988b: Metabolic and ventilatory responses during race pace at marathon. *Ann. Physiol. Anthropol.*, 7: 15-21.
- Tanaka, K., N. Takeshima, T. Kato, S. Niihata, and K. Ueda, 1989b: Critical determinants of endurance performance in middle-aged and elderly endurance runners with heterogeneous training habits. *Eur. J. Appl. Physiol.*, 59:443-449.
- Tesch, P.A., D.S. Sharp, and W.L. Daniels, 1981: Influence of fiber type composition and capillary density on onset of blood lactate accumulation. *Int. J. Sports Med.*, 2: 252-255.
- Wasserman, K., B.J. Whipp, and J.A. Davis, 1981: Respiratory physiology of exercise: metabolism, gas exchange, and ventilatory control. (In) *MTP International Review of Physiology — Respiratory Physiology III*. (Ed.) Widicombe, J.G., Baltimore, MD: University Park Press, pp.149-211.
- Wasserman, K., B.J. Whipp, S.N. Koyal, and W.L. Beaver, 1973: Anaerobic threshold and respiratory gas exchange during exercise. *J. Appl. Physiol.*, 35: 236-243.
- Wasserman, K., and M.B. McIlroy, 1964: Detecting the threshold of anaerobic metabolism in cardiac patients during exercise. *Am. J. Cardiol.*, 14: 844-852.
- Weltman, A., D. Snead, R. Seip, R. Schurrer, S. Levine, R. Rutt, T. Reilly, J. Weltman, and A. Rogol, 1987: Prediction of lactate threshold and fixed blood lactate concentrations from 3200-m running performance in male runners. *Int. J. Sports Med.*, 8: 401-406.
- Williams, C.G., C.H. Wyndham, R. Kok, and M.J. Von Rahden, 1967: Effect of training on maximum oxygen intake and anaerobic metabolism in man. *Int. Z. angew Physiol.*, 24: 18-23.
- Withers, R.T., W.M. Sherman, J.M. Miller, and D.L. Costill, 1981: Specificity of the anaerobic threshold in endurance trained cyclists and runners. *Eur. J. Appl. Physiol.*, 47: 93-104.
- Yoshida, T., Y. Suda, and N. Takeuchi, 1982: Endurance training regimen based upon arterial blood lactate: effects on anaerobic threshold. *Eur. J. Appl. Physiol.*, 49: 223-230.

(Received March 1, 1990)