Lactate Threshold Comparison in Anaerobic vs. Aerobic Athletes and Untrained Participants

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ABSTRACT

International Journal of Exercise Science 7(4) : 329-338, 2014. This study compared VO_2 max, lactate threshold (LT) and VO_2 at LT (VO_{2LT}) among aerobic athletes (ARA) (n=10), anaerobic athletes (ANA) (n=9) and untrained participants (UTS) (n=7). From a treadmill test to exhaustion, VO_2 max and LT (4 mmol·L^{-1} blood lactate concentration) were assessed. Analysis of variance showed VO_2 max (ml·kg^{-1}·min^{-1}) was significantly greater for ARA (67.6 ± 9.4) than ANA (53.4 ± 6.4) and UTS (44.9 ± 6.9), with ANA significantly greater than UTS. LT for ARA (82.9 ± 6.4) was not significantly different than ANA (77.5 ± 13.1). However, ARA and ANA were significantly greater than UTS (66.8 ± 5.4). VO_{2LT} (ml·kg^{-1}·min^{-1}) was significantly greater for ARA (55.9 ± 7.7) and ANA (41.5 ± 8.6) than for UTS (29.9 ± 4.1) with ANA significantly greater than UTS. Although used to establish groups, VO_2 max for ARA (vs. UTS) reflect aerobic training adaptations. Similarly high LT would be expected in ARA. Modest VO_2 max for ANA reflects only a mild stimulus to oxidative pathways (plausibly occurring during recovery from repeated high-intensity efforts). However, anaerobic training may provide a stimulus adequate to increase LT. Elevated LT with moderate changes in VO_2 max for ANA provide indirect evidence that differential mechanisms alter VO_2 max and LT. Still, VO_2 at LT would have the greatest implication with regards to aerobic performance. From a practical standpoint, training approaches may be enhanced with a greater understanding of the impact of anaerobic training on LT. Future research should more directly examine threshold-altering mechanisms between these groups of athletes.

KEY WORDS: Sprinters, inflection point, OBLA, training

INTRODUCTION

Various threshold measures may predict endurance performance success more effectively than VO_2 max, the criterion measure of aerobic fitness (1, 6, 9, 17, 27, 28, 31, 33). Weltman (29) thoroughly reviews factors influencing threshold values.

Perhaps the greatest factor altering threshold is training with values (expressed as % VO_2 max) of 65-80% in endurance athletes and at 50-60% in sedentary individuals (21, 31). Training for endurance as aerobic athletes do, tends to stimulate oxygen-dependent (i.e. aerobic) metabolic

pathways. Adaptations are evidence in elevated VO2 max values and enhanced LT values (21, 29, 31). When training principally stimulates oxygen-independent metabolic pathways, predictably, changes enhance those pathways (21). Threshold values of athletes whose training and competitions are dominated by oxygen-independent or 'anaerobic' metabolic pathways are however, less well-understood.

We previously compared respiratory compensation threshold (RCT) values (11) using the V-slope method of Beaver et al. (4) among aerobic and anaerobic competitors as well as untrained participants. Predictable responses were observed for aerobic competitors (VO$_2$ max: 67.2 $\pm$ 8.5, RCT: 76.3 $\pm$ 8.7 % VO$_2$ max) and untrained participants (VO$_2$ max: 43.8 $\pm$ 5.4, RCT: 62.5 $\pm$ 8.8 % VO$_2$ max). For anaerobic competitors VO$_2$ max values were 50.0 $\pm$ 6.5 with RCT being 80.6 $\pm$ 5.6 % VO$_2$ max. VO$_2$ max values for anaerobic competitors fell between aerobic competitors and untrained participants reflecting the modest training stimulus to the aerobic metabolic pathways in this group, similar to other investigations (14, 15, 18). However, an enhanced RCT was observed in those accustomed to repeated bouts of high intensity training. While exact mechanisms were not investigated in Green et. al. (11), it has been previously established that central cardiovascular system adaptations primarily enhance VO$_2$ max while peripheral adaptations at the level of the muscle have greater impact on threshold values (8, 12, 25, 30). The observation that differential mechanisms prompt change in these variables is also confirmed by the observation that threshold values may change independently of VO$_2$ max (7, 8, 10, 12, 20, 25, 26, 30).

MacDougall (16) demonstrated that anaerobic training stimulates peripheral adaptations such as changes in lactate dehydrogenase (LDH) with concomitant changes in VO$_2$ max. Conversely, Linossier et. al. (15) and Roberts et al. (24) found LDH changes in the absence of changes in VO$_2$ max. With differential mechanisms operating on these variables, training heavily dependent on oxygen-independent metabolism may generate positive responses in blood lactate threshold (LT) without considerable changes in VO$_2$ max. While previous research confirms this idea using RCT (11), respiratory measures do not always correspond with blood lactate threshold (5). This is confirmed also by the presence of a ventilatory threshold in patients with McArdle's disease in the absence of a lactate threshold (23). Lactate threshold in anaerobic athletes is not well-understood. The objective of the current study was to compare VO$_2$ max, LT, and VO$_2$ at LT of aerobic athletes (ARA), anaerobic athletes (ANA), and untrained participants (UTS). It was hypothesized that a) VO$_2$ max for ANA would be intermediate to ARA and UTS, b) LT would be similar between ARA and ANA both of which would be greater than UTS and c) VO$_2$ at LT would be incrementally lower from ARA to ANA to UTS.

METHODS

Participants
Males and females were recruited based on physical activity status and events in which they were competitive during the time of data collection. Participants were screened

using the Physical Activity Readiness Questionnaire (PAR-Q) (31) and a health status questionnaire with all participants stratified as low risk based on known risk factors and ACSM stratification procedures (2). Participants signed a written informed consent and procedures were approved by the appropriate Institutional Review Board for the protection of human participants prior to data collection.

Participants were classified as ARA, ANA, or UTS. Criteria for ARA were; currently active as a member of a collegiate cross-country team or VO_2 max $\geq$ 55 ml$\cdot$kg$^{-1}\cdot$min^{-1} (females) and 60 ml$\cdot$kg$^{-1}\cdot$min^{-1} (males) reflecting significant aerobic training. Criteria for ANA were; currently active as a collegiate athlete in competitions requiring repeated high intensity, short duration exercise bouts. This group included football players (n=3), sprinters/strength-trainers (n=4), softball players (n=1), and gymnast (n=1). Criteria for classification as UTS were; VO_2 max $\leq$ 40 ml$\cdot$kg$^{-1}\cdot$min^{-1} (females) and $\leq$ 50 ml$\cdot$kg$^{-1}\cdot$min^{-1} (males) or self-reported minimal engagement in physical activity (2). Both genders were represented in each group [ARA: males (n=7), females (n=3), ANA: males (n=7), females (n=2), UTS: males (n=4), females (n=3)].

Protocol
Following consent and screening, participants were assessed for height (m), body mass (kg) using a balance type scale (Detecto Scales Inc. Brooklyn. NY, USA). Body fat (%) was estimated using Lange skinfold calipers (Cambridge, MA) and a 3 site method (19) (males: chest, abdomen, thigh) (females: triceps, iliac crest, thigh). Participants then completed a treadmill test

to exhaustion. The protocol utilized three minutes stages with the following velocity (m/min) and grade combinations: 80:0%, 94:2%, 121:3%, 147:5%, 188:8%, 214:10%, 214:12%. Initial stages were intentionally easy as a rigorous protocol would have limited the number of data points for untrained participants making LT difficult to identify.

Data were collected at two institutions resulting in different models of metabolic systems and lactate analyzers being utilized. Participants were fitted with a heart rate monitor (Polar Electro, Kempele, Finland) an air-cushioned face mask (Vacu-med, Ventura, CA) or a Hans Rudolph (Kansas City, MO) mask. Oxygen consumption (VO_2) was assessed using 20 sec averages with a Vacu-med Vista mini cpx (Vacumed, Ventura, CA), or a Parvomedics Truemax 2400 (Parvomedics Inc., Sandy, UT). Units were calibrated with gas of known concentration (16% O_2, 4% CO_2) prior to each test. Calibration of ventilatory measures was completed using a 3L Hans Rudolph syringe (Hans Rudolph, Kansas City, MO, USA). Verbal encouragement was provided with tests terminated at volitional exhaustion. Criteria for achievement of VO_2 max were a) RER $\geq$ 1.1, b) heart rate at test termination $\geq$ 85% of age-predicted max, and c) RPE $\geq$ 18 on Borg's category RPE scale (21,31). A minimum two of three criteria was satisfied for each subject. VO_2 max was accepted as the highest observed value (as a 20 sec average) recorded during testing.

Capillary blood samples were taken from a fingertip the last 10 sec of each stage using a sterile, single-use lancet (BP Lancet CAT

3300, Ulster Scientific Incorporated, NY) and capillary tubes (Analox Inc., Boston, MA, USA, or Stat Sampler Capillary Tubes, Northwood, MA). Samples (25 microL each) were analyzed in duplicate with the average recorded as the value for each stage. Lactate concentration per sample was determined by electroenzymatic method using a YSI 1500 Sport (Yellow Springs Instruments, Yellow Springs, OH) or an Analox PGM-7 analyzer (Analox, Boston, MA). Analyzers were calibrated prior to initiation of each test. For all equipment, procedures were in accordance with manufacturer's instructions.

Following completion of maximal testing, LT was determined for each participant using a graphic plot of blood lactate concentration [La] (y-axis) vs. VO_2 (x-axis) (29). On each plot, the 4 mmol·L^{-1} lactate concentration point was identified on the y-axis. From that point, a horizontal line was drawn across the graph with a vertical line drawn straight down at the point where the lactate curve crossed the horizontal line. The vertical line crossed the horizontal axis at a VO_2 which was identified as the VO_2 at the 4 mmol·L^{-1} threshold. VO_2 at threshold was expressed as a percentage of VO_2 max and accepted as LT.

Statistical Analysis
Analyses were conducted using SPSS software version 14.0 (Somers, NY, USA). Age, height, body mass, and estimated body fat percent were compared among groups using an ANOVA for each dependent measure. Similarly, VO_2 max (ml·kg^{-1}·min^{-1}), LT, and VO_2 at LT (VO_{2LT}) were compared using a series of one-factor ANOVA's. When follow-up tests were required, an independent samples t-test was used for identifying between group differences. Results were considered significant at $p < 0.05$.

RESULTS

All data are presented as means and standard deviations. Descriptive characteristics of participants by group are presented in Table 1. Age and height were not significantly different among groups. Body mass was significantly greater for ANA vs. ARA with no significant difference for ANA vs. UTS or ARA vs. UTS. Estimated body fat percentage was significantly greater for UTS vs. ARA an ANA with no significant difference for ARA vs. ANA. Figure 1 shows VO_2 max (ml·kg^{-1}·min^{-1}) for ARA was significantly greater than ANA and UTS with ANA significantly greater than UTS. Lactate threshold (as % of VO_2 max) values are displayed in Figure 2. There was no significant difference for ARA vs. ANA (power = 0.030). ARA and ANA were significantly greater than UTS. Oxygen consumption at LT (VO_{2LT}) was significantly greater for ARA than ANA and UTS with ANA significantly greater than UTS as well (Figure 3).

Table 1. Means and standard deviations for descriptive characteristics aerobic athletes (ARA), anaerobic athletes (ANA) and untrained participants (UTS).

Variable	ARA (n=10)	ANA (n=9)	UTS (n=7)
Age (yrs)	22.9 ± 3.4	22.2 ± 2.7	21.6 ± 3.3
Height (m)	1.75 ± 0.10	1.78 ± 0.11	1.79 ± 0.07
Body mass (kg)	67.0 ± 8.3	81.8 ± 13.4**	72.8 ± 12.5
Body Fat (%)	11.6 ± 6.3	12.0 ± 5.4	18.9 ± 5.2*

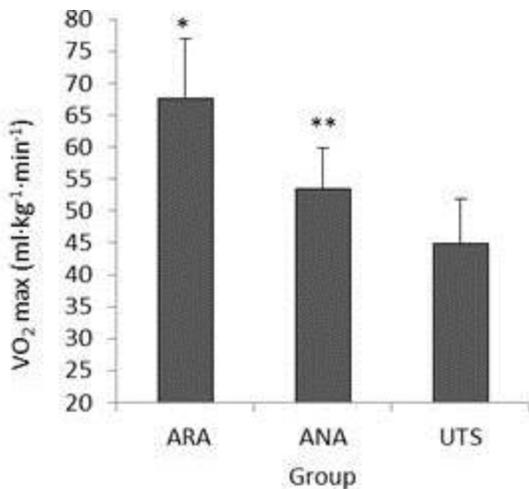* UTS significantly greater than ARA and ANA (p<0.05). ** ANA significantly greater than ARA (p<0.05)

Figure 1. VO_2max (ml·kg^{-1}·min^{-1}) of aerobic athletes (ARA), anaerobic athletes (ANA) and untrained participants (UTS). * ARA significantly greater (p<0.05) than ANA and UTS. **ANA significantly greater (p<0.05) than UTS.
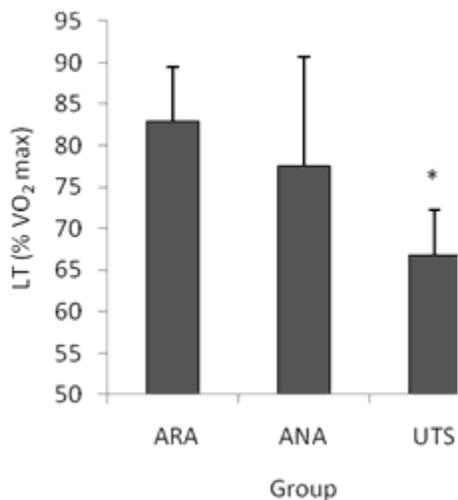

Figure 2. LT (as % VO_2 max) of aerobic athletes (ARA), anaerobic athletes (ANA) and untrained participants (UTS). * UT significantly less (p<0.05) than ARA and ANA.

DISCUSSION

Lactate threshold (LT) of aerobically-trained athletes and untrained participants are well-documented. However, for anaerobic athletes LT is not well understood. RCT as a percent of VO_2 max may be similar between aerobic and anaerobic athletes (11), yet ventilatory and blood lactate inflection points may not coincide (5). The current study compared VO_2 max, LT and VO_2 at LT among aerobic athletes, anaerobic athletes, and untrained participants. Based on a previous investigation from our lab (11) we anticipated VO2 max values for ANA would fall between ARA and UTS with LT values being similar for ANA and ARA. In general, these responses were observed.
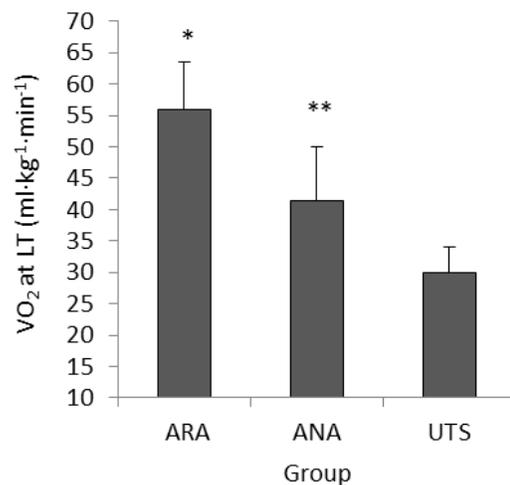

Figure 3. VO_2 (ml·kg^{-1}·min^{-1}) at LT for aerobic athletes (ARA), anaerobic athletes (ANA) and untrained participants (UTS). * ARA significantly greater (p<0.05) than ANA and UTS. **ANA significantly greater (p<0.05) than UTS.

Different mechanisms alter VO_2 max and threshold (7, 8, 10, 12, 20, 25, 26, 30). There is an obvious discordance between training for aerobic vs. anaerobic competitions. Aerobic athletes' training and competition is dominated by oxidative metabolism while anaerobic athletes depend more heavily on the phosphagen system and anaerobic glycolytic turnover in shorter duration, near maximal intensity efforts (21, 31). Improved VO_2 max is an obvious adaptation for aerobic athletes. Conversely, low reliance on oxidative metabolism is

unlikely to elevate VO_2 max for anaerobic athletes. Peripheral muscular adaptations are more likely to augment threshold (8, 12, 25, 30). It is reasonable that high intensity exercise improves buffering of acidosis, as the peripheral musculature is exposed to high levels of acid. Consequently, an exponential increase in [La] may be delayed to a higher percent of VO_2 max for ANA vs. UTS.

Current results reveal VO_2 max (ml·kg⁻¹·min⁻¹) was significantly greater for ARA than ANA and UTS (Figure 1). Even in consideration of error associated with open-circuit spirometry, observed values are obviously divergent among groups. Greater values for ARA were predictable because VO_2 max was used in defining groups and because of the influence of aerobic training on VO_2 max. However, VO_2 max was not used as a criteria defining ANA. Values for VO_2 max for ANA fell between ARA and UTS (Figure 1). Current results for VO_2 max (Figure 1) are similar to results from our previous investigation of aerobic competitors (67.2 $\pm$ 8.5), anaerobic competitors (50.0 $\pm$ 7.8) and untrained participants (43.8 $\pm$ 5.4) (11). In that study we attributed modest VO_2 max of ANA to a relatively minor contribution of oxidative metabolic pathways during training. Energy system contributions are dependent heavily on intensity and duration of a bout (10). We determined ANA training and competition was dominated by oxygen-independent pathways with comparatively less contribution from oxidative pathways. With minimal stimulus, there would be minimal response. We propose the greatest role of oxidative metabolism for ANA likely occurs during recovery periods between repeated high intensity bouts. The contribution of aerobic metabolism during a given bout increases with sequential repeated efforts during high intensity interval training (3, 22). Theoretically then, the stimulus to the aerobic system could systematically increase to a considerable level during repeated 'anaerobic' efforts for ANA. Even so, previous (11) and current VO_2 max results (Figure 1) indicate the stimulus fails to reaches a level comparable to that experienced by aerobic athletes. High intensity, short duration bouts associated with training/competition for ANA hinge principally on provision of ATP via oxygen –independent (anaerobic) pathways (21). We attribute the intermediate VO_2 max values for ANA in part to provision of only a marginal stimulus to pathways critical to oxidative energy production due to the nature of training/competition.

Another possible explanation for low VO_2 max values of ANA (vs. ARA) would be the genetic make-up of this group (31). Self-selection of athletes with a high percentage of fast-twitch muscle fiber into sports dominated by high intensity, shorter duration bouts is plausible. If ANA in the current study possessed a high percentage of fast-twitch muscle fibers, they would have a lower potential to enhance the oxidative capacity of their muscle, thus resulting in a lower ceiling for VO_2 max. It is emphasized that muscle biopsy information was not available for current participants and further, it is arguable whether 'elite' is an accurate descriptor for collegiate level athletes. Direct evaluation of muscle was beyond the scope of this study but should be mentioned as a potential factor influencing VO_2 max results. It is emphasized however that

attributing result of the current investigation to muscle fiber type in the current study is speculative. Future research is warranted to more closely examine a possible link.

Research shows threshold (as % VO_2 max) may continue to increase beyond a point where VO_2 max increases (7, 8, 10, 20, 26). This demonstrates there is at least some independence between the mechanisms prompting change in these two variables. Differential mechanisms may help explain current results. Endurance training for ARA certainly enhances VO_2 max. Further, Ivy (13) found strong correlations between threshold values and percentage slow twitch muscle fiber (r=0.70) and muscle fiber respiratory capacity (r=0.94) a function of mitochondrial density. Although not directly measured, this would also be expected for ARA. If high intensity training enhances threshold as evidenced in ANA (Figure 2), aerobic training alone may not result in LT occurring at its highest possible intensity within a given individual. High LT for ARA in the current study could be attributed to a combination of aerobic training as well as incorporation of high-intensity interval work as a part of training. Participation by ARA in interval training was not assessed but should be included in future investigations.

Because of the independence of VO_2 max and threshold responses, it is reasonable that LT could increase in absence of major changes in VO_2 max. Current results support this. Figure 1 shows VO_2 max for ANA was only modestly elevated compared to UTS while Figure 2 shows greater LT for ANA vs. UTS. Peripheral changes may have positively augmented LT

while central cardiorespiratory changes necessary to increase VO_2 max were largely absent. It is noticeable in Figure 2 that the standard deviation for LT for ANA (13.1) was greater than for other groups (ARA = 6.5, UTS = 5.4). Greater variation could be related to the diverse nature of training within ANA. That is, training, as well as competition for football players vs. basketball would be noticeably different although both are principally anaerobic in nature. Therefore, training stimuli for these athletes could have varied even among athletes within the ANA group however this is speculative in absence of detailed information regarding training (volume, intensity, duration). Training logs of participants would permit a more direct link between training type and adaptations and should be included in future investigations.

Blood lactate concentration is the difference between lactate production vs. removal. Consequently, training-induced changes in production or removal could alter LT. High intensity, short duration efforts enhance intramuscular lactate dehydrgogenase (LDH) (15, 16, 24). Although no direct data are available in the current study, this may have occurred in ANA. If so, favorable responses in LT could have been related to enhanced lactate turnover, a peripheral adaptation. Marcinik et al. (18) showed 12 weeks of strength training lowered blood lactate concentration at relative exercise intensities (55 - 75% peak VO_2) and significantly increased (12%) lactate threshold values with no significant changes in cycle or treadmill VO_2 max. Similar to Marcinik et. al. (18), current results show minimal central adaptations (i.e. VO_2 max)

implicating peripheral adaptations as responsible for changes influencing lactate kinetics. That is, for ANA, absence of central changes indicates LT improved due to peripheral changes. More in-depth analysis of LDH (including specific isozymes) in future studies may extend the understanding of adaptations to LT.

Various threshold values can predict success in endurance events (1, 6, 9, 17, 27, 28, 31, 33). However, care should be taken when interpreting current results within that paradigm. Lactate threshold is commonly expressed relative to VO_2 max (i.e. as a percentage). Consequently VO_2 max which sets the upper limit for endurance performance success must be considered in conjunction with LT. For this reason VO_2 at LT was analyzed separately in the current study (Figure 3) even though this variable is a calculation of two other dependent measures. Solely evaluating lactate threshold would potentially lead to the conclusion that an anaerobic athlete with a high LT (80% for example) would out perform an aerobic athlete who had a lower LT (75% for example). This presumption would obviously be false. Greater VO_2 max for the aerobic athlete would more than compensate for inferior LT. More prudently, endurance performance potential should be evaluated by assessing VO_2 max and lactate threshold concurrently.

In summary, this study shows anaerobic athletes possess VO_2 max values intermediate to aerobic athletes and untrained individuals. Conversely, LT for ANA, while having greater variation, are superior to UTS and may rival values for ARA. Training-induced adaptations for anaerobic athletes warrant additional research. From a practical application standpoint, current results re-emphasize inclusion of anaerobic training as part of endurance athlete's preparation.

The current investigation was limited in part by lack of information regarding precise training habits for ARA and ANA and somewhat small (uneven) sample sizes. Also, future investigations should seek to obtain training log information from participants to overcome poor control of training undertaken by participants. This could more definitively link LT with training type. Another limitation was direct measures of LDH which may be responsible for LT responses. As such, exact mechanisms elevating LT in ANA remain elusive. It is plausible that adaptations within muscle such as enzymatic changes or buffering capacity may play a role. These possibilities should be more rigorously investigated in future research. Current results indirectly add to the literature indicating mechanisms leading to changes in VO_2 max and LT are at least partially independent.

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