THE EFFECTS OF TRAINING INTENSITY ON BLOOD LACTATE BREAKPOINTS IN RUNNERS

by

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The purpose of this study was to determine the effects of an eight week running endurance training program at two different intensities on both aerobic (AeT) and lactate turn point (LTP) thresholds.

Twenty-nine male middle distance runners volunteered to participate in this study. Subjects were randomly assigned to 3 groups: two experimental and one control. The low intensity (LI) group trained at HR corresponding to the AeT. The high intensity (HI) group trained at HR corresponding to the LTP. The training contents of the control (C) group was not supervised: they combined mixed athletic training program with sports games. All subjects underwent an interval incremental running test before and after the training program in order to determine the changes if any in AeT, LTP and heart rates (HR) corresponding to these thresholds. Two breakpoints were determined in the lactate (LA) running velocity curve. The LTP was determined as the second abrupt increase of La around 4 mmol·l-1 by means of computer-aided linear regression break-point analysis. The AeT was defined as the first increase of LA above resting level.

The analysis of training induced changes revealed a significant increase in both AeT and LTP for HI group ($p<0.05$). The increase of LTP was a little greater than that of AeT (6,8% versus 5,5%). However, these thresholds did not change significantly in C or LI groups. No significant difference either in HR or LA variables in either of the groups was observed.

These results seem to indicate that only training program at the intensity of LTP produces an increase in both aerobic-anaerobic transition characterizing variables (AeT and LTP) while the training at the AeT intensity does not cause any changes of these thresholds in moderately trained runners.

Key words: lactate, aerobic threshold, lactate turn point, endurance training intensity, running.

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Introduction

Blood lactate (LA) measurements during exercise have become widely used to assess adaptation to endurance training. It is rather blood LA variables during incremental intensity tests rather than maximal oxygen consumption (VO2max) that determine endurance performance (Weltman A., 1995).

The relationship between blood lactate concentration and work rate during incremental exercise remains controversial. The models of threshold behavior (Beaver et al., 1985) or of continuous increase (Houghson et al., 1987) were proposed. In addition, the existence of a triphasic phenomenon in the transition from aerobic to anaerobic metabolism during increasing exercise intensity has been suggested (Kindermann et al., 1979; Skinner, McLellan, 1980; Cabrera , Chizeck, 1996). According to this model two parameters of aerobic performance – LA or aerobic threshold (AeT) and onset of blood lactate accumulation threshold (OBLA) or second LA turnpoint threshold (LTP) may be deduced from the relationship between blood La concentration and work rate. Despite the widespread use of AeT and OBLA, the techniques used for their detection vary. Assuming the triphasic model of LA changes the two breakpoint technique seems to be most appropriate (Davis et al., 1983).

Relatively few longitudinal studies have been devoted to comparing the influence of different training intensities upon AeT, and the results have been equivocal (Henritze et al., 1985; Poole et al., 1990; Golden, Vaccaro, 1984;Weltman et al., 1992). To our knowledge a comparison of response of two thresholds characterizing aerobic-anaerobic transition to continuous endurance training of different intensity has not been made.

The purpose of this study was to determine the effects of an eight week running endurance training program at two different intensities on both, aerobic (AeT) and lactate turn point (LTP) thresholds.

Methods

Subjects. Twenty-nine male middle distance runners volunteered to participate in this study. Table 1 presents the subjects' descriptive

characteristics. All subjects had undergone systematic endurance running training for at least 3 years (range 3-8 years).

Table 1. Descriptive characteristics of subjects

Group	Age (years)	Height (cm)	Weight (kg)	HRmax ($b \cdot min^{-1}$)	MAS ($m \cdot s^{-1}$)	V1km ($m \cdot s^{-1}$)
L (n=10)	19,5±6,2	176,5±5,9	63,6±7,1	198,9±6,1	4,50±0,57	5,37±0,35
H (n=11)	19,9±7,4	178,3±4,7	65,3±4,2	196,2±6,2	4,38±0,49	5,49±0,39
C (n=8)	19,3±3,2	177,1±5,7	63,4±4,9	182,5±6,8	4,51±0,39	5,36±0,29

Maximal aerobic speed (MAS) determination. The continuous incremental running test was performed on an indoor track. Each athlete ran at constant velocity in subsequent 200 m laps. Starting speed (2,65 $m \cdot s^{-1}$) was increased by 0,15 $m \cdot s^{-1}$ until exhaustion. The heart rate (HR) was measured continuously and stored at 5-s intervals (Accurex PLUS, Polar Electro, Finland). The individual running speed was controlled by a light leader. MAS was obtained by extrapolating the linear part of the HR speed curve to the HRmax and to the corresponding speed (according to Conconi et al., 1992; 1996).

Lactate and running speed relationship assessment. An interval incremental running test was performed on an indoor track. The test was preceded by a 15-20 min warm-up (stretching and 10 min of slow running) after then repeated running (3 min) and passive rest (4 min) intervals were performed. Starting speed (2,65 $m \cdot s^{-1}$) was increased by 0,25 $m \cdot s^{-1}$ during each interval. The test was continued until blood lactate concentration exceeded 4 $mmol \cdot l^{-1}$ after two consecutive laps.

Arterialised fingertip blood samples were obtained at third min of each rest interval. Using a micropipet, a blood sample of 0,1 ml was drawn and immediately analyzed by an enzymatic membrane method (Exan-G analyser, Kulis Y. et al., 1988). Prior to blood analyses, the analyzer was calibrated with standard solutions.

Two breakpoints were determined in the lactate running velocity curve. The lactate turn point (LTP) was determined according to Davis et al. (1983) as the second abrupt increase of La around 4 mmol·l-1 by means of computer-aided linear regression break-point analysis. Calculations were made

exclusively between aerobic threshold (AeT), which was defined as the first increase of LA above resting level, and maximal test speed.

Training program. Following the MAS test and prior to start of training, the subjects were grouped into triplets according to their MAS and 1 km best time, and then randomly assigned to either one of the threshold exercise groups or to a control group. One threshold exercise group trained at HR corresponding to the pre-training AeT (low intensity – LI group) for the duration of the program. The other threshold exercise group trained at HR corresponding to the pre-training LTP (high intensity – HI group). The training content of the control group (C) was not supervised: they combined mixed athletic training program with sports games.

All training took place on an indoor track of the Academy of Physical Education. The subjects were instructed to attend three training sessions per week for a period of eight weeks. An exercise leader supervised all training sessions. During each session after 10-15 min of warm-up the subject was running at the predetermined intensity for 45 min. The intensity was controlled using HR monitors (Polar Pacer, Polar Electro, Finland).

Protocol. Prior to the training program each subject was tested twice. First the MAS test was performed. Then within a week but not sooner than one day after MAS test the interval incremental running test with blood lactate determination was carried out. Immediately after the second testing the subjects of LI and HI exercise groups started their training program. The same interval incremental running test was performed within three days following the termination of an eight week training period.

Statistics. The mean value ± SD was calculated for all variables. For statistical evaluation nonparametric tests (Kruskal-Wallis ANOVA, Wilcoxon matched pairs test) were used. Statistical significance was set at $p<0,05$.

Results

Table 1 presents descriptive characteristics of the subjects. Pre-training and post-training means for all variables are presented in Table 2. The analysis of training induced changes revealed a significant increase in both AeT and LTP

for HI group ($p<0.05$). The increase of LTP was a little greater than that of AeT (6,8% versus 5,5%). This resulted in significantly greater difference between LTP and AeT after an 8 week training program in this group ($p<0,05$). However, these thresholds did not change significantly in the C or LI groups. No significant difference either in HR or LA variables in either of the groups was observed.

Table 2. Pre-training and post training means for each variable

Variable	Group	Pretraining Mean±SD	Post-training Mean±SD	Diff	% Diff
AeT ($m \cdot s^{-1}$)	LI HI C	3,34±0,41 3,43±0,29 3,63±0,22	3,37±0,39 3,62±0,30* 3,73±0,14	+0,03 +0,19 +0,10	0,9 5,5 2,8
La AeT ($mmol \cdot l^{-1}$)	LI HI C	2,52±0,56 2,51±0,61 2,05±0,62	2,38±0,68 2,30±0,85 1,91±0,32	-0,14 -0,21 -0,14	-5,6 -8,4 -6,8
HR AeT ($b \cdot min^{-1}$)	LI HI C	171,13±14,97 163,90±12,97 164,88±12,33	166,00±10,43 163,40±10,44 171,29±10,26	-5,13 -0,50 +6,41	-3,0 -0,3 3,9
LTP ($m \cdot s^{-1}$)	LI HI C	3,79±0,47 3,97±0,37 4,20±0,24	3,92±0,39 4,24±0,32* 4,25±0,11	+0,13 +0,27 +0,05	3,4 6,8 1,2
La LTP ($mmol \cdot l^{-1}$)	LI HI C	3,39±0,39 3,38±0,59 3,11±0,3	3,33±0,69 3,24±0,68 3,09±0,33	-0,06 -0,14 -0,02	-1,8 -4,1 -0,6
HR LTP ($b \cdot min^{-1}$)	LI HI C	181,94±11,05 178,88±10,45 179,38±11,17	179,57±8,63 178,01±9,67 182,14±8,19	-2,37 -0,87 +2,76	-1,3 -0,5 +1,5
LTP-AeT ($m \cdot s^{-1}$)	LI HI C	0,44±0,17 0,54±0,12 0,55±0,06	0,55±0,10 0,63±0,09* 0,53±0,05	+0,11 +0,09 -0,02	+25,0 +16,7 -3,6

Discussion

The major finding of this study was that only training at the intensity of LTP resulted in a similar increase of both aerobic-anaerobic transition

characterizing parameters (AeT and LTP) while the training at the AeT intensity did not cause any changes of these thresholds in moderately trained runners.

Similar lack of changes in AeT following two months of training at this threshold has been reported (Golden, Vaccaro, 1984; Sady et al., 1980). Sady et al. (1980) did not show any change in AT for their low intensity group, which trained below AeT. They did not train a group at an intensity equal to LTP as in the present study. The results of the study made by Golden & Vaccaro also indicated that training at the AeT couldn't increase AeT by moderately fit subjects. On the contrary, Fabre et al. (1997) observed improvement of ventilatory threshold (VeT) in elderly unfit subjects after three months of training at this threshold.

Our results agree with those of many investigators who found enhancement of AeT (or similar parameters) only in experimental groups which trained at higher intensity (Davis et al., 1979; Sady et al., 1980; Denis et al., 1982; Henritze et al., 1985;Weltman et al., 1992). Significant increases in AeT were found by Davis et al. (1979) and Sady et al. (1980) in their high intensity group. The subjects, however, were less fit. Davis et al. (1979) used as subjects sedentary, middle aged males, and Sady et al. (1980) used overweight, college age females. The fitness level of the subjects may, therefore, be a factor in any alterations in AeT resulting from this type of training. Meta-analysis of studies performed in this field showed that training at the intensity of AeT or VeT might provide enough training stimulus for sedentary subjects, but higher intensity might be necessary for conditioned subjects (Londeree, 1997).

It is worth to remember that changes in VeT and AeT are not always matched. Poole and Gaesser (1985) reported that the La and VeT responded differently to a particular training stimulus.

The changes of LTP related thresholds after training at the intensity corresponding to this threshold have also been reported. Significant increases in OBLA after 14 weeks of training at the intensity corresponding 4 mmol/l blood lactate level in well trained middle and long distance runners were observed (Sjodin et al., 1982). 40 weeks of training at the intensity of approximately LTP resulted in 18 % increase of 4 mmol threshold but in less (10-11 %) improvement in LT and VeT (Denis et al., 1982). The increase of respiratory compensation point was larger than that of AeT after high-intensity endurance

training at levels above AeT in middle-distance runners (Oshima et al., 1998). There was a tendency of greater increase in LTP (+6.8 %) in comparison with AeT (+5.5 %) in our study as well. This might be associated with the increase in the range of isocapnic buffering suggested by the authors cited above (Oshima et al., 1988).

Our subjects can be considered as moderately trained athletes. Although we did not measure maximal oxygen consumption directly, it can be predicted from MAS using equations recommended by ACSM (1985) and may reach approximately 55-57 ml kg min^{-1}. In addition, by the level of LTP our runners can also be considered as moderately trained ones (Ekes et al., 1990). So it appears that in the conditioned subjects a higher training intensity is required to produce a training effect on lactate variables.

At least theoretically the running speed at the AeT and LTP may be increased due to improved mechanical efficiency (Bunc et al., 1989). This unlikely to be the case in our study, because the subjects were experienced runners and the period of training was rather short. On the other hand if mechanical efficiency were to be improved, it should be observed in all the groups examined.

It is supposed that aerobic-anaerobic transition thresholds are mostly related to the aerobic metabolism properties of the working muscles (Jacobs, 1981; Foxdal et al., 1994). It has been reported that both the oxidative capacity and the fiber type distribution of the exercising muscle are related to the metabolic rate that defines these thresholds (Ivy et al., 1980).

Many underlying mechanisms responsible for inducing adaptations in muscle are not known. However, it is clear that muscles or muscle fibers must be recruited during exercise in order to adapt to the training program (Holloszy, 1967). The effect of increased training intensity on muscle adaptations can be attributed to the effect of intensity on muscle fiber recruitment (Dudley et al., 1982). As the training bouts become more intensive, more of the low oxidative type II fibers are recruited and become adapted to the training.

Possible mechanisms that might account for an increased AT after endurance training include an improved distribution of blood in the trained muscles, increased oxidative capacity at the cellular level, and an alteration in

the muscle fiber recruitment pattern resulting in an increased activation of the red, oxidative muscle fibers.

One possible reason for the increase in AeT and LTP might be due to the reduction in La at submaximal work rates (Hurley et al., 1985). It has been shown that endurance training enhances the clearance (Donovan, Brooks, 1985) and decreases the production (Favier et al., 1986) of La at a given metabolic rate.

The validity of the results presented depends upon the acceptance of a "threshold" phenomenon in the exercise blood lactate response. Hughson et al. (1987) reported that the lactate profile during incremental exercise could be described by an exponential plus constant mathematical function, thereby implying that a "threshold" did not exist. . It is supposed that La-kinetics during an incremental exercise fits the three-segment model (i.e., has two intersection points) better than the two-segment model (Skinner, McLellan,1980). Despite disagreements over the definition and causal mechanisms of the anaerobic thresholds, their practical importance has been adequately documented. It is well known, that each of LT or OBLA has a high correlation to various parameters, e.g., distance running performance and maximal aerobic power. AeT or LTP within its limits can be used for prediction of aerobic performance, evaluation of training effect, selection of exercise intensity for exercise prescription, and evaluation of exercise tolerance capacity for cardiac rehabilitation.

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