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Effects of moderate-intensity endurance and high-intensity intermittent training on anaerobic capacity and $\dot{V}O_{2\max}$

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The training protocol used in experiment 2 was first introduced by Kouichi Irisawa, who was a head coach of the Japanese National Speed Skating Team. The training has been used by the major members of the Japanese Speed Skating Team for several years.

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ABSTRACT

This study consists of two training experiments using a mechanically braked cycle

ergometer. First, the effect of 6 wk of moderate-intensity endurance training (intensity: 70% of maximal oxygen uptake ($\dot{V}O_{2\max}$), $60 \text{ min}\cdot\text{d}^{-1}$, $5 \text{ d}\cdot\text{wk}^{-1}$) on the anaerobic capacity (the maximal accumulated oxygen deficit) and $\dot{V}O_{2\max}$ was evaluated. After the training, the anaerobic capacity did not increase significantly ($P > 0.10$), while $\dot{V}O_{2\max}$ increased from $53 \pm 5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ to $58 \pm 3 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ($P < 0.01$) (mean \pm SD). Second, to quantify the effect of high-intensity intermittent training on energy release, seven subjects performed an intermittent training exercise $5 \text{ d}\cdot\text{wk}^{-1}$ for 6 wk. The exhaustive intermittent training consisted of seven to eight sets of 20-s exercise at an intensity of about 170% of $\dot{V}O_{2\max}$ with a 10-s rest between each bout. After the training period, $\dot{V}O_{2\max}$ increased by $7 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, while the anaerobic capacity increased by 28%. In conclusion, this study showed that moderate-intensity aerobic training that improves the maximal aerobic power does not change anaerobic capacity and that adequate high-intensity intermittent training may improve both anaerobic and aerobic energy supplying systems significantly, probably through imposing intensive stimuli on both systems.

During high-intensity exercise lasting more than a few seconds, adenosine triphosphate (ATP) is resynthesized by both aerobic and anaerobic processes (7). The ability to resynthesize ATP may limit performance in many sports. Thus, if possible, the training of athletes for sports involving high-intensity exercise should improve the athletes' ability to release energy both aerobically and anaerobically. The success of different training regimens can and should be evaluated by the athletes' performance. However, performance is influenced by other factors such as psychology. In addition, an adequate training regimen may have several different components, all of which may not improve the athletes' ability to resynthesize ATP. Training programs should therefore be evaluated by other means, e.g., by laboratory experiments.

The aerobic energy releasing system is conventionally evaluated by maximal oxygen uptake ($\dot{V}O_{2\max}$) (10), and there are many studies on the effect of training on $\dot{V}O_{2\max}$ (9). However, until recently methods for quantifying anaerobic energy release have been inadequate and thus information on the effect of training on anaerobic

capacity, i.e., the maximum amount of energy available from anaerobic sources, is incomplete. We have proposed that the accumulated oxygen deficit, first introduced by Krogh and Lindhard in 1920 (4), is an accurate measure of the anaerobic energy release during treadmill running (6) and bicycling (7). This principle may allow examination of the anaerobic capacity (3), taken as the maximal accumulated oxygen deficit during 2-3 min of exhaustive exercise (6,7). Therefore, the effect of specific training on the anaerobic capacity may be evaluated by measuring the maximal accumulated oxygen deficit before and after training. Generally, the more demanding the training, the greater the fitness benefits. Therefore, we were interested in learning whether the effects of training on anaerobic capacity are dependent on the magnitude of anaerobic energy release developed by the specific training. To study this issue, we compared two different training protocols: a moderate-intensity endurance training that is not supposed to depend on anaerobic metabolism and a high-intensity intermittent training that is supposed to recruit the anaerobic energy releasing system almost maximally.

MATERIALS AND METHODS

Subjects. Young male students majoring in physical education volunteered for the study (Table 1). Most were physically active and were members of varsity table tennis, baseball, basketball, football (soccer), and swimming teams. After receiving a detailed explanation of the purposes, potential benefits, and risks associated with participating in the study, each student gave his written consent.

	<i>N</i>	Age (yr)	Height (cm)	Weight (kg)	$\dot{V}O_{2max}$ (ml·kg ⁻¹ ·min ⁻¹)	Maximal Accumulated O ₂ Deficit (ml·kg ⁻¹)
Experiment 1	7	23 ± 1	169 ± 5	68.5 ± 7.5	52.9 ± 4.7	69.0 ± 6.1
Experiment 2	7	23 ± 1	172 ± 3	68.5 ± 5.9	48.2 ± 5.5	60.9 ± 8.6

Values are means ± SD. $\dot{V}O_{2max}$: maximal oxygen uptake. Maximal accumulated O₂ deficit is taken as the accumulated O₂ deficit during a 2- to 3-min exhausting bouts of bicycling. Maximal accumulated oxygen deficit and $\dot{V}O_{2max}$ are the pretraining values.

TABLE 1. Characteristics of the subjects.

Protocol. All experiments, as well as pretests, were done on a mechanically braked cycle ergometer (Monark, Stockholm, Sweden) at 90 rpm. Each test or high-intensity intermittent training session was introduced by a 10-min warm-up at about

50% of $\dot{V}O_{2\max}$.

Experiment 1. The subjects started training after their $\dot{V}O_{2\max}$ and maximal accumulated oxygen deficit were measured. They exercised $5 \text{ d}\cdot\text{wk}^{-1}$ for 6 wk at an intensity that elicited 70% of each subject's $\dot{V}O_{2\max}$. The pedaling rate was 70 rpm, and the duration of the training was 60 min. As each subject's $\dot{V}O_{2\max}$ increased during the training period, exercise intensity was increased from week to week as required to elicit 70% of the actual $\dot{V}O_{2\max}$. During the training, the maximal accumulated oxygen deficit was measured before, at 4 wk, and after the training. $\dot{V}O_{2\max}$ was determined before and after the training and every week during the training period.

Experiment 2. Subjects exercised for $5 \text{ d}\cdot\text{wk}^{-1}$ for 6 wk. For $4 \text{ d}\cdot\text{wk}^{-1}$, they exercised using exhaustive intermittent training. They were encouraged by the supervisor to complete seven to eight sets of the exercise. Exercise was terminated when the pedaling frequency dropped below 85 rpm. When they could complete more than nine sets of the exercise, exercise intensity was increased by 11 W. One day per week the subjects exercised for 30 min at an intensity of 70% $\dot{V}O_{2\max}$ before carrying out four sets of the intermittent exercise at 170% $\dot{V}O_{2\max}$. This latter session was not exhaustive. The anaerobic capacity was determined before, at 2 wk, and 4 wk into the training, and after the training. $\dot{V}O_{2\max}$ was determined before, at 3 wk, 5 wk, and after the training.

METHODS

Pretest. Each subject's oxygen uptake was measured during the last 2 min of six to nine different 10-min exercise sets at constant power. The power used during each set ranged between 39% and 87% of the $\dot{V}O_{2\max}$. In addition, the power that would exhaust each subject in 2-3 min was established. These pretests were carried out on 3-5 separate days.

$\dot{V}O_{2\max}$. After a linear relationship between exercise intensity and the steady-state oxygen uptake had been determined in the pretests, the oxygen uptake was measured for the last two or three 30-s intervals during several bouts of supramaximal intensity exercise that lasted 2-4 min. The highest $\dot{V}O_2$ was determined to be the

subject's $\dot{V}O_{2\max}$ (7,10).

Anaerobic capacity. Anaerobic capacity, the maximal accumulated oxygen deficit during a 2-3-min exhaustive bicycle exercise, was determined according to the method of Medbø et al. (6,7). The exercise intensity used to cause exhaustion within the desired duration (2-3 min) was established on pretests. On the day that anaerobic capacity was measured, the subjects exercised at the preset power to exhaustion (defined as when they were unable to keep the pedaling rate above 85 rpm).

Methods of analysis. Fractions of oxygen and carbon dioxide in the expired air were measured by a mass spectrometer (MGA-1100, Perkin-Elmer Cetus, Norwalk CT). The gas volume was measured by a gasometer (Shinagawa Seisakusho, Tokyo, Japan). Values are shown as means \pm SD. The data were compared using a paired *t*-test. The significance level for all comparisons was set at $P < 0.05$.

Calculations. For each subject linear relationships between the oxygen demand and power (experiment 1: $r = 0.997 \pm 0.001$, experiment 2: $r = 0.998 \pm 0.001$) were established from the measured steady state oxygen uptake at different power during the pretests. The regression parameters are shown in Table 2. The regression parameters did not change during training periods in either experiment 1 or 2.

	Y-Intercept ($\text{ml}\cdot\text{kg}^{-1}$)	Slope ($\text{ml}\cdot\text{min}^{-1}\cdot\text{w}^{-1}$)	$S_{y,x}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)
Experiment 1	6.0 ± 0.9	13.2 ± 0.4	0.7 ± 0.1
Experiment 2	6.1 ± 0.8	13.0 ± 0.3	0.6 ± 0.1

Values are means \pm SD. $S_{y,x}$ is the scatter around the regression line. Y-intercept and slope of the regression line was calculated from the relationship between the power (X axis: w) and oxygen uptake during the steady state submaximal bicycle exercise (Y axis: $\text{ml}\cdot\text{kg}^{-1}$).

TABLE 2. Regression characteristics of the subjects.

The oxygen demands of the 2-3 min of exhausting exercise were estimated by extrapolating these relationships to the power used during the experiment. The accumulated oxygen demand was taken as the product of the estimated oxygen demand and the duration of the exercise, while the accumulated oxygen uptake was taken as the measured oxygen uptake integrated over the exercise duration. The accumulated oxygen deficit was taken as the difference between these two entities.

RESULTS

Experiment 1. After the 6 wk of training, the anaerobic capacity did not change ([Fig. 1](#)) ($P > 0.10$). The $\dot{V}O_{2\max}$ increased significantly during the training ([Fig. 2](#)) ($P < 0.01$).

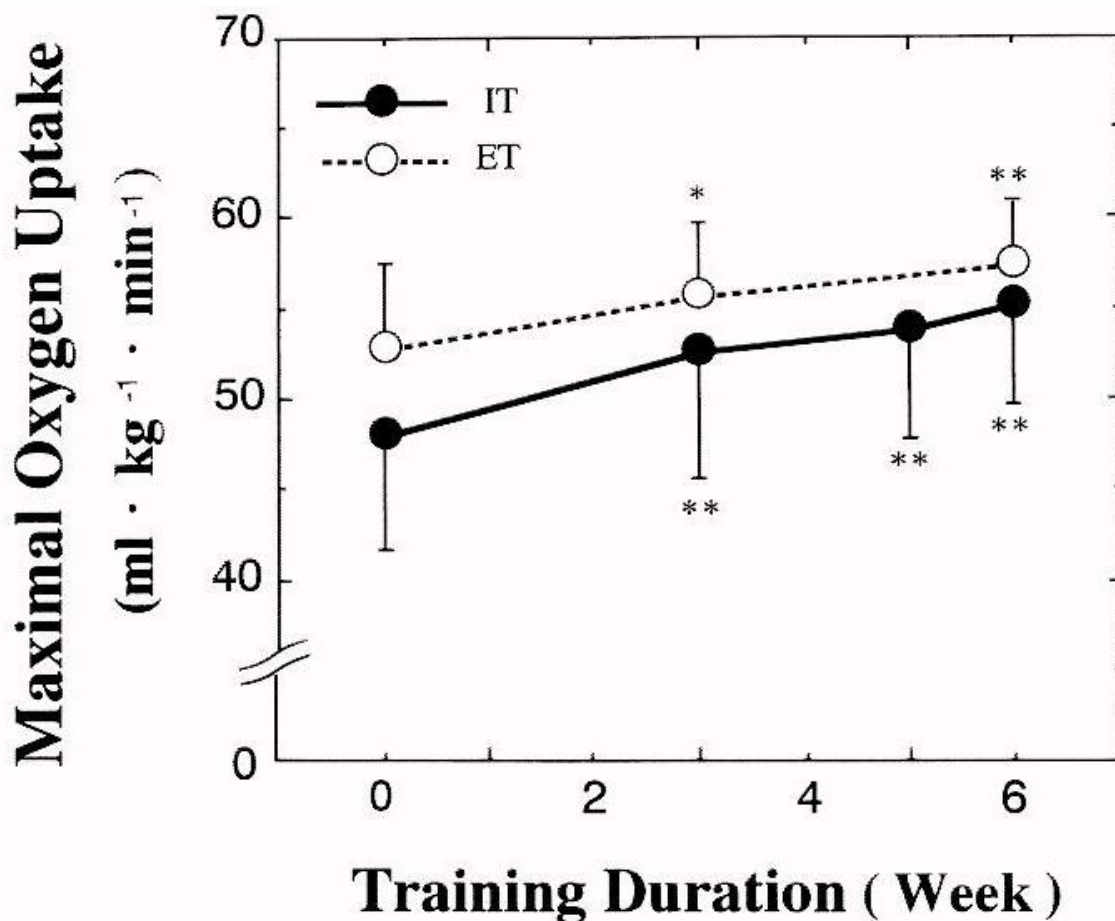


Figure 1-Effect of the endurance training (ET, experiment 1) and the intermittent

training (IT, experiment 2) on the anaerobic capacity. Significant increase from the pretraining value at $*P < 0.05$ and $**P < 0.01$; significant increase from the 2-wk value at $\#P < 0.05$.

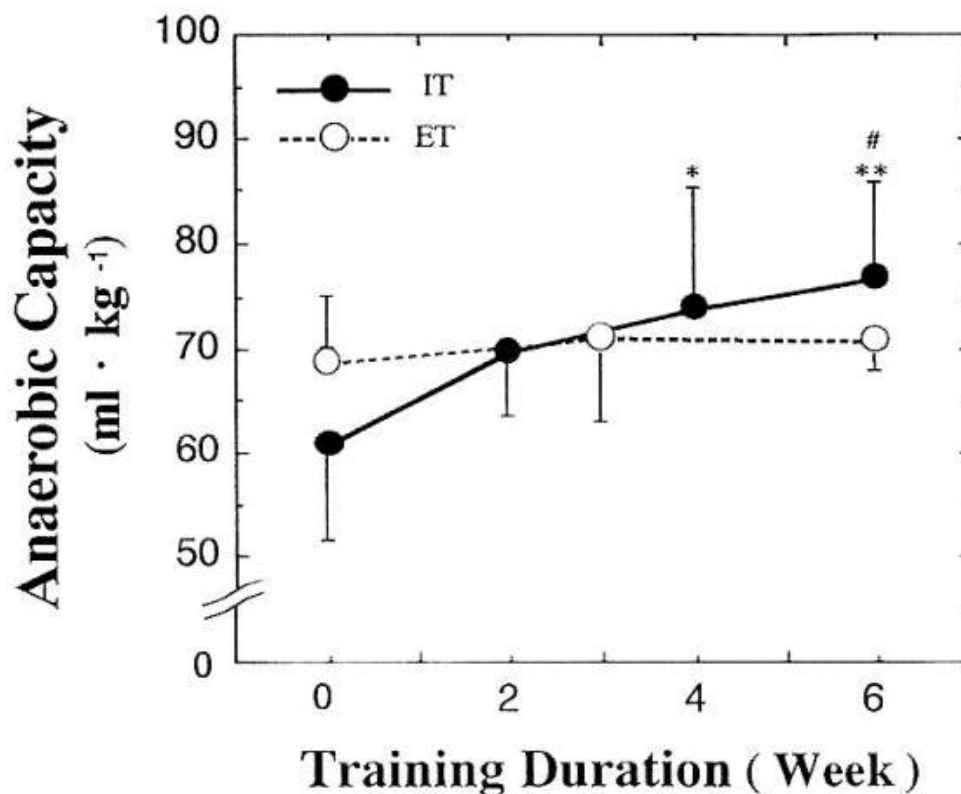


Figure 2-Effect of the endurance training (ET, experiment 1) and the intermittent training (IT, experiment 2) on the maximal oxygen uptake; significant increase from the pretraining value at $*P < 0.05$ and $**P < 0.01$, respectively.

Experiment 2. The anaerobic capacity increased by 23% after 4 wk of training ($P < 0.01$, [Fig. 1](#)). It increased further toward the end of the training period. After the training period, the anaerobic capacity reached $77 \pm 9 \text{ ml} \cdot \text{kg}^{-1}$, 28% higher than the pretraining capacity.

After 3 wk of training, the $\dot{V}O_{2\max}$ had increased significantly by $5 \pm 3 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ($P < 0.01$, [Fig. 2](#)). It tended to increase in the last part of the training period, but no significant changes were observed. The final $\dot{V}O_{2\max}$ after 6 wk of training was $55 \pm 6 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, a value of $7 \pm 1 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ above the pretraining value.

DISCUSSION

The main finding of this study was that 6 wk of aerobic training at $70\%\dot{V}O_{2\max}$ improved the $\dot{V}O_{2\max}$ by $5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ in moderately trained young men but that the anaerobic capacity, as judged by the maximal accumulated oxygen deficit, did not change. The second finding is that 6 wk of training using high-intensity intermittent exhaustive exercise improved $\dot{V}O_{2\max}$ by $7 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ and the anaerobic capacity by 28%.

The observation in experiment 1 that anaerobic capacity did not change after 6 weeks of moderate-intensity endurance training but that $\dot{V}O_{2\max}$ did increase has several implications. First, it shows the specificity of training; aerobic training does not change anaerobic capacity. Since lactate production accounts for about 75% of maximal anaerobic energy release (11), significant improvements in anaerobic capacity will probably require that the subjects can produce more lactate after training. Consequently, lactate production should be stressed to increase the anaerobic capacity during “anaerobic” training. However, since the blood lactate concentration during the exercise was low (about $2 \text{ mmol}\cdot\text{l}^{-1}$), the major part of anaerobic energy released during the exercise probably comes from the breakdown of phosphocreatine (PCr). Therefore, the training sessions in experiment 1 probably did not tax the lactate producing system much and therefore did not tax the whole anaerobic energy releasing system to any significant extent. Actually, the accumulated oxygen deficit during the first minutes of the exercise at $70\%\dot{V}O_{2\max}$ was only $37 \pm 6\%$ ($N = 7$) of the maximal accumulated oxygen deficit (data not shown).

Second, the results of experiment 1 support the idea that the accumulated oxygen deficit is a specific measure of the maximal anaerobic energy release. Due to the increased $\dot{V}O_{2\max}$ after the training period, the subjects could exercise for more than 6 min at the power used for the pretraining 2- to 3-min anaerobic capacity test. Therefore, the exercise power for the posttraining anaerobic capacity test was

increased by $6 \pm 3\%$ to exhaust each subject in 2-3 min. However, the accumulated oxygen deficit appeared unaffected by the higher power used at the posttraining test, suggesting that this entity is able to distinguish between aerobic and anaerobic energy release at different powers. The alternative interpretation, that there was a change in the anaerobic capacity but that this change was obscured by a bias in the accumulated oxygen deficit, cannot be ruled out, but the findings here suggest that the latter interpretation is less likely.

The high-intensity intermittent training in experiment 2 improved anaerobic capacity by 28%. Medbø and Burgers (5) reported that 6 wk of the intermittent training (their group B) increased the anaerobic capacity of untrained men by 16%. Since there are no clear differences in exercise intensity, exercise duration, and number of exercise bouts between the two studies, this quantitative difference in improving anaerobic capacity is probably explained by the difference between the two studies in magnitude of the anaerobic energy release during each training session. The peak blood lactate concentration after each training session in the previous study (5) was 69% of the peak blood lactate concentration after the 2-min exhaustive running. Therefore, anaerobic metabolism, and especially the lactate producing system, was probably not taxed maximally. In contrast, the peak blood lactate concentration after the intermittent training in this investigation was not significantly different from the value observed after the anaerobic capacity test that recruited anaerobic energy releasing systems maximally. In addition, our subjects exercised to exhaustion, but in the previous study, the subjects' rating of perceived exertion (1) was only 15 ("hard"). This difference may also reflect the recruited level of anaerobic energy release. Therefore, these results support our hypothesis that the higher the anaerobic energy release during each training session the higher the increase in anaerobic capacity after a training period.

In addition to anaerobic capacity, the intermittent training increased $\dot{V}O_{2\max}$ significantly in experiment 2. This is to our knowledge the first study to demonstrate an increase in both anaerobic capacity and maximal aerobic power. It should be emphasized that during the last part of each training session the oxygen uptake almost equaled each subject's maximal oxygen uptake (data not shown). High-intensity intermittent training is a very potent means of increasing maximal oxygen uptake (2). It is interesting to note that the increase in the maximal oxygen uptake

that we found is almost identical to that expected for intermittent training by Fox (2). Consequently, the protocol used in training in experiment 2 may be optimal with respect to improving both the aerobic and the anaerobic energy releasing systems.

The intensive bicycle training may have affected the efficiency of cycling, meaning that the relationship between power and $\dot{V}O_2$ may have changed. This change may affect the measurement of anaerobic capacity because the accumulated oxygen deficit is a calculated entity assuming a constant mechanical efficiency. However, our subjects were sufficiently familiar with bicycle exercise through repeated testing and experiments so that the relationship between the steady state oxygen uptake and power did not change during the training periods. Therefore, the pre- and posttraining data of the accumulated oxygen deficit should be comparable.

In summary, this investigation demonstrated that 6 wk of moderate-intensity endurance training did not affect anaerobic capacity but that 6 wk of high-intensity intermittent training (20 s exercise, 10 s rest; intensity $170\% \cdot \dot{V}O_{2max}$) may improve both anaerobic capacity and $\dot{V}O_{2max}$ simultaneously.

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