THE EFFECT OF CESSATION OF TRAINING ON SELECTED PHYSIOLOGICAL AND PERFORMANCE VARIABLES IN FEMALE RUNNERS

By

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We accept this thesis as conforming to the required standard

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ABSTRACT

The purpose of this study was to examine the effect of 15 days of cessation of training on maximal oxygen consumption ($\dot{V}O_2max$) and selected physiological functions as they relate to $\dot{V}O_2max$ in female middle distance runners with a training base of at least two years. Fifteen female runners participated in the study (20 (3) yrs, 57.3 (1.4) kg, 163.7 (1.0) cm, and 49.6 (0.8) $mL \cdot kg^{-1} \cdot min^{-1}$). One group ceased their training program for 15 days (CT, n=7) and one group maintained their training program (MT, n=8). The subjects were tested at day 0, 5, 10, and 15 to monitor body mass (kg), $\dot{V}O_2max$ ($mL \cdot kg^{-1} \cdot min^{-1}$), maximal heart rate (HR, bpm), cardiac output ($\dot{Q}$, $L \cdot min^{-1}$), stroke volume (SV, $mL \cdot beat^{-1}$), arterial venous oxygen difference (a-v O_2 difference, $mL \cdot 100mL^{-1}$) and plasma concentration (PC, $mL \cdot 100mL^{-1}$). Body composition (SOS, mm) was examined at day 0 and day 15 and a running performance task (2400 m) was tested before day 0 and at day 16 of the study. $\dot{Q}$, SV, and a-v O_2 difference were analysed via the CO_2 rebreathing method as described by Jones (1988). No significant changes in any of the variables were found at day 5. Ten days of no training resulted in a significant decrement in $\dot{V}O_2max$ of 3.8 $mL \cdot kg^{-1} \cdot min^{-1}$ in group CT. After fifteen days of no training, $\dot{V}O_2max$ was still significantly lower ($p < 0.05$) in comparison to the MT group. No significant changes were found in body mass, body composition, $\dot{Q}$, SV, HRmax, a-v O_2 difference, and PC after 15 days of no training. Running times increased significantly by 18.4 (7.1) seconds after 15 days which corresponded to the approximate 7.8% mean decrease in $\dot{V}O_2max$ $mL \cdot kg^{-1} \cdot min^{-1}$. These findings suggest that in female runners, performance decrements found after 15 days of CT are likely due to declines in $\dot{V}O_2max$.

Examiners:

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TABLE OF CONTENTS

List of Tables

List of Figures

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Dedication

To my parents Dorothy, and Don and Sylvia, and to my loving sister Siobhan. You all taught me to believe in myself, and to love and to cherish the good things around me. And to my friends, for always being there.

INTRODUCTION

Physiological adaptations to endurance training include increased maximal oxygen consumption ($\dot{V}O_2max$), increased cardiac output ($\dot{Q}$), and increased stroke volume (SV) (Saltin & Rowell, 1980). These increases contribute to improved performance by enhancing the delivery to and utilization of oxygen by the muscles. Exercise training programs are designed to promote changes in the aerobic energy system that will enhance performance. Factors such as injury, minor illness, scheduling, or climatic circumstances may cause interruptions of a few days to a few weeks in the training regimens of athletes. Enhanced physiological functions in response to training are not permanent and if the training stimulus is removed, function will decline. Research findings on the effects of short interruptions in training (2 weeks) are equivocal. Elite athletes with a training base of up to five years may experience some decreases in various physiological functions in as little as five days of no training; while some functions remain above pretrained levels (Costill, Fink, Hargreaves, King, Thomas, & Fielding, 1985; Houston ,Bentzen & Larsen, 1979). Due to equivocal findings in the literature and for the purpose of this study, breaks in training will be referred to as cessation of training. The amount of decrement in physiological function during cessation of training is important to both coach and athlete. If there are no changes in a short period of cessation of training then it may be beneficial for an athlete to cease training for a few days to a week to avoid fatigue or to aid healing of minor injuries.

Saltin, Blomqvist, Mitchell, Johnson, Wildenthal, and Chapman (1968) were some of the first investigators to examine the effects of cessation of training on exercise. Trained and untrained subjects were examined after 20 days of bed rest and $\dot{V}O_2max$ was found to decrease in both groups. Decreases in $\dot{V}O_2max$ were attributed to concurrent decreases in $\dot{Q}$, SV, and plasma volume (PV). When training programs were resumed, the previously trained athletes values all returned to the levels observed before bed rest faster than the untrained group. Although athletes today are unlikely to experience extended periods of bed rest even during minor illness or when inflicted with minor injuries, Saltin and co workers (1968) introduced the concern of how the physiological function of trained athletes would respond to cessation of training while maintaining a regular sedentary lifestyle.

Energy required for endurance events is provided primarily by aerobic metabolism and thus the success of endurance performance is related to the ability to transport oxygen to the exercising muscles and the muscles ability to extract and utilize the oxygen (Fox, Bowers & Foss, 1989; Hartley, 1992). The rate at which energy is supplied by aerobic metabolism is called aerobic power (Thoden , 1991) and maximal aerobic power ($\dot{V}O_2max$) is the maximal rate at which oxygen can be consumed and utilized by the tissues (Fox et al., 1989; Thoden, 1991). Athletes who perform activity for longer than two minutes generally have higher maximal oxygen uptake than those performing for lesser amounts of time, therefore the highest relative values for $\dot{V}O_2max$ are found in sports such as middle distance running (Hartley, 1992; Thoden, 1991). $\dot{V}O_2max$ is dependent on the ability of the tissues to utilize the oxygen - peripheral components, and the transport of oxygen to the tissue - central components (Hartley, 1992). Genetic factors also may have a limiting effect on maximal aerobic power, but to what extent is unclear (Thoden, 1991).

Maximal aerobic power increases with endurance training. If the training stimulus is removed, as when training is ceased, it would be expected that $\dot{V}O_2$ max would decrease and performances dependent on maximal aerobic power would also be expected to decrease (Henriksson & Reitman, 1977). Decreases in maximal oxygen consumption have been shown to be related to the central system as indicated by changes in cardiac output ($\dot{Q}$), stroke volume (SV) and blood volume (Coyle, Martin, Bloomfield, Lowry & Holloszy, 1985; Neufer, 1989). Significant decreases in $\dot{V}O_2$max of 6% to 7% within the first two weeks of cessation of training have coincided with decreases of 12% in stroke volume (Coyle, Martin, Sinacore, Joyner, Hagberg, & Holloszy, 1984; Coyle, Hemmert, & Coggan, 1986).

Reductions in $\dot{V}O_2$max during initial periods of cessation of training appear to be directly related to the changes in SV (Coyle et al., 1984) and decreases in blood volume. Coyle et al. (1986) found an increase in $\dot{V}O_2$max and SV when decreases in blood volumes following cessation of training were artificially elevated to trained levels. Coyle et al. (1984) also found respective decreases of 8% and 7% in $\dot{Q}$ and $\dot{V}O_2$max after 21 days of no training that coincided with a 9% decrease in a-v O_2 difference. These results indicated that decreases in cardiovascular function following a few weeks of cessation of training may be largely due to a decline in blood volume when training is stopped and imply that decrements in performance which are evident after a short period of cessation of training may be due to changes in these cardiovascular (central) components of $\dot{V}O_2$max.

The most rapid decreases in the oxygen transport system occur at the peripheral level. For example, oxidative enzymes of the muscles may decrease up to 50% of trained levels after one week of no training (Costill et al., 1985), however, these decreases do not coincide numerically with the decreases in $\dot{V}O_2$max which may be as little as 4% within

the first 15 days of CT (Houston et al., 1979). Glycolytic enzyme activity has been shown to be maintained for the first few weeks of cessation of training, while oxidative enzyme activity decreases quickly in the first two weeks. Decreases in succinate dehydrogenase (SDH) are reported after 4 and 6 weeks (Green, Thomson, Daub & Ranney, 1980; Henriksson & Reitman, 1977; Houston et al., 1979), while phosphofructokinase (PFK) remained high until 18 weeks of cessation of training (Costill et al., 1985; Simoneau, Lortie, Boulay, Marcotte, Thibeault, & Bouchard, 1987). Enzymatic decreases do not correlate with those in $\dot{V}O_2$max; for example it has been shown that after 12 weeks of cessation of training decrements in enzyme activity may no longer be evident (for example when activity reaches a new stable level) while $\dot{V}O_2$ max may continue to decrease to 14% below the trained level (Coyle et al., 1984; Green et al., 1980; Henriksson & Reitman, 1977; Houston et al., 1979). Although oxidative enzyme adaptations are correlated with both increases and decreases in $\dot{V}O_2$ max, $\dot{V}O_2$max is not dependent on these adaptations (Houston et al., 1979).

The effects of endurance training are fragile and will change during periods of no training. Changes in $\dot{V}O_2$max due to cessation of training are equivocal; for example decrements between 4-7% after 14-21 days of cessation of training have been reported (Coyle et al., 1984; Houston et al., 1979) while others have found no decrements until after 6 weeks of cessation of training (Ready & Quinney, 1982). The decreases in $\dot{V}O_2$max have been attributed to combinations of changes in stroke volume, blood volume, a-v O2 difference and muscle capillarization (both central and peripheral components) (Coyle et al., 1984 & 1986; Neufer, 1989). The rate and magnitude of change in central and peripheral physiological functions and the effects on sport performance are equivocal.

Statement of the problem

The purpose of this study was to examine the effects of 15 days of cessation of training on both the magnitude and rate of change in $\dot{V}O_2$max and in selected physiological variables that relate to $\dot{V}O_2$max in female middle distance runners with a training base of at least two years, and to determine the effect of the cessation of training period on their running performance.

Research questions

1) What is the rate and magnitude of decline in $\dot{V}O_2$max and selected physiological variables which contribute to $\dot{V}O_2$max of middle distance runners over 15 days of cessation of training?

2) If $\dot{V}O_2$max changes after 15 days of cessation of training, do changes occur centrally or peripherally?

3) Does fifteen days of cessation of training affect performance of a running task, and are the physiological changes associated with changes in performance?

Operational definitions

Cessation of training: The period in which the athletes refrain from any formal training. Acceptable activities are general everyday activities, for example walking and lifting.

Maintenance of training: The period in which athletes maintain their regular training program under the supervision of the coach. No alterations are made at this time to enhance performance.

RESEARCH METHODS

Subjects

Fifteen healthy female middle distance runners ($\dot{V}O_2max$ = 49.6 (0.8) $mL \cdot kg^{-1} \cdot min^{-1}$,2.84 (0.32) $L \cdot min^{-1}$, age = 20 (3) years) volunteered to take part in this study. Eight were junior varsity runners from the University of Victoria Vikes Racing Team; four were members of the Victoria Running Club; one was from the Royal Roads Military School Racing Team; and two were competitive runners from the School of Physical Education at the University of Victoria and were training independently. All had been training regularly for 2-6 years. The subjects were randomly assigned to two groups; a maintenance group (MT, n=8) which maintained their training for 15 days and a cessation of training group (CT, n=7) which ceased their training program for 15 days. Prior to the study, training averaged 5-6 days per week and approximately 30 - 45 miles per week for at least 18 weeks. Training regimens combined continuous and interval programs working from 70% to 90% of $\dot{V}O_2max$. Prior to testing all subjects were oriented to the lab, given verbal and written information about the demands, requirements and test protocols before agreeing to participate and signing an informed consent (Appendix A).

Physiological Testing

Subjects reported to the University of Victoria Sport and Fitness Center (SFC) five times during the course of the study. The first testing session was for lab familiarization and a treadmill test for assessment of submaximal loads for use in the $\dot{V}O_2max$/cardiac output ($\dot{Q}$) examination. The second testing date was the first of four assessments for hematology, $\dot{V}O_2max$ and $\dot{Q}$. Testing sessions were every five days (day 0 , day 5, day 10, and day 15). The CT group was asked to refrain from any physical activity for the

fifteen days of the study and the MT group continued their training programs under the supervision of their coaches. Anthropometric measures of height and body mass, and body fat from eight skinfold sites were taken at days 0 and 15 (Ross & Marfell-Jones, 1991). A coach-supervised 2400 meter time trial was done 1 week prior to the study and on day 16 of the study to examine any changes in sport specific performance. A time line of data collection is provided in Figure 1.

Table 1
Physiological characteristics of female middle distance runners over 15 day period; cessation of training group (CT, n=7) and maintenance of training (MT, n=8).

	CT		MT	
	Mean (SE)	Range	Mean (SE)	Range
Age (years)	21 (1.0)	19-23	19 (1.0)	16-25
Height (cm)	162.9 (1.1)	158.6-166.3	164.3 (1.7)	155.0-168.5
Body mass (kg)	57.5 (1.5)	52.6-64.1	57.1 (2.3)	48.1-67.1
SOS (mm)	89.4 (6.2)	73.8-115.0	80.8 (5.7)	57.1-109.3
$\dot{V}O_2max$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	49.7 (1.1)	46.0-54.4	49.5 (1.2)	43.9-53.7
$\dot{V}O_2max$ ($L \cdot min^{-1}$)	2.85 (0.09)	2.64-3.26	2.83 (0.14)	2.29-3.42

Figure 1. Time line of data collection for CT and MT.

Lab Orientaion	Performance test	Day 0	Day 5	Day 10	Day 15	Performance test
-intro to lab and expectations of study -$\dot{V}O_2$max test	-2400m (n=13) -400m (n=2)	-resting blood samples -body compositon -$\dot{Q}$ -$\dot{V}O_2$max	-resting blood samples -$\dot{Q}$ -$\dot{V}O_2$max	-resting blood samples -$\dot{Q}$ -$\dot{V}O_2$max	-resting blood samples -body compositon -$\dot{Q}$ -$\dot{V}O_2$max	-2400m (n=13) -400m (n=2)

$\dot{Q}$ = cardiac output test during the $\dot{V}O_2$max protocol.

Anthropometric measurements

Height, body mass, and the sum of eight skinfolds: triceps, biceps, subscapular, supra iliac, supra spinale, abdomen, thigh and calf, (Ross & Marfell-Jones, 1991) were taken at day 0 and day 15 by the same investigator.

Hematological analysis

Blood samples were taken by a trained technician on days 0, 5, 10 and 15. Subjects arrived at the SFC between 6:00 am and 9:00 am in a rested state (for example: no large breakfast and not riding bike to lab). After anthropometric measurements, subjects had seated rest for 15 minutes and 2 mL of blood were drawn via venipuncture from an antecubital vein. Blood samples were analysed for hemoglobin and hematocrit with a Coulter^{R} STKS analyzer. Plasma concentrations (PC) were calculated using hemoglobin and hematocrit values as described by Dill and Costill (1974).

Exercise testing: cardiac output ($\dot{Q}$) and $\dot{V}O_2max$

$\dot{V}O_2max$ was determined on a treadmill using a continuous, incremental protocol (Thoden, 1991). The protocol consisted of a two minute warm up at a submaximal load. Speed was then increased by 0.22 $m \cdot s^{-1}$ and maintained for 4-6 minutes to obtain steady state required for the CO_2 rebreathing procedure. Once the rebreathing procedure was complete, the subject continued running at the steady state speed for one minute. Speed was then increased (by approximately 0.44 $m \cdot s^{-1}$) to elicit a pace approximate to that of the subjects race pace. This was testing speed for determining $\dot{V}O_2max$. The subject ran for one minute at the testing speed after which speed was maintained and percent grade was increased 2% every minute until $\dot{V}O_2max$ was obtained. $\dot{V}O_2max$ was achieved when the following criteria were met: a plateau or decrease in oxygen consumption

despite an increase in percent grade of the treadmill; a respiratory quotient which exceeded 1.1; attainment of predicted maximal heart rate; or volitional fatigue. All subjects met the criteria and $\dot{V}O_2max$ was expressed as $mL \cdot kg^{-1} \cdot min^{-1}$ and $L \cdot min^{-1}$.

During the exercise test expired gases were collected by a two-way Rudolph valve with a breath-by-breath connection. Gases were analysed every 15 seconds by a Horizon metabolic measurement cart(MMC) (Sensormedics). The cart was calibrated before and after each test using primary standard gases. Heart rate was monitored every 30 seconds to note any changes within loads throughout the test using a Polar Vantage XL heart rate monitor.

Cardiac Output ($\dot{Q}$)

Cardiac output was determined during the 4-6 minute submaximal (approximately 60% of $\dot{V}O_2max$) load of the $\dot{V}O_2max$ protocol. This load was chosen since stroke volume is known to increase during submaximal exercise to a heart rate of approximately 120 beats per minute (about 25-40% of maximal heart rate). Once this level is reached there is little or no increase in SV with further increases in workload or heart rate (Coyle et al., 1984; Jones, 1988). Since direct measurement of maximal cardiac output is difficult to obtain, data from this load allowed for the calculation of maximal cardiac output by multiplying maximal stroke volume by maximal heart rate (SV · HRmax) (Coyle et al., 1984).

Once a steady state was reached , as indicated by a constant $\dot{V}_E \pm 1.0\ L \cdot min^{-1}$, a constant $\dot{V}O_2$ within $\pm 0.1\ L \cdot min^{-1}$, and an end tidal CO_2 ($P_{ET}CO_2$) which was equal to or 1.0 mmHg less than the previous reading, the rebreathing procedure was implemented. All subjects reached steady state. The subject would hyperventilate for 20 seconds from a 5L bag containing a mixture of 11%-13% CO_2 and O_2 balance (Jones, 1988; Bhambhani,

Norris, & Bell, 1994). The CO_2 mixture used was determined from a table of $P_{ET}CO2$ and $\dot{V}O_2$ $L \cdot min^{-1}$ values as described in Jones (1988).

The Advanced Exercise Testing Program supplied with the Horizon MMC was used to calculate cardiac output from the data collected during the rebreathing procedure. Values were applied to the indirect Fick equation $\dot{Q} = VCO_2 / C_vCO_2 - C_aCO_2$: where $\dot{Q}$ is cardiac output $L \cdot min^{-1}$; VCO_2 is the expired CO_2 content ($L \cdot min^{-1}$ STPD); C_vCO_2 is the CO_2 content of mixed venous blood ; and C_aCO_2 is the CO_2 content of arterial blood. VCO_2 was measured during steady state prior the rebreathing procedure. $P_{ET}CO2$ (from end tidal F_ECO2) was measured by the CO_2 gas analyzers and then P_aCO_2 (alveolar CO_2 pressure mmHg) was calculated. From P_aCO_2, the C_aCO_2 was derived from the equation $\log_e C_{CO_2} = [0.396 \times \log_e P_{CO_2}]$ (Jones, 1988).

C_vCO_2 was derived from P_vCO_2 which was estimated by extrapolation of the rise in $P_{ET}CO2$ during the rebreathing procedure. As the gas from the rebreathing bag mixed with the alveolar gas, PCO_2 plateaued (a difference of < 1 mmHg in the first 8 seconds), indicating that P_aCO_2 equaled P_vCO_2. The pressure (mmHg) of this equilibrium was taken as P_vCO_2 and C_vCO_2 was calculated from the equation used for C_aCO_2 (Jones, 1988). When gas concentrations are applied to the Fick equation to derive Q for the workload, the software incorporates corrections for blood pH and hemoglobin as described by Jones (1988) and Bhambhani et al.(1994). Stroke volume was calculated from the ratio of $\dot{Q}$ and HR, and arterio-venous oxygen difference (a-v O_2 difference) was calculated from the ratio of $\dot{V}O_2$ and $\dot{Q}$.

A review by Marks, Katch, Rocchini, Beekman and Rosenthal (1985) on the validity and reliability of cardiac output measured by CO_2 rebreathing, reported a validity coefficient range of 0.86 - 0.96 against the direct Fick method. Bhambhani et al. (1994) using the same method as the present study found a reliability coefficient of 0.89.

$\dot{V}O_2max$ and $\dot{Q}$ were examined during the same testing procedure to reduce the number of exercise tests the subjects were to perform. By having only one exercise test of 10-14 minutes long, the possibility of a training effect of testing for the CT group was decreased.

STATISTICAL ANALYSIS

A two by four factorial analysis of variance with repeated measures was used to determine if cessation of training produced group, time, and group interaction changes in the dependent variables. A Tukey's post-hoc analysis was used to identify means that differed. Significance was set at $p < 0.05$. All analyses were performed using SPSS PC program for IBM computers.

RESULTS

The mean body mass (kg) and body composition (SOS mm) did not change significantly over the fifteen days for either CT or MT groups (Table 2).

Cessation of training did not produce any significant changes after 5 days in mean $\dot{V}O_2max$ ($mL \cdot kg^{-1} \cdot min^{-1}$), however significant changes were found after 10 and 15 days of no training (Fig. 2). Ten days of CT produced a mean decrement of 3.8 $mL \cdot kg^{-1} \cdot min^{-1}$, and at fifteen days of CT $\dot{V}O_2max$ was still significantly different from the MT values.

For both CT and MT no significant changes were noted from day 0 to day 5, 10 and 15 for the following variables: $\dot{Q}max$ values for CT were 18.2 (0.6), 19.0 (0.6), 18.3 (0.9), and 18.5 (0.7) $L \cdot min^{-1}$, and for MT 19.0(1.3), 20.1(1.4) 19.8(1.1) and 19.2(0.9) $L \cdot min^{-1}$. Maximal SV values for CT were 93.5 (3.4), 96.7 (3.9), 92.7 (5.4), and 93.7 (4.5) $mL \cdot beat^{-1}$, and for MT were 96.7(7.1), 103.4(7.5), 102.9(5.5) and 98.9(5.5) $mL \cdot beat^{-1}$. CT values for HRmax were 195 (2), 197 (3), 198 (3), and 199 (3) $beats \cdot min^{-1}$, and for MT were 197(4), 195(4), 198(4) and 195(4) $beats \cdot min^{-1}$. CT values for a-v O_2 difference were 15.7 (0.3), 15.5 (0.3), 14.6 (0.4) and 15.8 (0.7) $mL \cdot 100mL^{-1}$, and for MT were 15.1(0.6), 15.1(0.7), 15.3(0.6) and 16.2(0.7)$mL \cdot 100mL^{-1}$. For PC, CT values were 61.0 (0.7), 61.0 (1.0), 61.5 (1.2) and 61.4 (1.3) $mL \cdot 100mL^{-1}$, for MT values were 59.6(0.4), 59.2(0.7), 60.2(0.4) and 59.6(0.6) $mL \cdot 100mL^{-1}$ (Fig. 3, 4, 5, 6, and Table. 3, respectively).

Neither central variables $\dot{Q}$max ($L \cdot min^{-1}$), maximal SV ($mL \cdot beat^{-1}$), HRmax (bpm), PC ($mL \cdot 100mL^{-1}$), nor peripheral variables a-v O_2 difference ($mL \cdot 100mL^{-1}$) changed significantly over the fifteen day period (Fig. 3, 4, 5, Table 3, and Fig 6 respectively).

Mean performance times of the running task were significantly slower after fifteen days of no training (Table 4). The 4 % slower running performance times coincided with the 7.8 % decrease in $\dot{V}O_2$max $mL \cdot kg^{-1} \cdot min^{-1}$.

Table 2

Mean (SE) body mass (kg) and body composition (SOS mm) over 15 days for CT and MT.

	Body mass (kg)		SOS (mm)	
	CT	MT	CT	MT
Day 0	57.5(1.5)	57.1(2.3)	89.4(6.2)	80.8(5.7)
Day 5	57.8(1.3)	57.2(2.7)		
Day 10	57.9(2.2)	58.0(2.2)		
Day 15	57.8(2.1)	56.6(2.1)	88.5 (5.6)	77.5(4.9)

Figure 2. Mean (SE) maximal oxygen consumption ($mL \cdot kg^{-1} \cdot min^{-1}$) for CT and MT groups at days 0, 5, 10, and 15 days. * CT values significantly different from day 0 and from MT ($p < 0.05$), a = significant difference between days 5-10, b = significant difference between days 10-15.

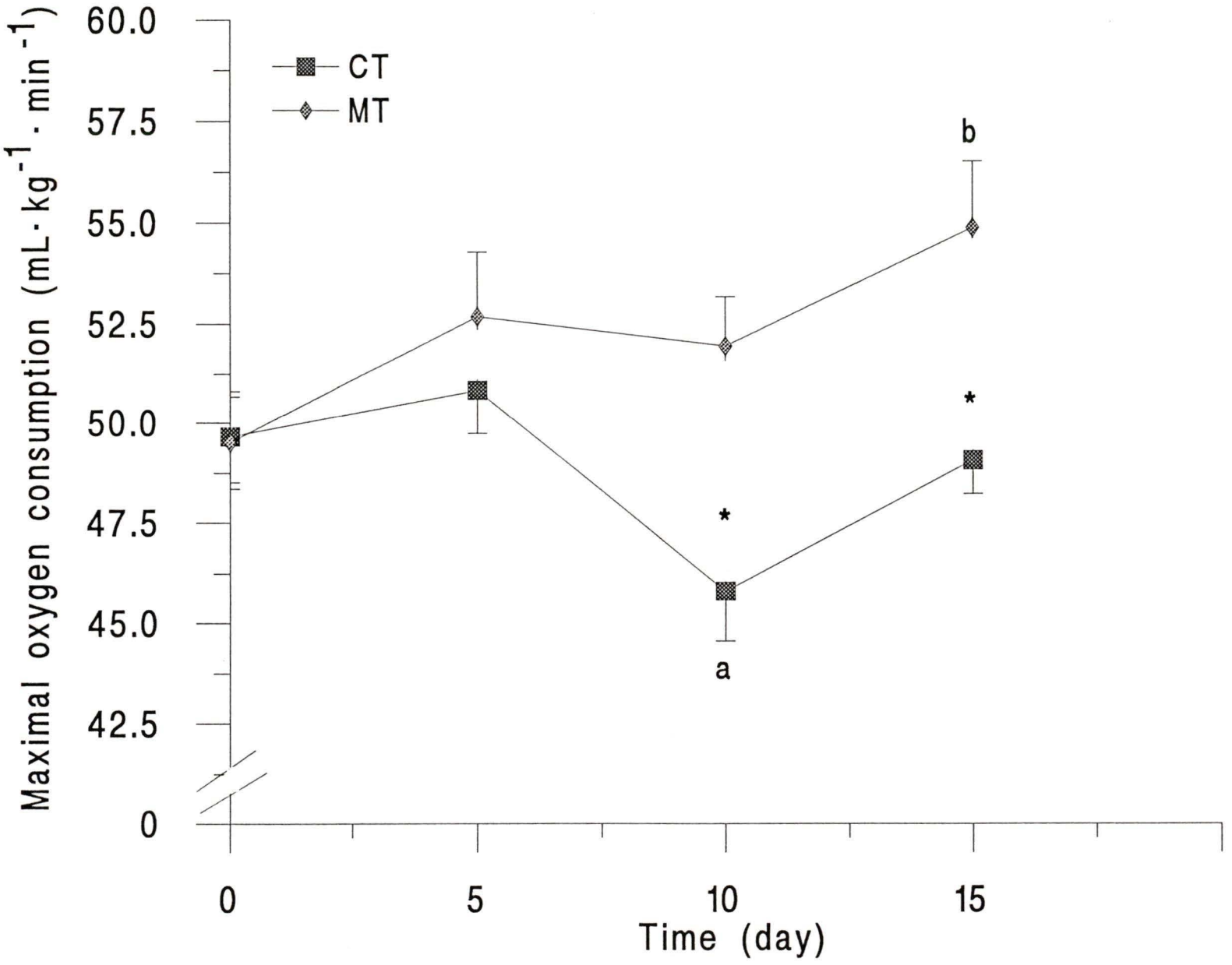
Maximal oxygen consumption (mL · kg-1 · min-1)
60.0
57.5
55.0
52.5
50.0
47.5
45.0
42.5
0
CT
MT
b
a
*
*
0
5
10
15
Time (day)

Figure 3. Mean (SE) maximal cardiac output ($L \cdot min^{-1}$) for CT and MT at days 0, 5, 10, and 15.

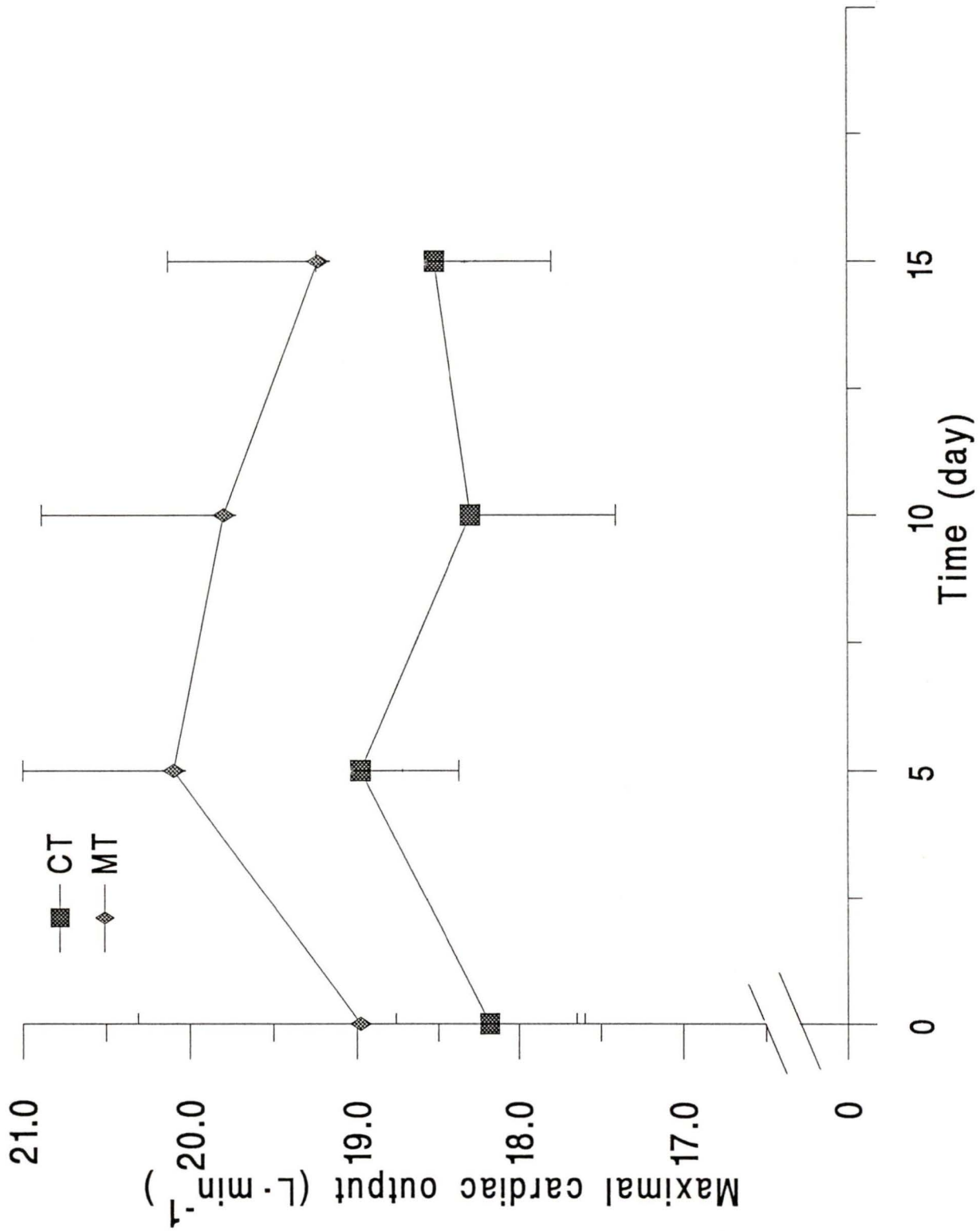
Maximal cardiac output (L·min-1)
21.0
20.0
19.0
18.0
17.0
0
0
5
10
15
Time (day)
CT
MT

Figure 4. Mean (SE) maximal stroke volume ($mL \cdot beat^{-1}$) for CT and MT at days 0, 5, 10, and 15.

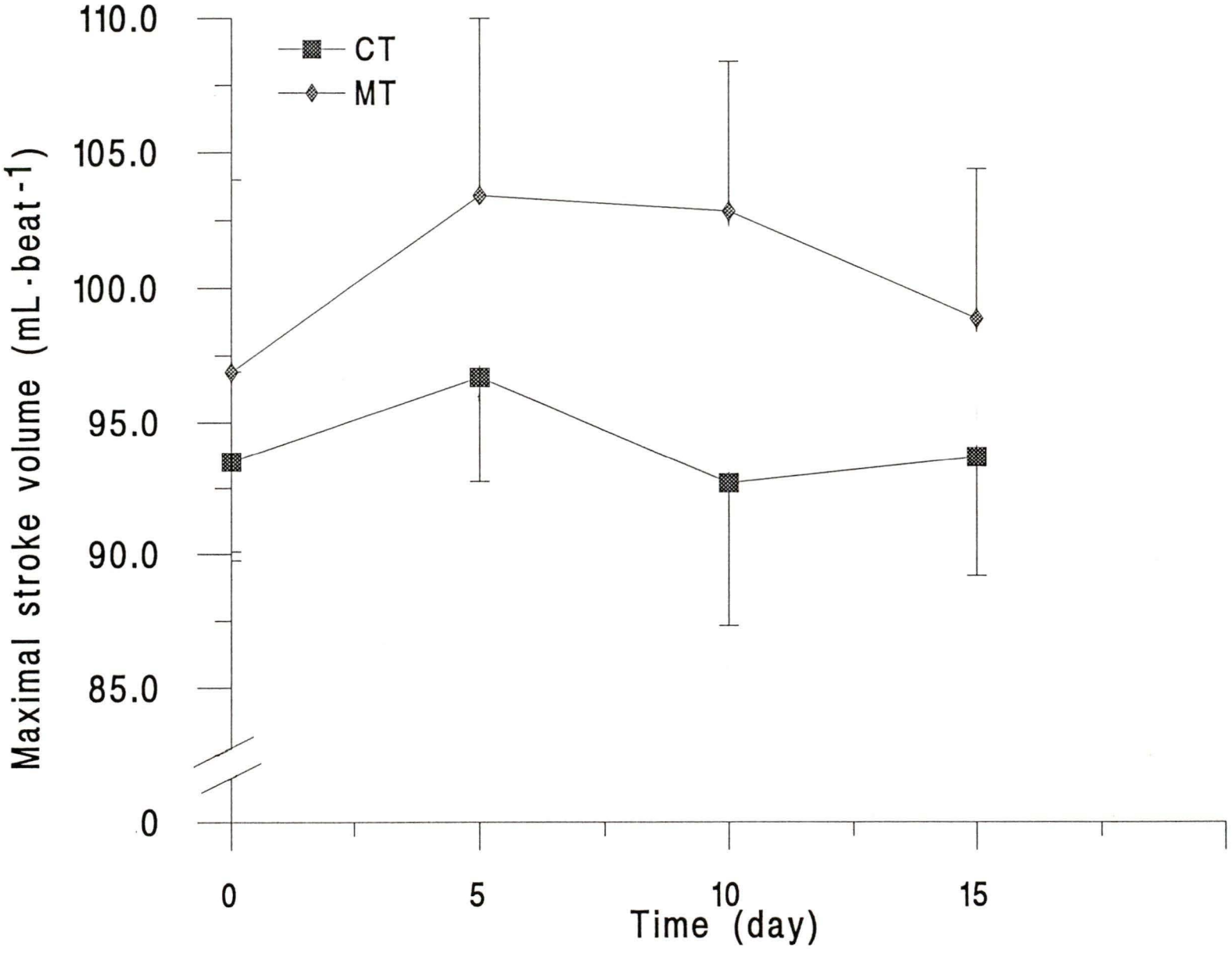
110.0
105.0
100.0
95.0
90.0
85.0
0
CT
MT
Maximal stroke volume (mL·beat-1)
0
5
10
15
Time (day)

Figure 5. Mean (SE) maximum heart rate (beats·min^{-1}) for CT and MT at days 0, 5, 10, and 15.

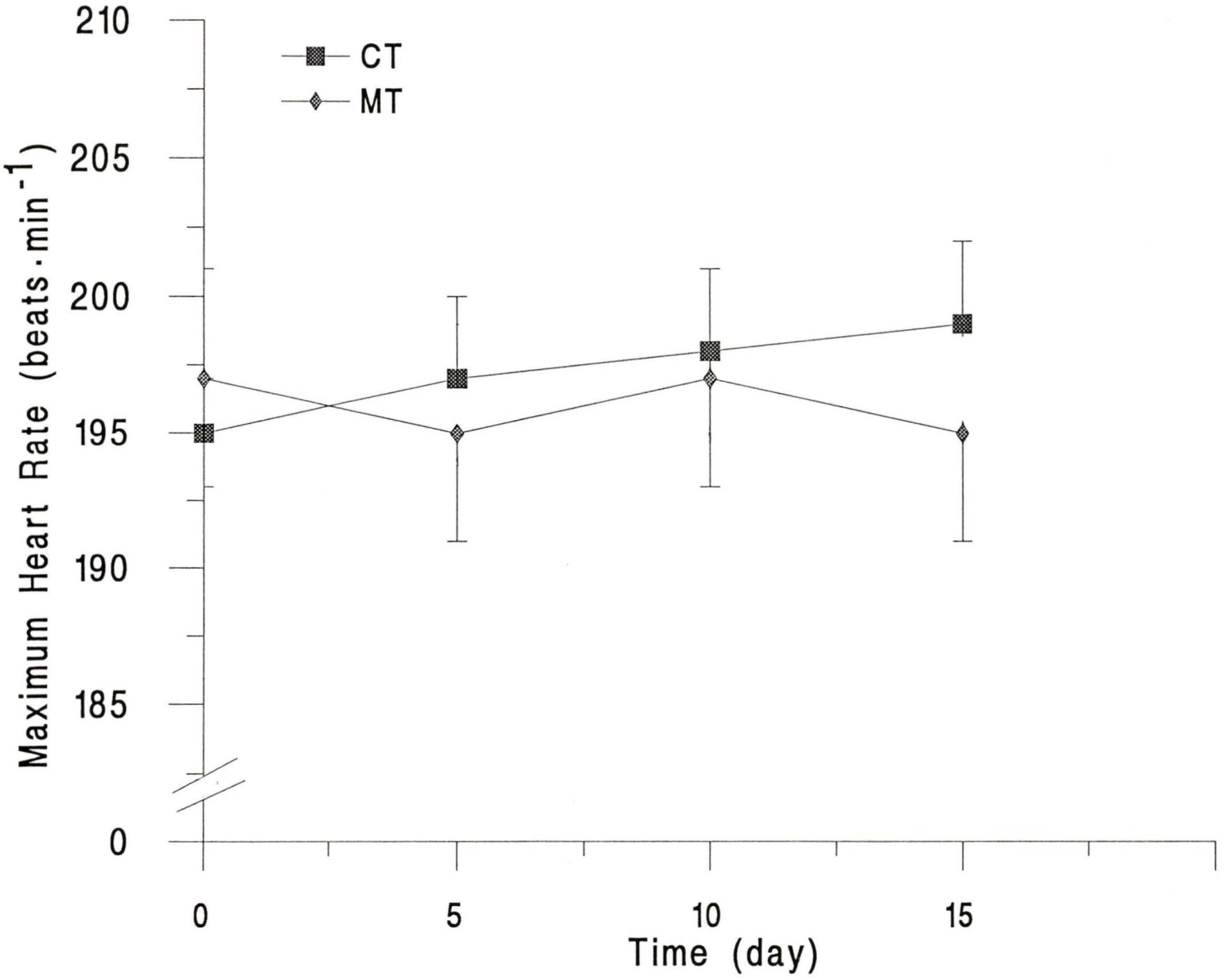
CT
MT
Maximum Heart Rate (beats·min-1)
210
205
200
195
190
185
0
0
5
10
15
Time (day)

Figure 6. Mean (SE) maximal arterio-venous oxygen difference (a-v O_2 difference) for CT and MT at days 0, 5, 10, and 15.

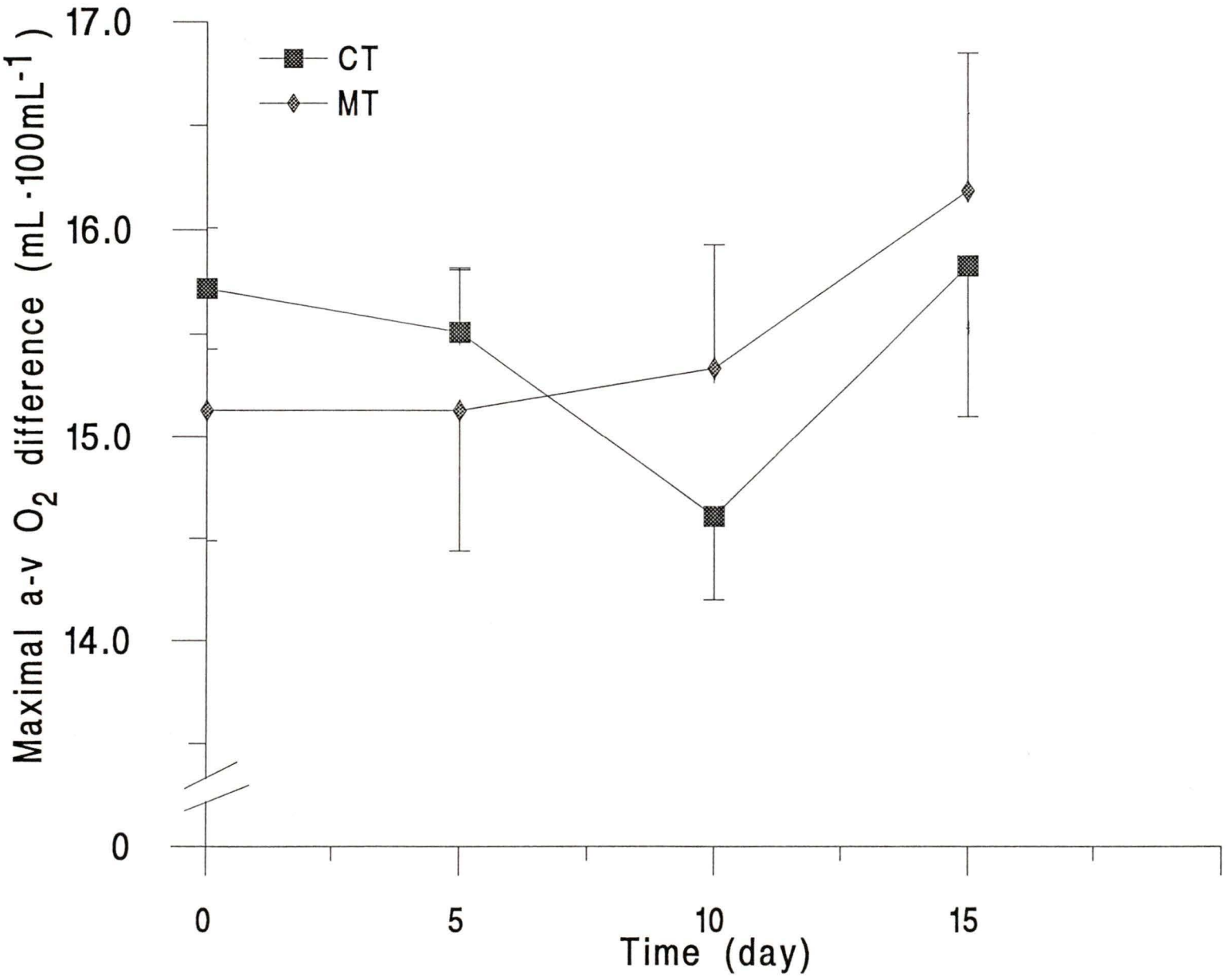
17.0
16.0
15.0
14.0
0
CT
MT
Maximal a-v O_2 difference (mL·100mL^{-1})
0
5
10
15
Time (day)

Table 3

Mean (SE) plasma concentration ($mL \cdot 100mL^{-1}$) for CT and MT at day 0, 5, 10, and 15.

	CT	MT
Day 0	61.0 (0.7)	59.6 (0.4)
Day 5	61.0 (1.0)	59.2 (0.7)
Day 10	61.5 (1.2)	60.2 (0.4)
Day 15	61.4 (1.3)	59.6 (0.6)

Table 4

Mean (SE) 2400m running performance times (s) before and after the study for CT and MT. *Significantly different from pre test ($p < 0.05$).

	Pre	Post	Change (s)
CT (s)	541.5 (9.4)	563.0 (6.9)	+18.4 (7.1) *
MT (s)	553.7 (15.2)	551.5 (12.8)	-2.0 (2.8)

DISCUSSION

For the present study, female middle distance runners were examined to investigate the effects of cessation of training for 15 days on: 1) the rate and magnitude of change in $\dot{V}O_2max$ and the physiological variables which contribute to $\dot{V}O_2max$, 2) whether decrements in these variables occurred centrally or peripherally, and 3) how cessation of training affected performance of a running task and if any changes in the running task were associated with changes in physiological variables.

Rate and magnitude of decline in physiological function during CT

Research has examined the effects of cessation of training on physiological variables as they relate to $\dot{V}O_2max$ and performance. Cessation of training of three weeks and more results in decrements in $\dot{V}O_2max$ and physiological variables contributing to $\dot{V}O_2max$. Drinkwater and Horvath (1972) found decrements of approximately 7 $mL \cdot kg^{-1} \cdot min^{-1}$ in female track athletes (14-17 years) after 3 months of cessation of training. Smith and Stransky (1976) found that $\dot{V}O_2max$ approached pretrained levels in 18-25 year old females after three weeks of cessation of training. Decrements in physiological variables contributing to $\dot{V}O_2max$ after 3 weeks cessation of training are well supported (Coyle, Hemmert, & Coggan, 1986; Coyle, Martin, Sinacore, Joyner, Hagberg, & Holloszy, 1984; Fringer & Stull, 1974; Green, Thomson, Daub, & Ranney, 1980; Henricksson & Reitman, 1977). Discrepancies exist in how soon after the onset of cessation of training that decrements occur. Equivocal results are found especially when focusing on well-trained athletes (a training base of a number of years) who stop training

for one to two weeks (1 - 15 days). Most studies have examined endurance trained male runners (long and middle distance), cyclists and swimmers with training bases ranging from 2 to 10 years and have often pooled data from the different athletic events. The present study examined female middle distance runners with 2-5 years of training experience and ages ranging from 16-25, and $\dot{V}O_2max$ values ranging from 43.9 - 53.7 $mL \cdot kg^{-1} \cdot min^{-1}$.

Moore, Thacker, Kelley, Musch, Sinoway, Foster, and Dickinson (1987) suggested that short term cessation of training (1-2 weeks) results in decrements in $\dot{V}O_2max$ and contributing variables may depend on the population studied. They also suggested that rapid declines in aerobic power are found in subjects who had a large initial aerobic power (> 62 $mL \cdot kg^{-1} \cdot min^{-1}$ Coyle et al., 1984; Houston, Bentzen & Larsen, 1978) and an extensive training background (> 2 years). However, Moore et al. (1987) also noted that three weeks of no training did not produce significant changes in subjects with a lower initial $\dot{V}O_2max$ (< 56 $mL \cdot kg^{-1} \cdot min^{-1}$; Henriksson & Reitman, 1977). In the present study, no significant changes in mean body mass (kg), and body composition (SOS mm) (Table 2) occurred after 15 days of cessation of training. However, maximal oxygen consumption ($\dot{V}O_2max$) decreased significantly ($p<0.05$) by 3.8 $mL \cdot kg^{-1} \cdot min^{-1}$ at 10 days in group CT and was still significantly different from trained and MT group values at 15 days (Fig. 2). Initial $\dot{V}O_2max$ values were 49.7(1.2) $mL \cdot kg^{-1} \cdot min^{-1}$, and training experience was 2-5 years. Houmard, Hortobagyi, Johns, Bruno, Nute, Shinebarger, and Welborn (1992) found similar results to the present study when examining males and females of the same age group with 2-8 years of training experience. Initial $\dot{V}O_2max$ values were higher than in the present study (61.6 (2.0) $mL \cdot kg^{-1} \cdot min^{-1}$) and decreased significantly (2.9 $mL \cdot kg^{-1} \cdot min^{-1}$) after 14 days of no training. Coyle et al., (1984) pooled results from male and female athletes and found a decrease in $\dot{V}O_2max$ of approximately

5 mL·kg^{-1}·min^{-1} after 12 days of no training. These results agree with Houmard et al. (1992) and the present study. Although the initial mean $\dot{V}O_2$max values (62.1 (3.3) mL·kg^{-1}·min^{-1}) and training experience (10 years) were higher than those in the present study, mean a-v O_2 difference were similar; 15.1(0.5) mL·beat^{-1} (Coyle et al., 1984) and 15.7 (0.3) for the present study. Also the female $\dot{V}O_2$max values in the Coyle (1984) study were similar to the highest initial values for this study; 54 mL·kg^{-1}·min^{-1} and 53.7 mL·kg^{-1}·min^{-1} respectively.

Cessation of training did not result in any significant changes after 15 days in the variables which contribute to $\dot{V}O_2$max; cardiac output ($\dot{Q}$) L·min^{-1}, stroke volume (SV) mL·beat^{-1}, maximal heart rate (HRmax) beats·min^{-1}, plasma concentration (PC) mL·100mL^{-1} of blood, and arterio-venous difference (a-v O_2 difference) mL·100mL^{-1}. The lack of change in variables that contribute to $\dot{V}O_2$max in the present study contradict findings in previous studies. Maximum heart rate has been found to rise approximately 9 beats·min^{-1} (Coyle et al., 1984; Houmard et al., 1992). Coyle et al. (1986) found SV to decrease significantly from 166 (8) to 146 (6) mL·beat^{-1} after 2-4 weeks of cessation of training. HRmax increased 11%, PV decreased 12% and there was no change in $\dot{Q}$. Coyle suggested that declines in central variables contributing to $\dot{V}O_2$max were due to changes in plasma volume. Using pooled male and female data, Coyle et al (1984) found significant changes in $\dot{V}O_2$max, SV, and HRmax after 12 days of no training and $\dot{Q}$ significantly changed after 21 days no training. Coyle suggested that decreases in SV and a-v O_2 difference contribute to the decreases in $\dot{V}O_2$max found after cessation of training.

Although many studies have supported decrements of approximately 4-7% in $\dot{V}O_2$max and other variables to varying degrees within 12 days of cessation of training, other research contradicts these findings. Koutedakis, Budgett, and Faulman (1990) found no decrements in athletes who stopped training for 3 -5 weeks. $\dot{V}O_2$max values

actually increased from 5.0 to 5.2 $L \cdot min^{-1}$, however these athletes were reported to be chronically fatigued apparently due to overtraining. Cullinane, Sady, Vadeboncoeur, Burke, and Thompson (1986) examined male long distance runners (61.3 (6.2) $mL \cdot kg^{-1} \cdot min^{-1}$) after 10 days of cessation of training and found no significant change in $\dot{V}O_2max$ or SV. However, changes were found in plasma volume (5% decline) and maximum heart rate (increased 9 (5) $beats \cdot min^{-1}$).

The present study found no significant ($p<0.05$) changes in the variables measured after 5 days of cessation of training. When examining the effects of tapering on the performance of endurance trained cyclists, a control group ceased training for four days and no significant changes were found in performance at ventilatory threshold (Neary, Martin, Reid, Burnham, & Quinney, 1992). These studies and those of Cullinane et al. (1986) and Koutedakis et al. (1990) suggest that in 5 days of cessation of training, little or no change is evident. However, if cessation of training continues, for example to 10 days, physiological variables such as $\dot{V}O_2max$ and those variables that contribute to $\dot{V}O_2max$ are at risk of decreasing to meet the new lower load. Adaptation to the removal of a training stimulus ultimately results in decrements in physiological function at the expense of the athlete's performance.

The present study and the supporting literature suggest that physiological function begins to decline within 10 days of cessation of training and the magnitude of decline in $\dot{V}O_2max$ is approximately 4 $mL \cdot kg^{-1} \cdot min^{-1}$ (Coyle et al., 1986; and Costill, Fink, Hargreaves, King, Thomas, and Fielding, 1985). Although $\dot{V}O_2max$ seemed to increase between day 10 and 15 in the CT group (see fig. 2) this increase was not significant. A successful taper effect results in the maintenance or increase in physiological variables and performance when training is reduced for a period of time (Houmard, Costill, Mitchell, Park, Hickner, & Roemmich, 1990; McConell, Costill, Widrick, Hickey, Tanaka, &

Gastin, 1993, Neary et al., 1992). Since a significant decrement occurred between day 5 and 10, removal of training in this study cannot be a taper but more appropriately a detraining effect.

The increase in $\dot{V}O_2$max at day 15 in CT group was a 5 % increase and was within the 2-7 % range which may be attributed to biological variability (Katch, Sady & Freedson, 1982). Coyle et al. (1984) found $\dot{V}O_2$max to decrease after 12 days and stabilize between 12 and 21 days of cessation of training; one subject followed the same pattern as in the present study increasing after an initial decrease. Coyle et al. (1984) suggested that this pattern was due to the exercise stimulus at day 12 which may have caused a training effect. In the present study, the longest exercise test duration was fourteen minutes with only 3-5 minutes being maximal intensity. Neufer, Costill, Fielding, Flynn and Kirwan (1987) found that reducing training to one day a week was not sufficient to maintain $\dot{V}O_2$max. Therefore it is unlikely that the 14 minute exercise bout once every five days caused an increase in $\dot{V}O_2$max at day 15 in the present study. In the CT group, $\dot{V}O_2$max values remained significantly different from MT group values at day 15. This may be due to a combined effect of cessation of training in CT group and the increase in $\dot{V}O_2$max in the MT group at day 15 due to a possible training effect. The increase in $\dot{V}O_2$max at day 15 in the CT group remains difficult to explain, but it is expected that values would drop if cessation of training continued to 20 or more days since the literature is consistent in reporting decreases in trained subjects after 2 - 4 weeks of cessation of training.

Do changes occur centrally or peripherally?

Central and peripheral variables contributing to $\dot{V}O_2max$ measured in this study are comparable to the values of female subjects in the literature. Initial values for both the CT group and the MT group in this study were not significantly different; 49.7 (1.2) and 49.5 (1.2) $mL \cdot kg^{-1} \cdot min^{-1}$ respectively. Kollais, Barlett, Mendes and Franklin (1978) found values of 59.5 $mL \cdot kg^{-1} \cdot min^{-1}$ for female middle distance runners but commented that their values were approximately 10 mL higher than values found in other studies. The highest maximal values in this study were 54.4 and 53.7 $mL \cdot kg^{-1} \cdot min^{-1}$ and are comparable to female athletes in the literature (Astrand, Cuddy, Saltin, & Stenberg, 1964; Coyle et al., 1984). The range of values in central variables is also comparable to those in this study. The range for cardiac output ($\dot{Q}$) is 18.5 - 20.2 (2) $L \cdot min^{-1}$ (Astrand et al., 1964; Kollais et al., 1978; Spina, Ogawa, Martin, Coggan, & Holloszy, 1992). Initial values for this study were CT 18.2(0.6) $L \cdot min^{-1}$ and MT 18.9 (1.3) $L \cdot min^{-1}$ (Fig. 2). Stroke volumes measured in this study were also within the ranges in the literature; 93 - 107 $mL \cdot beat^{-1}$ (Fig. 4) (Astrand et al., 1964; Kollais et al., 1978; Spina et al., 1992). Maximal heart rate was the only variable that differed. It was higher than those reported in the literature by approximately 4 $beats \cdot min^{-1}$ (Fig. 5). The only peripheral variable calculated in this study was a-v O_2 difference and it was also within the ranges in the literature; 14.3 - 15.4 $mL \cdot 100mL^{-1}$ (Fig. 6) (Coyle et al., 1984; Kollais et al., 1978; Spina et al., 1992).

After 10 days of cessation of training, $\dot{V}O_2max$ decreased approximately 3.8 $mL \cdot kg^{-1} \cdot min^{-1}$. Values were still significantly different at day 15 (Fig. 2). No significant

changes within or between groups were found after 15 days of no training for any of the central variables that contribute to $\dot{V}O_2max$ (Fig 3, 4, 5 and Table 3). The only change associated with the decrease in $\dot{V}O_2max$ was a decrease in a-v O_2 difference (Fig. 6). A change of approximately 1 $mL \cdot 100mL^{-1}$; 15.7 (0.3) to 14.6 (0.4) $mL \cdot 100mL^{-1}$ was found and although not statistically significant ($p=0.3$) within or between groups, physiologically it is the only variable in this study that changed concurrently with the decline in $\dot{V}O_2max$. The results of this study suggest that the change in $\dot{V}O_2max$ after 10 days of cessation of training in female runners is probably peripherally mediated.

After 10 days of cessation of training, Cullinane et al. (1986) found no significant change in $\dot{V}O_2max$ or SV. A decrease in plasma volume and an increase in HRmax were noted. Since body mass also decreased with no change in body composition, Cullinane attributed the decrease in plasma volume to fluid loss. The decrease in PV was compensated for by an increase in HR which in turn maintained SV. Since $\dot{Q}$ and $\dot{V}O_2max$ are closely coupled (Coyle et al., 1984) and SV was maintained, it was implied that $\dot{Q}$ was also maintained (Cullinane et al., 1986). Coyle et al. (1986) found that 2 -4 weeks of cessation of training resulted in a 6% decline in $\dot{V}O_2max$ which was accompanied by a 12% decrease in PV and 11% increase in HRmax. Stroke volume also decreased 166.0 (8) to 146.0 (6) $mL \cdot beat^{-1}$. No significant change was noted in submaximal $\dot{Q}$. When blood volume was artificially expanded with a dextran solution there was a reversal of the detrained results; SV, HR and $\dot{V}O_2max$ returned to trained levels. Thus Coyle suggested that declines in cardiovascular function in 2 - 4 weeks of cessation of training were due to decreases in PV. However, no change in PC was found after 15 days cessation of training in the present study.

The mechanisms responsible for training induced increases in PV are suggested to be increases in plasma albumin content, elevations in vasopressin and renin activity, and an

increased sensitivity of the aldosterone action (Convertino, 1991; Convertino, Brock, Keil, Bernauer, & Greenleaf, 1980; Gillen, Lee, Mack. Tomaselli, Nishiyasu, & Nadel, 1991). The exercise stimulus which is responsible for maintaining these factors is removed during cessation of training, and therefore the absence of this stimuli may account for the decrease in PV reported in the literature. The maintenance of PC in the present study may be due to an increase in aldosterone sensitivity which is suggested to occur in trained subjects (Convertino, 1991). It is possible that this sensitivity remains in some subjects when training is ceased for a short period of time. Gillen et al. (1991) found that a single 32 minute high intensity intermittent exercise stimulus was sufficient to cause an increase in PV. In the present study, the single bout of exercise performed every fifth day was at maximum 14 minutes in duration and only the last 3-5 minutes were high intensity. Therefore it is unlikely that the testing sessions every five days were adequate to be the stimulus for the maintenance of PC.

Coyle et al. (1984) examined trained athletes (male and female) at 12, 21, 56, and 84 days of no training. Significant decreases were found at day 12 in $\dot{V}O_2$max (7%) and SV (10%), and HRmax significantly increased (4%). $\dot{Q}$ did not significantly change until 21 days of no training (decreased 8%). Coyle et al. (1984) attributed the initial decline in $\dot{V}O_2$max to declines SV. Further declines in $\dot{V}O_2$max were attributed to significant declines in a-v O_2 difference which dropped from 15.1 (0.5) to 14.1 (0.5) $mL \cdot 100mL^{-1}$.

Since the subjects (Coyle et al., 1984) were only first tested after 12 days of no training, it is possible that $\dot{V}O_2$max began to decline at a similar rate to that in the present study, and that central variables may not have declined until 12 days. At 56 days, the subjects had a $\dot{V}O_2$max similar to the starting value of the subjects in the present study. Coyle found that subjects with the highest $\dot{V}O_2$max had the greatest decline during cessation of training ($r = 0.93$). Initial $\dot{V}O_2$max values in the present study are lower than

those in the Coyle study and it would be expected that the two groups would have different rates of decline. However, when $\dot{V}O_2$max values were similar in each study (for example at 56 days Coyle et al., 1984), the patterns of decline are comparable because when $\dot{V}O_2$max values were similar and cessation of training was continued, central variables were maintained while $\dot{V}O_2$max and a-v O_2 difference declined.

The decrement to 84 days (Coyle et al., 1984) was attributed to a decrease in a-v O_2 difference of approximately 1.0 (0.5) $mL \cdot 100mL^{-1}$. This is similar to the approximate 1.1 $mL \cdot 100mL^{-1}$ decrease which coincided with a 3.8 $mL \cdot kg^{-1} \cdot min^{-1}$ decrease in $\dot{V}O_2$max at 10 days in the present study. As $\dot{V}O_2$max increases with training, significant increases in a-v O_2 difference are found (Ekblom, Astrand, Saltin, Stenberg, & Wallstrom, 1968; Karpman, 1983; Kollais et al., 1978). Ekblom et al. (1968) attributed increases in $\dot{V}O_2$max to increases in $\dot{Q}$ and a-v O_2 difference. Coyle et al. (1984) suggested that when PV, SV, $\dot{Q}$ and HR are stabilized, decreases in maximal aerobic power observed after cessation of training are entirely due to a-v O_2 difference. Therefore, the Coyle et al. (1984) data supports the present study in that the decline in maximal oxygen consumption was associated with declines in peripheral factors represented by a-v O_2 difference.

Total peripheral resistance (TPR)has been found to increase after 2-4 weeks of cessation of training (Coyle et al., 1986). When plasma volumes were artificially increased with a dextran solution, changes in TPR were reversed to trained levels. The change in TPR was associated with increased HR and decreased PV, SV and $\dot{Q}$. Since no changes were found in PC, SV, $\dot{Q}$, or HR in the present study, it is unlikely that decreases in a-v O_2 difference were due to changes in TPR.

Peripheral variables that have been found to decrease as a result of cessation of training are muscle enzymes that represent different energy systems. Enzymes such as

phosphofructokinase (PFK) and lactate dehydrogenase (LDH) which represent glycolytic energy systems remain unchanged after 3 - 18 weeks of cessation of training (Costill et al., 1985; Green et al., 1980; Simoneau, Lortie, Boulay, Marcotte, Thibault, & Bouchard, 1987). Mitochondrial enzymes represent the capacity of muscle to extract and utilize oxygen as a metabolic fuel (Coyle, 1990). Oxidative enzyme activities are found to increase when the demand for oxygen consumption increases, for example with maximal exercise and training. These enzymes regulate the muscles use of metabolic fuels towards fat metabolism, sparing glycogen and delaying the onset of acidosis which may cause fatigue (Sjogaard, 1984). If the demand for oxygen is removed, as with cessation of training, then the stimulus to keep oxidative enzymes active is reduced and enzyme activity may decrease. Two common enzymes studied to examine changes at the muscle mitochondrial level are succinate dehydrogenase (SDH) and citrate synthase (CS) (Chi, Hintz, Coyle, Martin, Ivy, Nemmeth, Holloszy, & Lowry, 1983; Coyle et al., 1984; Klausen, Andersen, & Pelle, 1981). Muscle oxidative capacity has been found to decrease within the first 2 weeks of cessation of training (Henriksson & Reitman, 1977; Houmard et al., 1992; Houston et al., 1978). Costill et al. (1985) found the respiratory capacity of the deltoid muscle in swimmers to drop by 50% in the first week of cessation of training. Moore et al. (1987) also found CS activity to drop with 3 weeks of cessation of training, however, these declines were not related to changes in $\dot{V}O_2$max. This is also supported by Henriksson and Reitman (1977), and Houston et al. (1978). Klausen et al. (1981) also agreed that changes in oxidative capacity do not coincide with $\dot{V}O_2$max in the first 5-12 days of inactivity and suggested that the muscles respiratory capacity is not a limiting factor in the body's ability to utilize oxygen. In contrast, Coyle et al. (1984) examined CS and SDH at 0, 6, 12, 21, 56, and 84 days of no training. Significant decreases were found in both enzymes at 12 days of no training and reductions were observed up to 56 days.

No changes were observed between 56 and 84 days. Throughout the cessation of training period all values were well above sedentary control levels. A 40% decrease in mitochondrial enzyme activity coincided with a 7% decrease in a-v O_2 difference and a 14-18 % decrease in $\dot{V}O_2$max, which suggests the associated decrement in $\dot{V}O_2$max was attributable to the decrease in oxidative enzyme activity. The comparisons between values obtained in the Coyle (1984) study, those in the present study, and those of Costill et al. (1985) imply that the decrease in $\dot{V}O_2$max at 10 days of cessation of training is due in large part to decreases in a-v O_2 difference and muscle oxidative capacity.

Some evidence suggests that decreases in a-v O_2 difference may be due to decreased capillarization. Houston et al. (1978) found capillarization (capillaries per fiber) to decrease with 15 days of no training well-trained runners, however these results were from only two subjects. Klausen et al. (1981) found a decrease in capillary density (capillary per fiber) only after 8 weeks of cessation of training in previously untrained subjects. In contrast, Coyle et al. (1984) found that capillary density in skeletal muscle remained significantly higher in well-trained athletes showing no significant decline during 12 weeks of cessation of training. Therefore changes in capillary density do not seem to be the cause of changes in a-v O_2 difference during short periods (15 days) of cessation of training.

The ability to retain high levels of a-v O_2 difference and muscle oxidative capacity above those of sedentary controls seems to be a consequence of long term (years) training (Cullinane et al., 1986; Coyle et al., 1984; Houmard et al., 1992). The subjects in the present study had similar training experience to those in the literature. Cessation of training appeared to affect the subjects in this study peripherally, however, central changes were expected from evidence presented in the literature. The majority of the literature examined male athletes with extensive training experience or pooled results from female

and male subjects. Coyle et al. (1984) examined mainly male (one female) subjects and attributed changes in central variables to changes in PV. The present study examined females and found no changes in PC or central variables. Fortney, Beckett, Carpenter, Davis, Drew, LaFrance, Rock, Tankersley and Vroman (1988) suggested that estrogen may play a role in the stabilization of body fluid levels and these stable levels would influence the maintenance of central variables such as $\dot{Q}$ and SV. Therefore, it is possible that a gender difference may affect the responses of central and peripheral factors contributing to $\dot{V}O_2max$ during short term cessation of training. However, this remains to be determined.

Affects on Performance

Although there is much literature on the effects of cessation of training on maximal aerobic power and the physiological variables that contribute to it, the effects of change on actual sport performance are only implied. To our knowledge no study has examined the effects of cessation of training on sport specific performance in female middle distance runners. Performance measures have been analysed in the form of a treadmill timed run to exhaustion, and such studies have found that with cessation of training, time to exhaustion decreases indicating a decrement in performance (Coyle et al., 1986; Houston et al., 1978; Madsen, Pedersen, Djurhuus, & Klitgaard 1993). Each study examined endurance trained male athletes in which most decreases in performance were associated with decrements in $\dot{V}O_2max$. However, Madsen et al. (1993) found no significant change in $\dot{V}O_2max$ after 4 weeks of cessation of training but time to exhaustion decreased in association with decreases in substrate utilization. In the present study a 2400 m run was performed before cessation of training and after 15 days of cessation of training. The

maintenance group decreased their run time by 2 seconds while the cessation of training group increased by a mean of 18 seconds (Table 4). The decrement in performance in this study occurred concurrently with the declines in $\dot{V}O_2$max and a-v O_2 difference. If $\dot{V}O_2$max is indicative of performance as described by run time, then these results suggest that performance will decline as early as 10 days following cessation of training. Coyle et al. (1986) found that 2-4 weeks of cessation of training resulted in declines in $\dot{V}O_2$max and time to exhaustion on a treadmill test. Manipulating changes in PV resulted in improvements in $\dot{V}O_2$max after cessation of training, however performance was not improved. Artificial increases in PV lowered the hemoglobin concentrations which would reduce the oxygen carrying capacity of the blood and performance has been found to decline when hemoglobin is low (Kanstrup & Ekblom, 1984). Therefore decreases in performance were attributed to factors other than $\dot{V}O_2$max. Houmard et al. (1992) found no significant change in treadmill run time to exhaustion at 75% and 90% of $\dot{V}O_2$max after 14 days of cessation of training. Although muscle respiratory capacity decreased with in one week of cessation of training in trained swimmers, Costill et al. (1985) found no significant decreases in swim performance at one week or at four weeks of no training. In the present study no change was found in any of the variables measured after 5 days of cessation of training. These results and those presented in the literature suggest that this type of performance is not impaired for up to 5 days after cessation of training.

In summary, the present study showed that cessation of training for 15 days in trained female middle distance runners results in significant decreases in performance. Decreases in performance occurred concurrently with significant decreases in $\dot{V}O_2$max after 10 days of cessation of training and continuing to day 15.

CONCLUSIONS

1) Maximal oxygen consumption significantly decreased at 10 days of cessation of training by 3.8 $mL \cdot kg^{-1} \cdot min^{-1}$. No significant changes were found in physiological variables that contribute to $\dot{V}O_2max$.

2) No significant changes were found after 15 days of cessation of training in the central or peripheral physiological variables that contribute to $\dot{V}O_2max$. Although not significant, a 7 % decrease in the peripheral variable, a-v O_2 difference, occurred in conjunction with significant decrements in $\dot{V}O_2max$.

3) A significant decrease in performance was found after fifteen days of cessation of training. This decrease occurred concurrently with declines in physiological function represented by decreases in $\dot{V}O_2max$ and a-v O_2 difference.

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APPENDIX A

Informed Consent

INFORMED CONSENT - FOURTEEN DAY DETRAINING STUDY ON FEMALE RUNNERS

I _____, do acknowledge:
(please print)

___I consent to perform a submaximal exercise test to obtain ventilatory threshold prior to the detraining study. I understand that a mouthpiece will be worn and expired gases will be collected every 30 seconds for respiratory and metabolic factors.

___I consent to perform a maximal exercise test over four testing periods during which cardiac output will be measured at a submaximal exercise load and then the test will progress to obtain $\dot{V}O_2max$. I understand this test will require the use of a mouthpiece to collect expired gases as with the test mentioned above.

___I understand that a heart rate monitor will be attached across my lower chest to record my heart rate at rest, during submaximal and maximal testing.

___I understand that the CO_2 rebreathing method for measuring cardiac output will require me to breath a concentration of CO_2 for 30 seconds. During the rebreathing technique I may experience temporary dizziness which should pass upon the completion of the procedure.

___I understand that the maximal exercise test ($\dot{V}O_2max$) will involve exercise to a point of temporary exhaustion and that there is very little risk involved if I am a healthy active person. I regularly take part in strenuous exercise with my training at least as intense as these tests.

___I understand that 5 mL of blood will be drawn by venipuncture on three occasions during the study (pre, mid and post detraining). I understand that upon insertion of the needle I may experience some discomfort and a small amount of bruising may result at the puncture sight.

___I consent to a body composition assessment via caliper measurements.

___I understand that the study will consist of two groups; a detraining group that will cease all aerobic training for 14 days, and a maintenance group that will continue a maintenance program for the 14 days. I understand that I will be randomly assigned to one of these groups and I consent to adhere to the protocol assigned to me.

INFORMED CONSENT (cont'd)

___I understand that tests will be administered by qualified personnel under the direct supervision of the investigator(s).

___I understand that I may ask any questions or request further explanations or information about the procedures at any time before during or after the period of the study.

___I understand that I may withdraw from, reduce or modify my involvement in the study at any time and that the test may be terminated by the investigators upon observation of any symptoms of distress or abnormal responses.

___I understand that all my results are strictly confidential, that they will be cited using only an ID number or for the use in group means.

I hereby release the University of Victoria and its employees and any personnel involved in the research project from any and all liability that might otherwise arise as a result of my participation as a research subject in this study.

I acknowledge that I have read, understand and agree to the contents of this informed consent in its entirety. I agree to participate in this research project.

SIGNATURE:_____

DATE:_____

3

APPENDIX B

Review of Literature

Introduction

During a competitive season, athletes may encounter situations which require them to cease training or reduce training for a short period of time. Improvements or increases in physiological functions associated with training and enhanced performance may begin to decline during inactive periods and these periods are referred to as cessation of training (CT). During cessation of training, exercise stimulus is removed and the removal of exercise stimulus may cause physiological systems to readapt to the lesser load. The effects of CT are well documented; when the training stimulus is removed, selected physiological functions decline and ultimately performance will decline. However, the rates and magnitudes of decrements in physiological function and the effects on performance are equivocal. Adaptations to strength training have been shown to be maintained over several months of CT (Staron, Leonardi, Karapondo, Malicky, Falkel, Hagerman, & Hikida, 1991) while aerobic adaptations have been found to be more fragile and some decrements are found within the first week or weeks of CT (Coyle, Martin, Sinacore, Joyner, Hagberg, & Holloszy, 1985; Henriksson & Reitman, 1977).

The goals of training for athletic events that utilize aerobic metabolism as the main energy source are to develop the functional capability of central circulation and to enhance the aerobic abilities of specific muscles (McArdle, Katch, & Katch, 1991). The ability to perform physical work such as that required in the aerobic sport of endurance running depends on the ability of the muscle cell to transform food energy into mechanical energy for muscular work. This process depends on the ability to supply oxygen to the muscles and the ability for the muscle to extract and utilize the oxygen (Astrand & Rodhal, 1986, Coyle, 1990; McArdle et al., 1991).

Energy required for endurance events is primarily provided by the products of aerobic metabolism. The success of endurance performance is therefore related to the ability to transport oxygen to the exercising muscles and the muscles ability to extract and utilize the oxygen (Fox, Bowers, & Foss, 1989; Hartley, 1992). The rate at which energy is supplied by aerobic metabolism is called aerobic power (Thoden , 1991). Maximal aerobic power ($\dot{V}O_2$ max) is the maximal rate at which oxygen can be consumed and utilized by the tissues (Fox et al., 1989; Thoden, 1991). Athletes who perform activity continuously and at a high percentage of $\dot{V}O_2$max for longer than two minutes generally have higher maximal oxygen uptake than those performing high intensity activity for lesser amounts of time. Highest values for $\dot{V}O_2$ max are found in sports such as middle distance running and rowing (Hartley, 1992; Thoden, 1991). Maximal oxygen uptake is dependent on the ability of the tissues to utilize the oxygen (peripheral components), and the transport of oxygen to the tissue (central components) (Hartley, 1992). Maximal oxygen uptake is the product of cardiac output ($\dot{Q}$) and arteriovenous difference (a-vO_2 difference), both of which can increase with training (Blomqvist, 1983; Astrand, Cuddy, Saltin, & Stenberg, 1964; McArdle et al., 1991); genetic factors also may have a limiting effect on maximal aerobic power, but to what extent is not clear (Thoden, 1991).

Maximal aerobic power increases with endurance training. If the training stimulus is removed it would be expected that $\dot{V}O_2$ max would decrease and performance which depends on $\dot{V}O_2$max would also be expected to decrease (Henriksson & Reitman, 1977). The purpose of this review is to outline the effects of cessation of training (CT) on physiological functions of the central and peripheral components of maximal oxygen uptake ($\dot{V}O_2$max) and the implications on performance.

Maximal oxygen uptake

Maximal oxygen uptake ($\dot{V}O_2max$) has been defined as the maximum amount of oxygen consumed per unit time during activity which requires large muscle groups. When measuring this variable, intensity of activity (for example, running on a treadmill) is increased progressively until exhaustion (Thoden 1991). Maximal oxygen uptake is expressed either as the absolute volume of oxygen consumed per minute ($L \cdot min^{-1}$)or the volume of oxygen consumed per minute per kilogram of body mass ($mL \cdot kg^{-1} \cdot min^{-1}$)and is abbreviated as $\dot{V}O_2max$ (Thoden, 1991). Criteria for achieving $\dot{V}O_2max$ during a laboratory test are as follows: (1) a plateau or decrease in oxygen consumption despite an increase in work intensity; (2) volitional exhaustion; (3) attainment of predicted maximal heart rates and (4) respiratory exchange ratio greater than 1.10 (Hickson & Rosenkoetter, 1981; Thoden, 1991).

Changes in $\dot{V}O_2max$ during CT have been investigated, however, results are equivocal. Male runners with a training base of 4 years were examined after fifteen days of CT and $\dot{V}O_2max$ decreased by 4% (Houston, Bentzen & Larsen, 1979). Similar results were found when previously untrained females were trained for six weeks and then ceased training for two weeks; $\dot{V}O_2max$ decreased by 5% however this was not considered to be a significant decrease (Ready, Enyon, & Cunnigham, 1981). When examining 12, 21, 56, and 84 days of cessation of training in trained male runners, $\dot{V}O_2max$ declined 7% after 12 days and remained at that level from days 12 - 21, but from days 21 - 56 $\dot{V}O_2max$ declined to a level 14% below trained levels. In untrained males, significant decreases in $\dot{V}O_2max$ were not noted until after nine weeks of no training (Ready & Quinney, 1982).

In contrast, Cullinane, Sady, Vadenboncouer, Burke, and Thompson (1986) examined the effects of ten days of exercise cessation on male long distance runners and found no significant decrements in $\dot{V}O_2$max. Michael, Evert, and Jeffers (1972) examined young female track athletes over 23 weeks of CT and found no significant decrements in the first three weeks of cessation of training. Also, Koutedakis, Budgette, and Faulman (1990) examined athletes whos' performance was declining rather than increasing with training. These athletes rested for three weeks and mean $\dot{V}O_2$max values were found to increase.

These results indicate that percentage decrements in $\dot{V}O_2$max are similar in previously untrained subjects and subjects with a training base of 2 - 5 years. The levels of $\dot{V}O_2$ max attained by highly trained runners are not permanent nor are they more stable than subjects with less training when the training stimulus is removed. However, highly trained athletes do not decrease to pretrained levels as quickly as previously untrained subjects. Highly trained athletes may exhibit enhanced central and peripheral components which contribute to the maintenance of $\dot{V}O_2$max. Which of these factors is more limiting to $\dot{V}O_2$max is equivocal.

Central components: Cardiac output, stroke volume and heart rate

$\dot{V}O_2$max is the product of maximal cardiac output ($\dot{Q}$) and arterial venous difference (a-vO_2 difference) at maximum exercise (Ekblom, Astrand, Saltin, Stenberg, & Wallstrom, 1968). Cardiac output is the volume of blood pumped by the heart per minute, and is the product of heart rate (HR) (number of times the heart beats per minute) and stroke volume (SV) (the volume of blood ejected from the heart during ventricular

contraction) (Fox et al., 1989; Vander, Sherman, & Luciano, 1990). Cardiac output and the physiological functions which effect it are the central components of $\dot{V}O_2max$.

Central physiological components which are attributed to increases in $\dot{V}O_2max$ are increased in cardiac output ($\dot{Q}$) which is brought about by changes in stroke volume and heart rate (Saltin & Rowell, 1980). Training induced increases in $\dot{V}O_2max$ are accompanied by increases in $\dot{Q}$, SV, and arteriovenous difference of approximately 12%, 16% and 7% respectively (Spina, Ogawa, Martin, Coggan, Holloszy, & Ehsani 1992, Ekblom et al., 1968). Maximal values for $\dot{Q}$ are reported to be 20.9-27.4 $L \cdot min^{-1}$ for trained males, 18.5-20.2 $L \cdot min^{-1}$for trained females; stroke volume (SV) is 121-143 $mL \cdot beat^{-1}$ for trained males and 93-115 $mL \cdot beat^{-1}$ for trained females; and a-v O_2 difference for trained males is 14.2-16.7 $mL \cdot 100mL^{-1}$ and 14.3-15.4 $mL \cdot 100mL^{-1}$ for trained females (Astrand et al., 1968; Coyle et al., 1984; Kollais, 1978; Spina et al., 1992). Cardiac output directly affects the ability to circulate oxygen, and therefore low aerobic capacity is associated with a low $\dot{Q}max$ (McArdle et al., 1991). These indices of central increases in $\dot{V}O_2max$ are also found to be indices of the decrements found in $\dot{V}O_2max$ during CT.

Decreases in $\dot{Q}max$ are reported to coincide with decreases in $\dot{V}O_2max$ (8% and 7% respectively) in the first three weeks of CT (Coyle et al., 1984). These initial decreases in $\dot{Q}max$ are attributed to an 11% decrease in SV (Coyle et al., 1984). Cardiac output did not decrease to the same extent as SV. This may be due to an increase in HR which may be responsible for maintaining $\dot{Q}$ at 8% of trained values. Coyle et al. (1986) found decreases in $\dot{V}O_2max$ to be associated with a 12% decline in plasma volume (PV), and an 11% increase in maximal heart rate (HRmax). SV decreased approximately 20 $mL \cdot beat^{-1}$ and no significant change was noted in $\dot{Q}$.

Blood volume

Plasma volume and hemoglobin increase with endurance training, reflecting an increase in total blood volume (McArdle et al., 1991). Changes in blood volume can effect circulation by means of the Frank- Starling mechanisms which state that an increase in the volume of blood filling the ventricle will stretch the cardiac muscle causing the muscle to respond with a more forceful contraction, expelling more blood to the body (Fox et al., 1989; Vander et al., 1990). Changes in blood volume can therefore effect stroke volume and cardiac output; since these factors influence $\dot{V}O_2max$, ultimately changes in blood volume can also affect $\dot{V}O_2max$ (Gledhill, 1992).

Coyle et al (1984) noted decreases in SV after 12 days of inactivity and no further decrements after 3 months of inactivity. Coyle et al. investigated the effects of CT in male runners with a training base of 6 years to determine the contributing factors to the decreases in SV. Blood volume increases with training - an adaptation to enhance the oxygen delivery capacity during exercise (McArdle et al. 1991). Coyle et al., (1986) found that blood volume decreased by 9% over 2-4 weeks of detraining and suggested that the decrease was primarily due to a decrease in plasma volume of 12%. When blood volumes were artificially increased to trained levels, SV and $\dot{V}O_2max$ returned to near trained levels. These findings are supported by Kanstrup and Ekblom (1984).

In contradiction to Coyle et al. (1984), Coyle et al. (1986), and Kanstrup and Ekblom (1984), Cullinane et al. (1986) found no significant decreases in SV after 10 days of exercise cessation. Plasma volume did drop quickly and remained lower than the trained levels, while resting HR and SV did not change. The preservation of $\dot{Q}$ and $\dot{V}O_2max$ was attributed to higher exercise heart rate.

Cardiac mass

Cardiac mass in well trained runners is found to be larger than that of untrained individuals and is thought to contribute to increases in SV of athletes (McArdle et al., 1991). Ventricular mass has been found to increase between 6 - 19% with endurance training programs (Hickson, Foster, Pollock, Galassi, & Rich, 1985; Hickson, Kanakis, Davis, Moore, & Rich, 1982). During a one third and two third reduction in frequency of training for 15 weeks cardiac mass remained at trained levels. However, a reduction in training intensity by the same amount resulted in a decrease in mass after 15 weeks.

Cessation of training studies from 10 - 60 days have found no change in cardiac thickness in well trained athletes (Cullinane, et al., 1986; Pavlik, Bachl, Wollein, Langfy, & Prokop, 1986). The results of these studies support the suggestion by Coyle et al. (1986), that declines in cardiac mass are not readily evident because of the maintenance of SV when blood volume is increased to trained levels. If cardiac mass decreased, contractility may be affected and then SV would not be maintained.

Hemoglobin

Hemoglobin (Hb) is an oxygen binding pigment found in red blood cells (Vander et al., 1990). Each hemoglobin molecule is made up of four subunits called heme which bind to oxygen; thus each hemoglobin is capable of combining with up to 1.39 mL of oxygen (Fox et al., 1989; Gledhill, 1992; Vander et al., 1990). Variations in Hb concentration can affect the capacity of the blood to transport oxygen. The concentration of hemoglobin in the blood varies in males and females. At rest and sea level males have a hemoglobin concentration of approximately 16 grams per milliliter (g/mL) of blood ranging from 14.0 - 18.0 g/mL, and females 14 g/mL ranging from 12.0 - 16.0 g/mL (Fox et al., 1989;

Gledhill, 1992). Hemoglobin has been found to decrease with intense training in some subjects. For example, when Hb decreased 13 - 18%, physical performance capacity decreased by 30%, and $\dot{V}O_2$max decreased by 13 and 18% (Ekblom, Goldbarg, & Gullbring, 1972).

Hemoglobin has been reported to increase when training is reduced as in tapering or removed as in detraining. Yamamoto, Mutoh, & Miyashita (1988) followed collegiate swimmers during a competitive season and measured blood constituents during tapering phases. Hemoglobin was found to rise after seven days of tapering. When examining the effects of exercise cessation, hemoglobin concentration did not change significantly after three months of CT, however the subjects may not have been in a fully detrained state as they were allowed to participate in regular physical education classes during the three month term (Drinkwater & Horvath, 1972). Affects of CT or Hb on performance were not indicated.

Kanstrup and Ekblom (1984) found a decrease in $\dot{V}O_2$max and performance time when artificially lowering blood volume. When hemoglobin concentration was increased, blood volume increased slightly and $\dot{V}O_2$max and performance time also increased. These results suggest that the product of hemoglobin and blood volume may enhance performance time and $\dot{V}O_2$max. Coyle et al. (1986) noted that although $\dot{V}O_2$max increased when a detrained subject's plasma volume was artificially increased, time to fatigue decreased. The subjects commented that their legs fatigued much sooner than when plasma volume was not increased. These results suggest the importance of hemoglobin concentration as well as higher blood volumes and other hematological factors for maintenance of trained levels of $\dot{V}O_2$max.

Hematocrit

Hematocrit is defined as the percentage of total blood volume containing erythrocytes (red blood cells) (Vander et al., 1990). Mean hematocrit in healthy males is approximately 45% (± 5 %), mean hematocrit in women is approximately 42% (± 5 %) (Gledhill, 1992; Vander et al., 1990). Since hemoglobin is carried by the red blood cells, then the number of red blood cells represented by hematocrit is also important for oxygen carriage. If hematocrit is too low, then the oxygen carrying capacity is lower, affecting endurance type activities in which oxygen is the main fuel source. However, a higher than normal hematocrit does not necessarily benefit the oxygen transport system as the viscosity of the blood increases with increasing hematocrit (Ekblom et al., 1972; Gledhill, 1992; Kanstrup & Ekblom, 1984). As with hemoglobin, hematocrit has been reported to decrease with intense exercise training and to increase with tapering and detraining periods (Drinkwater & Horvath, 1972; Yamamoto et al., 1988).

Peripheral components

$\dot{V}O_2$max decreases in the first few weeks of CT; decrements are attributable to decreases in $\dot{Q}$max, stroke volume and blood volume (Coyle et al., 1984; Coyle et al., 1986; Kanstrup & Ekblom, 1984). In additional weeks of no training, no further decrements in stroke volume are noted, however $\dot{V}O_2$max will continue to decrease up to 14% below trained levels (Coyle et al., 1984). These findings suggest that other adaptations must account for the decreases in $\dot{V}O_2$max. Coyle et al.(1984) found concurrent decreases of 9% in $\dot{V}O_2$max and maximal a-vO_2 difference during 21 - 84 days of CT. Arteriovenous oxygen difference (a-vO_2 difference) represents how much

oxygen is extracted or consumed by the tissues from each 100mL of blood perfusing the tissues (Fox et al., 1989). Arteriovenous oxygen difference is a peripheral component of $\dot{V}O_2max$.

Arteriovenous oxygen difference increases with endurance training (Ekblom et al., 1968; Karpman, 1983; Kollais, 1978), and is limited by the ability to redirect blood during exercise. Increases in capillarization of the muscle expand the pathways for blood to reach the muscles; capillary density may increase with endurance training which would ultimately enhance oxygen extraction and utilization (Klausen, Andersen & Pelle, 1981). If capillary density increases, more blood can be accommodated in the muscle bed per unit time; therefore mean transit time would decrease and a more complete exchange of material would be allowed (Astrand & Rodhal, 1986). After 84 days of CT, Coyle et al. (1984) found no changes in capillary density with well trained subjects. However, Klausen et al. (1981) found decreases in capillary density after 8 weeks of CT in previously untrained subjects. These results suggest that increased capillary density in subjects with a training base of two or more years will be maintained longer during detraining than in subjects with a training base of less than one year.

Although capillary density is maintained over 84 days of CT, the changes in a-vO_2 difference may be attributed to muscle blood flow. Since muscle blood flow increases with training and coincides with increases in $\dot{V}O_2max$ and changes at the alveolar level, it is suggested that decreases in a-vO_2 difference after 12 weeks of CT may be related to decreases in muscle blood flow (Neufer, 1989). However, the response of muscle blood flow to CT is not well documented; changes in muscle blood flow can only be speculated.

Coyle et al. (1984) attribute the decrements in $\dot{V}O_2max$ after 3 months of CT to stroke volume and a-v O_2 difference; the peripheral factors (a-v O_2 difference) appear to be

associated with decreases in muscle mitochondria activity and other decrements in enzymatic activity. This is also supported by Neufer, Foster, Fielding, Flynn, and Kirwan (1987), Moore et al., (1987), and Kollais et al., (1978).

Muscle enzyme activity

In endurance activities, energy in the form of adenosine triphosphate (ATP) is provided mainly by the oxidation of glycogen in the skeletal muscle. The 39 moles of ATP are made available through aerobic glycolysis (3 ATP), the Krebs cycle (2 ATP), and electron transport (34 ATP) (Fox et al., 1989). The aerobic breakdown of fatty acids are through the process called beta oxidation, which yields a net production of 16 ATP (Fox et al., 1989). Different enzymes mark the metabolic activity of each of these systems. Commonly used markers to measure the changes in oxidative metabolism are the oxidative enzymes succinate dehydrogenase (SDH), 3- hydroxyacyl CoA dehydrogenase (HADH). SDH marks the activity of the Krebs cycle and changes in its activity can reflect changes in other enzymes; increases in SDH indicate that training has been successful in enhancing mitochondrial activity (Astrand & Rodahl, 1986). The activity of beta oxidation is marked by HADH. (Simoneau, Lortie, Boulay, Marcotte, Thibeault, & Bouchard, 1987). Glycolytic enzymes which mark the aerobic metabolism of glycogen are phosphofructokinase (PFK) and phosphorylase (PHOSP) (PHOSP indicates glycogenolysis). Lactate dehydrogenase (LDH) denotes lactate production from anaerobic glycolysis (Fox et al., 1989; Green, Thomson, Daub, & Ranney, 1980; Simoneau et al., 1987).

Various CT studies ranging from 2 to 18 weeks, have examined the time course of changes in the activity of these enzymes. Simoneau et al. (1987) and Green et al. (1980) examined the effects of CT on subjects who had been on a high intensity intermittent

training program. Simoneau et al. studied previously untrained subjects and found after 7 weeks of CT that oxidative markers decreased while glycolytic markers remained stable. This is in agreement with Green et al. (1980) and Coyle et al. (1985) who showed decreases in SDH and PHOSP after 3 weeks of CT while PFK and LDH remained higher until 6 -18 weeks of CT. Klausen et al. (1981) also had similar results with oxidative enzymes decreasing significantly after 4 and 8 weeks of CT, however PHOSP did not show decreases until 8 weeks. Houston et al., (1979) found similar decrements in previously well trained runners after 15 days of CT. SDH decreased by 24% and LDH decreased by 13%.

The results of these studies suggest that a training stimulus is required to maintain high oxidative activity while the effects of a few weeks CT on glycolytic enzymes are negligible. Maximal oxygen uptake in these studies did not decrease to the same extent as did the oxidative enzymes, for example with the trained runners $\dot{V}O_2$ max decreased by 4% while SDH decreased 24% in the same time frame (Houston et al., 1979). Such results suggest that adaptations in $\dot{V}O_2$max are associated with but not dependent on adaptations in skeletal muscle oxidative capacity.

Myoglobin

Myoglobin is an oxygen binding protein found in muscle fibers. It increases the rate of movement of oxygen into the muscle cells and provides a small storage site for oxygen (Vander et al., 1990). Although myoglobin content is sometimes thought to increase with training, its contribution to improvements in the aerobic system is minor. The main function of myoglobin is in aiding the delivery of oxygen from the cell to the mitochondria (Fox et al., 1989). Myoglobin concentration has been found to increase in some animals

with training, however in humans it seems that myoglobin concentration is unaffected by training or cessation of training (Jansson, Sylven, & Nordevang, 1982; Svedenhag, Henricksson, & Sylven, 1983). When examining the effects of CT on previously endurance trained athletes, no changes in myoglobin concentrations were found. Also, the myoglobin concentrations of the athletes studied were not significantly different than the concentrations found in the sedentary controls (Coyle et al., 1984). Thus, myoglobin does not appear to be a factor for the decrements found in $\dot{V}O_2$max during CT.

Performance

Although many studies have examined different parameters in physiological function during CT, few have analyzed the extent of these effects on sport specific performance on trained athletes. Costill, Fink, Hargreaves, King, Thomas, and Fielding (1985) examined male collegiate swimmers over four weeks of inactivity and found that changes in swim times, blood lactate, and pH values were small and only the changes in blood lactate were significant. In well trained runners with an average training base of 4 years, Houston et al. (1979) found performance (as measured by time to exhaustion on a treadmill run) to decrease relative to the magnitude of the decrease in SDH after 15 days of CT. However, these variables did not correspond with each other during retraining and no causal relationship could be drawn. After four weeks of CT (one exercise bout per week), Madsen, Pedersen, Djurhuus and Klitgaard (1993) found endurance time to decrease by 21%. Hickson et al. (1982) found no change in performance time of short term endurance after reducing training frequencies for 15 weeks. However, when intensity was reduced by one third and two thirds for the same time period, only the two third reduction group resulted in performance decrements and the decrements were only significant after 15

weeks. Although these results indicate some decrements in performance, the effects of short periods of CT (i.e. 2 weeks) is still equivocal.

Conclusion

Changes in a-v O_2 difference, capillary density and muscle enzymes during CT indicate that peripheral adaptations to training will remain at trained levels for a longer period of time than central adaptations. It has also been shown that subjects with a longer training base (2-5 years) will maintain peripheral values longer than previously untrained subjects and sometimes central values will also be maintained longer. Subjects with longer training base experience central decrements in the first few weeks of CT while peripheral components are maintained until approximately 4 weeks of detraining. Although decrements in central and peripheral physiological factors related to $\dot{V}O_2max$ do decline in well trained athletes in short periods of CT, the magnitude of decline and the influence of the declines on sport specific performance is still equivocal.

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