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Cross-training between cycling and running in untrained females

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Objective. **The purpose of the study was to evaluate the cross-training response between running and cycling in untrained females.**
Experimental Design. **The following study involved a pre-test, post-test, 3×3 factorial design.**
Setting. **Training (4 days-week^{-1}, 10 weeks, 70-80% heart rate reserve) occurred at the Center for Exercise and Applied Human Physiology. Exercise testing occurred at the Veterans Hospital, Exercise Laboratory.**
Patients and participants. **Subjects included healthy, untrained females aged 18-25 years, (N=18).**
Intervention. **Subjects were assigned to one of three (n=6) training groups (run=R, cycle=C, both run and cycle=RC) matched on pre-training CE $\dot{V}O_2$max results.**
Measures. **Graded treadmill run (TR) and cycle ergometer (CE) tests were performed on each subject to determine a mode specific $\dot{V}O_2$max and the lactate threshold (LT). Graded arm ergometer (AE) was performed to determine $\dot{V}O_2$max and heart rate and blood lactate at 20 and 40 Watts (W). Testing occurred prior to (0T), after 5 (5T) and after 10 weeks of training (10T). Body fat testing (hydrodensitometry at residual lung volume) was performed at 0T and 10T.**
Results. **TR and CE $\dot{V}O_2$max as well as TR and CE $\dot{V}O_2$ at the LT improved throughout the 10 weeks, regardless of training group. Although there were no changes in $\dot{V}O_2$max or blood lactate levels during AE, submaximal heart rates were significantly reduced over the 10 weeks, regardless of training group.**
Conclusions. **These results indicate that the aerobic benefits of either run, cycle or combined run and cycle training are similar in untrained females. The LT and AE heart rate data demonstrate that improvements in $\dot{V}O$max due to ten weeks of training are a result of pronounced peripheral and moderate central adaptations.**

KEY WORDS: Cross-training - Anaerobic threshold - Heart rate physiology - Ergometry - Lactates, blood - Exercise, physiology - Oxygen consumption - Running, physiology - Cycling, physiology.

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In recent years multi-event sports (e.g. biathlons, triathlons) have grown in popularity, and the interest in the potential for endurance run and cycle training to complement each other has been renewed. Past research has documented that a limited amount of cardiorespiratory improvement can transfer between cycle and run training (cross-training) when

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American College of Sports Medicine, Southwest Region Conference, November, 1991, San Diego, CA. Cross-Training Between Cycling and Running in Previously Sedentary Females. Presented by Brent C. Ruby.
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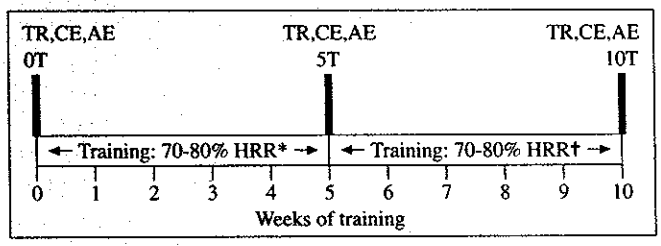

Fig. 1.—An illustration of the timing of treadmill (TR), cycle ergometer (CE) and arm ergometer (AE) testing during the 10 weeks of training. *HRR calculated from mode specific max HR and current resting HR; †HRR adjusted for any changes occurring in mode specific max HR and resting HR.

training in one of these exercise modes.[1-4] In contrast, Mutton *et al.*[5] showed that running or combined run/-cycle training improved treadmill run $\dot{V}O_2$max. However, the majority of these investigations were designed to answer questions of training specificity in moderately to highly trained individuals, and have neglected to specifically address the "cross-training" concept.

The increases in $\dot{V}O_2$max after cycle and run training programs have been found to be lower during the untrained exercise mode than the trained mode.[2 3] However, conflicting results have been reported for ventilatory (VT) and lactate threshold (LT) improvements for cycle and run trained individuals. Costa *et al.*[6] reported that the VT was similar for trained runners and cyclists during running and cycling, whereas other investigators have shown a higher LT [1 3] and VT [7] for the specifically trained mode of exercise than for the untrained mode.

In addition to cycle and run training, 0-10% improvements have been reported in AE $\dot{V}O_2$max in response to cycle training.[8 9] However, potential improvements in AE have not been reported following a similar run training program. Results of AE $\dot{V}O_2$max increases during leg training have been interpreted to indicate central training adaptations (increased maximal cardiac output and stroke volume).[10 11]

The aforementioned research has not been designed to specifically address the issue of cross-training, or whether endurance training improvements in either exercise mode are decreased when combining run and cycle exercise into a training program. Furthermore, the untrained female has not been adequately studied, and we know very little concerning the early training improvements in cycle and run exercise in women. Consequently, we investigated the cross training response in the most underrepresented group in exercise science research, the untrained female. The purpose of this study was to demonstrate the cardiorespiratory effects of 10 weeks of endurance run, cycle, and combined run and cycle training between three groups of untrained female subjects using cycle, run, and arm exercise tests.

Materials and methods

The subjects of this study were females not currently involved in an organized training program (N=18). The subjects were volunteers recruited from the undergraduate population at the University. Prior to the investigation, each subject read and signed an informed consent approved by the University Human Research Review Committee.

Procedures

Prior to training, all subjects completed tests for VO_2max and the lactate threshold (LT) during treadmill running (TR) and cycle ergometry (CE), and a test for $\dot{V}O_2$max on an arm ergometer (AE). The variables of $\dot{V}O_2$max and the blood samples for detection of the LT were obtained from the same test. Subjects were then assigned to either a run, cycle, or run and cycle training group matched for the pre-training cycle $\dot{V}O_2$max results. Training consisted of 4 sessions week^{-1}, 45 minutes session^{-1} for 10 weeks at an intensity equivalent to 70-80% mode specific heart rate reserve (HHR), (Fig. 1). Mid-point testing occurred after weeks 5, and involved TR, CE, and AE tests of $\dot{V}O_2$max and TR and CE LT. Post training tests of TR and CE $\dot{V}O_2$max and LT, and AE $\dot{V}O_2$max were conducted following week 10.

Treadmill run $\dot{V}O_2$max and lactate threshold

The TR $\dot{V}O_2$max was completed with an intermittent, walk-run protocol using a Jager™ (Laufergotest) treadmill and Jaeger™ (Intercarepulmobile) cardiopulmonary metabolic system. Prior to each test the Jaeger™ pneumotachograph was calibrated at varied volumes and flows, and the oxygen and carbon dioxide gas analyzers were calibrated

against medically certified gases of known concentration. The initial TR speed was 6 km·hr^{-1} at 0% grade with speed increments of 1 km·hr^{-1} every three minutes until a speed of 10 km·hr^{-1} was reached. Thereafter, the percent grade increased 1% every 3 minutes until uditional exhaustion. All subjects received verbal encouragement throughout the tests. Respiratory gas exchange variables were displayed in 15 second intervals continuously during the test. $\dot{V}O_2$max was chosen by averaging at least four (1 minutes worth of data) of the highest recorded values of oxygen uptake observed during the exercise test. A plateau in $\dot{V}O_2$ and/or a respiratory exchange ratio greater than 1.1 was used as the criteria for the attainment of $\dot{V}O_2$max. These criteria were met in all subjects.

Venous blood samples were collected at rest and during the last 45 seconds of each exercise stage from an indwelling 20 gauge catheter in an anticubital vein kept patent by a continuous saline drip. A portion of each sample (0.5 ml) was deproteinized in 7% perchloric acid (1 ml) and centrifuged. The supernatant was refrigerated at -4°C for subsequent determination of the lactate concentration using an enzymatic, spectrophotometric method.[12]

To minimize subjective bias, the LT was determined using a two segment linear regression program integrating lactate concentration and exercise time. The lactate and time data were subjected to a least squares fit that selected a two segment linear regression model having the least error. The lactate threshold was defined as the point at which the two regression lines intersected.

Cycle egometer $\dot{V}O_2$max and lactate threshold

To determine $\dot{V}O_2$max during CE, and electronically braked ergometer was used (Jeager, Ergotest™). The initial work rate consisted of three minutes with no load cycling at a self selected cadence between 70-90 rpm. The work rate subsequently increased by 25 W every two minutes until a cadence of 40 rpm could not be maintained. The CE protocol was developed to allow subtle progression between stages beginning at a low intensity to increase the ability to detect the LT. Venous blood samples were obtained during the last 30 s of each stage. Oxygen consumption, blood sampling, and LT determination were performed using the aforementioned procedures described for TR testing.

Arm ergometer $\dot{V}O_2$max, HR and lactate measures

The arm ergometry test began approximately 30 minutes following the TR test of $\dot{V}O_2$max and was performed using and electronically braked ergometer (Jeager, Ergotest™). The subject was positioned behind the ergometer and strapped into a chair in such a manner that the contribution of the upper torso to pedal cranking was limited. The protocol was initiated with the subject cranking against zero resistance at a self selected cadence above 50 rpm during the first 3 minutes of the test. Thereafter, a self selected cadence above 50 rpm was maintained with the resistance increasing by 10 W every 2 minutes until a cadence of 40 rpm could not be maintained. Increments of 10 W per stage were selected to allow subtle progression from stage to stage and due to the relatively small active muscle mass used during AE. $\dot{V}O_2$max was determined as previously described. Blood samples (15 µl) were obtained from a hyperemized ear lobe [13] at rest, after 20 and 40 W, and after volitional exhaustion. The blood samples were deproteinized in 7% perchloric acid (100 µl) frozen and assayed for lactate as previously described.

Percent body fat assessment

Percent body fat was calculated using underwater hydrodensitometry and corrected for residual lung volume. Each subject was given as many trials as needed to obtain three readings within 100 g. Residual volume was measured outside the tank at least three times using a helium dilution technique (Collins Modular Lung Analyzer, P-1260-RV module, Greesboro, NC). During each residual volume trial, subjects were in a seated position similar to the position assumed during the hydrostatic weighing procedure. Percent body fat was calculated from body density using the Brozek equation.[14]

Training

The subjects were instructed to train only in their assigned exercise, and to avoid recreational involvement in the opposing exercises. Training was supervised by trained assistants and occurred four times per week for 45 minutes per session for a total of then weeks. The training intensity was set at 70-80% of HRR (as determined by the respective mode specific maximal tests) throughout the first 5 weeks

of exercise (Fig. 1). Training heart rate was adjusted for the last 5 weeks based on changes in max and resting heart rate obtained during the mode specific 5T testing session. Cycle training was performed in an indoor exercise center with the use of cycle ergometers (Bodyguard™ Egometer 990). Run training was performed outside on a 0.5 mile running track. The run/cycle training group was allowed to perform two days of cycling and two days of running each week in a self-selected order at the intensities shown above. Training intensity was countinously monitored using heart rate monitors (UNIQ™ CIC, model # 8799). Following the tenth week of training, the previously described tests (TR, CE, and AE) were repeated for each subject, resulting in a total of three testing dates to evaluate training improvements (a total of nine test sessions per subject, Fig. 1).

Statistical analysis

For each dependent variable (TR, CE and AE $\dot{V}O_2$max; TR and CE $\dot{V}O_2$ at LT; AE HR at 20 and 40 W; AE lactate at 20 and 40 W) a two way mixed design ANOVA with repeated measures was run between groups (cycle, run, both) and across the training times (0T, 5T and 10T). An additional two way mixed design ANOVA with repeated measures was run for the variable of body fat (measured at 0T and 10T). Post hoc calculations were performed as needed using the Tukey HSD test. Each hypothesis was tested at the $p<0.05$ level. However, due to the use of ten independent ANOVA's performed on the same data set, the alpha level was adjusted to maintain the same family wise error rate of $p<0.05$. Alpha was adjusted using the Bonferroni procedure (0.05/10 tests=0.005).

Results

Training adherence

Descriptive data for the three groups of subjects are presented in Table I. Subject training compliance was extraordinarily high. For each of the 18 subjects a total of 46 training and/or testing sessions were required, totaling 828 sessions. Of these, only 5 subjects missed between 2 to 5 sessions, resulting in a subject training compliance exceeding 95%.

TABLE I.—*Descriptive data of the subject and changes in body fat within each training group (mean±SE).*

Parameters	Run	Cycle	Both
Age	20.3±0.9	20.5±1.0	21.3±0.6
Weight (kg)			
pre	58.2±3.3	61.6±3.6	62.4±3.0
post	57.0±3.3	61.2±2.6	62.8±3.0
Height (cm)	160.0±7.4	165.4±1.2	166.1±1.9
% Fat			
pre	20.6±2.2	23.5±1.8	28.0±3.1
post	18.5±2.0	22.1±1.5	27.7±3.0
n	6	6	6

TABLE II.—*Maximal oxygen consumption ($L \cdot min^{-1}$) data for the three training groups during each exercise mode, and training/testing time (mean±SE).*

Test Type/Time	Training group		
	Run	Cycle	Both
TR			
0T	2.32±0.07	2.32±0.01	2.37±0.09
5T	2.45±0.10	2.41±0.11	2.47±0.13
10T	2.57±0.10*†	2.49±0.10*†	2.63±0.15*†
CE			
0T	2.16±0.04	2.07±0.09	2.22±0.14
5T	2.24±0.11*	2.22±0.10*	2.48±0.11*
10T	2.30±0.12*	2.34±0.09*	2.49±0.16*
AE			
0T	1.19±0.07	1.26±0.06	1.21±0.07
5T	1.24±0.07	1.24±0.09	1.28±0.10
10T	1.23±0.09	1.28±0.13	1.24±0.07

* main effect of time, $p<0.05$ (from 0T); † main effect of time, $p<0.05$ (from 5T).

TR, CE and AE $\dot{V}O_2$max

For the variable of TR $\dot{V}O_2$max, there was a significant main effect for time ($F=17.105$, $p<0.0001$). Tukey post-hoc analyses revealed that TR $\dot{V}O_2$max increased significantly from 0T at the 10T time points and from 5T to 10T. However, there was not a significant increase shown from 0T to 5T. For the variable of CE $\dot{V}O_2$max, there was a significant main effect for time ($F=15.98$, $p<0.0001$). Tukey post-hoc analyses revealed that CE $\dot{V}O_2$max increased significantly from 0T at both the 5T and 10T time points. However, there was not an additional increase in CE $\dot{V}O_2$max from 5T to 10T. AE $\dot{V}O_2$max did not significantly improve over the 10 weeks of training. For each testing mode at all time points, the main effect of training group was not significant. $\dot{V}O_2$max results are presented in Table II.

TABLE III.—*Oxygen consumption at the lactate threshold (L·min⁻¹) for the three training groups during each exercise mode, and training/testing time (mean±SE).*

Test Type/Time	Training group		
	Run	Cycle	Both
TR			
0T	1.73±0.07	1.77±0.09	1.87±0.16
5T	2.08±0.05*	2.03±0.12*	2.06±0.16*
10T	2.14±0.60*	2.04±0.10*	2.21±0.14*
CE			
0T	1.51±0.11	1.32±0.10	1.50±0.08
5T	1.57±0.05	1.51±0.11	1.61±0.07
10T	1.65±0.08*†	1.71±0.13*†	1.85±0.13*†

* main effect of time, $p<0.05$ (from 0T); † main effect of time, $p<0.05$ (from 5T).

TABLE IV.—*Heart rate (bpm) and blood lactate (mmol) during AE for the three training groups during each testing time (mean ±SE).*

Test Type/Time	Training group		
	Run	Cycle	Both
Heart Rate (Bpm)			
0T			
20 W	139±5.4	130±6.5	132±5.7
40 W	156±3.8	152±7.7	154±6.4
5T			
20 W	126±4.5*	124±4.0*	125±3.3*
40 W	145±5.6*	144±5.0*	144±2.3*
10T			
20 W	125±4.4*	123±6.1*	124±1.4*
40 W	144±3.8*	138±7.0*	144±2.5*
Blood Lactate (mMol)			
0T			
20 W	3.6±0.2	3.6±0.4	2.6±0.4
40 W	4.7±0.3	4.0±0.4	4.6±0.4
5T			
20 W	3.2±0.3	3.0±0.3	5.0±1.6
40 W	4.3±0.4	5.3±0.6	5.1±0.6
10T			
20 W	4.1±0.5	3.2±0.6	2.8±0.4
40 W	5.1±0.5	5.3±0.6	4.9±1.0

* main effec of time, $p<0.05$ (from 0T).

TR and CE $\dot{V}O_2$ at LT

For the variable of TR $\dot{V}O_2$ at LT, there was a significant main effect for time ($F=24.69$, $p<0.0001$). Tukey post-hoc analyses revealed that TR $\dot{V}O_2$ at LT increased significantly from 0T at both the 5T and 10T time points. However, there was not an additional increase in CE $\dot{V}O_2$ at LT from 5T to 10T. For the variable of CE $\dot{V}O_2$ at LT, there was significant main effect for time ($F=16.02$, $p<0.0001$). Tukey post-hoc analyses revealed that CE $\dot{V}O_2$ at LT increased significantly from OT at 10T and from 5T to 10T. However, the increase from 0T to 5T was not. For each testing mode at all time points, the main effect of training group was not significant. $\dot{V}O_2$ at LT results are presented in Table III.

AE HR and lactate at 20 and 40 W

For the variable of AE HR at 20 W during AE revealed a significant main effect for time ($F=8.01$, $p=0.002$). Tukey post-hoc analyses revealed that AE HR at 20 W decreased significantly from 0T at both the 5T and 10T time points. However, there was not an additional decrease in the AE HR at 20 watt from 5T to 10T.

Similarly, for the variable of AE HR at 40 W, there was a significant main effect for time ($F=12.84$, $p<0.0001$). Tukey post-hoc analyses revealed that AE HR at 40 W decreased significantly from 0T at both the 5T and 10T time points. However, there was not an additional decrease in the AE HR at 40 watt from 5T to 10T. Although submaximal AE HR was reduced over the training time, there was not a significant difference between the training groups (main effect for group). AE HR data are presented in Table IV.

Although there was a significant reduction in submaximal HR during AE testing, there was not a significant decrease in blood lactate concentration at 20 or 40 W. There were also no significant differences in submaximal lactate between training groups. AE lactate data are presented in Table IV.

Body fat

Percent body fat was assessed using hydrodensitometry at residual lung volume prior to and following the then weeks of training. Because the alpha level was adjusted from 0.05 to 0.005 due to multiple analyses, the minor reduction in percent body fat (main effect of time - 24±6.4 and 22.7±6.5 for the 0T and 10T time points, respectively) was not significant ($F=8.232$, $p=0.0112$). The change in percent body fat and total body weight is presented in Table I.

Statistical power estimate

Statistical power was calculated for the between group sum of squares and degrees of freedom from the $\dot{V}O_2$ and $\dot{V}O_2$LT statistical designs. Using a significance level of 0.05, the sample size of 6, and assuming the sample variance to equal the population variance, statistical power equaled 0.6. When the population variance was assumed to equal the sample variance (0.75 – a more realistic expectation), power equaled 0.68.[15] The power results indicate that although the design did not attain an ideal power level (0.8), it was sufficiently close given the nature of invasive experimental research involving human subjects.[15]

Discussion

To our knowledge, no previous research has applied LT and $\dot{V}O_2$max tests on untrained female during and following a training program of either running, cycling, or both running and cycling. Consequently our results can only be compared and contrasted to trained and untrained data collected on males.

Past research evaluating run and cycle training on measures of $\dot{V}O_2$max and either the LT or VT during run and cycle exercise, respectively, have used one of three common designs; training individuals in either running or cycling,[1-3 16] comparing individuals already highly trained in either running and cycling,[6 17 18] and individuals trained in both running and cycling (i.e.: triathletes).[4 19] Some of these studies have used both male and female athletes,[20 21] while others have focused on the gender differences in energy metabolism between moderately trained to well trained athletes.[22-25] Studies have also investigated CE and AE $\dot{V}O_2$max differences resulting from cycle and arm training.[8-11 26 27]

The methodology of this study adds to previous investigations due to the presence of tun, cycle and run-cycle training groups, the inclusion of an arm ergometer test within the same design, and the use of untrained female subjects. Due to the multifaceted design of this investigation, the discussion well be divided into sections specific to each of the methodologies used.

$\dot{V}O_2$max comparisons

$\dot{V}O_2$max is approximately 0.2 L·min^{-1} (5-10%) greater during TR than CE in trained and/or recreationally active individuals.[28 29] The difference in $\dot{V}O_2$max has been attributed to a smaller actively recruited muscle mass for cycling compared to that of running. Nevertheless, $\dot{V}O_2$max values have been shown to be significantly higher for CE than TR well trained male cyclists.[29] These findings demonstrate the importance of the specificity principle, the metabolic differences between cycle and run exercise, and the fact that muscle mass may not be the crucial determinant of $\dot{V}O_2$max during these two modes of exercise.

The data from this investigation supported previous findings of a larger TR $\dot{V}O_2$max than CE $\dot{V}O_2$max (Table II), and demonstrated that this principle is upheld for untrained females. Consequently, a training specific improvement in $\dot{V}O_2$max was not identified. That is, all subjects improved similarly in both modes of exercise. In studies using male subjects, run training increased both run and cycle $\dot{V}O_2$max, but cycle training only increased cycle $\dot{V}O_2$max.[2] In addition, Robert *et al.*[15] reported similar findings to Pechar *et al.*[2] yet used the test of work capacity (PWC_{150}) rather than $\dot{V}O_2$max. The differences between these studies and our findings could be explained by the variation in exercise training, or indicate that 10 weeks is not long enough for untrained females to develop adaptations specific to their training mode.

LT Comparisons

Previous investigations have shown that the LT and VT are more sensitive than $\dot{V}O_2$max in identifying training specific endurance adaptations in well trained individuals.[6 25] For example, Costa *et al.*[6] and Wither *et al.*[7] revealed that trained runners had a significantly ($p<0.05$) higher VT on the treadmill compared to a similar test on the cycle ergometer. In contrast, the VT in trained cyclists did not differ significantly during TR or CE testing. Specificity data have also been obtained from trained triathletes. Schnieder *et al.*[4] reported similar findings to Withers *et al.*[7] using highly trained triatheletes ($\dot{V}O_2$max=75.4 and 70.3 ml·kg^{-1} min for TR and CE, respectively). However, Albrecht *et al.*[19] reported that the VT and LT results did not differ significantly between exercise modes in moderately trained triathletes ($\dot{V}O_2$max=57.6 and 56.3 ml·kg^{-1}·min for TR and CE, respectively). It is likely that higher levels of endurance fitness are necessary before mode

specificity in $\dot{V}O_2$max and LT measures become pronounced.

Our findings for untrained females do not support the presence of mode specific differences in the LT. Subjects from each group significantly increased $\dot{V}O_2$ at the LT from pre-training values after both 5 and 10 weeks of training, demonstrating no differences between the three training groups (Table III). Although it is interesting that untrained females show significant improvement in both $\dot{V}O_2$max and $\dot{V}O_2$ at the LT in exercise modes in which they are untrained, their initial level of fitness should be considered.

The $\dot{V}O_2$max and $\dot{V}O_2$ at the LT data may indicate that the initial adaptations to endurance training do not show the exercise mode specificity characteristic of well trained endurance athletes, which is inderectly supported by the findings of Albrecht *et al.*[19] It is interesting to speculate on the physiological mechanisms that may have caused our results. Highly trained subjects not only have increased cardiovascular and muscular adaptations, but also further developed neuromuscular recruitment patterns and movement mechanics for specific modes of exercise. Perhaps our previously untrained females were not able to develop the specific mechanical movements that further differentiate cycle form run exercise. Therefore these individuals could not be expected to show maximal and submaximal metabolic differences between the two exercise modes. Regardless, it appears that a low to moderate level of initial fitness and a limited history of prior training decrease the effects of training specificity.

In agreement with the $\dot{V}O_2$max data, the $\dot{V}O_2$ at the LT was also greater for TR compared CE, amounting to 0.48, 0.32, and 0.36 L·min^{-1} for the run, cycle and run-cycle training groups, respectively. In addition, when expressed as a percentage of $\dot{V}O_2$max, $\dot{V}O_2$ at the LT was also greater for TR compared to CE, amounting to 11.2%, 8.6%, and 9.6% for the run, cycle and run-cycle training groups, respectively. This demonstrates that the differences in $\dot{V}O_2$ at LT between TR and CE were not simply due to a higher $\dot{V}O_2$max during TR. As the LT is determined by the kinetics of lactate production and removal during graded exercise, it is likey that lactate metabolism (production and removal) during TR and CE varies as a function of active muscle mass. Aside from the active muscle mass explanation, previous research has not attempted to directly investigate the mechanisms causing the differences in $\dot{V}O_2$ at LT between TR and CE.

Arm ergometry (AE) comparisons

In the present investigation, none of the three training groups showed significant improvements in AE $\dot{V}O_2$max following 10 weeks of run and/or cycle training. Our AE $\dot{V}O_2$max results are in contrast to previous research on males which showed improvements in AE $\dot{V}O_2$max following cycle training.[8,9] Lewis *et al.*[9] reported a 9% ($p<0.01$) increase in AE $\dot{V}O_2$max after 11 weeks of cycle training. However, the results of Klausen *et al.*[30] revealed no significant reductions in submaximal blood lactate accumulation during AE following 5 weeks of cycle training. Clausen *et al.*[10] demonstrated no increases in AE $\dot{V}O_2$max or AE submaximal heart rates following 4 weeks of cycle training.

It is interesting that our data revealed significant reductions in submaximal heart rates during AE for all three training groups. These data were supported by the findings of McKenzie *et al.*,[11] who reported lower submaximal heart rates during AE following 5 weeks of cycle training. It should be recognized that past research was performed using male subjects and not untrained females, and that our results are conclusive due to the large number of subjects who demonstrated this response ($n=18$, $p=0.0016$ and $p<0.0001$ for 20 and 40 W, respectively).

Central and peripheral adaptations

Direct methods used to evaluate central training adaptations have included measurements and/or calculations of cardiac output and stroke volume using either the CO_2 rebreathing method [21,26,31] or echocardiography.[32-34] Peripheral training adaptations have most often been documented from muscle biopsy, and subsequent assays of mitochondrial enzyme activities or histological staining.[35] Nevertheless, indirect strategies to evaluate central and peripheral training adaptations may include the present methodology using $\dot{V}O_2$max and LT techniques during exercise with trained (TR and CE) and untrained (arm ergometry) musculature. It is assumed that if endurance exercise training increases $\dot{V}O_2$max during the trained and untrained exercise mode with no change in the subsequent LT,

then central cardiovascular adaptations most likely have occurred. Con-sequently, if $\dot{V}O_2$ at LT shows an increase in the trained and untrained exercise mode with limited improvements in $\dot{V}O_2$max, then peripheral adaptations best explain the response.

Although there were significant improvements in TR and CE $\dot{V}O_2$max regardless of training group, neither of the training groups showed an improvement in AE $\dot{V}O_2$max. If a significant improvement had been detected in AE $\dot{V}O_2$max following the 10 weeks of training, the development of central adaptations could be better explained. Although AE $\dot{V}O_2$max did not change, the HR response at 20 and 40 W are in agreement with the data of McKenzie.[11]

Based on the concept that submaximal cardiac outputs show limited change as a result of endurance training, the lower submaximal heart rate during AE may have reflected a slight increase in stroke volume. Consequently, the present submaximal AE heart rate data may indicate the presence of minor central adaptations. Previous data by Pelliccia *et al.*[33] had indicated both a lower left ventricular mass and a smaller left ventricular wall thickness in elite level female athletes as compared to males. Whether these data indicate a limitation in central cardiovascular adaptation in female athletes remains inconclusive.

The change in $\dot{V}O_2$ at the LT relative to $\dot{V}O_2$max during TR and CE for all three training groups provides indirect evidence for peripheral training adaptations. Each training group not only increased $\dot{V}O_2$max and $\dot{V}O_2$ at the LT during TR and CE, but also increased the relative intesity of exercise at the LT. Consequently, the peripheral ability to consume oxygen and the metabolism of lactate better explain the significant increase in $\dot{V}O_2$max during TR and CE, and may further indicate the preponderance of peripheral rather than central adaptation in this population.

Energy metabolism

Research has documented a lower glycolytic potential and greater involvement of lipid metabolism in female subjects compared to males of equal training status.[23,24] Interestingly, the maximal blood lactate accumulation (La max) of the female in this study showed a slight increase during TR from 0T to 5T and 10T for each group (from 5.8±0.3 to 6.8±0.4 and 6.6±0.5 mmol·l^{-1}, respectively; N=18). However, no training specific increase in La max occurred in either TR or CE. As the increase in blood La accumulation during TR in each training group was small, it appears that endurance training in previously sedentary females does not appreciably alter their ability to accumulate lactate in blood during exercise to volitional exhaustion, and therefore was not reflective of an increased glycolytic capacity of the muscle.

The combined LT and La max data either indicate a different muscle metabolic response to TR than CE in previously untrained females, or that the variation in active muscle mass influences the LT. The fact that La max showed a slight increase in TR but not in CE from training, and that blood La was greater in TR than CE, support the potential influence active muscle mass may have on the measurement of LT. However, additional research should attempt to clarify this concept.

Conclusions

The results of this investigation indicate that previously untrained females can increase TR and CE VO_2max from either run, cycle, or combined run and cycle training. These increases can be detected after 5 weeks of training, and show further improvement with an additional 5 weeks of training. Unlike previous studies using either recreationally active or well trained male subjects, the current study did not demonstrate a training specific increase in LT (expressed as $\dot{V}O_2$ at the LT).

The stability of AE $\dot{V}O_2$max during the entire 10 weeks of training when both TR and CE $\dot{V}O_2$max increased indicates that central training adaptations were minimal. However, the decrease in the submaximal heart rate response during AE after 5 and 10 weeks of training may indicate minor central adaptations. The increase in $\dot{V}O_2$ at the LT that was not specific to training group indicates that the overall increase in $\dot{V}O_2$max is primarily a result of peripheral training adaptations.

The main recommendation from these data is that previously untrained females can train in either of run, cycle, or run and cycle regimens and derive similar aerobic benefits for up to 10 weeks. Consequently, the initial gains in aerobic adaptation to endurance exercise do not appear to have mode

specificity in this population, and the selection of an exercise regimen need not be biased towards either run or cycle exercise.

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Maximum 365.8 m Swim

Groups	Time #1 (sec)	Time #2 (sec)	La #1 (mmol/L)	La #2 (mmol/L)
S	252.9 ± 3.0	250.2 ± 2.3	12.1 ± 0.7	9.7 ± 0.7*
D	239.3 ± 3.7	233.4 ± 2.4*	13.6 ± 0.8	11.6 ± 0.6*
F	274.1 ± 7.1	269.7 ± 6.2*	10.5 ± 0.9	7.1 ± 0.9*

*p <.05 vs #1

Submaximal 365.8 m swim La also decreased for all groups pre- and post-training, with the largest decrease in the D group. The submaximal and maximal La responses were significantly lower for the F than D or S groups. Despite lower blood La for the S group post-training, no 365.8 m performance improvement was detected. SV increased from pre- to post-training for F across all splits and for D between splits 5–8, indicating greater endurance. The results indicate the limits of submaximal and maximal La testing for the detection of swim performance improvements. The results may also indicate the need for a separate training schedule early in the season for different distance and gender swim specialties.

CROSS TRAINING BETWEEN CYCLING AND RUNNING IN PREVIOUSLY SEDENTARY FEMALES

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Previously sedentary females (n = 18) were assigned to one of three (n = 6) training groups (run = R, cycle = C, both run and cycle = B). Training occurred 4 days·week^{-1} for a total of 10 weeks at 70–85% HRR. Tests for VO_{2max} were performed on each subject during treadmill running (TR), cycle ergometry (CE), and arm ergometry (AE). VO_{2max} tests were performed prior to training (OT), and after training for 5 (5T) and 10 weeks (10T). During the CE and TR tests, venous blood samples were obtained for determination of the lactate threshold (VO_2 @ LT).

VO_2 (ml·kg^{-1}·min^{-1})	Run	Cycle	Both
0T TR	40.81 ± 1.39	37.84 ± 0.99	38.12 ± 0.66
5T TR	43.27 ± 1.76†	39.73 ± 1.47	39.13 ± 0.90
10T TR	45.38 ± 2.24*	40.78 ± 1.69*	41.89 ± 0.85*‡
0T CE	37.62 ± 2.21	34.10 ± 1.80	35.87 ± 2.05
5T CE	39.06 ± 2.45	36.85 ± 2.00†	39.46 ± 0.94†
10T CE	40.62 ± 2.25*	38.36 ± 1.63*	39.49 ± 1.45*

*p <0.05 (0T vs 10T), †p <0.05 (0T vs 5T), ‡p <0.05 (5T vs 10T)

VO_2 @ LT was improved after 10T for the C and B groups during both CE and TR. The R group improved VO_2 @ LT after 5T and 10T during TR with no improvement during CE. There were no significant improvements in AE over the 10 weeks for either of the three groups. Due to the AE and LT results, cross training in previously sedentary females appears to result from peripheral adaptations rather than central improvements over 10 weeks of continuous training.

EFFECT OF CUTANEOUS ELECTRICAL STIMULATION ON QUADRICEPS AND HAMSTRING ISOMETRIC TORQUE PRODUCTION IN HEMIPARETICS

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Five male cerebrovascular accident patients with hemiparesis ($\bar{x}$ age, 64 ± 4 yr) participated in this pilot study to determine the effect of cutaneous neuromuscular electrical stimulation (NMES) on quadriceps (Q) and hamstring (H) peak isometric torque (IsomT) production. Subjects performed unilateral voluntary (VOL) isometric Q and H contractions with the nonimpaired and impaired leg with and without NMES for 5 sec each at 60° of knee flexion on a Cybex II device. Tests were balanced