

# Neural, Metabolic, and Performance Adaptations to Four Weeks of High Intensity Sprint-Interval Training in Trained Cyclists

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## Abstract

The purpose of this study was to investigate the effects of short-term, high-intensity sprint training on the root mean squared (RMS) and median frequency (MF) derived from surface electromyography (EMG), as well as peak power, mean power, total work, and plasma lactate levels in trained cyclists when performed concurrently with endurance training. Seventeen trained cyclists were randomly assigned to a sprint training (S) group ( $n=10$ , age  $25 \pm 2.0$  y) or a control (C) group ( $n=7$ , age  $25 \pm 0.5$  y). Sprint training was performed bi-weekly for four weeks, comprising a total of 28 min over the training period. EMG measurements were taken before and after training during a series of four 30-s sprints separated by four minutes of active recovery. Plasma lactate, peak power, mean power, and total work were measured during each sprint bout. Following sprint training a significant increase occurred in the RMS of the vastus lateralis with a decrease in MF of the same muscle. Values for the

vastus medialis did not change. Pre training exercising plasma lactate values were higher ( $p < 0.05$ ) in C compared to S, but did not change with training. Exercising plasma lactate values increased ( $p < 0.05$ ) from pre to post training in S, but were not different from C post training. Total work output increased from pre to post in S ( $p = 0.06$ ). Peak power, mean power, and  $\dot{V}O_2\text{max}$  increased ( $p < 0.05$ ) pre to post training in S and C, indicating C was not a true control. In conclusion, these data suggest that four weeks of high-intensity sprint training combined with endurance training in a trained cycling population increased motor unit activation, exercising plasma lactate levels, and total work output with a relatively low volume of sprint exercise compared to endurance training alone.

## Key words

Electromyography · motor unit activation · plasma lactate concentration · peak power · total work · recovery · cycling

## Introduction

Cycling is a physically demanding sport requiring high levels of both aerobic and anaerobic power. The aerobic power characteristics of cyclists are well documented [3, 5, 25]. Less documented are their anaerobic power characteristics. Anaerobic power is a major component of competitive cycling and plays a significant role in many aspects including breakaway attempts, hill climbing, and final sprints during competitive bicycle races [24]. In an effort to enhance anaerobic power, cyclists may perform sprint-interval training. Studies involving untrained subjects have shown sprint training improves anaerobic power output, buffer

capacity, muscle oxidative and glycolytic enzyme activity, and  $\dot{V}O_2\text{max}$  [6, 9, 10, 13, 21].

Neural adaptations derived from sprint training may also contribute to enhanced performance. During strength training neural adaptations have been shown to precede morphological adaptations [12, 16, 20]. These neural adaptations include increased muscle fiber recruitment, firing rate, and motor unit synchronization [8, 12, 15] resulting in the ability to exert more force. Paavolainen et al. [17] found that simultaneous explosive-type strength training, including sprint training, and endurance training led to improved 5K run times in well-trained endurance ath-

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Accepted after revision: May 10, 2003

## Bibliography

Int J Sports Med 2004; 25: 92-98 © Georg Thieme Verlag Stuttgart · New York · ISSN 0172-4622

letes in the absence of improvements in  $\dot{V}O_2\text{max}$ . Although not directly measured, it was suggested that these improvements might be due to improved neuromuscular characteristics that transferred into improved maximal anaerobic velocity and running economy.

Sprint training studies have confirmed increases in muscular power as measured by a 45-s maximal cycle ergometer test [21], a 15-s cycle ergometer power test [23], a 3-s cycle sprint test [10], a dynamic knee extensor test [1], and various sprints [1,6]. Of these studies, only Sleivert et al. [23] and Zhou and associates [27] collected electromyography (EMG) data to investigate possible neural adaptations associated with sprint training. Unfortunately, they failed to show any differences in EMG activity following a period of sprint-interval training. In both cases, however, EMG was measured during maximal isometric contractions instead of a more cycling specific exercise.

According to Coyle et al. [4], improvements in peak torque following resistance training at different velocities were velocity specific. These data suggest that training should be event specific. In a similar manner, testing should also be performed in a manner that specifically mimics the performance action to most accurately assess possible adaptations. In the present study, EMG recordings were taken during the actual sprint testing in an attempt to qualify the contribution of neural adaptations to sprint training.

Little data are available on the neural effects of performing sprint training; furthermore, this is the first study to our knowledge to measure EMG variables during an actual sprint exercise. In light of the importance of neural adaptations in the improvement of other forms of power activities [8,12,16,20] and in view of the fact that previous studies that cite neural adaptations resulting from sprint training failed to measure EMG in a sprint-like manner [23,27], it is important to obtain more information on the effects of short-term, high-intensity sprint training on the neuromuscular system in trained cyclists to determine if those adaptations factor into improved sprint performance. Therefore, the primary purpose of this study was to investigate the effects of short term, high-intensity sprint training performed in conjunction with endurance training on the root mean squared (RMS) and median frequency (MF) derived from EMG, peak power, mean power, total work, and plasma lactate levels during a series of 30-s maximal sprints compared to endurance training alone in trained cyclists.

## Material and Methods

Seventeen trained cyclists [11] from the local population were randomly selected to a sprint training (S) group ( $n=10$ , age  $25.1 \pm 2.3$  y, height  $178.5 \pm 7.0$  cm, weight  $69.0 \pm 5.2$  kg, body fat  $10.1 \pm 4.0\%$ ,  $\dot{V}O_2\text{max}$   $3.9 \pm 0.3 \text{ l} \times \text{min}^{-1}$ , training  $5.0 \pm 1.1 \text{ h} \times \text{wk}^{-1}$ ) or a control (C) group ( $n=7$ , age  $24.5 \pm 0.5$  y, height  $178.3 \pm 7.5$  cm, weight  $68.9 \pm 5.9$  kg, body fat  $9.0 \pm 3.4\%$ ,  $\dot{V}O_2\text{max}$   $4.1 \pm 0.5 \text{ l} \times \text{min}^{-1}$ , training  $8.0 \pm 2.0 \text{ h} \times \text{wk}^{-1}$ ). Subjects had at least two years of cycling experience and had been involved in previous training programs.

All subjects were tested pre and post training, with testing being performed over a seven day period before and within 48 hours after completing training. Testing included body composition analysis,  $\dot{V}O_2\text{max}$  determination, and a repeated sprint test. Furthermore, a minimum period of 48 hours was allowed between tests and training bouts to allow for adequate recovery. During the study one subject from C was excluded because of injury. All subjects were informed of risks and benefits of the investigation prior to signing an informed consent letter. This study was approved by the Human Subjects Institutional Review Board of Brigham Young University.

## Anthropometric measurements

Each subject's age, height, and weight were recorded prior to any experimental testing. Body composition was determined by air displacement plethysmography (Bod Pod, Concord, CA).

## Aerobic power measurement

To determine aerobic power, subjects performed a graded exercise test protocol to determine maximal oxygen consumption ( $\dot{V}O_2\text{max}$ ). All training and testing was performed on an electromagnetically braked cycle ergometer (Lode Excalibur, Lode, Groningen, The Netherlands). Ergometer seat height was adjusted and subjects were allowed a five minute warm up period at an intensity of 100 watts (W). Immediately following the warm-up, subjects began testing by cycling at 150 W for three minutes; with resistance being raised by 25 W each additional minute until  $\dot{V}O_2\text{max}$  was achieved. Three criteria were used to determine  $\dot{V}O_2\text{max}$ ; reported rate of perceived exertion (RPE) of 20, respiratory exchange ratio (RER)  $> 1.5$ , and a plateau in  $\dot{V}O_2\text{max}$  readings. Expired gases were measured and analyzed for the determination of expired ventilation (VE), oxygen consumption, carbon dioxide production, and RER using a metabolic cart (Consentius Technologies Inc., Salt Lake City). Oxygen and  $\text{CO}_2$  analyzers were calibrated prior to each test using standard gases of known concentration. Heart rate was monitored continuously by radiotelemetry (Polar Electro Inc., Port Washington, NY) and recorded at the end of each stage along with RPE. Ventilatory threshold (VT) was determined from a non-linear increase in VE during the incremental test [22].

## Anaerobic power measurements

Anaerobic power testing included performing a series of four, 30-s maximal sprints separated by four minutes of active recovery at 50 W ( $\leq 75$  RPM). Prior to sprint one, subjects were prepared for EMG electrode placement and had their right knee fitted with a potentiometer calibrated to track joint angle from minimum ( $0^\circ$ , top of the pedal stroke) to maximum ( $180^\circ$ , bottom of the pedal stroke) throughout the pedal stroke. After the ergometer seat height had been adjusted, a blood sample was drawn and the subject began a four-minute warm-up at 100 W with the ergometer set in hyperbolic mode, allowing for work to be performed at an absolute wattage. Upon completion of the warm-up, the subject performed a series of four sprints with the ergometer set in linear mode. In this mode, resistance was dictated by pedal cadence in place of working against an absolute work load. For each sprint, work (kJ) was recorded at five-second intervals, and total work was recorded at the end of the 30-s period. From these values peak power and mean power output were calculated. Fatigue index was calculated by taking

the ratio of the lowest and highest 5-s power outputs. Total work was recorded as the number of kJ accumulated in the 30-s period. Subjects were required to remain seated throughout the sprint test.

### Electromyography procedures

EMG signals of the right vastus lateralis (VL) and vastus medialis (VM) were recorded using a Noraxon Telemetry System (Noraxon USA Inc., Scottsdale, AZ) during repeated sprint testing. The EMG signals were recorded with bipolar Ag/AgCl circular surface electrodes (Noraxon Dual Electrode, Product #272) with a one cm diameter-recording surface and a fixed two cm (center to center) inter-electrode distance. The electrodes were aligned parallel to the muscle fibers over the belly of the muscle. An electrode positioned mid-shaft on the medial side of the tibia served as a ground. The position of each electrode was marked with a small dot and transferred along with other marks (angiomas and/or scars) on the subject's skin to transparent sheets. These sheets were used to ensure proper electrode placement for subsequent testing. Impedance at the electrode site was reduced by shaving the site, abrading the site with a pad to remove oil and dead skin, and cleansing the site with isopropyl alcohol.

Electromyographic signals were recorded with a sampling frequency of 1000 Hz using a Dell Latitude P-450 computer interfaced to a Keithley-Metrabyte KPMCIA-16, 12-channel, 16-bit analog-to-digital converter (Keithley Instruments Inc., Cleveland, OH). The EMG signals were differentially amplified with a gain of 1000 and a bandwidth of 16–500 Hz at –3 dB using the Noraxon Telemetry System previously described. The system amplifiers have an input noise below 1  $\mu$ V root mean squared (RMS) and an effective common mode rejection ratio of 85 dB. Root mean squared and MF values of each muscle derived from each pedal stroke were summed and averaged to obtain a mean value of these variables for the 30-s sprint period.

### Median frequency

The MF was computed from the raw EMG signals using a 200 point short-term Fast Fourier Transform. Each EMG segment was multiplied by a Blackman taper window and zero padded to obtain a frequency resolution of 2 Hz for each interval.

A power spectral analysis of each muscle was computed from the raw EMG signals using a 200 ms point Fast Fourier Transform. The MF of the power density spectrum was then calculated [14].

### Blood lactate measurements

For blood sampling procedures, a flexible Teflon catheter was inserted into a forearm vein under sterile conditions. The skin was cleansed with isopropyl alcohol and investigators wore latex gloves throughout the procedure. Blood samples were taken with the subject on the bike and the catheterized arm extended. A three ml sample of blood was taken under sterile conditions from the antecubital vein prior to sprint testing and three minutes following each sprint bout, with additional samples taken at six and nine minutes after the final sprint. To keep the line patent the catheter was flushed with one ml of sterile saline after each blood sample was taken.

Each whole blood sample was centrifuged and the plasma was transferred to a separate tube for subsequent lactate analysis. Samples were stored at –80°C. Plasma lactate concentration was analyzed in triplicate using an Analox Micro-Stat GM7 analyzer (Analox Instruments, Lunenburg, MA).

### Training protocol

Subjects in the S group performed two sprint training sessions per week separated by at least 48 hours. Ergometer seat height, determined during initial sprint testing, was maintained for each subject prior to training. Each training session began with a five-minute warm-up at 100 W. Upon completion of the warm-up each subject completed a series of 30-s all out sprints separated by four minutes of active recovery. Active recovery was performed at 50 W at a cadence  $\leq$  75 RPM to assist in recovery of power output between sprints by increasing blood flow to the exercised muscle [2]. Similarly to the sprint trials, warm-up and recovery periods were performed with the ergometer set in hyperbolic mode, allowing for work to be performed at an absolute work load. Sprints were performed with the ergometer in linear mode in which resistance is determined by pedal cadence. Week one included four 30-s sprints per training session, with two additional sprints added to each training session every week. By week four a total of ten sprints were performed per training session. A total of 28 minutes of sprint training was completed over the training period.

Average power output and total work from each sprint trial were recorded to quantify training and track progress throughout the training period. Subjects in both the S and C groups were required to maintain their pre-study level of endurance training throughout the course of the study. Each subject was given a training log to record training durations. Training data were reported on a weekly basis.

### Statistical analysis

A  $4 \times 2 \times 2$  repeated measures design with six dependent variables was employed. The factors of sprint trial, group, and pre-post condition were the independent variables with the dependent variables being RMS, MF, peak power, mean power, total work, and fatigue index. For plasma lactate values, a  $6 \times 2 \times 2$  repeated measures design with the dependent variable being plasma lactate concentrations following exercise and recovery was employed. Ventilatory threshold and  $\dot{V}O_{2\max}$  values were analyzed using a  $2 \times 2$  repeated measures design. Peak power, mean power, total work, fatigue index, RMS, and MF were analyzed for each condition using a three factor ANOVA with repeated measures on all three factors. Training volumes, resting lactate concentrations, and pre  $\dot{V}O_{2\max}$  values were compared using a t-test.

In the event of interaction a Tukey's post-hoc analysis was used to make all pairwise comparisons to locate significant differences. For all statistical analysis in this study, a p-value  $< 0.05$  was used to establish significant differences. Results are reported as the means  $\pm$  standard deviation (SD) unless otherwise indicated.

## Results

### Electromyography

Four weeks of intense cycle sprint training led to an overall increase ( $p < 0.05$ ) in muscle fiber recruitment as demonstrated by elevated RMS values in the VL of S (Table 1). In addition, when values from all four sprint trials were averaged, in pre and post conditions, MF of the VL in S declined over the course of training ( $p < 0.05$ ). Root mean squared and MF values in the VM of S did not change with training. Likewise, no changes were detected in EMG measurements in either muscle in C. Furthermore, there were no differences in RMS or MF in either muscle between sprints during pre or post testing.

### Sprint performance variables

Values for peak power, mean power, and total work are displayed in Table 2. There was a main effect for peak and mean power when the means for each sprint were combined, indicating a significant increase in peak (6%, 4%) and mean power (6%, 3%) from pre testing to post testing in S and C. In addition, all subjects responded similarly in power output during sprint testing, showing decrements from sprint 1 to sprint 2 and sprints 2 to 3, ( $805 \pm 105.0$  to  $694 \pm 73.5$  to  $628 \pm 80.5$ , respectively) ( $p < 0.05$ ) with no decrement between sprints 3 and 4 ( $628 \pm 80.5$  to  $589 \pm 85.3$ ). Fatigue index for the repeated sprint test for all subjects was different only between sprint 1 ( $66 \pm 13.5\%$ ) and sprint 4 ( $74 \pm 13.4\%$ ) ( $p < 0.05$ ), and was unaffected by the training protocol. No interaction occurred between groups for peak or mean power.

In response to the four weeks of sprint training, mean total work derived from the four sprint trials increased 7% in S ( $p = 0.06$ ) (Table 2). Pre testing total work in C was greater than S ( $16.49 \pm 2.5$  to  $15.54 \pm 2.4$ ) ( $p = 0.06$ ), however, total work values in C did not change.

### Aerobic power characteristics

Subjects did not differ in  $\dot{V}O_{2\max}$  prior to the study, however, there was a general increase in  $\dot{V}O_{2\max}$  for both groups following the four week training period ( $4.0 \pm 0.4$  to  $4.2 \pm 0.4 \text{ l} \times \text{min}^{-1}$ ). When combined, mean ventilatory threshold values occurred at a higher percentage of maximum heart rate in C compared to S ( $89.1 \pm 3.5$ ,  $92.2 \pm 2.1\%$  MHR) ( $p < 0.05$ ), however, VT did not change over the course of the study in either group.

### Plasma lactate measurements

Plasma lactate values are represented in Table 3. Resting plasma lactate values were not different between groups. As expected with repeated sprint performance, there was an overall time effect for increased plasma lactate levels from pre to post exercise in each group ( $p < 0.05$ ). Sprint training resulted in a general increase in the mean exercise plasma lactate concentrations in S from pre to post training ( $p < 0.05$ ). Pre testing revealed greater mean exercise plasma lactate values in C vs. S ( $20.0 \pm 3.7$  to  $18.2 \pm 3.1 \text{ mmol} \times \text{l}^{-1}$ ) ( $p < 0.05$ ), but no changes occurred in C between pre and post testing. Furthermore, there were no changes in post lactate values between the two groups.

**Table 1** Changes in root mean squared (RMS) and median frequency (MF) activity in the vastus lateralis (VL) and vastus medialis (VM) of the Sprint training (S) and control (C) groups prior to and following 4 weeks of cycle sprint training

Group	VL		VM	
	Pre	Post	Pre	Post
S RMS	848.0 ± 142.2	1083.2 ± 98.1*	1411.4 ± 183.7	1312.3 ± 203.9
C RMS	1113.8 ± 140.9	1116.6 ± 127.9	936.6 ± 120.7	1081.4 ± 159.2
S MF	95.1 ± 9.1	79.1 ± 4.9*	97.6 ± 5.8	93.2 ± 5.8
C MF	89.06 ± 11.6	87.4 ± 8.4	99.8 ± 3.4	98.4 ± 4.0

Values represent average of all four sprint trials in pre and post conditions.

\* Denotes pre different from post in the VL ( $p < 0.05$ ).

**Table 2** Changes in peak power, mean power, and total work in S and C following 4 weeks of cycle sprint training

	Peak power (W)	%Δ	Mean power (W)	%Δ	Total work (kJ)	%Δ
	S pre	646.5 ± 74.5		515.0 ± 36.5		15.5 ± 1.1
S post	690.0 ± 74.5*	6.0%	544.8 ± 36.7*	6.0%	16.5 ± 1.3†	6.0%
C pre	682.5 ± 98.3		549.6 ± 58.7		16.5 ± 1.8**	
C post	712.5 ± 99.9*	4.0%	563.5 ± 60.5*	3.0%	16.9 ± 1.8	2.0%

Values represent the average of all four sprint trials in pre and post conditions.

\* Denotes pre different from post ( $p < 0.05$ ), \*\* denotes C pre was different than S pre ( $p = 0.06$ ); † denotes S pre was different from S post ( $p = 0.06$ ).

### Training volume

Training outside the lab was maintained at  $5.0 \pm 1.1 \text{ h} \times \text{wk}^{-1}$  in S ( $4.0$  to  $7.0 \text{ h} \times \text{wk}^{-1}$ ). Total training volume in C averaged  $8.0 \pm 1.7 \text{ h} \times \text{wk}^{-1}$  ( $6.2$  to  $10.5 \text{ h} \times \text{wk}^{-1}$ ), and was significantly higher than S ( $p < 0.05$ ). For S, actual sprint-interval training in the lab increased from 4–10 min over weeks 1–4 for a total of 28 min, and accounting for an increase of 1.7–3.3% in total training volume, respectively. Similar to testing, sprint training bouts were separated by four minutes of active recovery at 50 W. During recovery, subjects pedaled at a cadence of  $\leq 75$  RPM. Based on pre training  $\dot{V}O_{2\max}$  workloads, recovery periods represented a load of approximately 20% of  $\dot{V}O_{2\max}$ .

### Discussion

The purpose of this study was to investigate neural, metabolic, and performance adaptations to intense sprint interval training in trained cyclists. This population was selected to increase the applicability of the results obtained from introducing a short-term sprint-training stimulus into endurance training performed by trained cyclists. The results of the present study demonstrate that performing a total of only 28 minutes of intense sprint-interval training in conjunction with regular endurance training over a four-week period was sufficient to increase motor unit recruitment, plasma lactate values, and total work over a series of 30-s sprint bouts compared to performing endurance training alone in trained cyclists.

**Table 3** The effect of consecutive sprint bouts on plasma lactate levels. Samples were taken prior to exercise (Pre), 3 minutes following the completion of each sprint (P1, P2, P3, P4), and at 6 and 9 minutes following sprint 4 (R1, R2)

	Pre	P1	P2	Plasma lactate concentration (mM)		R1	R2	Mean
				P3	P4			
S pre	1.4±0.4	14.1±2.8	17.4±2.4	19.6±2.2	20.2±2.0	19.9±2.6	18.0±2.2	18.2±2.4
S post	1.8±0.6	14.3±4.6	18.5±3.9	20.8±3.0	22.0±2.7	21.0±2.2	20.0±2.5	19.4±3.1*
C pre	1.7±0.3	14.2±1.9	18.7±2.8	20.8±1.4	23.1±3.5	22.5±2.5	20.8±2.1	20.0±2.4**
C post	1.6±1.0	13.2±1.4	17.6±2.0	20.9±0.7	21.5±0.7	20.9±1.1	19.6±3.2	18.9±1.5

Mean values represent the exercising plasma lactate values (P1–R2). \* represents a significant difference in exercising plasma lactate values from pre to post training in S ( $p < 0.05$ ). \*\* denotes a difference in pre mean exercising plasma lactate values in C vs. S ( $p < 0.05$ ).

Amplitude of the VL RMS increased in S from pre to post testing. Over the same period, MF decreased in the same muscle. These data demonstrate that the sprint stimulus applied was sufficient to elicit neuromuscular changes and are consistent with other studies that have shown increases in motor unit recruitment, firing rate, and synchronization [8, 12, 15] in response to training. Synchronization of motor units yields a simultaneous increase in the amplitude and decrease in the frequency of the EMG signal [19, 26], resulting in force potentiation, which improves efficiency and coordination. Improved efficiency, due to neural alterations, may have delayed the onset of fatigue, enabling the S group to tolerate the increased levels in plasma lactate production (Table 3). Furthermore, neural adaptations observed in S (Table 1) may have also contributed to improvements in power and work production by means of increased motor unit recruitment. Similar results have been reported in elite runners who have combined explosive-strength training, including sprint training, with regular endurance training [17]. In these runners, 5-km run times were improved compared to a control group performing endurance training alone. Although no EMG measurements were made, improvements were suggested to be neuromuscular, leading to improvements in peak velocity and running economy.

In contrast to our current findings, two previous studies involving EMG measurements in combination with sprint cycle training detected no changes in EMG, although power variables such as peak power and total work improved in both studies [23, 27]. In each case, EMG was measured during isometric contractions performed both pre and post sprint training. It was suggested that the inability to detect changes in EMG was due to a lack of transfer from cycle sprint training to maximal isometric contractions [23]. It has also been suggested that isometric contractions may be too insensitive to detect changes induced by cycle sprint training [27]. In the present study, EMG measurements were taken during the actual sprint exercise in an attempt to more accurately determine neural adaptations in response to sprint training. Due to the dynamic nature of the pedaling motion, attempts were made to minimize the possible confounding effects of muscle velocity and length changes [7] by analyzing the EMG from a 200 ms fixed segment of the pedal stroke (50 ms prior to the minimum knee joint angle, represented as the top of the pedal stroke, to 150 ms into knee extension during the down stroke). This portion of the pedal stroke was selected because it was the point at which the least amount of muscle shortening occurs and where muscle contraction velocity is slowest.

Although changes occurred in the VL, no changes occurred in RMS or MF of the VM. Pincivero and associates [18] studied the effects of performing a series of nine progressively more difficult isometric contractions on the MF of the VL, VM, and the rectus femoris (RF) muscles. Overall MF over the course of the contractions was increased in the VL, with negligible changes occurring in the VM and RF muscles. It was suggested that muscle fiber homogeneity may exist between the VM and RF muscles preventing notable changes from occurring with increased contraction intensity, whereas the VL may possess greater morphological variability, contributing to the changes reported. Although VL MF was shown to decrease in the present study, changes in EMG variables were unique to the VL as opposed to the VM. These data suggest that increased fiber type variability in the VL compared to the VM may have enhanced the ability of the VL to respond to a short term, intense training stimulus. Reasons for the discrepancy between the present MF data and that reported by Pincivero et al. [18] could possibly be explained by increased motor unit synchronicity derived from sprint training as mentioned previously.

Blood and muscle lactate values tend to increase when measured after cycle sprint training [9, 21]. Currently, C pre test exercising plasma lactate values were higher than S, however, following sprint training exercising plasma lactate values increased in S. Post training exercise plasma lactate values remained consistent in C. An increase in plasma lactate concentration post training coincided with increased peak power, mean power, and total work in S. Sharp et al. [21] reported an increase in blood lactate levels and total work performed during a 45-s maximal cycle sprint after eight weeks of intense sprint training in untrained subjects. These data were reported in conjunction with an increase in the glycolytic enzyme phosphofructokinase (PFK), suggesting that increased lactate and total work values were due to improved glycolytic output. Increases in glycolytic enzymes such as hexokinase and PFK due to sprint training have been shown as well [13], indicating that increases in plasma lactate and total work values in the current population may be due to improved glycolytic function. Interestingly, Sharp and colleagues [21] saw no change in muscle pH in spite of increased lactate levels suggesting an increase in buffer capacity, which may have also contributed to increases in total work in the current population in spite of increased plasma lactate levels.

Total work increased from pre to post training in S ( $p = 0.06$ ) with no apparent change in fatigue index. Total work performed by C during pre testing was higher compared to S by the same probability level; however, C did not change from pre to post training. Increases in total work due to sprint training have been reported in studies ranging from 7–8 weeks of training, and involving 30-s, 45-s, and repeated 30-s maximal cycle sprint bouts [9,13,21,27]. Interestingly, improvements in the current subjects occurred in only four weeks. Reasons for improved work outputs over these brief periods of intense exercise include improved glycolytic enzyme activity, oxidative enzyme activity, buffer capacity, and  $H^+$  clearance [9,13,21]. In the present study, total work improvements were seen in connection with increased motor unit recruitment and plasma lactate levels.

Fatigue index did not change with training in either group, suggesting that the training protocol may not have effectively attenuated fatigue, however, four weeks of high-intensity sprint-interval training did improve work production in S, indicating the ability to produce more work prior to fatigue.

Further improvements in S include increases in peak power, mean power, and  $\dot{V}O_{2\max}$ , with VT being unaffected by training. Interestingly, these increases were also seen in C making the control group a limiting factor in this study. The purpose of the applied control in this study was to allow for greater generalization to a cycling population, however, performance improvements in C clearly indicate that an overload training stimulus occurred. In an attempt to control for these improvements subjects were instructed to maintain their pre-study training volume for the duration of the study. Despite a greater weekly training time in C compared to S, statistical analysis demonstrated that the weekly hours of training remained consistent throughout the study period within both groups. These data suggest that the cyclists were in a maintenance phase of training, indicating no addition or subtraction from pre-study weekly training volume, thus making an overload stimulus in C unlikely regardless of differences in training time between groups. In the case of C, consideration must be given to the nature of exercise performed. The control group was training out of doors on bicycles where many factors are involved compared, for example, to a resistance training setting in which case administrators may closely monitor volume and load. Training out of doors and over long distances increases the likelihood of confounding training variables including, wind direction and speed, terrain, and temperature, all of which are factors that could have led to an overload stimulus but would go unreported in an hourly reporting format. In spite of efforts to control for the endurance stimulus, improvements in C may have limited our ability to identify true adaptations in S. Results of this study are presented in light of the several alterations in C.

In spite of the failure of C to be a true control, improvements were seen in motor unit recruitment, plasma lactate production, and total work in S. These data suggest that the incorporation of a sprint training protocol into regular endurance training did lead to changes in S.

In conclusion, this is the first investigation, to our knowledge, to utilize EMG during a dynamic exercise to assess neural adapta-

tions to sprint training. Results from the present investigation suggest that four weeks of high intensity sprint training combined with endurance training in a trained cycling population increases neural activation, plasma lactate concentrations, and total work during a series of high intensity sprint bouts. In the four week period a total of 28 min of sprint training was performed, representing an increase of only 1.7 – 3.3% of total training volume. These data demonstrate that a relatively small, high intensity sprint stimulus improves neuromuscular and metabolic function leading to improvements in repeated sprint work performance in trained cyclists.

## Acknowledgements

These data were previously published in abstract form:

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