Changes in Acute Skeletal Muscle Oxygenation and Blood Flow in Trained and Untrained Males During 10RM Resistance Protocol Measured by NIRS.

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 observe muscle oxygenation and blood volume changes and half time to full O_2 saturation elicited by 10RM biceps exercise in trained (ages 23.8 ± 3.5 years, sum of 4 arm skinfolds 15.2 ± 3.7 mm, arm girth 35.7 ± 3.0 cm) and untrained subjects (ages 25.3 ± 3.6 years, sum of 4 arm skinfolds 16.6 ± 3.2 mm, arm girth 28.4 ± 3.1 cm). 22 males volunteered for the study and were self-assigned to a trained (N=13) or untrained (N=9) group based on training experience. Each subject completed 3 sets of a 10RM exercise protocol using biceps curls, and a base-line muscle ischaemia protocol on separate days that were randomly determined. Blood volume, tissue oxygenation and half-time to full O_2 saturation of the right biceps brachii muscle were monitored non-invasively using a near-infrared spectrophotometer (NIRS) in both protocols. The NIRS probe was positioned approximately at the mid-point of the belly of the biceps brachii muscle. Both the trained and untrained subject groups showed decreases in blood volume and tissue oxygenation during each set of exercise of the 10RM protocol. No set-wise differences were seen within the trained and untrained subject groups during the 3 sets of right arm biceps curl exercise at a load of 10RM. In addition, no differences were seen between trained and untrained groups in a set-wise comparison of muscle oxygenation and blood volume. However, a statistical significant difference was seen between the group mean percentage change in muscle oxygenation of the untrained subject group and the warm-up set and the 3 sets of 10RM for the trained subject group. No set-wise differences were found within the trained and untrained subject groups in the half-time to full O_2 saturation during the exercise protocol. In

addition, no differences were seen between trained and untrained groups in a set-wise comparison of the half-times to full O_2 saturation. It was concluded that blood volume and oxygenation decrease during resistance exercise but the decrease appears to be independent of the training status of the subjects.

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DEDICATION

I dedicate this thesis to my children Mason and Paige, my Sun and my Moon, who help me make sense of the world around me and for who I'll make a better world.

INTRODUCTION

Improvements in athletic power (force x velocity) and strength are of great importance to athletes and coaches as they aid in the development of optimal performance in sport (Sale, 1992). Resistance training (RT), modeled after body-building RT, is a common protocol used to increase athletic power, thereby, enhancing sport performance (Armstrong, Warren, & Warren, 1991). These RT protocols typically consist of 10 repetitions at approximately 70-75% of 1 maximal concentric contraction (MVC) for 3 sets (Poliquin, 1990). Muscle tissue displays adaptability to various stimuli brought about by RT, such as hypertrophy (an increase in muscle cross sectional area). However, the adaptations of muscle tissue is dependent on the type of stimulus it receives, usually related to load, repetition, time of contraction and recovery. It is, therefore, important to identify the physiological stimuli which will bring about desirable physiological adaptations in muscle tissue that enhance the physical development of athletes.

Muscle fibers exist in a continuous spectrum based on the force production, the duration of muscle contraction and the predominant bio-energetic system. Type I muscle fibers or slow twitch (ST) are characterized by low tetanic force production over an extended period of time and driven primarily by high concentrations of oxidative enzymes. In contrast, Type IIb fibers or fast twitch (FT) are characterized by large tetanic force productions of short duration and fueled primarily through glycolytic enzymes (Hintz, Chi, Fell, Ivy, Kaiser & Lowery, 1982). An individual may increase their absolute strength through resistance training (RT) emphasizing a moderately high load and a high number of repetitions (Armstrong, Warren & Warren, 1991). Type IIa or

fast–twitch oxidative gylcolytic muscle fibers (FOG) lie between the two polar extremes of ST and FT fibers. FOG are characterized by moderate force productions of long duration and high levels of both oxidative and glycolytic enzymes (Young & Lowry, 1983). RT increases muscle contractile protein content primarily in Type IIb muscle fibers through an increase in cross-sectional area, thus increasing strength and enabling a higher level of athletic performance (Sale, 1992).

MacDougall, Sale, Elder and Sutton (1982) and Tesch and Karlsson (1985) have shown a fiber type shift (FTS) from FT to FOG in elite male bodybuilders and attributed it to prolonged RT. Normally, oxidative adaptations are associated with endurance training not RT. RT would decrease the oxidative capacity of muscle by decreasing the relative capillary and mitochondria densities and increase O_2 diffusion gradients as a result of increased muscle size due to hypertrophy (Tesch & Larsson, 1982). Despite the above biochemical and anatomical changes, a shift in the oxidative capacity of Type II muscle is still considered a result of RT (Tesch, Thorsson & Kaiser, 1984).

These fiber type shifts may have direct implications to athletes who require muscular power for successful performance (i.e. rugby, hockey and football players). Although RT will improve the absolute strength of an athlete through muscle hypertrophy, a FTS from FT to FOG may not result in any beneficial gains in athletic power, as oxidative muscles tend to produce lower forces at reduced contraction speeds (Eiken, Sundberg, Esbjornsson, Nygren, & Kaijer, 1991). As a result, traditional RT programs, employed by athletes to enhance power, may in fact impede or decrease the muscular power of an athlete as oxidative adaptations are also associated with lower and slower rates of force production (Hintz *et al.*, 1982).

FTS has been attributed to a state of anoxia, due to circulation restrictions during RT (MacDougall *et al.*, 1982; Tesch & Karlsson, 1985). Recently, Tamaki, Uchiyama, Tamura and Nakano (1994) hypothesized that FTS are the result of a training regimen emphasizing moderate loads and a high number of repetitions per set typically employed by bodybuilders. The duration of this type of RT would create a prolonged state of muscle anoxia as a result of blood occlusion associated with muscle contractions of greater than 60% MVC. To "noninvasively" test his theory Tamaki *et al.* (1994) directly monitored and measured the level of muscle oxygenation during RT using near-infrared spectroscopy (NIRS). The relative changes in optical density absorbency (OD) were measured and reflected the volume changes in oxygenated hemoglobin ($\Delta[HbO_2]$), deoxygenated hemoglobin ($\Delta[Hb]$) and blood volume ($\Delta[HbO_2+Hb$) of the biceps brachii muscle in adult men of various training levels. Similar changes in OD for $[HbO_2]$, [Hb] and $[HbO_2+Hb]$ were seen during RT exercise when the blood flow to the biceps brachii muscle of the subjects was partially occluded via tourniquet. During the 10RM protocol, the trained subjects exhibited significantly higher levels of oxygen desaturation and significantly higher levels of $[HbO_2+Hb]$ than the untrained subjects. Thus, based on the observations obtained by Tamaki *et al.* (1994), the trained subjects may be more efficient at mobilizing and utilizing oxygen during RT due to a FTS. The results obtained by Tamaki *et al.* (1994) also seem to support observations by Eiken *et al.* (1991), Gollnick and Saltin (1982), Terjung, Mathien, Erney and Ogilvie (1988) and Jansson and Kaijser (1978) who have also reported an increased percentage in the muscle cross sectional area (CSA%) of slow twitch muscle when muscle tissue was under ischaemic conditions.

$\Delta[HbO_2]$, $\Delta[Hb]$ and $\Delta[HbO_2+Hb]$ (Table 1). The trained subjects also displayed a net deficit in skeletal muscle O_2 saturation during RT. This may have been the result of a greater utilization of both vascular O_2 and myoglobin stores of O_2. These results are contrary to observations noted by Lind & Willams (1978), who concluded that blood flow is occluded by mechanical restrictions during muscular contractions of greater than 60% MVC and are not dependent on the training state of the individual. The RT protocol in the Tamaki *et al.* study (1994) had all subjects perform one arm curls exercise at 75% of their 1RM. Therefore, no significant differences in $\Delta[HbO_2]$, $\Delta[Hb]$ and $\Delta[HbO_2+Hb]$ should be observed between trained and untrained subjects. In addition, the rate at which the dependent variables changed over time reflected a greater utilization of O_2 in the trained group during "anaerobic" muscular contractions.

Table 1: Sample size (N), mean peak optical density (OD), standard deviations (SD) and probability of two independent sample T-test (P) of trained (T) and untrained (U) subject groups for oxygenated hemoglobin ($\Delta[HbO_2]$), deoxygenated hemoglobin ($\Delta[Hb]$) and blood volume ($\Delta[HbO_2+Hb]$) optical density absorbency during 10 repetition maximum curl exercise (data obtained from Tamaki, 1994).

	$\Delta[HbO_2]$-T	$\Delta[HbO_2]$-U	$\Delta[Hb]$-T	$\Delta[Hb]$-U	$\Delta[HbO_2+Hb]$-T	$\Delta[HbO_2+Hb]$-U
N	6	3	6	3	6	3
OD	0.52	0.27	0.38	0.26	0.14	0.01
SD	0.045	0.029	0.021	0.012	0.026	0.010
P	(0.00010)		(0.0020)		(0.0000)	

In order for RT to be an anoxic stimulus for FTS, an anoxic state must be present in the untrained subjects. However, untrained subjects do not experience the same relative level of anoxia during RT as trained subjects. Blood occlusion during RT may not provide the stimuli for muscle FTS as theorized by Tamaki *et al.* (1994). Therefore, the purpose of this study was to: 1) measure the muscle oxygenation, blood flow and the

half-time to full O_2 saturation of trained and untrained subjects during 10RM one arm curl exercise using NIRS, 2) statistically compare the muscle oxygenation, blood flow and the half-time to full O_2 saturation of trained and untrained subjects during 10RM one arm curl exercise, 3) determine if the training status of the subjects has an effect on the muscle oxygenation, blood flow and the half-time to full O_2 saturation of trained and untrained subjects during 10RM one arm curl exercise and 4) to assess if the above dependent variables as credible indicator or predictor of muscle fiber type shift as the result of RT training.

STATEMENT OF THE PROBLEM

Four problems have been identified:

1. To identify the effects of RT (10RM x 3 sets) on muscle oxygenation.
2. To observe OD changes in hemoglobin/myoglobin oxygenation and muscle blood flow in the biceps brachii muscle during 3 sets of 10RM one arm curls.
3. To identify differences in muscle oxygenation during RT between trained and untrained subjects.
4. To monitor anoxic changes in muscle tissue due to RT (10RM x 3 sets).

HYPOTHESES

The following hypotheses were tested:

H_0 1: In the trained subjects the resistance training protocol (10RM x 3sets) will have no effect on:

H_0 1a: The OD of hemoglobin/myoglobin oxygenation.

H_0 1b: The OD of total hemoglobin

H_0 1c: Muscle O_2 supply (Blood flow)

H_0 2: In the untrained subjects the resistance training protocol (10RM x 3sets) will have no effect on:

H_0 2a: The OD of hemoglobin/myoglobin oxygenation.

H_0 2b: The OD of total hemoglobin

H_0 2c: Muscle O_2 supply (Blood flow)

H_0 3: No differences will be identified between trained and untrained subjects for the following as a result of RT:

H_0 3a: OD of hemoglobin/myoglobin oxygenation.

H_0 3b: OD of total hemoglobin

H_0 3c: Muscle O_2 supply (Blood flow)

ASSUMPTIONS

1. NIRS is a valid and reliable measure of muscle oxygenation represented by changes in hemoglobin and myoglobin saturation.
2. Oxygen is consumed during metabolic re-synthesis of ATP as the result of skeletal muscle activity.
3. Bodybuilding RT protocol (10RM x 3 sets with 1 min rest between sets, 1.5s concentric phase: 1.5s eccentric phase) is representative of training programs used by athletes to increase strength and athletic power.
4. 60 mmHg is sufficient pressure to occlude venous blood without affecting arterial blood flow.
5. 250 mmHg is sufficient pressure to occlude both venous and arterial blood flow.
6. Comparing skeletal muscle oxygenation consumption during cuff occluded dynamic RT exercise is a valid determinant of muscle anoxia.
7. The experimental protocols will not elicit a training affect in the untrained subjects as a result neural adaptations, resulting in increased muscular strength.
8. Two years of RT training will have resulted in FTS.

DELIMITATIONS

1. Only college-aged males were used in the study.
2. Muscle oxygenation of forearm flexor muscle during RT may not be reflective of other muscle groups in the body.

3. Bodybuilding RT protocol (10RM x 3 sets with 1 min rest between sets, 1.5s concentric phase: 1.5s eccentric phase) is representative of training programs used by athletes to increase strength and athletic power.

LIMITATIONS

1. Distribution of muscle fiber types between subjects will be different, affecting the response to the experimental conditions.
2. NIRS data cannot be taken repetitively from the same muscle sample due to inadvertent movement of the NIRS light probes during dynamic exercise.
3. Subjects may not be able to complete the assigned experimental condition.
4. Untrained subjects may not be able to perform 10RM for 3 sets.
5. The changes in OD signals from NIRS are primarily from forearm flexor muscles.

OPERATIONAL DEFINITIONS

Anoxia:

An absence or reduction of oxygen reaching the muscle tissue.

Arm Flexors:

Biceps brachii, biceps brachialis and brachioradialis muscles.

Blood Pooling:

Gradual increase in muscle blood volume and a decrease in O_2 perfusion, due to nondescript factors restricting venous blood outflow to the heart from skeletal muscle.

Blood Flow:

The rate at which blood flows through muscle tissue expressed as millilitres per 100 ml of tissue per min determined by changes in total hemologbin concentrations via NIRS.

Fiber type shift (FTS):

An apparent change in muscle biochemistry and function due to RT, typically seen as a shift in muscle fiber from Type IIb to Type IIa.

Hemoglobin (Hb):

The chromophore molecule responsible for binding and carrying oxygen in the circulating blood.

Concentric Contractions:

The development of muscle tension with a change in muscle length.

Maximum Voluntary Contraction (MVC):

The maximum amount of weight which can be lifted during one concentric repetition.

Muscle Oxygenation:

The dynamic supply of oxygen to muscle regulated by changes in oxygen delivery and oxygen consumption.

Myoglobin (Mb):

The chromophore molecule responsible for binding and carrying oxygen in muscle tissue.

Near infrared spectroscopy (NIRS):

Noninvasive and indirect technique to measure muscle oxygenation during dynamic exercise by assessing the change in reflected visible light optical density.

Repetition Maximum (RM):

Represents the maximum number of concentric contractions for a given load.

Set:

A collection or group of consecutive contractions.

Tourniquet:

An external device for stopping the flow of blood through a blood vessel.

Trained subjects:

Individuals that have participated in regular weight-lifting for two consecutive years.

Untrained subjects:

Individuals that have not participated in any form of strength training.

Δ[Hb/MbO_2]:

Change in oxygen bounded hemoglobin/ myoglobin represented as optical density measured by NIRS.

Δ[Hb/MbO_2+Hb/Mb]:

Change in total hemoglobin/ myoglobin concentration represented as optical density measured by NIRS.

METHODOLOGY

Subjects

Twenty two healthy adult men participated in the study (n=22). Both trained and untrained males were recruited as volunteer subjects. Approval from the University of Victoria Human Ethics committee was obtained prior to the commencement of data gathering. All subjects were briefed on the testing procedures. Subjects signed a written informed consent form prior to participation in this study. Subjects of good health and who were eligible to participate in the study were self-selected as either, trained (n=13) or untrained (n=9). Trained subjects were defined as individuals who had participated in regular weight lifting for two consecutive years. Untrained subjects were defined as individuals that had not participated in any form of strength training. One repetition maximum (1RM) and 10RM were determined in during a introductory session. In addition, the age (yrs.), the sum of the biceps brachii muscle skinfolds of the anterior, posterior, medial and lateral aspects of the upper arm (mm), relaxed arm girth (cm) of the subject.

Measure of Muscle Oxygenation

NIRS was used to assess muscle oxygenation during all experimental procedures. NIRS is based on the linear relationship between the light absorption characteristics of hemoglobin (Hb) and myoglobin (Mb) in the near infrared spectra (700-1000nm), expressing absorbencies as changes in Hb/Mb optical densities under physiological conditions (Tamaki *et al.*, 1994). At a wavelength of 850nm, both oxygenated and deoxygenated forms of Hb/Mb absorb light, whereas deoxygenated forms primarily

absorb light at 760nm. Infrared light can only pass through the tissue of blood vessels of less than 1 mm in diameter. Larger diameter blood vessels tend to have thicker vessel walls, which do not permit the passage of light. As a result, NIRS assesses tissue oxygenation at the level of small blood vessels, capillaries and intracellular sites of oxygen uptake which are more representative of muscle oxygenation. Thus, NIRS can distinguish qualitative changes in oxygenated Hb/Mb (Hb/MbO_2) and deoxygenated Hb/Mb (Hb/Mb) content at the level of the muscle tissue simply and non-invasively (Chance, Dait, Zhang, Hamaoka & Hagerman, 1992). A strong relationship between blood flow, determined by strain-gauge plethysmography, and NIRS was found ($r=0.93$; $P<0.01;n=11$). However, NIRS consistently underestimated BF. This may have been due to the fact that NIRS only monitors small blood vessels and BF from larger blood vessel are undetectable by NIRS (Mancini *et al.*, 1994).

NIR signal.

NIR signal was obtained from a Runman, NIM O_2 monitor. Details of the technique have been personal computer (Bhambhani, Maikala, & Buckley, 1998; Bhambhani, Buckley & Susaki, 1999). Data obtained from each subject was stored and analyzed after all subjects had completed testing.

Two light probes were placed approximately 10cm proximally and 3cm laterally from the medial epicondyle of the humerus over a layer of cellophane wrap to prevent moisture contamination (Bhambhani *et al.*, 1998). Changes in the 760nm and 850nm signals emitted by the NIRS were used to assess changes in deoxygenated Hb/Mb (Δ[Hb/Mb]) and total Hb/Mb ($\Delta[Hb/MbO_2+Hb/Mb]$) and were monitored every 1.5sec. The NIRS

data were numerically expressed as millivolts (mV) and graphically displayed for the entire duration of all four phases for each subject group and was stored on 1.25 inch floppy disks. The following equations were used to convert the stored mV reading into OD values for the 760nm and 850nm signals.

$$OD760 = \log_{10}(I760_{calibrated} / I760_{measured});$$

$$OD850 = \log_{10}(I850_{calibrated} / I850_{measured});$$

Peak OD values during each exercise set for $\Delta[Hb/MbO_2]$, $\Delta[Hb/Mb]$ and $\Delta[Hb/MbO_2+Hb/Mb]$ were averaged within subject group for each experimental phase (personal communication, Bhambhani, 1998).

Experimental Protocol.

NIRS was used to measure the optical density (OD) from which changes in $\Delta[Hb/MbO_2]$ and total $\Delta[Hb/MbO_2+Hb/Mb]$ were estimated. Ten-repetition maximum (10RM) for one forearm flexion was determined during the introductory session prior to performing the experimental conditions. Experimental procedures consisted of 10RM x 3 sets with 3 min passive rest between sets. The tempo of contractions was standardized at 1.5 sec for each concentric and eccentric muscle action. Each exercise session was preceeded by one warm-up set of 10 repetitions at 50% of 1RM. Each subject participated in three experimental conditions: an exercise session consisting of 10RM one-arm curls (Condition1) and a control protocol (Condition 2).

Experimental Methods.

Each subject was required to perform forearm flexion with their "writing" arm while seated on a standard "preacher curl bench" (with the arm flexed 45^0 at the shoulder joint). The angle of forearm flexion was between 180^0 and 45^0 at the elbow determined with a goniometer prior to testing. Each subject was land-marked with non-erasable ink where the NIRS optical probe was placed on the surface of the biceps brachii muscle to ensure intra-subject consistency in the placement of the probes.

Description of Experimental Conditions.

Condition 1: Exercise – Each subject from both groups completed 3 sets of a 10RM protocol. This experimental condition was performed to observe and measure the changes in muscle oxygenation during a typical body-building RT regimen. Each subject performed the experimental procedure with a resistance that would limit the subject to 10 repetitions. The subject actively moved his arm through the angle of forearm flexion (180^o-45^o) at a tempo of 1.5 sec for concentric and eccentric actions per repetition, until the tempo could not be maintained or the subject could not move the resistance (Fig. 1).

Figure 1: Diagramatic representation of Condition 1, Exercise $\Delta[\chi]_n$

Condition 2: Control - The subject performed the experimental procedure without a resistance load. This was done to validate the NIRS standardization procedure. A test-

retest validation experiment, which had been completed prior to this experiment, showed the NIRS technique to be a "robust" and reliable measure of muscle oxygenation and blood flow (Chance *et al.*, 1992; Bhambhani *et al.*, 1997). A tourniquet was placed at the proximal end of the dominant arm of the subject using a pressure cuff. The pressure of the cuff was set at 250mmHg and the pressure was maintained for 7 minutes, which was considered to be sufficient to occlude venous blood out-flow and arterial inflow to the muscle (Mancini, Bolinger, Lui, Kendrick, Chance & Wilson, 1994).

Statistical Analysis.

Independent t-tests were conducted using SPSS version 10.0 to determine the main effects within and between the means of the trained and untrained groups for the 10RM exercise protocol. A Scheffé test was conducted to determine if any differences occurred within and between trained and untrained subjects in a set-wise comparison for the 10RM exercise protocols. Significance was set at $p<0.05$.

RESULTS

Muscle oxygenation

Raw data for muscle oxygen desaturation during the 10RM exercise protocol for a typical subject are represented in Figure 2. Values in Figure 2 are expressed in mV. Muscle oxygen desaturation increased as indicated by the negative slope of the NIRS recorded signal in mV during the 30 sec. work phase of the warm-up set, Set 1, Set 2 and Set 3 of the exercise protocol. This trend was seen in all subjects for both subject groups. After the cessation of work, the oxygen saturation of the muscle tissue increase as indicated by the positive slope of the NIRS recorded signal during the rest period between the work phases of the warm-up set, Set 1, Set 2 and Set 3. Between sets and after the completion of the 10RM protocol, muscle oxygen exceeded baseline values. Both trends were also seen in all subjects for both groups.

The set-wise comparison of muscle oxygenation between trained and untrained groups for the exercise protocol is shown in Figure 3. Changes in muscle oxygenation were determined by finding the range in mV between the lowest level of desaturation, occurring at the initiation of exercise, and the highest level of desaturation, occurring just prior to the cessation of exercise. Muscle desaturation ranges were determined for the warm-up set and for the three exercise sets for all subjects in both subject groups. The individual subject ranges were then expressed as a percentage of the cuff occlusion range of each subject, which determined the standard measure of maximum muscle tissue desaturation. The percent ranges were averaged set-wise within subject group and compared between subject groups. During the work phases of the warm-up set, Set 1, Set

2 and Set 3, both the trained and untrained subject groups showed increases in muscle deoxygenation.

Average percent values and standard error of the mean for both subject groups for the Warm-up set, Set 1, Set 2 and Set 3 are represented in Table 3. Muscle deoxygenation ranged from 78.3% to 83.1% in the trained group and from 69.5% to 71.42% in the untrained group (Table 3) for the 10RM protocol.

Significant differences in O_2 desaturation were found between untrained subjects during the Warm-up and trained subjects during Warm-up, Set 1, Set 2 and Set 3 ($P < 0.05$). However, no statistically significant differences were found between the set-wise comparisons for the trained or the untrained groups, nor were significant differences found between trained and untrained subject groups among Set 1, Set 2 and Set 3 (Fig. 3, Table 3).

Table 2: Mean physical characteristics (SD), age (years), and sum of biceps brachii skinfold of anterior, posterior, medial, and lateral aspects of the upper arm (mm), relaxed arm girth (cm) and 10RM (lbs) of trained (N=13) and untrianed (N= 9) collage-aged males.

Group	Age	Skinfold	Arm Girth	10RM
Untrained	23.8(3.5)	15.2(3.7)	28.37(3.1)	28.24(3.9)
Trained	25.3(3.6)	16.6(3.2)	35.7(3.0)	39.81(10.5)

Figure 2: An example of Hb/MbO_2 trends (760nm NIRS signal; Channel 2) of the forearm flexors in one male subject during one Warm-up set (10 repetition at 50% 1RM) and 3 sets of 10RM with 3min rest between sets, expressed in mV. The raw signal was sampled at 60Hz and averaged over 3-s intervals using a 3-point moving average.

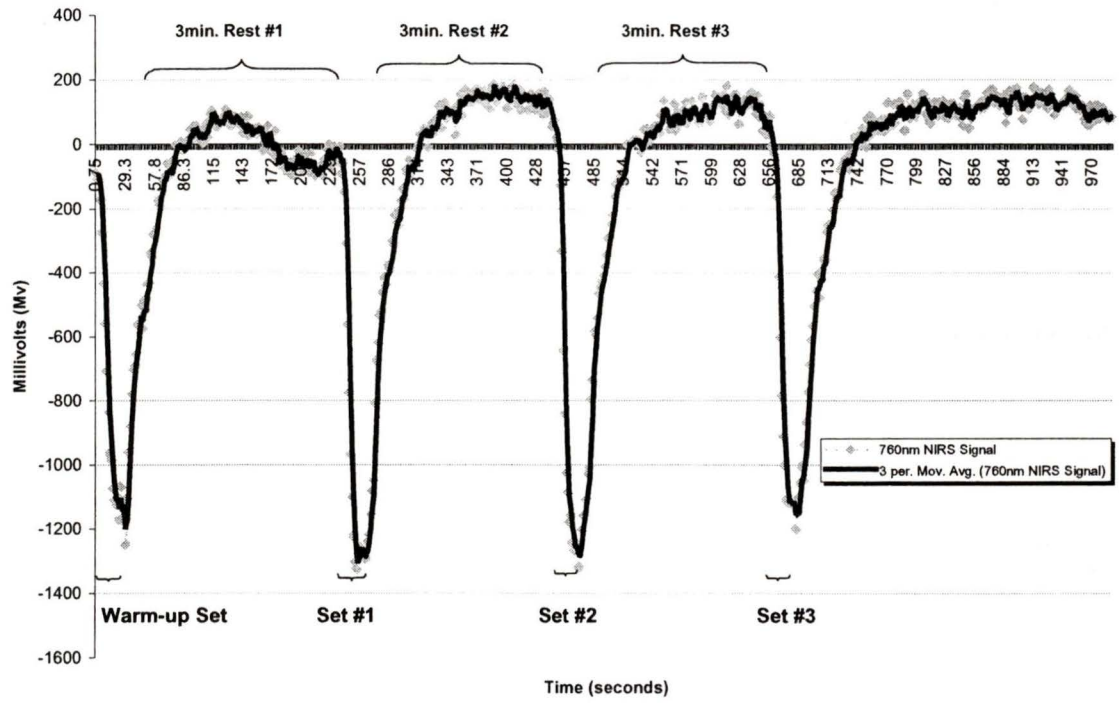

3min. Rest #1
3min. Rest #2
3min. Rest #3
400
200
0
-200
-400
-600
-800
-1000
-1200
-1400
-1600
Millivolts (Mv)
0.75
29.3
57.8
86.3
115
143
257
286
343
371
400
428
485
542
571
599
628
656
685
713
742
770
799
827
856
884
913
941
970
760nm NIRS Signal
3 per. Mov. Avg. (760nm NIRS Signal)
Warm-up Set
Set #1
Set #2
Set #3
Time (seconds)

Figure 3: Mean change (%) in oxygenation values (SEM) for trained (N=13) and untrained (N=9) subject groups for each set of the 10RM exercise protocol. Significant differences (*) were found between untrained subjects during the Warm-up and trained subjects during Warm-up, Set 1, Set 2 and Set 3 ($P < 0.05$).

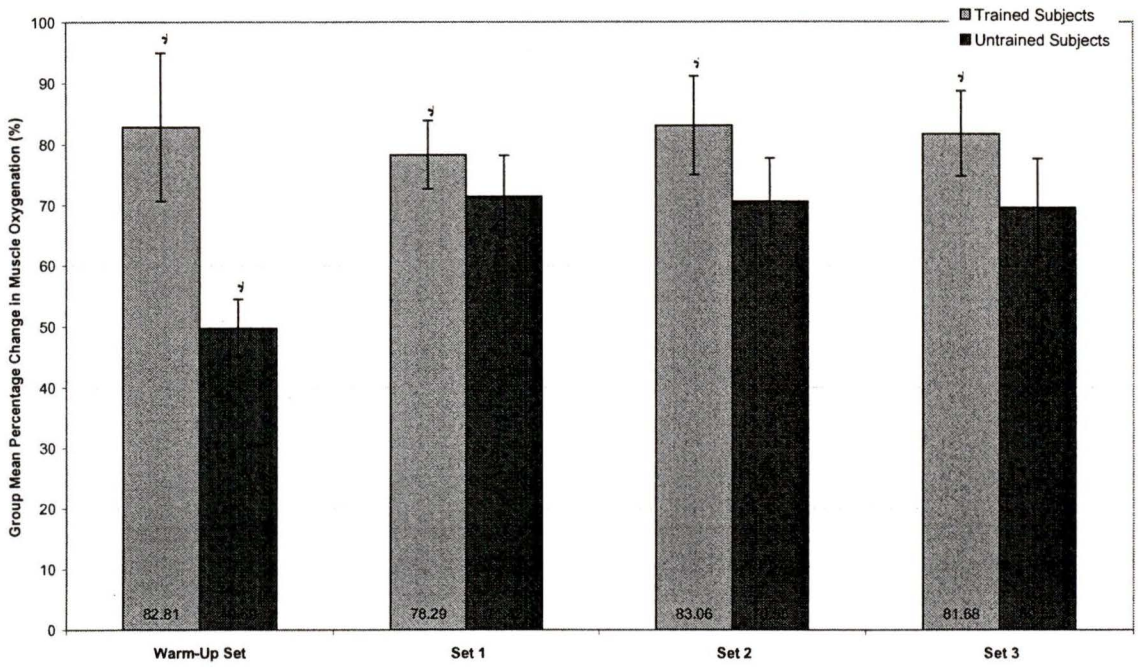

Trained Subjects
Untrained Subjects
Group Mean Percentage Change in Muscle Oxygenation (%)
100
90
80
70
60
50
40
30
20
10
0
82.81
78.29
83.06
81.68
Warm-Up Set
Set 1
Set 2
Set 3

Table 3: Mean (SEM) values for relative muscle [Hb/MbO_2] change for trained (N=13) and untrained (N=9) groups during Warm-up and 10RM exercise protocol. These values were expressed as a percentage of the range of the cuff occlusion protocol.

Group		Set		
	Warm-up	1	2	3
Trained				
[Hb/MbO_2]	62.2(12.7)	78.3(5.6)	83.1(8.1)	81.7(7.6)
Untrained				
[Hb/MbO_2]	49.7(4.8)*	71.4(6.7)	70.6(7.1)	69.5(8.0)

* Significant differences were found between untrained subjects during the Warm-up and trained subjects during Warm-up, Set 1, Set 2 and Set 3 ($P < 0.05$).

Blood Volume

Raw data for muscle blood volume during 10RM exercise protocol for a typical male subject are represented in Figure 4. Values in Figure 4 are expressed in mV. Blood volume decreased as indicated by the positive slope of the recorded NIRS signal during the 30 second work phase of the Warm-up set, Set 1, Set 2 and Set 3 of the exercise protocol. This trend was seen in all subjects for both subject groups. Between the work phases of the Warm-up set, Set 1, Set 2, and Set 3, muscle blood volume increased, as indicated by the positive slope of the NIRS signal. Muscle blood volume exceeded baseline values during the rest interval. Both trends were also seen in all subjects for both trained and untrained groups.

The set-wise comparison of muscle blood volume between trained and untrained groups for the exercise protocol is shown in Figure 5. Muscle blood volume ranges were determined for the warm-up set and of the three exercise sets for all subjects in both groups. Changes in muscle blood volume were determined by finding the range in mV between the highest blood volume value, occurring at the initiation of exercise, and the lowest blood volume value, just prior to the cessation of exercise. The individual ranges were then expressed as a percentage of the cuff occlusion blood volume range of each subject, which was used as a standard measure of maximum muscle blood volume. The percent ranges were averaged set-wise by subject group and compared set-wise. During the work phases of the warm-up set, Set 1, Set 2 and Set 3 both the trained and untrained subject groups showed decreases in muscle blood volume.

Average percent values and standard mean error for both subject groups for the Warm-up set, Set 1, Set 2 and Set 3 as shown in Table 4. Muscle deoxygenation ranged

from 54.5% to 62.2% in the trained group and from 22.3% to 46.2% in the untrained group for the 10RM protocol.

Significant differences in muscle blood volume were found between untrained subjects during the Warm-up and trained subjects during Warm-up, Set 1, Set 2 and Set 3 ($P < 0.05$). Significant statistically differences were not found between the remaining set-wise comparisons for the trained or the untrained groups, neither were significant differences found between trained and untrained subject groups among sets (Figure 5).

The comparisons between muscle oxygen saturation and muscle blood flow for a typical male subject is shown in Figure 6. Both muscle oxygen saturation and muscle blood flow are expressed as a percentage of cuff occlusion muscle oxygen saturation and muscle blood flow.

Figure 4: An example of [Hb/MbO_2+Hb/Mb] trends (850nm NIRS ignal; Channel 3) of the forearm flexors in one male subject during one warm-up set (10 repetition at 50% 1RM) and 3 sets of 10RM with 3min rest between sets, expressed in mV. The raw signal was sampled at 60Hz and averaged over 3-s intervals using a 3-point moving average for the duration of the test.

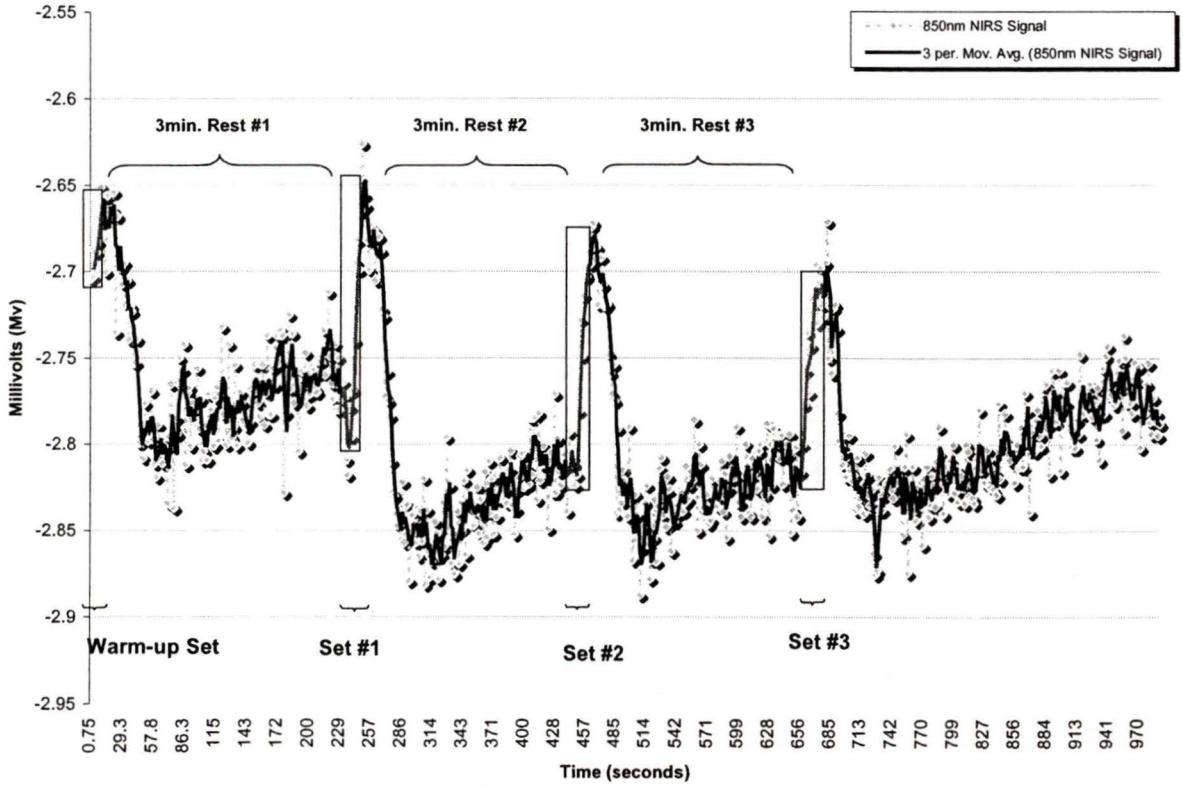

850nm NIRS Signal
3 per. Mov. Avg. (850nm NIRS Signal)
3min. Rest #1
3min. Rest #2
3min. Rest #3
Warm-up Set
Set #1
Set #2
Set #3
Millivolts (Mv)
-2.55
-2.6
-2.65
-2.7
-2.75
-2.8
-2.85
-2.9
-2.95
0.75
29.3
57.8
86.3
115
143
172
200
229
257
286
314
343
371
400
428
457
485
514
542
571
599
628
656
685
713
742
770
799
827
856
884
913
941
970
Time (seconds)

Table 4: Mean (SEM) values for relative blood volume (BV) change for trained (N=13) and untrained (N=9) males during Warm-up and the 10RM exercise protocols. Values are expressed as a percentage of the cuff occlusion protocol.

Group	Set			
	Warm-up	1	2	3
Trained				
BV(%)	62.1(12.7)	54.5(7.2)	61.7(9.2)	58.1(8.1)
Untrained				
BV (%)	22.3(6.6)*	42.9(11.3)	46.2(7.9)	40.8(8.8)

* Significant differences were found between untrained subjects during the Warm-up and trained subjects during Warm-up, Set 1, Set 2 and Set 3 ($P < 0.05$).

Figure 5: Mean change (%) in [Hb/MbO_2+Hb/Mb] values (SEM) for trained (N=13) and untrained (N=9) subject groups for each set of the 10RM exercise protocol. Significant differences were found between untrained BV during the Warm-up and trained BV during Warm-up, Set 1, Set 2 and Set 3 ($P < 0.05$).

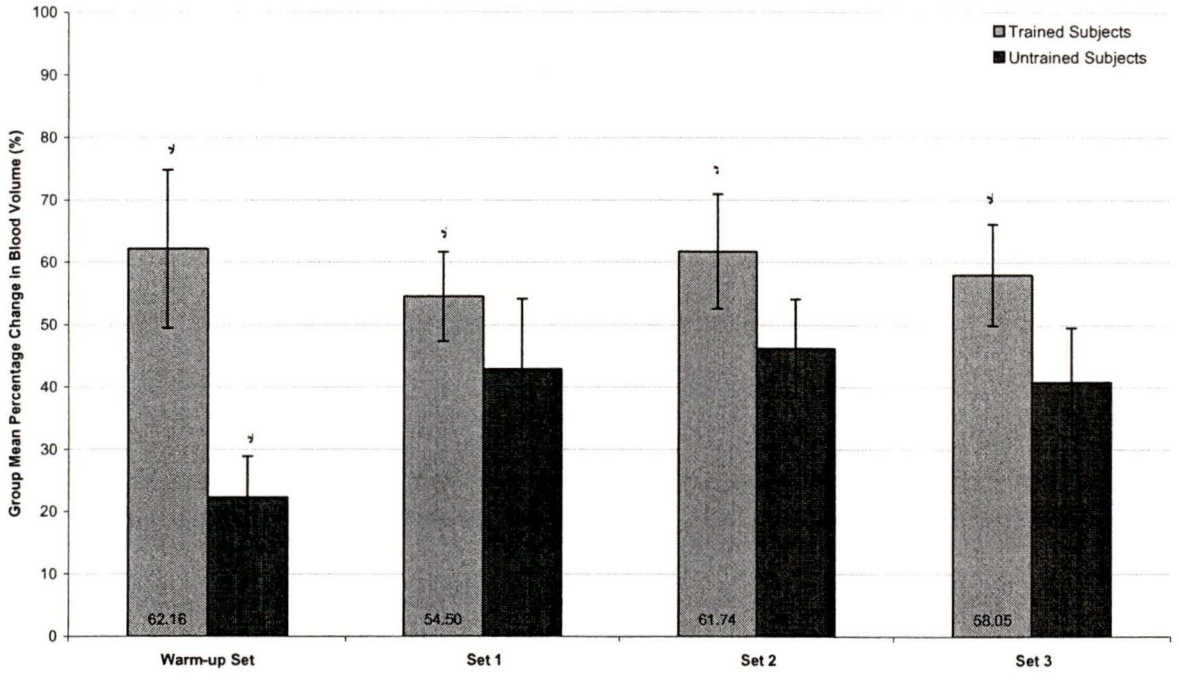
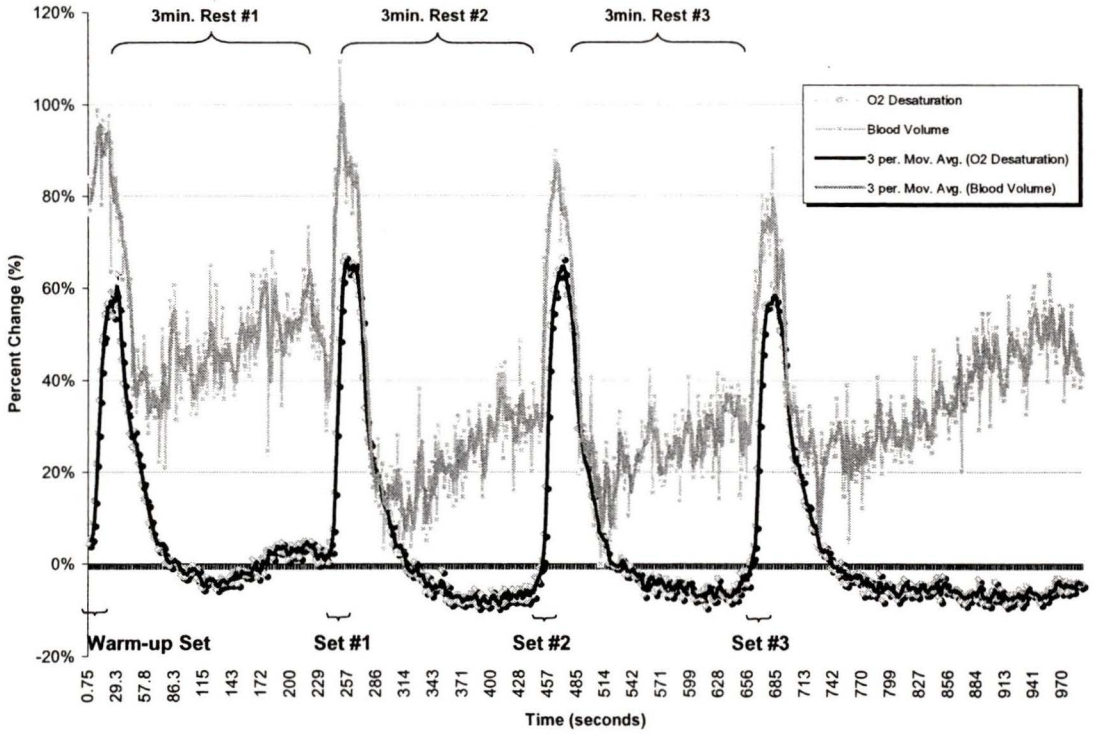

Trained Subjects
Untrained Subjects
Group Mean Percentage Change In Blood Volume (%)
100
90
80
70
60
50
40
30
20
10
0
62.16
54.50
61.74
58.05
Warm-up Set
Set 1
Set 2
Set 3

Figure 6: Percent change in [Hb/MbO_2+Hb/Mb] (850nm; Channel 3) and [Hb/MbO_2] utilization (760nm; Channel 2) of the forearm flexors relative to a blood occlusion control in one male subject during one warm-up set (10 repetition at 50% 1RM) and 3 sets of 10RM with 3min rest between sets. Variability was reduced by 3-point moving average for the duration of the test.


3min. Rest #1
3min. Rest #2
3min. Rest #3
O2 Desaturation
Blood Volume
3 per. Mov. Avg. (O2 Desaturation)
3 per. Mov. Avg. (Blood Volume)
Percent Change (%)
120%
100%
80%
60%
40%
20%
0%
-20%
Warm-up Set
Set #1
Set #2
Set #3
0.75
29.3
57.8
86.3
115
143
172
200
229
257
286
314
343
371
400
428
457
485
514
542
571
599
628
656
685
713
742
770
799
827
856
884
913
941
970
Time (seconds)

Half Time to Full Saturation

The set-wise comparison of the half-time to full saturation (T_{50}) between trained and untrained groups for the exercise protocol is shown in Figure 7. T_{50} were determined by finding the total time in seconds between the highest level of desaturation, just prior to the cessation of exercise and the lowest level of desaturation, occurring at the initiation of exercise. Values for T_{50} were expressed in seconds. T_{50} values were determined for the Warm-up set and of the three exercise sets for all subjects in both trained and untrained groups. T_{50} times were averaged set–wise within subject groups and compared between subject groups set-wise. The percent ranges were averaged set-wise by subject group and compared set-wise. During the work phases of the warm-up set, Set 1, Set 2 and Set 3 both the trained and untrained subject groups showed increases in muscle deoxygenation.

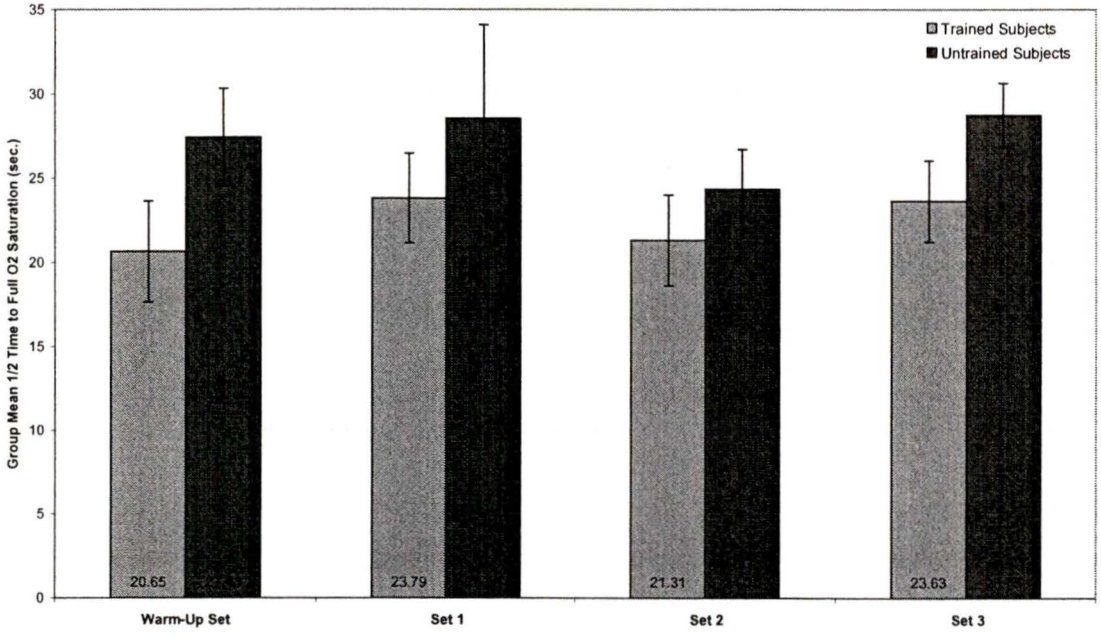
T_{50} and standard error of the mean values for both trained and untrained groups for the Warm-up set, Set 1, Set 2 and Set 3 are found in Table 4. T_{50} ranged from 21.3 seconds to 23.8 seconds in the trained group and from 24.4 seconds to 28.8 seconds in the untrained group for the 10RM protocol.

No significant differences were found between trained and untrained subjects during Warm-up, Set 1, Set 2 and Set 3.

Table 5: Mean values (SEM) of half-time to full saturation (T_{50}) for trained (N=13) and untrained (N=9) subject groups during Warm-up and for each set of the 10RM exercise protocol, expressed as a percentage of the range of the cuff occlusion protocol.

Group		Set		
	Warm-up	1	2	3
Trained				
T_{50}	20.7(3.0)	23.8(2.7)	21.3(2.7)	23.63(2.4)
Untrained				
T_{50}	27.4(2.9)	28.6(5.6)	24.4(2.4)	28.8(2.0)

Figure 7: Mean values (SEM) of half-time to full saturation (T_{50}) for trained (N=13) and untrained (N=9) subject groups for each set of the 10RM exercise protocol. No significant differences were found between untrained and trained subjects.


Group Mean 1/2 Time to Full O2 Saturation (sec.)
35
30
25
20
15
10
5
0
Trained Subjects
Untrained Subjects
20.65
23.79
21.31
23.63
Warm-Up Set
Set 1
Set 2
Set 3

DISCUSSION

Decrements in athletic power performance have been attributed to an oxidative FTS as the result of RT (Eiken *et al.*, 1991). Tamaki *et al.* (1994) has proposed that skeletal muscle blood occlusion creates an anoxic environment which results in this FTS.

Muscle Blood Flow

Several studies have investigated blood flow during skeletal muscle contractions. Baker and Davis (1975) studied skeletal muscle blood flow and volume changes during muscle electrical stimulation of isolated dog gracillus muscle at 2-15Hz for 15-60sec to investigate increases in total tissue volume after repetitive muscular contractions. Circulating blood volumes were measured by constant infusion technique using RBC-^{51}Cr or albumin-^{131}I. Volume changes were measured by plethymography and changes in total muscle volume by radioactivity. Blood volume increased during all muscle contractions. Total muscle volume initially decreased during muscular contractions, but increased after the cessation of the electrical stimulation. Increases in intramuscular pressure caused the movement of fluid into the surrounding vascular system. Minimal loss of labeled albumin during muscular contraction suggests that fluid loss was not due to capillary permeability but due to vasodilatation and recruitment of new vascular units. As the intensity of exercise increased, the distribution of RBCs was restricted as they were too large to access small capillaries, in contrast the blood plasma was free to flow throughout the vascular system. This may have been due to: (1) capillaries being partly occluded due to increases in tissue pressure that exceed capillary pressure caused by

contracting muscle, or (2) the orientation of the capillaries were altered compared to the parent blood vessel.

Qvarfordt, Eklof, Ohlim, Plate and Saltin (1984) measured the blood flow of the anterior tibial muscle and the deep posterior compartments in nine patients with chronic vein obstructions and venous claudication in both legs, at rest and during exercise, to investigate the effect of intramuscular pressures on muscle blood flow. Intramuscluar pressures in the anterior tibial muscle and the deep posterior compartments were higher in the leg with iliac obstruction than in the contralateral leg. Muscle blood flow, determined by ^{133}xenon clearance, was lower in the venous obstructed leg than in the control leg during exercise. Increased interstitial pressure may have compressed the distal end of capillaries, resulted reduced capillary flow from parent arteries. In addition, lactate levels were significantly higher in the obstructed leg as compared to the control leg.

Tamaki *et al.* (1994) reported increases in blood volume during resistance training of the bicep brachii muscle in nine male subjects during 3 consecutive sets of 10RM, separated by 1 min rest period, of dominant single forearm flexion. Total blood volume increased in each set during exercise and did not return to the resting state during the recovery period of each set. In addition, blood volume did not return to the resting rate for more than 90 s after the third set. The observed results were attributed to restricted arm blood flow that decrease venous blood out flow but did not exclude arterial inflow. As a result, the blood volume gradually increased during the exercise periods.

The results of the present study are not in agreement with the above literature. In the 10-RM arm curl exercise protocol in the present study, both the trained and the

untrained subjects showed the same relative changes in skeletal muscle blood volume during all sets and rest periods. Skeletal muscle blood volume temporally increased at the start of the 10-RM protocol and then rapidly decreased at the end of the exercise. However, the intensity in this study was approximately 73% MVC, which is much greater than previously reported. Lind and Williams (1978) observed similar results in the skeletal muscle blood flow of 11 human volunteer subjects during brief isometric hand-grip exercise at varying intensities from 10 to 100% of the maximal voluntary contraction (MVC) and from 2 to 12 sec. in duration. Blood flow increased linearly with tension up to 60% MVC but tensions above 60% MVC did not have an effect on blood flow measured by plethysmography. A direct relationship between "force-time integral" (the duration of contraction x peak tension) and blood flow, expressed as $mL.min^{-1}.100^{-1}$, was found during brief isometric contractions. This may indicate that a graded level of blood flow in response to intermittent isometric contractions is the result of metabolic vasodilatation dependent on exercise intensity.

Measures of blood flow by NIRS in the present study are representative of the blood flow found in small 3^{o} capillary beds in the skeletal muscle of the biceps brachii muscle (Hampson & Piantadosi, 1988). In contrast, measures of blood flow from plethysmography may not correlate well with skeletal muscle blood flow because the NIRS signals are predominately from skeletal muscle rather than changes in total muscle volume derived from plethysmography which would reflect changes in the total muscle volume, including bone and skin blood flow. De Blasi, Ferrari, Natali, Conti, Mega and Gasparetto (1994) investigated the forearm blood flow and $\dot{V}O_2$ of eleven healthy subjects by NIRS and plethysmography during two experimental protocols in order to

evaluate NIRS as a valid and reliable measurement technique. All subjects performed five 30 sec 50-mmHg venous occlusion protocol induced by a pressure cuff positioned proximally on the arm. In addition, the subjects performed 60 consecutive handgrip exercises. Blood flow was measured simultaneously by NIRS and plethysmography during the occlusion and exercise protocols. The measurement techniques agreed well for both blood flow and $\dot{V}O_2$ measurements, however more variability was seen during the rest period after the handgrip exercise stopped and during higher rates of blood flow. In addition, the blood flow values were found to be lower with NIRS.

The results obtained in the present study can be explained as follows. During high intensity dynamic isometric muscular contractions arterial flow decreased due to the increased tissue pressure. With each muscular contraction the blood volume decreased, as blood was forced out of the capillary bed with each repetition. Blood volume subsequently decreased with each muscular contraction over the duration of the exercise set. No inter-set differences in blood flow were seen within subject groups or between subject groups for the remaining set-wise comparisons. Both training groups had similar relative responses in the skeletal muscle vascular circuits over the course of the 10-RM protocol.

During the rest period, the blood flow of both the trained and untrained subjects increased in the present study, which is in agreement with the previous studies. The increase in blood volume after exercise has been attributed to reactive hyperameia. Hyperameia has been observed following occlusion (Hampson & Piantadosi, 1998) and following intense dynamic isometric exercise (Belardinelli, Barstow, Porszasz, & Wasserman, 1995; Tamaki *et al.*, 1994).

Baker, Menninger, Schoen, and Sutton (1976) studied the skeletal muscle blood flow and volume changes of isolated dog gracillus muscle during varying venous pressures to investigate the redistribution of blood flow and capillary dynamics in skeletal muscle during reactive hypereamia. Volume changes and blood flow were compared between averaged constant venous pressure at 3mm Hg and a range of tissue pressure from 5-40 mm Hg. The changes in total tissue blood volume, circulating blood and mobilized vascular volumes were calculated and monitored by plethysmography and by changes in total muscle radioactivity using RBC-^{51}Cr or albumin-^{131}I. Tissue volumes increased with increments of venous pressure but plateaued at a venous pressure of 25 mm Hg. When returning to control levels a significant amount of radio labeled red cells-Cr^{52} were not accounted for. This suggests, that increased tissue volume is due to (1) distention of opened capillaries and veins, (2) a distension of any recruited vessels that retained blood or (3) increased blood flow in previous low flow opened capillaries and veins where red blood cells gain access as a result of reduced plasma skimming during reactive hypereamia. This is in contrast with rhythmical contracting muscles, in which plasma skimming is increased and results in reduced or occluded tissue blood flow.

Significant differences in blood flow during the 10-RM arm curl exercise protocol were found between untrained subjects during the Warm-up and trained subjects during the Warm-up and Set 1-3 ($p \leq .05$). This result was unexpected as the Warm-up load was approximately 55% MVC for both the trained and untrained. The greatest level of relative blood flow was expected at this intensity as it would result in the least vascular resistance (Lind & Williams, 1979).

It has been established that RT does not influence anatomical adaptation in capillary neo-formation or redistribution in response to strength training (Tesch & Larsson, 1982; Tesch & Karlsson, 1985; Tesh, Thorsson, & Kaiser, 1984). Based on the results of this present study, it appears that no relative physiological adjustments have been made in response to RT as measured by NIRS.

Muscle Oxygenation

In the 10-RM arm curl exercise protocol, muscle blood oxygenation rapidly decreased at the start of the 10-RM protocol, then rapidly increased at the cessation of work and exceeded baseline values. Significant differences were seen between the untrained warm-up and the trained Warm-up and Sets 1-3. No other significant differences were seen within each subject group or between subject groups among sets.

Several studies have investigated skeletal muscle oxygenation during intense exercise in normal and clinical subjects. Cheatle, Potter, Coope, Delpy, Coleridge Smith, and Scurr (1991) measured the skeletal muscle blood flow and tissue oxygenation in 38 subjects, 21 normal controls with no vascular disease and 17 patients with peripheral vascular disease, using NIRS to explore: (1) the possible relationship between oxygen consumption and blood flow, and (2) to evaluate NIRS as a clinical technique in determining the presence or absence of arterial disease. The rate of muscle oxygen consumption was measured during 5 min of tourniquet-induced ischaemia. The median oxygen consumption values in subjects with peripheral vascular disease was 0.10mL 100g tissue^{-1}min^{-1} while in the control group it was 0.2mL 100g tissue^{-1}min^{-1} ($p \leq .03$). The post ischaemia reoxygenation was also measured. Patients with peripheral disease

demonstrated an increase in oxidative capacity determined by fiber type as the result of intermittent claudication, however vascular disease may prevent recruitment of additional vascular units during the post-ischeamic conditions. Therefore, mitochondrial oxygen consumption is directly dependent on substrate availability and differences between groups may be the result of a reduced availability of high-energy compound, such as phosphocreatine.

Belardinelli, Barstow, Porszasz and Wasserman (1995) measured the NIRS signal from the vastus lateralis and gas exchange monitored by breath by breath in eleven adult subjects during incremental cycle ergometry to investigate the relationship between progressively increasing work rate exercise and muscle oxyhemoglobin and oxymyoglobin saturation. Tissue O_2 saturation decreased throughout the exercise protocol for all subjects, but the rate of the decrease was most notable near the end of the exercise protocol, occurring at approximately 80W. Immediately at the beginning of recovery, tissue O_2 saturation increased to levels above baseline due to reactive hyperemia. The changes in tissue O_2 were attributed to a change in HbO_2 desaturation below lactate threshold calculated by the $\dot{V}O_2/\dot{V}CO_2$ ratio measured during breath-by-breath collection of the subjects. The changes in tissue O_2 were attributed to changes in MbO_2 desaturation above lactate threshold. Liberation of O_2 from myoglobin was attributed to the increased PO_2 gradient between the capillaries and cells at high workloads due to decreasing PO_2 as $\dot{V}O_2$ increases.

The level changes in muscle oxygation for this study were consistent with previously published studies. However, the significant differences found between the untrained and trained subjects were not consistent. These results may be explained by

observations noted by Kahn, Jouanin, Bussiere, Tinet, Avrillier, Ollivier and Monod (1998) while investigating the relative muscle oxygenation of the brachioradialis muscle by NIRS during isometric forearm flexion up to the isotonic part of the isometric contraction (IIC) and beyond that time for 120s (anisotonic part of the isometric contraction) in 32 trained male subjects. During IIC each subject maintained relative isometric forces of either: 25% and 70% MVC, 50% and 100% MVC, or 40% and 60% MVC. Muscle oxygenation was expressed as a percentage of the reference value obtained by measuring maximum muscle oxygen desaturation during 6 min forearm cuff occlusion ischemia protocol and the maximal reoxygenation following the release of the cuff. During IIC at 25% MVC, muscle oxygenation decreased to 17% of resting O_2 values then leveled off at 25% of resting O_2 levels and reoxygenation was weak after the cessation of exercise. During IIC at 40%, 50%, 60% and 70% MVC, the lowest muscle oxygenation values were obtained after 15-20s of contractions and deoxygenation values were -18±6%, -59±12% -31±6% and -29±6% of resting O_2 values, respectively. During 100%MVC, the lowest oxygenation, -19±9%, was observed while force was decreasing (69% MVC) and reoxygenation was seen during the isotonic part of the isometric contraction at 50% MVC. Increased muscle oxygenation at 50% MVC may be explained by: 1) increased deoxygenation of the brachioradialis tissue due to neural activation of type I, type IIa and type IIb muscle fibers, 2) blood flow in the upper arm during 50% MVC isometric contractions may not be totally interrupted, and 3) blood flow is distributed heterogeneously with the deepest part of the muscle being affected first. Muscle deoxygenation at high forces (60%, 70% and 100%) was lower than during 50%

MVC isometric contractions, however desaturation values were lower as compared to 6 min. occlusion protocol.

Kahn *et al.* (1998), therefore, demonstrated that there is not a linear relationship between external force, muscle oxygenation and maximal muscle deoxygenation. During the 10-RM protocol, the load for the Warm-up used by the untrained subjects may have been below 50% due to increased perceived exertion resulting from a lack of RT. It is also likely that the reduced Warm-up load still recruited a large portion of the muscle fibers but without a large reduction in blood flow in the upper arm during the isometric contractions. The increase in skeletal muscle blood flow resulted in a greater muscle deoxygenation as compared to Set 1-3 in both the trained and the untrained subject Warm-up set.

Half-Time to Full Saturation

In the 10-RM arm curl exercise protocol, the T_{50} to full saturation was within agreement with previously published studies.

The T_{50}, as measured by NIRS, was investigated by Sahlin (1992). Four subjects (aged between 18 and 29 years) performed two experimental protocols, (1) a 20 min occlusion protocol of the leg, induced by a pressure cuff positioned proximal to the measuring probe on the upper thigh and inflated to a pressure of 250mm Hg and (2) an exercise protocol under arterial occlusion, at a cuff pressure of 250mm Hg, where each t subject performed sustained static contractions of the knee extensors at 66% MVC while seated in a chair with the knee at 90 degrees. During the occlusion protocol all subjects experienced a decrease in O_2 saturation, with a half-time to full occlusion occurring at

2.3 ±0.2min. During recovery, oxygenation increased rapidly to a level above pre occlusion tissue saturation with peak full recovery occurring at 2.6min and an average half-time to full saturation occurring at 24 ± 2s. The intensity of the sum signal (blood volume) increased slightly, indicating a decrease in blood volume or a decrease in penetrating depth. During recovery, blood volume increased indicating hyperemia with the average half-time of the increase in blood volume occurring at 10.8 ±0.9s, which was less than the reoxygenation volume. During the exercise protocol, after 1 min arterial occlusion, there was an additional decrease in O_2 saturation, which was partially reversed when the contraction stopped, despite the maintenance of arterial occlusion. This suggests a partial reoxygenation during arterial occlusion or a change in muscle geometry during muscular contractions influencing the light penetrating depth (Sahlin, 1992).

No significant differences were found between trained and untrained subjects, which was unexpected. Cheatle *et al.* (1991) also measured post ischaemic reoxygenation after 5 min of tourniquet-induced ischaemia. The half time to full reoxygenation was 20 sec in the control subjects and 40 sec in the patient with peripheral vascular disease. Therefore, mitochondrial oxygen consumption is directly dependent on substrate availability and differences between groups may be the result of a reduced availability of high-energy compounds, such as phosphocreatine.

However, in this study the trained subjects showed a trend for having shorter T_{50}. The lack of statistical difference may have been due to the selection of the subjects rather than lack of sensitivity of the NIRS. Subjects were selected on the basis of resistance training experience and were self selected as untrained or trained. A wide range of experience and training levels within these two groups could have occurred. This may

have attributed to the wide range of half-time to saturation observed. In addition, endurance athletes were classified as untrained. It is known that aerobic training enhances peripheral aerobic adaptations (Holloszy & Booth, 1976; Anderson & Henriksson, 1977; Gollnick & Saltin, 1982). It is therefore possible that the endurance trained individuals may have contributed to the wide distribution in recorded OD values during he 10RM exercise protocol, resulting no statistically differences in BV and muscle oxygenation between trained and untrained groups.

Fiber Type Shifts

Oxidative changes in muscle fiber distribution have been observed as a result of endurance training (Anderson, & Henriksson, 1977a; Jansson, & Kaijser, 1977) and have also been observed in claudicate patients (Henriksson, Chi, Hintz, Young, Kaiser, Salmons & Lowry, 1986). Oxidative fiber-type shifts have also been observed as the result of resistance training (Staron, 1991).

Tesch and Larsson (1982) investigated the relationship between selective hypertrophy of fast twitch muscle (FT) on the distribution of fiber types and fiber area as the result of weight-training in the vastus lateralis of three high caliber competitive bodybuilders, eight elite power lifters, and fifty physical education students. For the bodybuilders the percentage of FT was less, mean fiber area was smaller and selective FT fiber hypertrophy was not evident in muscle tissue samples of the other two groups. The percentage of FT was less and mean fiber area obtained were similar to the non-trained individuals and were found to be less than values obtained in powerlifters (Tesch & Larsson, 1982). The muscle fibers in the three bodybuilders resembled values obtained

in endurance athletes. The bodybuilders did exhibit relatively high muscular endurance, which may explain the low percentages of the type IIb glycolytic fibers. Therefore, Tesch and Larsson (1982) concluded that bodybuilding training is a combination of both strength as well as endurance, which would favor a higher percentage of ST fibers.

However, the current nomenclature for fiber typing is based on the qualitative analysis of skeletal muscle myosin ATPase (mATPase). Biochemical assays that utilize varying pH are used to discern between what has been termed type I and type II muscle fibers. Under alkaline conditions, type I fibers exhibit low mATPase activity or are termed alkaline labile. Under acidic conditions, type I fibers exhibit high mATPase activity and are termed acid stable. Type II fibers activities are the reverse of type I fibers being acid labile and alkaline stable, respectively. There are subclasses of type II fibers showing varying levels of mATPase activities during various levels of pH (Anderson & Henriksson, 1977a). The current nomenclatures used to describe the fibers of this subclass are in order of alkaline stability and is as follows: type I, IIA, IIB, and IIC. The mATPase activities of all fiber types have been show to be stable during *in vitro* experiments throughout an approximate pH range of 4.7 to 10.0 (Staron, 1991). This is well under physiological pH conditions during *in vivo* experiments, in which skeletal muscle pH was found to range from approximately 7.0 at rest to 6.2 immediately after dorsiflexion exercise measured by nuclear magnetic spectroscopy of the gastrocnemius muscle (Staron, 1997). It has been well documented that human type II mATPase activity is 2-3 times higher the than type I mATPase activity, however histochemical analysis of skeletal muscle fibers is a qualitative method of distinguishing muscle fiber groups and does not asses the amount of mATPase activity. Type I muscle fibers are the

most oxidative and type II the least, there exists a wide range of metabolic enzyme activity within each specific mATPase based fiber type. This suggests metabolic changes can occur without a change in myosin heavy chain protein or its associated mATPase activity (Staron, 1997).

CONCLUSION

The 10RM protocol elicited decreases in muscle oxygenation and reduced blood flow in the biceps brachii muscle in both the trained and untrained groups. No set-wise differences were seen between or within groups. However, the measures taken to compare blood flow and muscle oxygenation are relative to each physiological range of the subject and do not reflect the absolute change in either muscle blood flow or muscle oxygenation. Differences may well exist between trained and untrained subjects but by expressing these differences as relative measures any differences may have been masked.

In addition, there is no established correlation between skeletal muscle blood flow and changes in muscle fiber typing. The relationship between skeletal muscle blood flow and muscle fiber type claimed by Tamaki *et al* (1994) is based solely on myosin heavy chain isoform fiber type shifts observed in claudicate patients. Although claudicate patients do have an increased number of oxidative type I fibers as compared to sedentary individuals, claudicants have a lower concentration of oxidative enzymes than sedentary individuals (Anderson & Henriksson, 1977a). Endurance training at low speeds increases peripheral blood flow yet promotes an oxidative shift in skeletal muscle isoforms and concentration increases in oxidative enzymes (Goldspink, 1992). Similar oxidative fiber type shifts are also seen as the result of intermittent high intensity, interval training,

which has been shown to decrease peripheral blood flow (Henriksson & Reitman, 1996). In addition, no significant differences were seen in muscle blood flow and muscle oxygenation elicited by power-lifting exercises (4RM one-arm flexion) and bodybuilder type exercises (10RM one-arm flexion) measured by NIRS (Campbell, Harrison, & Docherty, 2000). However, power lifters do not exhibit the same oxidative fiber type shifts and enzyme profile as body builders (Tesch & Larsson, 1982). Finally, micro-gravity studies have shown conversions in skeletal muscle fast-type myosin isozymes in slow fibers (hybrid I/II fibers) and by the increased expression of fast type II fiber types, although blood flow was near normal and not occluded (Fitts, Riley, & Widrick, 2001). As stated by Staron (1997), skeletal muscle fiber typing should not be used as the definitive indicator for evaluating the oxidative state of skeletal muscle. Therefore, it is questionable that there is a direct relationship between blood flow and muscle fiber type shifts as claimed by Tamaki *et al.* (1996).

DIRECTIONS FOR FUTURE RESEARCH

Although this study gives a glimpse at muscle oxygenation and muscle blood flow there are some limitations to this study. Future work using NIRS should focus on quantifying oxygen utilization and skeletal muscle blood flow, either by developing new NIRS methodology and technology or using a combination of nuclear magnetic spectroscopy and NIRS. Histochemical analysis of oxidative/glycolytic metabolic enzymes, myosin heavy and light isoforms, and capillary distribution should also be done in addition to measures of muscle oxygenation and muscle blood flow. Further, the force and velocity of skeletal muscle needs to be measured if inferences are to be made

between the relationships between fiber types, metabolic enzyme profiles and performance.

Future research questions related to changes in FTS in this area could include: What is the effect of single vs. multiple sets? What is the effect of different rest intervals between sets? What is the effect of lifting tempo? What is the effect of load? What is the effect of different number of repetitions? What are the differences of going to failure and not going to failure? What are the effects of training interference? What is the effect of aerobic fitness on weight training?

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APPENDIX A: REVIEW OF LITERATURE

Review of Literature
Non-Invasive Measurement of Muscle Oxygenation During Resistance Training Using Near Infrared Spectroscopy.

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INTRODUCTION

Improvements in athletic power (Force x velocity) and strength are of great importance to athletes and coaches as they aid in the development of optimal performance in sport (Sale, 1992). The loading parameters of resistance training (RT) used by body-builders are often used to increase athletic power in order to enhance sport performance (Armstrong, Warren, & Warren, 1991). Such RT protocols typically consist of 10 repetitions at approximately 70-75% of 1 maximal concentric contraction (MVC) for 3 sets (Poliquin, 1990). However, the adaptations of muscle tissue is dependent on the type of stimulus it receives, usually related to load, repetition, time of contraction and recovery.

It has been proposed that blood occlusion occurs during some RT protocols that results in oxidative adaptations in muscle tissue (Tamaki, Uchiyaina, Tamura & Nakano, 1994). Although many RT protocols will produce an increase in strength, oxidative adaptations may reduce muscular power, which is often the way strength is expressed in sport (Hintz, Chi, Fell, Ivy, Kaiser & Lowery, 1982; Eiken, Sundberg, Esbjomsson, Nygren & Kaijer,1991). It is, therefore, important to identify the physiological stimulus that produces the desired physiological adaptations to enhance the specific physical development of athletes. The purpose of this review of literature is to investigate the phenomenon of muscle fiber shifts, and the assess the viability of measuring muscle oxygenation by near infrared spectroscopy (N1RS). In addition, this paper will address the rational the acute and chronic changes in muscle oxygenation as the result of RT and possible theoretical muscle oxidative adaptations due to RT.

MUSCLE FIBER SHIFTS

Muscle Classification

Muscle fiber exist in a continuous spectrum based on the force of production, and can be categorized by the duration of muscle contraction and predominant bio-energetic system used for the generation of chemical energy for muscular contractions. Type I muscle fibers (ST) are characterized by low tetanic force production over a extended period of time and fuelled primarily through oxidative enzymes. In contrast, type IIb fibers (FT) are characterized by large tetanic force productions of short duration and fuelled primarily through glycolytic enzymes (Hintz et al., 1982). An athlete may increase absolute strength, without consideration of contraction speed, by employing a RT program that emphasizes moderately high loads performed at a high number of repetitions (Armstrong *et al.*, 1991). Type IIa muscle fibers (FOG) lie between these two polar extremes. FOG are characterized by moderate force productions of long duration and high levels of both oxidative and glycolytic enzymes (Young & Lowery, 1983). RT is thought to increase muscle size by the circumferential and longitudinal addition of muscle contractile protein content, primarily in Type IIb muscle fibers though muscle hypertrophy. Muscle hypertrophy would, therefore, increase strength and achieve a higher level of athletic performance (Sale, 1992).

Muscle Fiber Shifts & Resistance Training

MacDougall, Sale, Elder and Sutton (1982) and Tesch *et al.* (1984) have also noted a fiber type shift (FTS) from FT to FOG a result of RT. Normally, oxidative

adaptations are associated with Endurance training not RT (Holloszy, 1967). Theoretically, RT would decrease the oxidative capacity of muscle by decreasing the relative capillary and mitochondrial densities. Which would result in an increase 02 diffusion gradients due to increased muscle size as a result of muscle hypertrophy (Tesch & Larsson, 1982). Despite the above biochemical and anatomical changes, a shift in the oxidative capacity of Type II muscle occurs as a result of RT (Tesch, Thorsson & Kaiser, 1984).

These fiber type shifts may have direct implications to athletes who require muscular power for successful performance (i.e. rugby, hockey and football players). Although RT will improve the absolute strength of an athlete, a FTS from FT to FOG may not result in any beneficial gains in power, as oxidative muscles tend to produce lower forces at reduced contraction speeds (Eiken *et al.*, 1991). Traditional RT programs, employed by athletes to enhance power, may in fact impede or decrease physical development of an athlete as oxidative adaptations are also associated with lower and slower rates of force production (Hintz *et al.*, 1982).

Validity of Muscle Fiber Shift

The apparent fiber type shift may also be due to methodological oversimplification and subsequent misinterpretation of histochemical staining techniques (Staron *et al.*, 1997). Both MacDougall *et al.* (1982) and Tesch *et al.* (1984) qualitatively assessed human muscle tissue fiber based on the general ATPase activity and the modulating effects of proteins containing sulfhydrl (-SH) groups as described by Padykula, (1954). This histochemical staining technique distinguishes muscle fibers based on the oxidative

capacity of the muscle tissue as a result of enzyme activity. Although fiber typing using ATPase histochemical staining is a proven diagnostic technique, classification of muscle fiber based only on oxidative characteristics does not accurately describe the contractile properties of muscle, usually associated with various myosin ATPase isoforms (Staron *et al.*, 1997). Therefore, the oxidative adaptations observed by MacDougall *et al.* (1982) and Tesch *et al.* (1984) as a result of RT may be due to increases in oxidative enzyme activity and not a change in myosin ATPase isoform. This implies that the apparent fiber type shifts induced by RT may not adversely affect athletic power and other factors may influence the decrement in athletic power as noted by Eiken *et al.* (1991). Little research has been done to simultaneously identify oxidative enzyme activity and myosin ATPase isoforms in relation to RT.

MUSCLE OXYGENATION AND MUSCLE FIBER SHIFTS

Anoxia & Muscle Fiber Shifts

Oxidative muscle fiber shifts have been noted under conditions other than endurance training. Eiken *et al.* (1991) has reported increased percent muscle cross sectional area (CSA%) of slow twitch muscle as a result of intensive ischaemic training of short-term, anaerobic experiments. Saltin and Gollnick (1982) and Terjung, Mathien, Erney and Ogilvie (1988) have reported that low local oxygen concentrations in the muscle or some other factor related to ischaemia seems to be "one independent and adequate" for the increased oxidative metabolic capacity of the muscle. Jansson,

Johansson, Sylven, and Kaijser (1988) have also reported that chronic ischaemia gives rise to an increase in oxidative enzyme activity in leg muscles of patients with intermediate claudication. These studies suggest that exposure of working muscle to anaerobic conditions when there is a restriction of blood flow (BF) and anoxia (i.e. insufficient oxygen supply) may cause an oxidative fiber type shift (Tamaki *et al.*, 1994).

Resistance Training and Muscle Fiber Shifts

Lind and Nicole (1966) noted a saturation of BF of muscular contractions over 60% of one maximal concentric contraction (MVC). At these high loads, it is hypothesized that blood pressure exceeding 120 mmHg creates a state of blood occlusion. Blood occlusion is created by an increased rate of arterial BF to active muscles during RT, with a restriction or a mismatch in venous return. As a result, BF is reduced and pools (De Blasi, Quaglia, Gaspairetto & Ferrari, 1992). Without adequate venous blood out flow, oxygen within the blood is consumed without being replenished due to the stagnation of BF creating a local state of muscle anoxia (Tamaki *et al.*, 1994).

RT protocols employed by athletes to enhance strength through muscle hypertrophy, typically require muscle contractions of 70-75% MVC for several consecutive contraction over multiple sets (Poliquin, 1990). Tamaki *et al.* (1994) hypothesized that muscle contractions over 60% MVC would create a state of anoxia in working muscles during RT. This anoxic state would then provided the stimulus for a muscle FTS, in favour of Type I muscle fiber (Tamaki *et al.*, 1994). This could explain the increased CSA in bodybuilders. This would suggest that FTS is closely related to BF and oxygen supply in the contracting muscles. However, research by Tamaki *et al.*

(1994) provides the only information concerning oxygen consumption and not BF in human contracting muscles, during weight-lifting exercises, assessed by near infrared spectroscopy.

NEAR INFRARED SPECTROSCOPY

Understanding the relationship between blood flow (BF), oxygen supply ($[O_2]$) and oxygen consumption (VO_2) are important factors in understanding the physiology of muscle oxygenation during exercise (De Blasi *et al.*, 1992). The majority of information on factors influencing muscle oxygenation is derived from isolated enzymes and mitochondria. However, from a physiological perspective, " in vitro" experimentation is limited. The metabolism of an intact biological system depends on the complex interplay of several factors that only "in vivo" studies can adequately describe (De Blasi, Ferrari, Natali, Conti, Mega & Gasparetto, 1994).

NIRS & Muscle Oxygenation

The accurate measurement of skeletal muscle oxygen consumption requires the study of muscle blood flow (BF) and tissue arteriovenous O_2 difference during maximal exercise. Traditional techniques used to study oxygen consumption at the level of the muscle are invasive, limiting their practical application. Technical developments in near infrared spectroscopy (NIRS) have made it possible to measure dynamic "in vivo" changes in BF, $[O_2]$ and $\dot{V}O_2$. NIRS is based on the linear relationship between the light absorption characteristics of hemoglobin and myoglobin in the near infrared spectra (700-

1000nm), expressing absorbencies as changes in hemoglobin optical densities (De Blasi *et al.*, 1992; Mancini, Bolinger, Li, Kendrick, Chance & Wilson, 1994; Tamaki *et al.*, 1994). Light transmitted by the NIR probe is either scattered or absorbed by the skeletal muscle. Infrared light can only pass through the tissue of blood vessels, which are less than 1 mm in diameter. Larger diameter blood vessels tend to have thicker vessel walls that do not permit the passage of light. As a result, NIRS can asseses tissue oxygenation at the level of small blood vessels, capillaries and intracellular sites of oxygen uptake (Mancini *et al.*, 1994) which are more representative of muscle oxygenation. The light that is reflected or incident light is monitored by the photo receptors of the NIRS probe (Belardinelli, Barstow, Porszasz, & Wasserman, 1995). The associated energy of transmitted light is absorbed by iron-porphyrin complexes found in hemoglobin (Hb), myoglobin (Mb) and chromophores complexes in the mitochondria. By utilizing different wavelengths of transmitted light, the variability in the absorption spectra between the oxygenated and deoxygenated forms of Hb/Mb and chromophores can be measured using the Lambert-Beer law (Sahlin, 1992; Hampson & Piantadosi, 1988). According to Beer's Law, the number of light absorbing molecules is logarithmically proportional to the concentration of the substance, its molar extinction coefficient and the optical path length of the incident light (Maikala & Bhambhani, 1999). At a wavelength of 860nm, oxygenated forms of Hb/Mb are absorbed whereas deoxygenated forms of hemoglobin primarily absorb light at 760mn. Blood flow can also be inferred by the sum of the absorbency at 760nm and 860nm (Belardinelli, Barstow, Porszasz & Wasserinan, 1995; De Blasi *et al.*, 1992). Thus, NIRS can distinguish qualitative changes in muscle

tissue Hb/MbO_2 and Hb/Mb simply and non-invasively based on the quantity of transmitted and incident light (DeBlasi *et al.*, 1994).

Muscle Oxygenation During Maximal Muscle Contractions

The majority of studies employing NIRS to measure muscle oxygenation have been concerned with oxygen utilization during aerobic exercise or ischaemia in claudation patients. As a result, little is known about muscle oxygenation during muscle contractions above 30% MVC. The study conducted by Tamaki *et al.* (1994) describing muscle oxygenation during 10RM arm-curl of 9 male subjects provides the only data into muscle oxygenation during maximal contractions.

Subjects in the Tamaki *et al.* study (1994), experienced rapid increase in deoxy-hemoglobin and a rapid decrease in oxy-hemoglobin as a result of local oxidative cellular metabolism during one set of a typical RT regimen (Fig. 1). Deoxy-hemoglobin and oxy-hemoglobin returned to pre exercise levels after approximately 30seconds. After 30 seconds, subjects experienced a supra oxidative state, in which oxy-hemoglobin was maintained above pre exercise levels and below pre exercise deoxy-hemoglobin levels. This state lasted approximately 3 min until returning to pre-exercise levels. Subjects also experienced an increase in total Hb up to the termination of exercise. This is attributed to an increase in local blood flow to the working muscle.

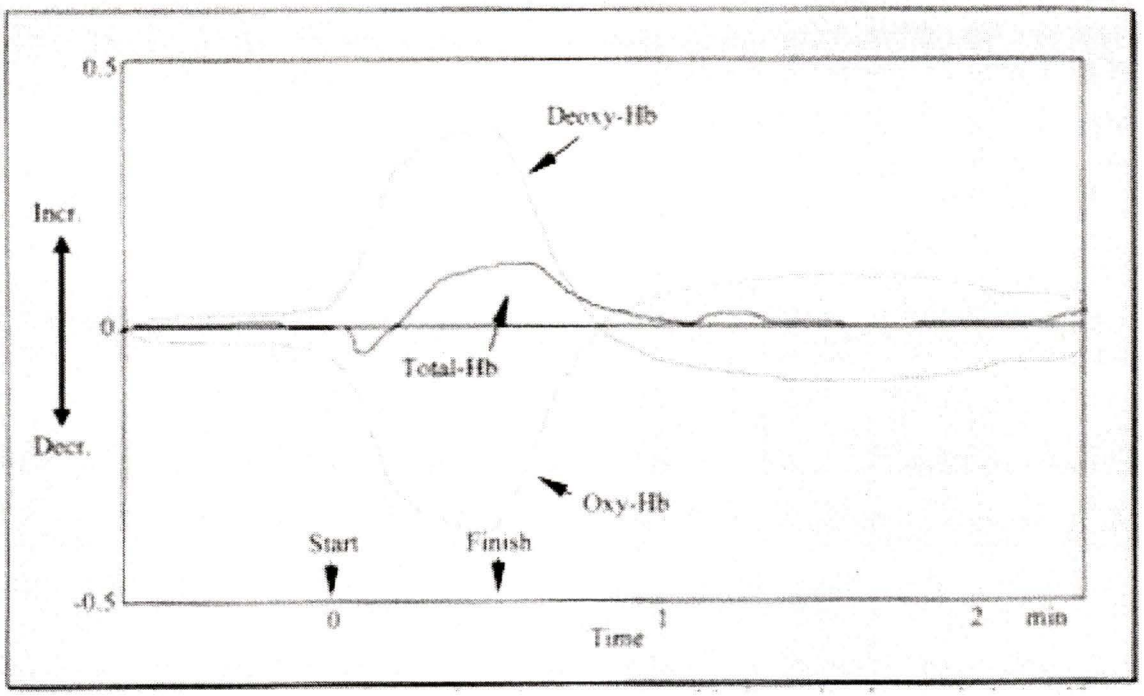

Figure 1: Diagrammatic representation of muscle oxygenation of the biceps brachii muscle during 10 *repetitions of 70% MVC (Tamaki, 1994).*

VALIDITY OF NIRS

NIRS is a relatively new technique in the study of muscle oxygenation. As a result, there are many differences of opinion about the validity of NIRS as a tool to assess muscle oxygenation.

Muscle Blood Flow

De Blasi *et al.* (1994) examined the validity of NIRS to estimate BF by comparing total hemoglobin derived from NIRS with plethysmography, the traditional method of monitoring dynamic changes in BF due to muscle volume changes. A strong relationship between BF, determined by strain-gauge plethysmography, and NIRS was found ($r=0.93$; $P<0.01$;$n=11$). However, NIRS consistently underestimated BF. This may have been

due to the fact that NIRS only monitors small blood vessels and BF from larger blood vessel are undetectable by NIRS (Mancini *et al.*, 1994). Lower hematocrit values in smaller blood vessels could explain the differences between NIRS and plethysmography (De Blasi *et al.*, 1994).

Plethysmography has also been criticized as a method of measuring BF. Measurement from this technique depends on the assumption that changes in muscle volume are directly related to BF. BF calculations do not take into account changes in bone BF and contribution of skin BF which both increase dramatically during exercise. This inflation in BF caused by skin and bone BF estimated by plethysmography may explain the underestimated BF derived from NIRS (De Blasi *et al.*, 1994). In contrast, NIRS is unaffected by changes in skin BF due to the small volume of skin relative to muscle being sampled (Mancini *et al.*, 1994). Although severely criticized, plethysmography is still accepted as the "gold standard" measure of BF (De Blasi *et al.*, 1994).

Changes in Muscle Oxygenation

Typical light emission of N1RS is between 700-1000nm. This range of light frequencies is absorbed by Hb/Mb and chromophores of the mitochondria. NIRS, therefore, measures the saturation of both O_2 carrying molecules and cannot be distinguished. Changes in skeletal muscle Hb/MbO_2 have been studied using NIRS during occlusion studies and exercise studies. Sako, Hamaoka, Higuchi, Kurosawa & Katsumura (2001) measured the post exercise $\dot{V}O_2$ of the finger flexor muscles during 15 min arterial occlusion using ^{31}P-MRS and NIRS. Immediately after arterial occlusion was initiated Hb/MbO_2 decreased linearly for 5-6 min and then remained unchanged for

the remainder of the protocol. During exercise Hb/MbO_2 decreased rapidly at the onset of exercise, rapidly increased immediately after exercise ceased, and then decreased further after post exercise arterial occlusion. Statistically significant correlation between NIRS and MRS indicate and Hb/MbO_2 trends were attributable to muscle oxidative ATP production.

Contribution of Myoglobin to Changes in Muscle Oxygenation

Another concern about the application of NIRS to RT, is that the contribution of myoglobin to muscle oxygenation has not been studied during muscle contractions under conditions of heavy resistance. Hb/Mb and HbO_2/MbO_2 have identical absorbencies in the near-infrared light range and as a result, measures of muscle oxygenation are a product of both Hb/Mb and HbO_2/MbO_2 (Hampson & Piantadosi, 1988). The exact contribution of Mb is unclear. Claims have been made that myoglobin has minimal influence on oxygen desaturation during exercise (Mancini, Bolinger, Li, Kendrick, Chance & Wilson 1994; Wilson, Mancini, McCully, Ferraro, Lanoce & Chance, 1989; De Blasi *et al.*, 1994). However, these studies observed muscle oxygenation during aerobic exercise. Myoglobin has a higher affinity for O_2 than hemoglobin at sites of O_2 utilization. During aerobic exercise, O_2 partial pressure (PO_2) would not drop sufficiently for myoglobin to desaturate. A rapid decrease in oxygenation above lactic threshold has been observed, the point of increased involvement of anaerobic metabolism (Belardinelli, Barstow, Porszasz & Wasserinan, 1995). It would be expected that loads experienced during RT would well exceed 30% MVC and, therefore, myoglobin would influence NIRS. Mancini *et al.* (1994) examined myoglobin desaturation during

progressive resistance loading up to 30% MVC while subjects performed wrist curls. Resistance did not exceed 30% MVC and subject training status was not specified. One subject did experience oxygen desaturation of myoglobin and this was attributed to the training status to the subject (Mancini *et al.*, 1994). Wilson *et al.* (1989) suggested that myoglobin desaturation did not occur until a partial pressure of oxygen below 20 mmHg so Mb would not desaturate at all during low to moderate exercise and therefore not contribute to the NIR signal. In addition, when the contribution of Mb to the NIR signal was decreased by ethyl hydrogen peroxide, no significant differences was seen. It was concluded that changes in Hb contributed more to the overall NIR signal. When NIRS and nuclear magnetic spectroscopy (NMS)were used to monitor muscle oxygenation during exercise greater amounts of Hb deoxygenation were observed than Mb deoxygenation (Mancini et al. (1994). However, Tran, Sailasuta, Kreutzer, Hurd, Chung, Mole, Kuno & Jue (1999) demonstrated similar desaturation kinetics between Mb measured by NMS and the observed kinetics of deoxygenation as measured by NIRS, suggesting that the NIRS signal is primarily derived from Mb desaturation during exercise. As a result of the lack of consistent evidence, most studies cite Mb as a minor contributor to the NIR signal, because Mb has similar absorbencies to Hb although the influence Mb has not been fully resolved (Belardinelli *et al.*, 1995; Wilson *et al.*,1989; Sahlin, 1992; Chance *et al.*, 1992).

It is theorized that myoglobin will contribute to muscle PO_2 when venous PO_2 becomes considerably lowered as a result of cellular oxidative metabolism (De Blasi *et al.*, 1992). These conditions are seen under anoxic conditions or during periods of exhaustive exercise (Belardinelli *et al.*, 1994). Hemoglobin desaturation is, therefore, not

an accurate indicator of muscle anoxia as myoglobin will facilitate muscle oxygenation at the level of the mitochondria, which will be undetectable or not distinguishable from hemoglobin because Hb and Mb absorb light in the same spectral range (Mancini *et al.*, 1994). New methodology or simultaneous NIRS with magnetic resonance spectroscopy of myoglobin at greater than 30% MVC must be conducted to enable proper assessment of muscle anoxia.

CONCLUSION

In conclusion, NIRS provides a non-invasive method of monitoring tissue oxygenation. However, the major limitation of NIRS is that it can only provide qualitative data (De Blasi *et al.*, 1992; Mancini *et al.*, 1994; Tamaki *et al.*, 1994). As the light path length is not consistent between or within subjects, only changes in OD can be obtained via NIRS (De Blasi *et al.*, 1994). As a result of the varying light absorption levels, NIRS cannot yield absolute levels of deoxygenated hemoglobin.

ADAPTIVE STRATEGIES TO ANOXIA

Athletes and coaches use a variety of behavioral and physical training to elicit a sportspecific adaptations in the attempt to improve physical performance for a specific sporting environment. RT is a common training strategy employed by power athletes and coaches to manipulate the rate and direction of muscle physiological and biochemical function to enhance muscle power.

Only the study by Tamaki *et al.* (1994) provides support for anoxia as the stimuli for FTS during RT. However, no theoretical physiological or biochemical mechanisms were posed to support the conclusion of the study.

Whether or not anoxia contributes to muscle FTS, the mechanism behind the oxidative adaptations as a result of RT are still unknown. Theories and mechanisms on blood flow, O_2 supply and consumption only attempt to explains FTS on the physiological level. It is apparent that oxidative adaptations as a result of RT differ from adaptations due to endurance training. Increases in capillary and mitochondria density minimizes the effects of anoxia experienced by muscle during endurance type training. These adaptations are not seen in response to RT. In fact, the decreases in capillary and mitochondria density as a result of hypertrophy would only serve to reduce the oxidative capacity of muscle. Despite these contradictory adaptations oxidative FTS is still seen in muscle in response to RT.

SUMMARY

In conclusion, the apparent FTS as a result of RT many have significant implications for the development of athletic performance in power athletes. Due to limited research in the area no theoretical physiological nor biochemical mechanism have been posed to explain FTS in response to RT. However, this does provide an opportunity to address fundamental research questions and to conduct original research in this area.

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APPENDIX B: INFORMED CONSENT

Informed consent form for the research projects:

Near infrared estimation of muscle oxygenation during 4RM and 10RM weight lifting exercise

And

Acute skeletal muscle oxygenation during resistance training using NIRS

The purpose of these studies is to determine the influence of 4 repetition maximum (4RM) and 10 repetition maximum (10RM) resistance exercise protocols on skeletal muscle oxygenation and blood volume and to examine changes in acute muscle oxygenation and blood volume during acute resistance training that may act as a stimulus for a muscle fiber type shift.

Total duration of the study will be three days including an orientation session. During the orientation session, you will be instructed on proper lifting technique, have 4RM and 10RM values determined and introduced to the Near infrared spectrometer (NIRS; a surface probe used in the determination of muscle oxygenation). All exercise involves seated biceps curls performed on the preacher curl bench. Six exercise variations will be performed over the course of the three testing sessions. Prior to each exercise protocol a baseline will be established. This consists of a pressure cuff applied to the arm at 250 mmHg, occluding blood flow to the arm for a period of 8 minutes. Single set 4 repetition and 10 repetition contractions without a load will be completed as will a set unloaded 10 repetitions where blood flow is occluded throughout the exercise. As well, loaded 4RM, where four repetitions are performed to failure, and 10RM, where ten repetitions are performed to failure, will be performed. You will also complete a 10RM set with occluded blood flow. Trained subjects and untrained subjects will participate in both exercise protocols.

Muscle oxygenation and blood volume data will be collected using a portable near infrared spectrometer probe (NIRS). NIRS utilizes white light to analyze oxygen content of blood. It is a non-invasive procedure and poses no danger to the subject.

I acknowledge that the research procedures described in this form have been explained to me to my satisfaction. I am aware that muscle oxygenation will be measured for 10RM and/or 4RM protocols non-invasively with a NIRS probe. I understand that the 4RM and 10RM protocols may result in some discomfort in the form of delayed onset muscle soreness. I am aware that I can terminate my participation in any research procedures at any point without penalty and that any data collected will be destroyed within a period of five years. I understand that my participation or non-participation has no effect on my grades or standing and acknowledge no coercion of any kind has been used. I have been guaranteed anonymity as a subject and assured that all data will be kept confidential in a secured file.

I voluntarily consent to participate in this project and hereby release the University of Victoria and all personnel involved in and around this research project from any and all liability for any injury that may result from my participation in this study.

Participant name: Participant signature:

Participant Phone: Witness:

Researchers: Sean Campbell (250) 385-4967
David Harrison (250) 592-8325
Supervisor: Dr. D. Docherty (250) 721-8375

APPENDIX C: RAW DATA

Raw data 1: Raw data expressed as optical density (OD) for trained (1) and untrained (2) subjects for the maximum (Max) and minimum (Min) muscle blood flow and the muscle blood flow range (Ra) for the warm up set (50 % of 10RM) and Sets1, 2 and 3 of the 10RM exercise protocol, measured by NIRS.

Subject	Trained	Warmmax	Warmmin	WarmRa	Set1Max	Set1min	Set1Ra	Set2Max	Set2Min	Set2Ra	Set3Max	Set3Min	Set3Ra
1	1	-0.187	0.283	0.47	-0.358	0.454	0.812	-0.347	0.675	1.022	-0.265	0.631	0.896
2	**1**	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
3	1	-0.342	0.145	0.487	-0.577	0.375	0.952	-0.463	0.465	0.928	-0.452	0.537	0.989
4	1	-0.105	0.442	0.547	-0.127	0.639	0.766	-0.105	0.574	0.679	-0.028	0.51	0.538
5	1	-0.302	0.755	1.057	-0.223	0.795	1.018	-0.116	0.852	0.968	-0.149	0.979	1.128
6	1	-0.202	0.333	0.535	-0.21	0.46	0.67	-0.153	0.439	0.592	-0.093	0.57	0.663
7	1	-0.311	0.646	0.957	-0.434	0.773	1.207	-0.344	0.68	1.024	-0.27	0.896	1.166
8	1	-0.097	0.393	0.49	-0.123	1.048	1.171	-0.056	1.023	1.079	-0.02	1.061	1.081
9	1	-0.229	0.607	0.836	-0.193	0.836	1.029	-0.157	0.807	0.964	-0.125	0.787	0.912
10	1	-0.148	0.456	0.604	-0.16	0.716	0.876	-0.174	0.593	0.767	-0.116	0.691	0.807
11	1	-0.096	0.482	0.578	-0.108	0.619	0.727	0	1.27	1.27	0.022	1.112	1.09
12	1	-0.074	0.782	0.856	-0.143	0.78	0.923	-0.029	0.78	0.809	0.044	0.904	0.86
13	1	-0.25	0.3	0.55	-0.428	0.696	1.124	-0.344	0.536	0.88	-0.36	0.446	0.806
14	1	-0.324	0.824	1.148	-0.327	0.87	1.197	-0.274	0.716	0.99	-0.254	0.565	0.819
1	2	-0.066	0.424	0.49	-0.018	0.914	0.932	0.017	0.734	0.717	0.0099	0.86	0.8501
2	2	-0.3	0.586	0.886	-0.276	0.874	1.15	-0.166	0.639	0.805	-0.188	0.5	0.688
3	**2**	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
4	2	-0.129	0.522	0.651	-0.236	0.573	0.809	-0.266	0.781	1.047	-0.245	0.564	0.809
5	2	0.052	0.576	0.524	-0.016	0.548	0.564	-0.05	0.564	0.614	-0.007	0.71	0.717
6	2	-0.537	0.329	0.866	-0.519	0.477	0.996	-0.452	0.333	0.785	-0.465	0.475	0.94
7	2	-0.381	0.512	0.893	-0.56	0.612	1.172	-0.426	0.767	1.193	-0.434	0.659	1.093
8	2	-0.097	0.695	0.792	-0.025	0.923	0.948	-0.062	0.766	0.828	0.02	0.673	0.653
9	2	-0.228	0.354	0.582	-0.463	0.636	1.099	-0.407	0.828	1.235	-0.436	0.52	0.956
10	2	-0.055	0.252	0.307	-0.083	0.547	0.63	0.022	0.813	0.791	0.059	0.949	0.89

Raw data 2: Raw data expressed as optical density (OD) for trained (1) and untrained (2) subjects for the maximum (Max) and minimum (Min) muscle oxygenation and the muscle oxygenation range (Ra) for the warm up set (50 % of 10RM) and Sets1, 2 and 3 of the 10RM exercise protocol, measured by NIRS.

Subject	Trained	Warmmax	Warmmin	WarmRa	Set1Max	Set1min	Set1Ra	Set2Max	Set2Min	Set2Ra	Set3Max	Set3Min	Set2Ra
1	1	-0.042	0.546	0.588	0.04	1.42	1.38	-0.042	1.307	1.388	-0.081	0.633	0.714
2	1	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
3	1	-0.028	0.368	0.396	0.009	0.988	0.979	-0.151	0.791	0.961	-0.17	0.63	0.8
4	1	-0.085	0.61	0.695	-0.0469	0.919	0.9659	-0.0439	1.186	1.2519	-0.0659	1.275	1.3409
5	1	-0.013	1.177	1.19	-0.058	0.643	0.701	-0.045	0.577	0.618	-0.041	0.546	0.587
6	1	-0.059	1.091	1.15	0.115	1.254	1.139	0.031	1.133	1.086	0.047	1.041	0.994
7	1	-0.031	0.628	0.659	0.08	1.164	1.084	0.024	0.363	0.422	-0.059	0.48	0.539
8	1	0.003	1	0.997	-0.026	1.48	1.506	-0.076	1.649	1.728	-0.079	0.877	0.956
9	1	-0.061	1.407	1.468	0.003	1.144	1.141	-0.011	0.986	0.999	-0.013	0.608	0.621
10	1	-0.003	0.807	0.81	-0.034	0.191	0.225	-0.036	0.356	0.414	-0.058	0.182	0.24
11	1	0.018	0.514	0.496	-0.064	0.932	0.996	-0.097	0.414	0.506	-0.092	0.384	0.476
12	1	-0.015	0.966	0.981	0.141	1.515	1.374	0.095	0.51	0.488	0.022	0.782	0.76
13	1	-0.014	0.655	0.669	-0.027	1.805	1.832	-0.079	1.971	2.062	-0.091	1.439	1.53
14	1	-0.028	0.719	0.747	-0.035	0.696	0.731	-0.075	0.409	0.503	-0.094	0.517	0.611
1	2	0.013	0.996	0.983	-0.096	0.628	0.724	-0.096	0.827	0.859	-0.032	1.145	1.177
2	2	0.03	0.597	0.567	-0.109	0.466	0.575	0.02	1.063	1.188	-0.125	0.346	0.471
3	2	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
4	2	-0.126	0.23	0.356	-0.039	1.267	1.306	-0.092	0.145	0.163	-0.018	0.195	0.213
5	2	0.009	0.496	0.487	-0.042	0.953	0.995	-0.039	1.333	1.442	-0.109	1.154	1.263
6	2	-0.096	0.195	0.291	-0.003	0.646	0.649	-0.143	0.194	0.388	-0.194	0.213	0.407
7	2	0.023	0.736	0.713	0.015	1.356	1.341	-0.106	0.554	0.697	-0.143	0.533	0.676
8	2	-0.059	0.565	0.624	-0.068	1.01	1.078	-0.068	1.183	1.395	-0.212	1.052	1.264
9	2	-0.054	0.457	0.511	-0.096	0.973	1.069	-0.131	0.988	1.137	-0.149	1.102	1.251
10	2	-0.042	0.709	0.751	0.014	1.201	1.187	-0.007	1.681	1.704	-0.023	1.03	1.053

Raw data 3: Descriptive raw data of the trained (1) and untrained (2) subjects for weight (kg), age (years), 10RM (lbs.), relaxed arm girth (cm), flexed arm girth (cm) and the anterior (Ant), medial (Med), posterior (post), lateral (Lat) and the sum of then anterior and posterior (Ant+Post) skin fold measurement (mm).

Subject No.	Trained	Weight	Age	10RM	Relaxed	Flexed	Ant	Med	Post	Lat	Ant+Post
1	1	71.5	25	22.5	26	29.5	2.8	2.2	2.4	2.2	5.2
2	1	N/A	23	30	28.4	31.1	2.6	2.3	6.4	4.6	9
3	1	74	24	30	29	31.8	3	2.4	6.6	5	9.6
4	1	N/A	28	35	33.4	36.4	2.2	2	4.2	6.2	6.4
5	1	N/A	32	25	25	28	2.4	2.8	3.2	6.8	5.6
6	1	N/A	21	30	29	32	2.6	2.6	5.6	3.4	8.2
7	1	N/A	21	32.5	28	32	2.4	2.4	4.4	5.2	6.8
9	1	N/A	27	25	23	26	2	1.8	3.2	3.2	5.2
8	1	N/A	23	25	30.4	32.4	3.8	3	4.8	10	8.6
10	1	N/A	29	27.35	31.5	34.5	2.8	2.7	9.6	4.2	12.4
1	2	92	27	55	37	41.6	2.6	2.4	4	4.8	6.6
2	2	84.6	31	45	36.1	38.8	2.6	2.6	3.6	7.2	6.2
3	2	N/A	18	30	31	34.5	2.8	2.6	6.6	6.4	9.4
4	2	124.6	27	65	44	48	2.6	2.8	3.8	5.4	6.4
6	2	82.1	21	32.5	33	36	2.6	2.4	5	7.4	7.6
7	2	93	23	40	35	39.4	3	3.2	3.6	7.8	6.6
8	2	87.2	24	42.5	35.7	38.5	2.5	6.4	6.6	3.2	9.1
9	2	N/A	23	40	35	37	3	4	4.2	6.8	7.2
10	2	N/A	19	35	34	45	2.8	3	5	6.6	7.8
11	2	N/A	23	30	36.5	37.5	2.2	2.4	3.8	3.2	6
12	2	N/A	23	40	37	40.5	2.8	2.2	3.8	5.6	6.6
13	2	73.9	25	30	35	37.8	2.6	3.2	8.1	10.2	10.7
14	2	N/A	26	32.5	34.5	38	2.6	2.5	3.6	4.6	6.2

VITA

DAVID MICHAEL HARRISON

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EDUCATION

1997 - Present	University of Victoria, Victoria, BC. Master's of Science, 2001. Recipient of Internal Grant Funding for research, 1998. • School Nomination for Excellence in Teaching by a Graduate Student 2000 • Graduate Student Bursary 2000 • Graduate Teaching Fellowship 1997-98, 1998-1999, 1999-2000. • Recipient of Internal Grant Funding for research, 1998.
1990 - 1997	University of Victoria, Victoria, BC. Bachelor of Science Degree - Kinesiology, Honours.
1987 - 1990	Diamond Jenness Secondary School, Hay River, NWT. High School Diploma, 1990. Recipient of Sport North Athletic Scholarship, 1990.

RELEVANT WORK EXPERIENCE

2001- Present	**Research Assistant, University of Victoria, Victoria, BC.**
1999 - 2000 Sept. - May	**Anatomy Co-ordinator and Instructor, University of Victoria, Victoria, BC.** • Laboratory budget and purchasing of lab materials. • Organising and scheduling of weekly Laboratory periods and after Laboratory study times. • Supervising Instructors and Laboratory Assistants. • Classroom teaching and individual instruction. • Developing content for weekly Laboratory. • Writing and marking weekly Laboratory exams. • Facilitation of classroom environment, test evaluation and grading. • Implementing and maintaining course listserv • Developing interactive Anatomy web-site.

1997 - 2001 Sept. - April **Anatomy Laboratory Instructor, University of Victoria, Victoria, BC.**

- Classroom teaching and individual instruction.
- Writing Laboratory exams.
- Facilitation of classroom environment, test evaluation and grading.

1996 September - present **DreadKnotWeb Productions, Victoria, BC.**

- Sole proprietor of web production home based company
- Design layout, programming, graphics and maintenance of interactive web sites.
- Design and maintain on going projects with the University of Victoria (Athletics and Recreation web site and Women's Studies home page).
- Co-founder of North West Climbing Resource an interactive climbing web site.

1996 July - August **Tree Line, Hay River, NWT.**

- Joint reforestation project with TreeLine and the Government of the NWT Department of Natural Resources.

1996 & 1999 May - June **Celtic Reforestation, Prince George, BC.**

- Reforestation

1995 July - August **University of Victoria, Athletic and Recreation, Victoria, BC.**

- Developed personalised weight and fitness programs
- Weight room attendant and personal supervisor
- Overseer of safety and equipment maintenance

1994 - 1997 Sept. - April **Anatomy and Physiology Teacher's Assistant, University of Victoria, Victoria, BC.**

- Classroom teaching and individual instruction.
- Facilitation of classroom environment, test evaluation and grading.

1993 May - July

Government of NWT, Ministry of Natural Resources
Evergreen Fire Fighting Company, Hay River, NWT.

- Fire suppression, radio operation, commissary ordering, time keeping

1988-1989 May - August

Town of Hay River, NWT.

- Lifeguard
- Supervision and safety of patrons
- Knowledge of pool chemicals and filter systems to meet public health standards

AWARDS AND SEMINARS

- School Nomination for Excellence in Teaching by a Graduate Student 2000
- Graduate Student Bursary 2000
- Graduate Teaching Fellowship 1997-98, 1998-1999, 1999-2000.
- Recipient of Internal Grant Funding for research, 1998.
- Conducted seminar on Tutorials and Small Group Discussions during the 6th annual Graduate Teaching Assistant's Training Day, 2000
- Internet and Higher Education (Seminar)
- Introduction to the Web (Seminar)
- Education Technology Conference Series

SPECIAL SKILLS AND AWARDS

- RLSSC National Lifeguard Service
- St. John Basic First Aid
- CPR Level C
- Computer Experience: Macintosh, IBM compatible
- Win 3.1 and 95, 98 Word 7.0, Clarise Works, Cricket Graph, Excel, SPSS 8.0, Photoshop 4.0, FrontPage, WS-FTP

EXTRACURRICULAR ACTIVITIES

- Trainer/ player for Velox Rugby Team 1996 - present.
- UVic Men's Varsity Rowing Team, 1994 - 1996.
- Recruited participant in the 1995 Olympic development camp.
- Recipient of BC Athletic Scholarship for rowing 1995-96.
- Player on UVic Men's Varsity Rugby Team 1992-1994.

REFERENCES

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Dr. Kathy Gaul work number: 721-8380

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Title of Thesis:

Changes in Acute Skeletal Muscle Oxygenation and Blood Flow in Trained and Untrained Males During 10RM Resistance Training Protocol Measured by NIRS.

Autho[redacted]

David Michael Harrison

01.25.02