The impact of a single high volume exercise stimulus incorporated into a taper on 2000m ergometer performance

by

James Brotherhood
Bachelor of Physical Education, University of Alberta, 2006

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of the Requirements for the Degree of

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in Kinesiology in the School of Exercise Science, Physical and Health Education

Supervisory Committee

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Abstract

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The purpose of this experiment was to examine the efficacy of implementing a high intensity, high volume workout into the late stages of a taper, to identify if there was a performance enhancing effect beyond that of an intensity maintained, reduced-volume taper. Eleven male collegiate rowers (age 21.0 ± 1.9 years, VO_2max 60.9 ± 5.8 ml/kg/min) completed 23 days of progressively overloaded training, followed by 5 days of reduced training volume. Participants were matched and randomly assigned to either a high intensity-low volume or high intensity-high volume treatment workout approximately 48 hours prior to an indoor rowing competition. Other than the treatment workout, all prescribed training was identical. Both tapers resulted in significant improvements in 2000 m ergometer performance; however there was no statistically significant difference between these groups (Low volume: 5.4 ± 2.7 seconds High volume 4.0 ± 3.3 seconds) Post race blood lactate tended to be higher following taper, however it did not reach significance ($p = 0.06$) and there was no difference between groups. There were no differences throughout training and taper for hemoglobin (Hb), hematocrit (Hct.), and plasma volume in either group. Mean corpuscular volume (MCV), increased with training and increased

further with taper in both groups; conversely, red cell distribution width (RDW) decreased with training and decreased further with taper in both groups. Jump height did not change from pre-taper to competition; however, there was a decrease in dip depth and a corresponding increase in peak acceleration and rate of force development in both groups. There was also a reduction in fatigue at competition compared to week 2 as measured by the Profile of Mood States questionnaire. These physiological and psychological adaptations may in part explain the observed combined 1.8% improvement in 2000m ergometer performance compared to pre-taper test times, however we were unable to discern any differences in any measured parameters between the higher volume and low volume treatment groups. The changes in hematological parameters may be indicative of decreases in erythrocyte age; and the adaptations to acceleration / rate of force development suggest potential improvements under the broad theme of movement economy. This study found that employing a 5 day reduced volume taper improved performance, however, implementing a higher volume, high intensity stimulus 48-52 hours prior to competition resulted in no added benefit and a potentially meaningful (1.4 seconds) yet not significant reduction in performance response to taper.

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Acknowledgments

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Dedication

To my family, for your unwavering support in everything I do. Any and all successes I have in life are a testament to the incredible support network I am so fortunate to have.

Chapter 1 Introduction

As elite athletes prepare for competition, a universal practice with both anecdotal and scientific support is the use of a taper. A taper is a period of reduced training in the days leading up to competition with the intention of full recovery; to realize the adaptations made from training and optimize performance (Mujika and Padilla, 2003). It is characterized by a reduction in training volume, allowing both physiological and psychological recovery and often results in performance enhancements between 0.1 and 8% (Mujika and Padilla, 2003).

It has been shown consistently that during taper, training intensity must remain high, and a volume decrease of greater than 50% is needed to allow full recovery (Mujika and Padilla, 2003; Bosquet, Montpetit, Arvisais & Mujika, 2007). In most sports, there is a level of "feel" and comfort, thus it is usually desirable to maintain or only slightly drop training frequency (Mujika, Goya, Ruiz, Grijalba, Santisteban & Padilla, 2002).

The positive effects of a taper have been investigated for a multitude of physiological variables. Accompanying increases in performance, researchers have seen increases in VO_2max, (Neary, Martin, & Quinney,,2003; Mujika, Padilla, Pyne & Busso, 2004) and large improvements in movement economy (Houmard & Anderson, 1994; Johns et al., 1992), purportedly due to elevations in muscle mitochondrial capacity, neural, structural and biomechanical factors (Mujika et al., 2004).

Changes in hematological parameters have also been observed; including increases in total blood volume, hemoglobin (Hb) and hematocrit (Hct). These have been attributed to a decreased hemolysis from training, and an overshoot in hematopoiesis resulting in a net increase in erythrocytes (Mujika 1998; Mujika et al., 2004).

During taper, muscle glycogen concentration has been shown consistently to increase by as much as 34% (Neary Martin, Reid, Burnham, & Quinney, 1992). This finding is supported by other investigations, showing increases between 13 and 29% in muscle glycogen concentration with concomitant improvements in performance of 2.2 to 8%. (Neary et al., 2003; Shepley, Macdougall, Cipriano, Sutton, Tarnopolsky & Coates, 1992; Walker, Heigenhauser, Hultman & Spriet, 2000).

Peak blood lactates have been shown to increase, likely related to a mass action effect due to increased post-taper muscle glycogen (Houmard et al., 1994; Mujika et al., 2004) and sub maximal blood lactates have been shown reduced, which compliments the aforementioned research on movement economy (Kenitzer 1998; D'Acquisto, Bone, Takahashi, Langhans, Barzdukas & Troup, 1992; Costill, King, Thomas & Hargreaves, 1985; Steinacker et al., 2000).

Because the taper has such a profound impact on the physiology and psychology of each athlete, it is difficult to ascertain the contribution of each adaptation to performance. The holistic nature of taper leaves many questions regarding the optimal taper strategy. Therefore, many variations are explored in attempts to maximize the benefits of the taper and optimize performance.

One strategy that is beginning to appear in some endurance sports with anecdotal efficacy is a high intensity, low volume taper, with a single high intensity, high volume workout, incorporated two to three days out from the competition. The physiological rationale for this is speculative, yet, with anecdotal evidence of success, it may play a role in accentuating the positive adaptations of the taper resulting in additional performance benefits.

Physiological Rationale

Positive adaptations that may result from a higher volume workout amidst a taper include an exercise induced plasma volume expansion, which has been shown to occur in both trained and untrained populations when unaccustomed loads are, applied aerobically (Warburton, Gledhill & Quinney, 2000). This plasma volume expansion has been shown to increase VO_2max and improve endurance performance in untrained and moderately trained participants, however it has not shown significant increases in VO_2max or differences on high intensity endurance performance in elite cyclists (Warburton et al., 2004; Zavorsky 2006).

Enzymatic and metabolic properties at the muscle such as citrate synthase activity, myofibrillar ATPase, succinate dehydrogenase, Beta hydroxyacyl CoA dehydrogenase and cytocrome oxidase have been shown to increase with a taper (Shepley et al., 1992; Mujika et al., 2004). Often there are increases in performance without increases in VO_2 and it is likely that these metabolic and enzymatic adaptations play a large role in the enhanced performance (Mujika et al., 2004).

It is conceivable that the single high volume workout could stimulate further upregulation to these properties while still providing adequate recovery time prior to the performance. Researchers have recently investigated the short term training adaptations of high intensity exercise on Na+ -K+ - ATPase maximal activity and found that in trained athletes, there is a preferential upregulation of Na+ -K+ -ATPase α – isoform mRNA expression (Green, Barr, Fowles, Sandiford & Ouyang, 2004; Aughey et al., 2007). Other metabolic enzymes such as citrate synthase have also been shown to be highly responsive to high intensity exercise bouts (Burgomaster, Heigenhauser & Gibala, 2006). An upregulation of metabolic enzymes in

response to acute intensification and increased volume of exercise could play a functional role in minimizing muscular fatigue and improve athletic performance (Aughey et al., 2006).

Conversely metabolic properties may be put at risk by the higher volume session. Neuromuscular fatigue has been associated with decreases in resting sarcoplasmic reticulum Ca^{2+} ATPase uptake and activity, resulting in reduced sarcolemmal excitability, thus reducing excitation contraction coupling (Tupling, Green, Roy, Grant, Ouyang, 2003; Holloway et al., 2005).

The single higher volume treatment will could result in greater glycogen depletion at the muscle than a low volume, high intensity workout which, depending on nutrition and rest could have important ramifications on performance. The insulin response to exercise may stimulate an overshoot in muscle glycogen storage, resulting in a carbohydrate loading effect. In contrast, if this glycogen storage depleting session leads to reduced stores on race day, there could be a negative effect on performance.

The hormonal milieu plays a role in recovery, and depending on the effects of the differing sessions, the effects may play a role in altering performance 48 hrs later. The pituitary gland may be stimulated to release greater levels of growth hormone, resulting in positive adaptation (Crewther, Keogh, Cook & Cronin, 2006). Conversely, the high volume session could lead to elevated cortisol levels, attenuating recovery by decreasing protein synthesis, increasing protein degradation, and reducing circulating levels of other anabolic hormones such as testosterone and growth hormone (Crewther et al., 2006; Kraemer & Ratamess, 2005; Deschenes, Kreamer, Maresh & Crivello, 1991)

The impact of a high intensity high volume session during taper is unknown and whether these adaptations can impact performance is entirely speculative. Because both taper and high

intensity exercise have profound impacts physiology, it is conceivable that proper manipulation of these variables could result in performance enhancement.

Purpose

The purpose of this project is to determine whether a high intensity low volume taper, with a high intensity, high volume workout 2 days out from competition has the ability to enhance performance to a greater extent than a high intensity, low volume taper in a control group in rowers. A secondary objective was to observe a broad spectrum of physiological variables to provide insight to the consequences of each taper in collegiate level male rowers.

Delimitations

Participants were volunteers from the University of Victoria Men's Rowing Team, the skill, technique, experience and fitness level necessary to train and compete at the required frequency and intensity limited the availability of participants to this specific population.

Limitations

The inability to blind participants was an inherent limitation to this study. To minimize any psychological effects, both groups were openly explained the benefits of each taper protocol with emphasis on the possible performance enhancing benefits of each taper strategy.

Because performance was measured in actual competition, many exogenous variables such as presence of support, race lane assignment, and other similar variables were not able to be controlled for and may have had an impact on the results

Due to limitations imposed by the coaching staff, the study was limited to 4 weeks during a pre-competitive phase of training. It has been consistently shown that the amount of training prior to taper has a large impact on the necessary duration needed (Mujika et al 2004), and therefore, the ecological validity of the results is limited.

Chapter 2 Methods

Participants

Twenty-one college level rowers were recruited from the Victoria area for this study. All participants were male and between the ages of 18 and 26 years. Each participant provided written informed consent and was verbally reminded each day of their right to withdraw from the study without future consequence. Ethical approval was obtained from the University of Victoria Human Research Ethics Committee and University of Victoria Biohazard Safety Committee.

Experimental Design

The experimental design was a balanced assignment, repeated measures. The participants underwent 23 days of progressively overloaded training, followed by a 5 day taper prior to the Western Canadian Indoor Rowing Championships. All ergometer training and testing was done on Concept 2 Model D ergometer (Concept 2, Vermont USA). Prescribed training and taper was identical for both groups other than one high intensity, high volume workout two days (day 26) prior to the competition (day 28).

Athletes were ranked according to the test result of their week 3 ergometer test time, and subsequently paired in order from fastest to slowest. An individual external to the investigation randomly assigned one athlete from each pairing to the high volume treatment group and the other to the low volume treatment group. The athletes were not told of their group assignment until arrival at the exercise session on day 26.

Performance comparisons were made between the 2000m ergometer test at the end of week 3 (Day 20) and performance at the Indoor Rowing Championships. Hematological status was measured at baseline, prior to the pre-taper ergometer test, 12-14 hours prior to the treatment

session and at competition. Neurological fatigue was assessed at each ergometer test. And mood state was measured at baseline, the end of week 2, pre-taper, and at competition.

Each participant was given a nutrition journal and instruction to record dietary intake over the 2 days preceding and morning of both the pre-taper 2000m ergometer test and competition. The dietary intakes were subsequently analyzed for macronutrient content (Food Processor Version 8.6.0, Oregon, USA) and compared.

Pre-experimental protocol

Each participant reported to the laboratory during the week of November 12-15 2007, for post fall competitive season testing. These measurements were taken to asses the magnitude of detraining over an unstructured period of training due to exams and holidays. Anthropometric measurements, arm and thigh girths were taken and a progressive maximal oxygen consumption test on a rowing ergometer was conducted.

Two to five days prior to day 1 of the experiment, the participants reported to the same laboratory and underwent the same testing schedule; in addition the baseline measurement of "Profile of mood states" questionnaire was taken at this time.

Participants attended the lab within 4 days following competition for their post-study anthropometric measurements and VO_2max test which followed the same procedures as the prior two tests.

Approximately 5 ml of venous blood was extracted to be analyzed for a complete blood count 3 days prior to the beginning of the experiment. No participants completed their baseline testing session on this day. Each participant observed 20 min of stasis in an upright seated

position prior to blood sampling, and other methodological controls as outlined in a subsequent section of this methodology.

Anthropometric measurement

Skinfolds were measured in triplicate with skinfold callipers (Harpenden, John Bull British Industries Ltd., England) at seven sites (bicep, triceps, subscapular, iliac, abdominal, front thigh and calf).The median value was used to calculate the sum of skinfolds.

Body mass was recorded to the nearest 100 g, and height to the nearest 1 mm using the stretch method (CPAFLA, 2003).

VO_2max measurement

Following a 5 minute standardized warm-up on a rowing ergometer at 200-225 watts, participants completed an incremental VO2max test. Depending upon their competitive weight category, participants started their test at either 190 watts (lightweights) or 220 watts (heavyweights) and increased power output by 30 watts every 2 minutes until exhaustion. Expired gas samples were collected into a mounted face-mask and measured by a True One metabolic cart (Parvo Medics, USA) which was calibrated prior to each trial according to standard laboratory procedures. Heart rate was monitored continuously throughout the incremental test using a telemetric heart rate monitor (Polar, Finland). At least 2 of the following criteria was met for the determination of VO_2max: (1) attainment of predicted maximum heart rate (220-age); (2) a rise in VO_2 of less than 2 $ml/kg^{-1}/min^{-1}$ with an increase in workload; (3) a respiratory exchange ratio (RER) greater than 1.15 (4) volitional exhaustion.

Ventilatory threshold assessed subjectively by 2 trained personnel and was considered the point where V_E/V_{O2} ratio increased while V_E/V_{CO2} remained relatively constant (Caiozzo, Davis, Ellis, Azus & Vandagriff, 1982; Bentley, McNaughton, Roberts, Vleck, Fairbanks & Marinaki, 2007; Amann, Subudhi, Walker, Eisenman, Shultz & Foster, 2004).

Training

All training prior to taper was prescribed by the coaching staff. It consisted of seven aerobic training sessions per week on the water in singles and pairs, two high intensity ergometer sessions, and two strength training sessions per week. The taper was constructed by the investigators and the coaching staff, but volume restrictions were imposed due to training phase concerns.

Ergometer training

The high intensity ergometer work was a 1:1 work rest design with 3 minutes of work at < 90% of peak power attained on their VO_2max test and 3 minute rest intervals. Ergometer sessions were scheduled twice a week and progressed from three work intervals to six work intervals over the course of training.

Each athlete was given a training log at each training session in which they recorded distance, and average 500m split time for each interval. Intensity was calculated retrospectively by comparing average interval power output to the power output associated with VO_2max measured post study.

On-water training

On water training was predominately high volume-low intensity aerobic training and each session was approximately 90 minutes in duration. Training distances were measured in km, and calculated by coaching staff and principal investigator who was present at each training session. The body of water where the training sessions took place was clearly marked by bouys each 250m over a 2 kilometer stretch. GPS tools and satellite imaging enabled accurate measurements of other sections of the lake (Google Earth, USA).

On-water training intensity was measured by subjective assessment by the coaching staff and principal investigator according to the outlined definitions of a 6 category intensity scale commonly used in rowing (Appendix C)

Strength training

The weight training program consisted of 3 different full body workouts that were used in rotation. 2 workouts were completed per week on Tuesdays and Thursdays. Each workout was similar in demands although the exercises differed slightly. A detailed example is shown in Appendix D. The training was progressively overloaded with sets ranging from 2-5. The number of repetitions were prescribed to develop maximum strength and ranged between 3 and 6 repetitions. Intensity was not specifically controlled, however athletes were encouraged to select the maximum weight they could life to near failure given the prescribed repetitions. Coaching staff and the principal investigator were present at each weight training session to ensure proper and safe lifting practices were employed.

Training volume was recorded in training journals and calculated as $\sum$ (Weight * Reps * sets). Due to complexities in calculating load in body weight exercises such as sit-ups and push-ups, these are not included in the calculated total training load.

Taper

The taper was 5 days in duration for both the control and experimental group. “On water” training volume was not substantially reduced due to restrictions imposed by the coaching staff on volume decreases. The 5 day on-water training volume reduction was 25% in a linear reduction. Prescribed weight training volume was reduced by 40 and 60% each day, respectively.

All prescribed workouts were identical for both groups during training and taper other than the ergometer treatment session on day 26. Following a standardized warm-up, the higher volume treatment group completed three 3 minute work bouts with 3 minutes active recovery, followed by 5 min active recovery, then 2 x 500m at maximal intensity, with 3 minutes active recovery between.

In contrast, the control group did 2 x 1000m with 3 minutes active recovery in between. These 1000m intervals were done at race pace and took between 3 to 3.5 min depending on fitness level and size of the participant.

Pre-ergometer test procedures

Participants arrived at the testing site 90-120 minutes prior to their scheduled ergometer test time. Each participant sat stationary in an upright seated position for 20 minutes prior to blood letting. The athletes subsequently filled out the “Profile of Mood States questionnaire” (POMS) (McNair et al 1971) (Appendix E) and performed 6 maximal countermovement jumps while attached to the Gym Aware monitoring system (Kinetic performance technology, Australia).

Performance

The primary dependant variable for the study was 2000m ergometer test performance. (Coefficient of variation = 0.79% Intraclass r = 0.989) The pre-taper ergometer test took place in a gymnasium similar to the location of the Western Canadian Indoor Rowing Championships. Athletes were "seeded" by coaching staff and the principal investigator based on prior ergometer tests and seated next to athletes of similar speed. This is done at the indoor championships and helped replicate the competitive environment of the indoor championships. Additionally, while not racing, participants were allowed to verbally motivate their team-mates.

At the indoor championships individuals raced in their respective categories with approximately 20 athletes in each race. A crowd of supporters was present, as well as television monitors stationed in front of the athletes informing them of their real-time status in the race.

Prior to competing all athletes were given a 27-30 minute standardized warm-up (Appendix G).

Visual feedback

During the pre-taper 2000m test, the feedback the athletes received visually from the monitor was: pace per 500m, stroke rate, meters remaining, and duration (time).

During the 2000m test pulled at the Indoor Rowing Championships, the athletes received the same feedback on the ergometer screen as during the previous test. Additionally, there were television screens showing the real-time placing of each athlete over the course of the 2000m.

Post Race Lactate Analysis

Blood lactate samples were obtained 2 minutes following the conclusion of the 2000m race. The participants remained seated on the ergometer, not exercising until the completion of the sample. Blood samples were obtained via finger prick using a lancet device (Softclick Pro, Roche, Germany) under sterile conditions and analyzed for blood lactate using a Lactate Pro Portable Lactate Analyzer (Lactate Pro, Arkray Inc., Japan).

Hematology

Hematological variables were measured on 4 occasions for a complete blood count: 3 days prior to the investigation, pre-taper, pre-treatment, and at competition. All measurements were completed within 24 hours of sampling. From a complete blood count the following variables were selected for observation: red cell volume, white blood cell count, hemoglobin, hematocrit, mean corpuscular volume, and red cell distribution width

On the first 3 occasions, measurement was done on a XE-2100 Sysmex Hematology Analyzer (Sysmex Corporation, Japan). Due to scheduling difficulties measurement on the final day was performed on a Beckman Coulter 750 LH (Beckman Coulter Inc., USA) hematology analyzer at a different private laboratory. The analyzers have been shown to be highly agreeable in all components necessary for complete blood counts (Sandhaus et al., 2002; Johnson et al., 2002)

Plasma volume was calculated using the Dill and Costill equation for determining changes in plasma volume (Dill and Costill 1974) (Appendix G).

Counter Movement Jumps

Counter movement jumps were measured prior to ergometer testing for jump height, dip depth, velocity, acceleration, and rate of force development. Body weight was measured to the nearest 0.1 kg. Following a 3 minute standardized warm-up, participants performed 6 maximal counter-movement jumps.

The jumps were measured by a position transducer that measures vertical displacement and time. It also has corrects for slight horizontal displacement (GymAware, Kinetic, Australia). The position transducer was positioned on the floor between the legs of the athlete, with attachment via a wire from a belt tightened slightly above the hips at the narrowing of the abdominal region to avoid any slippage. The participants placed their hands on their hips during all jumps to help isolate changes in leg power and remove the skill of jumping from the measurement. Each participant had completed 4 familiarization trials in the weeks preceding the tests, and were comfortable with the measurement system.

Psychology

The Profile of Mood States (POMS) questionnaire has been established as a valid and reliable measure of global mood and attitudes (McNair et al., 1971). The 65 item questionnaire was completed under private settings and an investigator was present to answer any questions the participants had. The test was administered at baseline, at the end of week 2, and prior to the pre-taper and competition ergometer tests.

Statistical Analysis

A repeated measures analysis of variance was used to determine any main or interaction effects for all comparisons. Tukey's Post hoc analysis was used to further establish any differences between groups where necessary. A Chronbach's alpha for each of the 6 categories of the POMS questionnaire was calculated for internal validity. Type I error was protected at 5%. All statistical analysis were done using Statistica 6 (Statsoft, USA)

Chapter 3 Results

Subject Characteristics

An initial subject pool of 21 male collegiate rowers was recruited for this study. Due to circumstances beyond the control of the investigators, only 11 participants completed the requisite training volume of greater than 50%; 4 participants were forced to miss multiple weeks of training due to illness, 2 participants suffered major injuries, a rib stress fracture, and a pinched nerve, 2 participants voluntarily withdrew due to the required time commitment, and an additional 2 participants did not meet the minimum 50% training requirement. There were 6 participants in the high volume treatment group, and 5 in the low volume treatment group.

Five participants characterized themselves as lightweight rowers. There were no weight restrictions imposed for laboratory testing, however it did change the power output at which they began their VO2max test at. For the lightweight rowers there was a competition weight restriction of 75 kg for both ergometer tests. The mean age of the participants was 21.0 ± 1.9 years of age and experience rowing was 4.1 ± 1.8 years.

There were no physical or physiological differences between groups, however, as anticipated, due to various seasonal factors, some physical and physiological variables changed over the course of the investigation. Table 1 describes the physical and physiological characteristics immediately following the fall competitive rowing season, a 6 week period of reduced training, and following 1 month of training, culminating in the completion of the study.

Table 1 Physical and physiological characteristics measured over the course of the investigation

Variable	Post Competitive season	Pre-Study	Post-Study
Height	185.1 ± 6.2	184.8 ± 6.1	184.6 ± 6.0
Weight (kg)	81.7 ± 9.4	81.5 ± 8.8	81.6 ± 9.0
Sum of 9 skinfolds (mm)	91.3 ± 37.5	96.1 ± 33.8	90.2 ± 27.4
Flexed Bicep Girth (cm)	33.6 ± 1.9	33.5 ± 1.6	33.8 ± 1.7
Thigh Girth (cm)	53.8 ± 3.1	53.6 ± 3.0	53.9 ± 2.9
VO_2max (L/min)	4.8 ± 0.7	4.6 ± 0.6*****	5.0 ± 0.6
VO_2max (ml/kg/min)	59.6 ± 8.1	56.2 ± 7.5*****	60.9 ± 5.8
Maximum Heart Rate (b/min)	196 ± 9	198 ± 10	196 ± 9
Power Output at VO2max (Watts)	354 ± 45	326 ± 45*****	362 ± 43
Ventilatory Threshold (L/min)	4.2 ± 0.5	4.1 ± 0.7	4.4 ± 0.4 †
Power Output at VT (Watts)	281 ± 40	284 ± 44	305 ± 40

***** Denotes statistical significance from the preceding trial ($p \leq 0.05$). † denotes significance between pre and post study ($p \leq 0.05$). Data reported as mean ± standard deviation

.

Training Load

There was no difference between the groups for training volumes or intensity, however, both groups experienced a wide range of attendance, and therefore there is a large range in training load between individuals. Table 2 describes the mean and ranges for training volumes and intensity over the course of the study. Distance covered in scheduled ergometer sessions was significantly greater ($p \leq 0.05$) in weeks 3 and 4 than week 1. Also, the average intensity of scheduled ergometer training in week 4 was significantly higher ($p \leq 0.05$) than weeks 1, 2, and 3. On the water, week 2 was significantly greater ($p \leq 0.05$) in training distance (km) than weeks 1 and 4 and the intensity of week 3 on the water was higher ($p \leq 0.05$) than weeks 1 and 2. For total aerobic training volume, week 1 was statistically different ($p \leq 0.05$) from week 2, week 2

was different from weeks 1 and 4, and week 3 was statistically the same as all weeks. Weight training volume was significantly lower in week 4 than week 1. (Table 2)

Figure 1 displays the daily training volume for on water, ergometer, and weight training. The values are averaged from the participants who attended the training sessions, which ranged from 2 participants attending to full attendance of 11 over the course of the study.

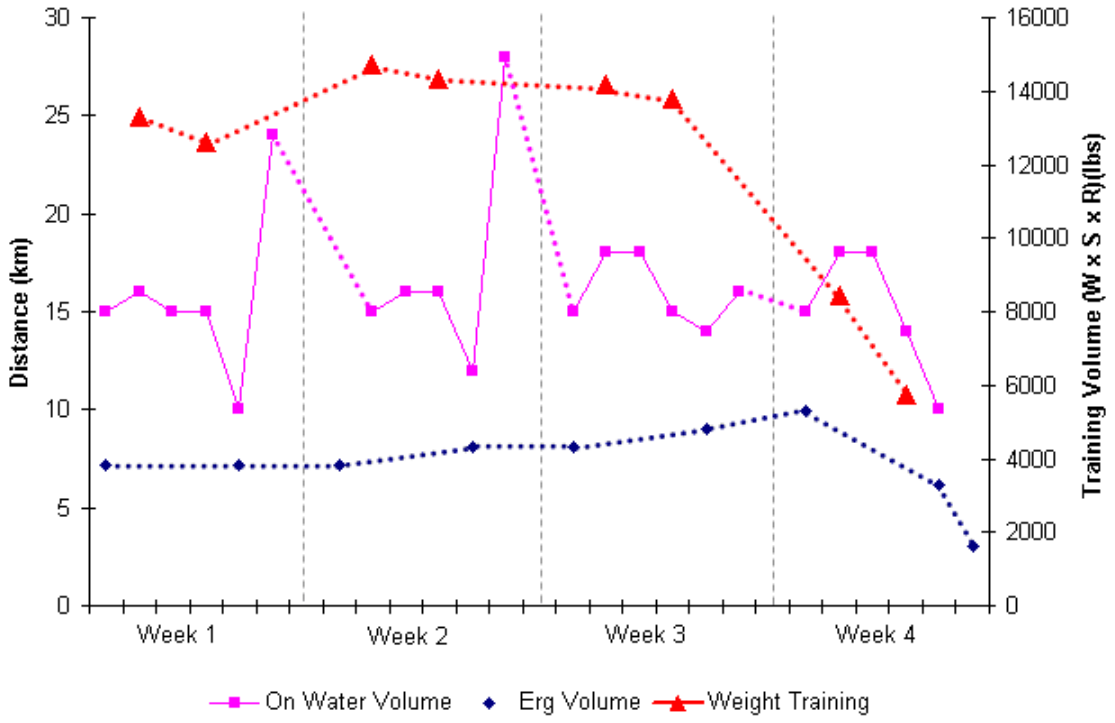

Figure 1 Mean daily training volume for participants who attended the training session

The treatment session on day 26 was different between groups in terms of volume (8.17 ± 0.14 km vs. 5.5 ± 0.0 km) ($p \leq 0.05$) but the intensity relative to peak power output was similar (102 ± 5.3% and 103 ± 2.8%).

Table 2. Mean and ranges for training volume parameters.

		Week 1	Week 2	Week 3	Week 4	Total
Scheduled Ergometer training (km)	Mean	6.9 ± 3.6	8.6 ± 3.3	10.0 ± 3.9	11.4 ± 2.3 *	36.9 ± 7.4
	Max	10.1	11.1	13.0	14.0	47.2
	Min	4.5	4.5	4.5	8.0	29.6
Scheduled Ergometer training Intensity (% PPO)	Mean	92 ± 4%	95 ± 5%	93 ± 5%	99 ± 3% *	95 ± 4%
	Max	99%	101%	99%	103%	101%
	Min	85%	86%	86%	93%	88%
On water (km)	Mean	49.5 ± 29.2	72.6 ± 22.0 *	57.3 ± 25.0	45.6 ± 25.2	225.0 ± 82.1
	Max	95.0	102.0	96.0	80.0	373.0
	Min	15.0	40.0	30.0	0.0	149.0
On Water Intensity	Mean	4.1 ± 0.7	3.6 ± 0.4	3.1 ± 0.7	3.8 ± 0.7	3.7 ± 0.3
	Max	5.0	4.0	4.1	5.0	4.2
	Min	3.0	2.7	2.0	2.6	3.1
Total aerobic training including additional ergometer training (km)	Mean	59.3 ± 27.9	86.3 ± 20.8 *	75.0 ± 29.6	63.2 ± 25.9	283.7 ± 83.2
	Max	105.1	113.1	117.4	108.4	437.2
	Min	21.9	49.9	34.5	22.7	211.6
Weight Training Volume (Weight (lbs) x sets x reps)	Mean	26,121 ± 7,881	18,489 ± 12,719	18,458 ± 13,030	10,078 ± 6,583 *	73,147 ± 33,994
	Max	37,970	38,295	33,140	17,159	118,448
	Min	14,003	0	0	0	19,937
Attendance	Mean	7.2 ± 2.4	8.3 ± 2.1	7.3 ± 3.4	7.4 ± 2.6	30.1
	Max.	11.0	11.0	11.0	12.0	42.0
	Min.	4.0	5.0	3.0	4.0	21.0

* denotes statistical difference from week 1 ($p < 0.05$) Data reported as mean ± standard deviation

Performance

2000m ergometer test time was not significantly different between groups on either trial ((p > 0.05) E.S. = 0.46). Both groups significantly improved their ergometer scores between pre-taper and competition (p = 0.0008). The low volume (control) group improved by 5.4 ± 2.7 seconds compared to the high volume (treatment) group, which improved by 4.0 ± 3.3 seconds (Table 3).

Capillary Blood Lactate

Capillary blood lactate values were the not statistically different between the high volume treatment and low volume control groups (p = .068), nor were the blood lactate values different after the ergometer tests (p = .065). Performance and associated blood lactate values are described in Table 3.

Table 3 2000 meter ergometer performance and associated capillary blood lactate measurement 120 seconds post ergometer test

Group	Performance (Seconds)			Blood Lactate (mmol/L)		
	Pre-Taper	Competition	Significance	Pre-Taper	Competition	Significance
Low Volume (Control)	392.3 ± 9.6	386.9 ± 8.7 *	0.015 *	13.3 ± 0.8	14.7 ± 0.9	0.31
High Volume (Treatment)	405.0 ± 20.2	401.0 ± 20.4 *	0.046 *	14.6 ± 1.6	15.4 ± 0.7	0.67
Combined	399.2 ± 16.9	394.6 ± 17.0 *	0.0009 *	14.0 ± 1.4	15.1 ± 1.0	0.06

* Statistical significance (p ≤ 0.05). Data reported as mean ± standard deviation

Neuromuscular tests

Countermovement jumps were measured for select variables by a position transducer. Calculated mean values for the 6 jumps and the single maximum values are reported in Table 4.

There were no significant differences between the groups for any of the selected variables so the results shown are collapsed data for the two groups. Both mean and maximum rate of force development increased following the taper. Dip depth decreased significantly in week 4.

Table 4 Mean and maximal values for maximal counter movement vertical jumps as measured by a position transducer between trials

	Pre-Taper		Competition	
	Mean	Max	Mean	Max
Jump Height (cm)	0.44 ± 0.05	0.46 ± 0.06	0.43 ± 0.03	0.46 ± 0.03
Concentric Mean Velocity ($m \cdot sec^{-1}$)	1.59 ± 0.13	1.65 ± 0.14	1.55 ± 0.12	1.66 ± 0.15
Concentric Peak Velocity ($m \cdot sec^{-1}$)	3.16 ± 0.23	3.28 ± 0.24	3.10 ± 0.29	3.28 ± 0.27
Concentric Peak Acceleration ($m \cdot sec^{-2}$)	15.7 ± 2.7	18.3 ± 4.2	20.6 ± 6.7 *	24.6 ± 8.3 *
Rate of Force Development (kN/s)	32.61 ± 10.9	42.4 ± 14.7	44.18 ± 12.4 *	61.3 ± 19.5 *
Dip	-0.52 ± 0.09	-0.55 ± 0.09	-0.46 ± 0.10 *	-0.42 ± 0.23 *

* denotes statistically significance between trials ($p \leq 0.05$) Reported as mean values ± standard deviation. "Mean" is the average of 6 countermovement jumps, "max" reports the highest value of the 6 jumps.

Hematology

White Blood Cells and differential

Figure 2 displays total white blood cell count and the differential for the 2 types of cells that primarily make up the white blood cell count. There were no differences between the two groups, and therefore the data presented is collapsed for both groups. White blood cell count was significantly lower ($p \leq 0.05$) at pre-taper and at competition compared to baseline and treatment days. Neutrophil count was different each collection session than the previous session and

lymphocytes significantly lower on competition day than at any other measured time throughout the study.

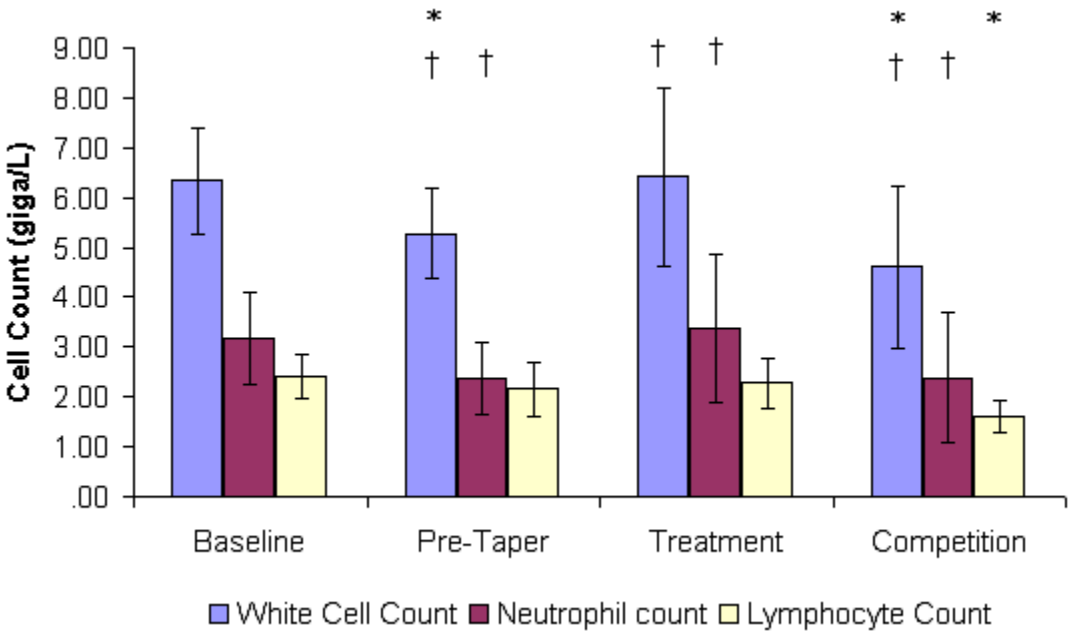

Figure 2 White blood cell count and differential measured by a complete blood count at select intervals throughout the study

* denotes statistical significance compared to pre-study values ($p \leq 0.05$). † denotes a statistically significant difference compared to the prior session.

Red blood cells

Collapsed group data for Hb, Hct, MCV, RDW and changes in plasma volume are reported in table 5. There were no correlations between changes in plasma volume and training volume, or between changes in plasma volume and relative VO_2max. Figure 3 shows the changes in Hb, Hct, MCV, and RDW for each group over the course of the study.

Table 5 Variables associated with red blood cells measured throughout the study by a complete blood count

Variable	Pre-Study	Pre-Taper	Treatment	Post-Taper
Red Cell Count	4.88 ± 0.24	4.84 ± 0.25	4.80 ± 0.26	4.73 ± 0.25
Hemoglobin (g/L)	149.82 ± 6.37	148.18 ± 7.48	147.36 ± 7.74	149.55 ± 10.18
Hematocrit	0.43 ± .02	0.44 ± 0.02	0.43 ± 0.02	0.43 ± 0.02
Mean Corpuscular Volume (fl)	88.09 ± 2.81 †	90.27 ± 2.69 *†	90.18 ± 2.99 *†	91.64 ± 2.94 *
Red Cell Distribution Width (%)	12.75 ± 0.35 †	13.04 ± 0.45 *†	13.01 ± 0.46 *†	12.25 ± 0.49 *
Change in Plasma Volume from baseline		1.2 ± 3.6%	0.6 ± 3.3%	-1.3 ± 3.7%

* denotes statistical significance from Pre-Study values
† denotes statistical significance from Competition values

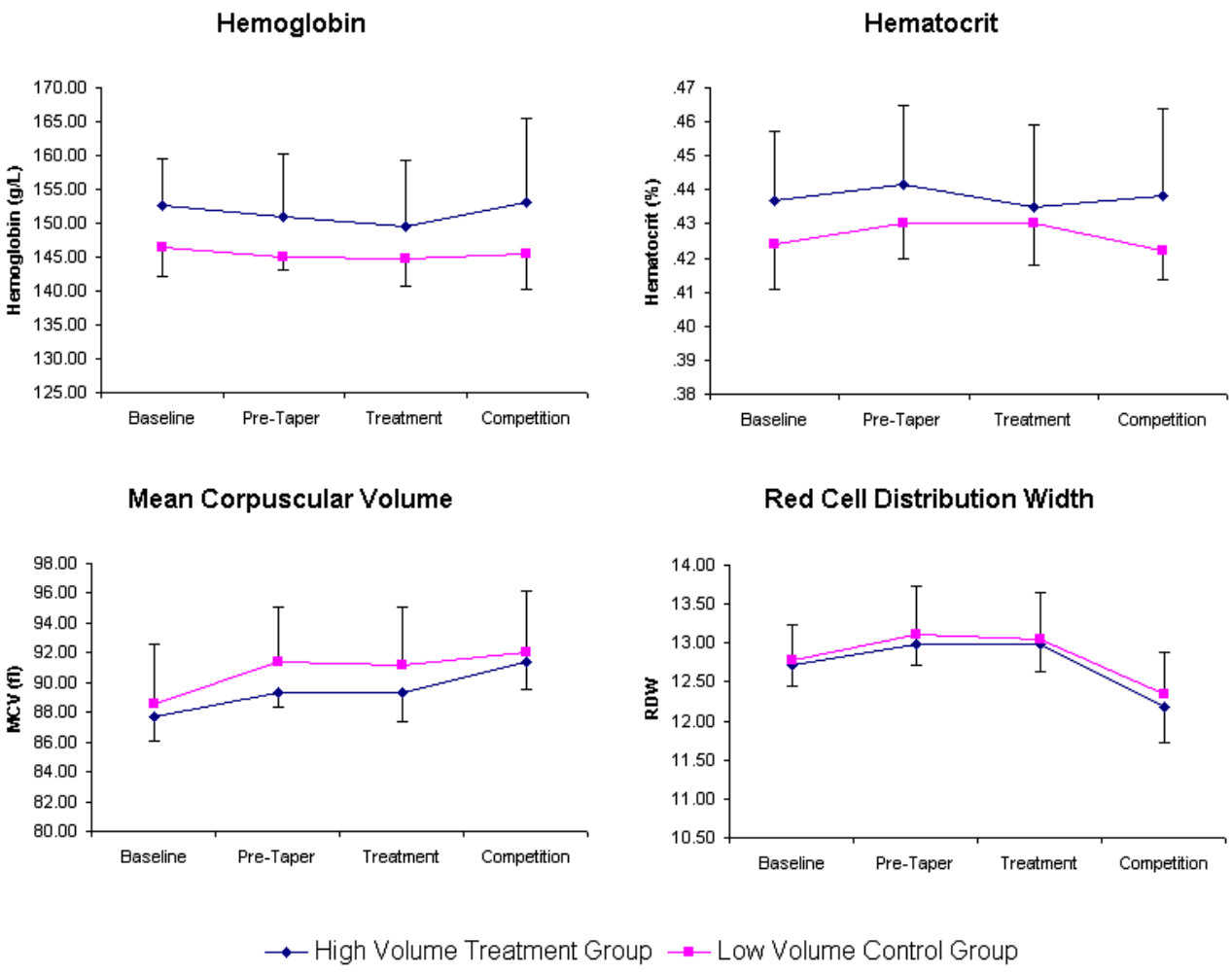

Figure 3 Changes in hemoglobin, hematocrit, mean corpuscular volume and red cell distribution width for each group over the course of the study

There were no differences between groups for any variable. Data shown as means, error bars are set as the standard deviation of each group for that measurement session

Profile of Mood States

There were no differences between the groups for any of the measured variables. Over the course of the study total mood disturbance was significantly increased compared to baseline and for the duration of the study. Weeks 2, 3, and 4 were not different from each other for total

mood disturbance (Pre-Study 47.5 ± 15.7, Week 2: 60.5 ± 21.9, Pre-taper: 63.7 ± 29.5. Post-taper: 62.9 ± 23.0).

Figure 4 shows the changes for the 6 constructs that the profile of mood states questionnaire interprets. There were no significant changes in depression, vigor, or confusion over the course of the study. Tension and anger were significantly elevated pre-taper and at competition compared to pre-study values. Fatigue was significantly lower at competition in comparison to week 2 levels, but not compared to week 3 or baseline values. Internal consistency for each of the subscales was (Tension $\alpha = 0.78$ Depression $\alpha = 0.85$ Anger $\alpha = 0.87$ Vigor $\alpha = 0.85$ Fatigue $\alpha = 0.86$ Confusion $\alpha = 0.62$)

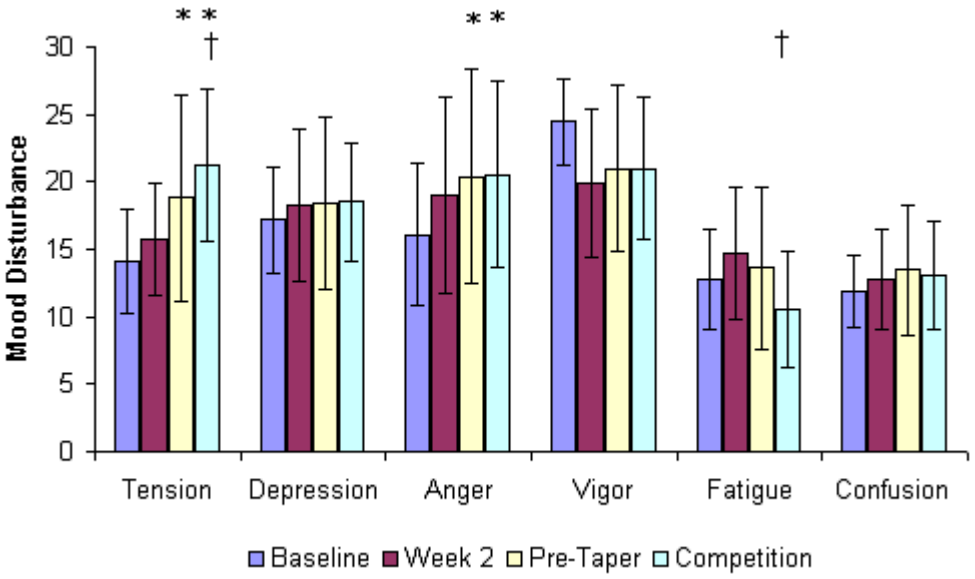

Figure 4 Graphical representation of mood disturbance for the constructs of the Profile of Mood States questionnaire

* denotes statistical significance ($p \leq 0.05$) within each construct from baseline values, † denotes significance within each construct from week 2.

Nutrition

Macronutrient consumption during the 2 days prior and morning of ergometer tests were not different between groups or ergometer test. Caffeine was consumed in the form of coffee in three individuals, and was consistent between trials.

Chapter 4 Discussion

The primary purpose of this study was to assess the performance effect of this higher volume treatment with measurement of a broad spectrum of variables associated with taper. The aim was to identify the potential physiological and psychological ramifications of each respective taper. The main finding of this study was that both taper protocols produced a similar improvement in 2000m rowing ergometer performance, and there was no consequence to performing a higher volume workout 48-52 hours prior to 2000m rowing competition amidst a 5 day taper. Consistent with many other investigations on taper, our findings suggest reducing volume and maintaining or slightly increasing intensity in the days preceding competition is beneficial, and can result in improved performance (Mujika et al., 2004; Bosquet et al., 2007).

Results indicate that a decrease in RBC age, neurological adaptations, and changes in psychological / psychophysical variables coincided with the 1.2 ± 0.8% improvement observed by both groups. We recognize however, that many other variables likely contributed to the improved performance and that the relationships are not inherently causal.

This study was constrained by limitations that commonly afflict applied sport science. Restrictions imposed by coaching staff in terms of how much control over the training plan and the given window for taper manipulation provides a major barrier to the external validity of this study. These limitations were founded in seasonal related factors, and that dropping volume to the magnitude suggested by current literature, greater than 50%, would compromise the over-all training effect on the team for the spring season.

The two groups were not significantly different with respect to any physiological or training variables; however the large variability in the amount of training completed in each group is a confounding factor in answering the research questions. Injury, illness, and motivation

plagued this investigation and the team during the winter months of training. Adherence was not expected to be perfect; however the 225.7km range between the highest volume completed and the 50th percentile was much larger than anticipated.

Athletes who were unable to attend the long continuous aerobic workouts during afternoon sessions were expected to make up the workouts on ergometers on their own time; however many athletes neglected to do this or instead completed an abbreviated distance. High volumes of aerobic training are traditionally the dominant form of rowing training and it is suggested that aerobic performance is compromised if training volume drops below 100km per week (Steinacher et al., 1993).

Baseline and Detraining

There were approximately 6 weeks between post fall season testing and baseline testing in which training was encouraged. During this time exams, holidays, and regeneration were a higher priority for many athletes and voluntary activity levels were likely reduced. Many reported participating in activities such as cross country skiing, running, weight training, snow shoeing and racquet sports, however training volumes were not recorded during this time.

The implications of short term periods of reduced training on highly trained athletes have been shown to manifest as alterations to both the cardiovascular system and energy metabolism. Decreases in blood volumes, plasma volume and cardiac dimensions, have been shown to effect stroke volume, heart rate, and cardiac output during exercise(Mujika et al., 2000). Reduced insulin sensitivity, muscle glycogen levels, and metabolic enzyme activity have also been shown to be reduced after 6 days of inactivity (Vukovich et al., 1996; Mujika et al., 2000). These adaptations are typically indicated by increases in respiratory exchange ratio at submaximal

levels, increased submaximal blood lactate, and a reduced lactate threshold (Godfrey et al., 2005; Petibois et al., 2003).

The effects of detraining were not in question and therefore a multitude of variables were not controlled. This makes it impossible to accurately make statements regarding this dimension of the study, although certain results are consistent with previous detraining literature (Mujika et al., 2000).

In this study there were no large increases in plasma volume between baseline and training. Although this could be taken to imply plasma volumes were maintained throughout the period of reduced training, this is likely not the case. For many participants, their baseline laboratory sessions could have acted as a training stimulus to acutely expand plasma volume. Additionally, although formal training had not begun, individuals likely increased their activity levels in preparation for the upcoming season, which may also have been a stimulus to increased plasma volume.

A factor not quantified in this study, that likely played a significant role in reducing baseline test scores, was a decrease in the "skill" of rowing on an ergometer. Movement economy has been shown to play a major role in detraining, and is reflected by an increased oxygen cost for a given power output (Godfrey et al., 2005). Even if participants had completed enough training to avoid detraining physiologically, it is possible that power outputs at VO_2max could be reduced due to reduced efficiency (Riveria-Brown et al., 1998). This is particularly highlighted in our results, as we see an average decrease of 4.1% in VO_2max and a 7.1% decrease in power output at VO_2max, yet no change in power output at ventilatory threshold.

Performance

The current body of taper-literature suggests that taper is usually accompanied by an increase in performance between 0 and 8%. These improvements are not always statistically significant, yet are of important consequence to athletes and coaches. Mujika et al., (2002) calculated the mean percentage difference in swim time for all events at the Sydney Olympic Games and found that the difference between a gold medal and 4th place was 1.2 ± 0.8%, and from 4th to 8th was another 2.0 ± 0.8%.

Hopkins et al., (1999) suggests that a performance improvement of one half of the normal within subject variation should be considered worthwhile. Within this definition, 2000m ergometer performance for college aged males an improvement greater than 1.6 seconds would be worthwhile (C.V. = 0.79 %). With respect to our data, both of the tapers employed have efficacy for implementation, however the additional 1.4 second improvement in the lower volume group is negligible.

Only two other published studies on taper have used rowers as their population of interest. Smith et al., (2000) observed 500m sprint performance before and after a 1 week taper to find no significant or applicably relevant difference in performance. Training volumes were only dropped by 25%, which combined with the reduced ability to detect changes due to the duration of the test selected, can explain the ineffectiveness of this taper. Jurimae et al., (2003) observed elite rowers following three weeks of overload training and two weeks of taper. They found a 0.8% (3.3 seconds) improvement in 2000m ergometer performance, which is a similar impact to the tapers in our study. The participants completed 12 training sessions per week and had similar training volumes to the few in the present study who completed the full prescribed training volume of 11 training sessions per week.

Both modeling and meta-analysis have determined that approximately 2 weeks is the optimal duration of a taper prior to major competition but it is highly dependent on the amount of work done prior to competition (Banister et al., 1975, Busso et al., 1994; Bosquet et al., 2007 Thomas et al., 2008). In the present study, a full 2 weeks may be an inappropriate length given the training load; however a greater reduction in volume likely would have allowed the participants enhanced recovery as well as further isolation of the physiological effects of the differing treatment workouts.

Blood Lactate

Post race blood lactate levels for the 2 groups combined neared statistical significance at competition ($p = 0.06$). Increases in post race blood lactates have been consistently shown with taper, often with an athletes highest blood lactate score correlating with their best performance (Mujika et al., 2004). Blood lactate measurement has excellent reliability ($r = 0.993$) (Pyne et al., 2002), and therefore it is proposed that if there was a difference between the trials we would have needed to increase the sample size to detect it. Based on the present data, an increase of two participants would increase the statistical power to 0.80, and if all 21 participants had completed the study statistical power increases to 0.95.

In this investigation motivational / psychological factors cannot be excluded as potentially confounding factors regarding effort because the competitive and motivational environment changed, including the amount of external motivation at the competition compared to the pre-taper ergometer test. Verbal encouragement has been shown to play a significant role in how hard individuals push themselves. Andreacci et al., (2002) provided encouragement at either 20 second, 60 second, or 180 second intervals during maximal testing on untrained

participants and found a significant improvement in VO_2max and post test blood lactate values. Other studies on elite athletes have observed increases in time to exhaustion and maximal blood lactate without increases in VO_2max (Moffat et al., 1994). It is proposed that the verbal encouragement provides a distraction from the pain associated with maximal exercise (Andreacci et al., 2002), and highlights the role of the central nervous system in maximal exercise (Weir et al., 2006).

The slightly higher intensity work during taper or a combination of intensity and recovery may have resulted in an increase in the ability of the muscle to remove lactate. Na / H+ exchanger isoforms and lactate transporters NEH1 have been shown to increase as a result of short term elevation of training intensity in trained athletes (Iaia et al., 2008). The exact mechanism by which metabolic by-products affect subsequent energy production are not well understood, however homeostatic balance within the intramuscular environment has a recognized importance in understanding limitations to endurance performance (Midgley et al., 2006). Whether an increase in blood lactate concentration is an example of adaptation to buffering capacities, due to a possible increase in blood pH or increased RBC's, or is due to an increased muscle lactate efflux is not clear and remains to be fully elucidated. Additionally, whether or not increased lactate production has a positive or negative effect on performance is also debated. Evidence presented for the positive effects of increased muscle lactate include that lactate and hydrogen ion do not interfere with excitation-contraction coupling, and decreasing muscle pH will decrease Chloride permeability in the T-tubules, allowing action potentials to be propagated regardless of an increased intra-cellular potassium build-up (Lamb, 2006; Bangsbo & Juel, 2006). Potassium build-up is suggested to be a main contribution to metabolic fatigue (Allen et al., 2008). Conversely, several experiments have demonstrated the positive effects of

altering blood pH through sodium citrate or sodium bicarbonate ingestion, which likely alter the ability to buffer hydrogen ions implying that muscle and blood pH play an important role in limiting strenuous exercise (Bangsbo & Juel., 2006).

Hemoglobin is also recognized as an important buffer, (Powers & Howley., 2001) and increases in blood lactate observed with taper may in part be due to an observed increase in RBC's and hemoglobin. In this study we did not see increases in red cell count, which tended to decline throughout the study, or hemoglobin and therefore cannot attribute any increase in lactate production to erythrocytic adaptations.

High volumes of aerobic training have been associated with glycogen depletion, and during periods of reduced training, such as in a taper, these stores are replenished (Shepley et al., 1992). Therefore, increased substrate availability, either from glycogen super-compensation, or glycogen store recovery may increase blood lactate production through a mass action effect (Houmard et al., 1994). Muscle glycogen stores were not measured in this investigation and therefore, we cannot determine if this or other factors played a role in performance or lactate production.

Hematology

In this study we did not find any differences in hematological variables between groups. This may have been due to the small sample sizes, the sensitivity of measures and the small effect size of these variables. Additionally, the duration of the taper and treatment stimulus may have not been ideal to elicit adaptations to these particular variables. Changes in plasma volume have been shown after single bouts of exercise; however the volume of these bouts is typically much larger than employed in this study; 8 x 4 min at 85% VO_2max with 5 minutes rest between

sets, (Gillen et al., 1991; Nagashima et al., 1999). In order to elicit a plasma volume expansion, the treatment stimulus must be beyond normal training volumes and intensity (Gillen et al., 1991). It is plausible that in a longer duration taper, the treatment volume employed in this study would have been isolated enough to be a substantial deviation from previous training volumes and caused a plasma volume expansion, this remains to be determined.

There was a significant increase in MCV from baseline, stability between pre-taper and treatment values and an elevated MCV at performance. It has been suggested that an increased MCV with regards to exercise is reflective of a younger, larger RBC population within the blood (Green et al., 1991). This is a common finding amongst athletic populations during heavy volume training. Older RBC's have been shown to be stiffer than young RBC's and this may make them susceptible to fragmentation and premature death from increased pressures and turbulence during exercise (Robinson et al., 2005; Green et al., 1991). This training-induced hemolysis subsequently stimulates erythropoises to maintain the oxygen carrying capacity of the blood repopulating the circulatory system with younger RBCs (Green et al., 1991; Robertson et al., 1988). Commonly shown, taper will utilize this training induced hematopoises, and decreased hemolysis, creating a positive red cell balance at performance. (Shepley et al., 1992; Mujika et al., 2004; Mujika et al., 2000).

Contrary to these studies, this study did not find an increased red cell volume, Hb or Hct as a result of either taper. Also, a positive balance between Hb and hemolysis would result in an increase in mean cell age; however these results indicate a younger red cell population at performance.

There is marginal benefit to having younger RBC's, such as increased 2-3 Diphosphoglycerol (2-3 DPG) favourably shifting the oxy-hemoglobin disassociation curve,

however this would not dramatically effect the ability of the cardiovascular system to deliver oxygen, nor the ability of the muscle cells to utilize oxygen, which are recognized as much greater limitations to performance (Mairbaurl et al., 1983; Robinson et al., 2006 Basset et al., 2000; Green et al., 1991). It has been shown that in elite athletes a limitation to performance is a reduced ability to diffuse CO_2 and bind O_2 in the lung due to the speed at which the blood is circulating (Hopkins et al., 1994; Zavorsky et al., 2002). It is therefore hypothesized that an elevated 2-3 DPG content could impact maximal exercise such as a 2000m ergometer test, however this was not measured and remains to be elucidated.

Several factors may explain the discrepancies between the hematological data of this study and the adaptations found in other investigations. These include: different physiological consequences of our taper compared to those employed in other studies, methodological constraints and the diurnal / circadian rhythm effects on hematological parameters, the large inter-individual variance in training volume, and limitations of using mean corpuscular volume as an indices of RBC age. Additionally, the timecourse of functional adaptation to red blood cells calls into question the present interpretations.

As mentioned previously, intense exercise can create a hypoxic environment and has been shown to stimulate erythropoietin production. Erythrocytes develop from stem cell to red blood cell in approximately 6-7 days and live for approximately 120 days (Powers and Howley, 2001). Therefore, even if the drop in volume in the taper were substantial, realization of benefits of positive red cell balance would likely not have fully manifested. Reticulocytes were not measured in this study, and would have been able to provide evidence of this hypothesis.

Training variability could be logically assessed a the main contributor to the apparent decrease in MCV at competition, however “change in MCV” between pre-taper and competition

was negatively correlated with change in training volume from weeks 3 to 4, ($r = -0.67$). This suggests that the reduction in training volume was not sufficient to reduce training induced hemolysis, and that the slight increase in intensity may have induced further RBC destruction. Intensity has been shown to be a more potent stimulus for training related hemolysis, particularly in non-contact or impact sports such as cycling and swimming (Robinson et al., 2006).

Red cell distribution width (RDW) is the coefficient of variation of MCV (Bessman et al., 1983). Deficiencies in iron, folate, or vitamin B12 will increase RDW, which make it a useful tool in identifying some anaemia's. Reduced iron stores, and iron deficiency often coincides with chronic endurance training as a result of the increased RBC destruction (Smith and Roberts, 1994). The observed increase in RDW with training is consistent with the proposed idea of training induced hemolysis and decreased RDW with taper implies that less abnormal sized cells were circulating, which would be consistent with decreased red cell destruction.

There are limitations to using changes MCV and RDW to suggest relationships to observations. Red cell creatine content has been determined to be the best measure of changes in red blood cell population age (Robertson et al., 1988). Red cell size, which both MCV and RDW measure, are sensitive to fluid shifts compromising the utility of these measures (Robertson et al., 1988). In this experiment hydration status was not controlled prior to blood letting and therefore this may have impacted the measurements of MCV and RDW.

Due to scheduling limitations, blood measurements were susceptible to diurnal and circadian rhythm variability. Blood measurements were taken between 14:30 and 17:00 at baseline and in the afternoon prior to the treatment workout, and between 07:30 and 10:00 am at post taper and competition measurement days. Pocock et al., (1989) observed a weak declining trend of red cell count, Hb and Hct, through the daytime. They determined that the percentage

variance due to time of day was 2.1% for Hct, 1.7% for Hb, 1.6% for red cell count and 0.6% for MCV.

Diurnal variation may have also contributed to the differences found with regards to WBC associated variables. This is the most likely explanation for the observed fluctuations. Figure 2 clearly shows there was no difference between measurements taken in the afternoon (baseline and treatment) and the morning (pre-taper and competition). Pocock et al., (1989) found WBC count increased throughout the day to a peak around 17:00 with 2.2% variation throughout the day. These data are similar to Haus., (1985) who also reported significant fluctuations with circadian rhythms.

Mujika et al., (1996) investigated the effects of taper on blood leukocyte populations in elite swimmers, and found a decrease in polymorphonuclear neutrophils, but no other significant responses to a 4 week taper following 22 weeks of training.

Changes in peripheral white cell populations have been related to circulating cortisol levels and both chronic and acute exercise have been shown to have an impact on WBC circulation. Training responses, therefore, cannot be ruled out as the cause of fluctuations; however, given that no clinically relevant changes in leukocytes occurred we are confident they had little to no impact on performance outcomes.

Profile of Mood States

POMS has consistently been shown to be a good predictor of training stress and has been utilized in taper literature as a measure of well being (Hooper et al., 1999). Our results showed a positive change in total mood disturbance with taper similar to previous investigations (Hooper et al., 1998; Berger et al., 1999).

Many athletes displayed an iceberg profile, with higher vigor scores than college-aged norms. This "iceberg profile" is a common phenomenon amongst athletes in comparison to age specific norms, where a relatively flat line is common (Rowley et al., 1995; McNair et al., 1971). In this study, the iceberg profile was most pronounced at baseline, however although slightly depressed from training, vigor scores were statistically the same at all trials. With taper, all values with the exception of tension should revert back to near baseline levels (Hooper et al., 1998). Tension was increased for both ergometer tests, which is commonly seen prior to competition and attributable to increases in pre-race anxiety (Hooper et al., 1998).

Fatigue scores were significantly lower at competition than week 2 values and although not significant, were lower at competition than at baseline as well. Although training was prescribed to increase volume during week 3, due to variability in attendance mean aerobic training distance was approximately 11 km lower in week 3 than week 2. The changes in POMS fatigue levels coincided well with training load and indicate that it is a highly sensitive to changes in training volumes.

Neuromuscular

Countermovement jump has been recognized as a valid measure of neuromuscular fatigue (Nicol et al., 2006). Various adaptations to force development during this task may be reflective of neurogenic and myogenic properties, and provide a window into further understanding of the complex balance between fatigue and recovery.

At competition, dip depth, maximum acceleration, and rate of force development were significantly increased in both groups. Despite no increase in jump performance, adaptations in these variables may be reflective of enhanced movement economy, which are commonly

observed with taper. With no change in jump height for either group there was no apparent improved ability to produce leg power as a result of either taper, however the changes observed may be suggestive of a broader theme of improved movement economy.

Measurement of countermovement jumps does not allow identification of the specific mechanisms of adaptation, however through methodological controls, sites of potential sites of adaptation can be proposed. It has been suggested that dip depth can be altered within a reasonable range without having an effect on jump height performance (Linthorne et al., 2007). However, it has also been shown that humans will tend to naturally select the optimal depth to elicit their highest jump (Ronglan et al., 2006)

Fatigue is very complex and is exhaustively studied, yet due to the interaction of neurogenic, myogenic, and endocrine systems, fatigue cannot be fully explained by single mechanisms (McKenna et al., 2008). Fatigue can manifest due to both acute and chronic changes in central and peripheral environments and structures. The observed alterations in acceleration and rate of force development are not due to changes in ATP and creatine phosphate availability, nor due to an accumulation of metabolic by-products because these return to near resting levels within 15 to 60 minutes (Allen et al., 2008). The longer term (multiple hours) role of calcium release, or calcium sensitivity is not entirely clear, however, the fact that we did not see a reduced force production, only an alteration in how force was achieved suggests calcium related variables did not play a role (Allen et al., 2008).

Neural signalling failure and excitation of the sarcolemma can also be ruled out. Nerve stimulation experiments have shown that electrical stimulation of a motor nerve in individuals with training induced fatigue results in no change in M-wave indicating that signal transmission

is not interrupted or altered as a result of residual fatigue (Allen et al., 2008; Edwards et al., 1977).

One possible neurogenic explanation for the increase in acceleration observed following taper in this study would be improved motor unit synchrony and/or enhanced recruitment of fast twitch muscle fibers with taper. Adaptations such as increased action potential firing, particularly increases in doublet firing have been shown to increase the rate of force development and may have played a role in the present observations (Dutchateau, 2006).

Muscle fiber shortening velocity is dependent on the rate of cross bridge cycling, which is determined by myosin heavy chain isoforms (Allen et al., 2008). The strength training employed in this study likely induced muscle damage to all fibers, however due to the contractile properties of type II fibers, recovery to this fiber type may play a more significant role in altering acceleration and rate of force development. Therefore, recovery of exercise induced muscle damage is a hypothesized to be the main reason for the observed improvement in the ability to produce greater acceleration.

Consistent with the suggestion that altered depth and acceleration may be related to taper, Trappe et al., (2000) extracted muscle biopsies from the deltoid muscles of elite swimmers before and after a 3 week taper. They found a 30% increase in peak isometric force, 67% and 32% increase in shortening velocity of type IIa and Type I fibers respectively, and a 250% increase in absolute fiber power following a 3 week taper.

Although alterations in central fatigue cannot be completely ruled out, the presented evidence suggests that recovery of contractile proteins could result in improved efficiency of contractions and be associated with the improvements in movement economy commonly associated with taper.

It is highly likely that reducing the training load concomitantly reduces the load placed on the neuromuscular system and plays a significant role in the improved performance that typically accompanies taper (Trappe et al., 2000). We did not measure jump height and associated variables at baseline and therefore cannot determine if chronic training had any effect over the three weeks of training.

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Conclusion

This study found no evidence, physiological or performance to validate the practice of incorporating a higher volume, high intensity workout 48-52 hours prior to competition in comparison to a reduced volume taper. Performance was improved in both taper groups and there were no significant differences in any of the hematological, biochemical, neurological or psychological indices collected. The gathered data collectively suggest that the sensitivity of the measures may have been compromised due to a lack of statistical power and limitations associated with applied research. The combined group 4.6 second improvement in performance observed in this study may be partially explained by decreased red blood cell age, favourable neurological adaptations, and improvement in mood state. The complex interactions of physiological adaptations from training and fatigue, and subsequent effect on performance will be continuously scrutinized and manipulated by sport scientists, coaches and athletes in an effort to gain a competitive advantage. Our results show that a high volume workout amidst a 5 day taper 48-52 hours prior to competition leads to a similar, yet slightly lower performance enhancement than a lower volume treatment. (4.0 ± 3.3 second vs. 5.4 ± 2.7 seconds respectively).

There are substantial limitations to the external validity of this data that must be recognised; the substantial loss of participants, large variability in training volume, and seasonal considerations all likely play confounding roles. Additionally, due to the small yet potentially important effect of a high volume stimulus during a low volume taper, it is likely that the present study did not isolate the treatment enough to determine the true response. If anecdotal evidence for this practice remains strong, research studies should be designed to better replicate the training and taper practices employed in these cases, and if possible, measure enzymatic changes to the muscle to better understand the implications of the high intensity, high volume work-bout.

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Appendix A Review of Literature

This review is organized into 2 major sections. The first will highlight findings relevant to the taper, including duration, intensity, volume, and physiological impact. The second will explore the physiological implications of high intensity exercise.

Taper has recently been defined as "a progressive non-linear reduction of the training load during a variable period of time, in an attempt to reduce the physiological and psychological stress of daily training and optimize sports performance" (Mujika and Padilla 2000). This definition encompasses and recognizes the many factors that may influence elite performance.

Taper and Athletic Performance

Both anecdotal evidence and scientific investigations have show that the effect of taper on athletic performance is usually an improvement between 0.5 and 8% (Mujika et al., 2003). Small improvements in performance may not reach statistical significance yet can have profound impacts on performance. Mujika et al. (2002) observed an average of 2.2% improvement in swimming performance during the final 3 weeks of training prior to the Sydney Olympic Games across all events.

Often for elite athletes, the margin that separates the entire final is only a few percent. The aforementioned analysis of swimmers prior to the Sydney 2000 Olympics found that the difference between the gold medal winner, and 4^{th} place, was $1.62 \pm 0.8\%$, and $2.02 \pm 0.81\%$ from 3^{rd} to 8^{th} place (Mujika et al., 2002). These statistics highlight the importance of the taper, and that if done effectively one could go from being 5^{th} or 6^{th} in a race, to a medal winner. Similar investigations have been done in track and field events, suggesting that as distance

increases, the margin of victory slightly widens however at the highest levels in sport these differences are still quite small. The 100m sprinting Gold medal final in the Barcelona Olympics was separated by 0.9%, compared to 1.5% in the 5000m running event (Hopkins et al., 1999).

Put in perspective of performance, Hopkins et al. (1999) suggests that improvements of 0.4 to 0.7 % of the normal with-in subject variation should be considered worthwhile for elite athletes. This was calculated by taking into account both within athlete variation and between athlete variations.

At the highest levels of sport, the margin that separates the best in the world is minimal, and therefore, it is advantageous for each athlete to participate in any legal strategy that will result in practical enhancements in performance.

Taper Intensity, Volume, and Duration

It has been consistently shown that training intensity during the taper must be high and sport specific to competition pace or higher in order to achieve optimal performance. It has been suggested that intensity is a necessary stimulus for the maintenance of blood volume, and to maintain a high level of enzymatic activity within the muscle and avoid detraining (Mujika et al., 2004).

Shepley et al. (1992) investigated high intensity, low volume taper, a low intensity, moderate volume taper, and a rest only taper. They found that VO_2max was not effected by any of the 3 tapers, but run time to fatigue increased 22% after a high intensity low volume taper, and was unaffected by a low intensity moderate volume, (6%) and rest only taper (-3%).

In a series of 3 studies, Hickson et al. (1981, 1982, & 1985) demonstrated that for maintenance of training induced adaptations, intensity is the most important variable.

In the first publication, Hickson et al. (1981) observed the impact of changes in frequency. Following 10 weeks of training they found reducing training from 6 to 4 or 2 sessions per week for 15 subsequent weeks had no impact on VO_2max, however the 15 weeks of reduced frequency training did not improve VO_2max at all, implying that more sessions per week, or a change in other parameters such as intensity would be necessary to improve aerobic fitness.

The second study had a similar design and observed the ramifications of reducing volume. The volume reductions of 33 and 66% for 15 weeks after 10 weeks of training resulted in no changes in VO_2max, short term endurance performance, and left ventricular mass following 15 weeks. Long distance endurance performance was also maintained after 15 weeks of endurance training in the training group that reduced volume by 33%, but was reduced in the 2/3's reduction group which experienced a 10% decrement in time to exhaustion cycling at 80% VO_2max (Hickson et al.,1982).

The third study by Hickson et al. (1985) compared reductions in intensity with the same design of the previous 2 studies. Training intensity was reduced by 1/3 and 2/3rds following 10 weeks of training and training volume was held constant. VO_2max was not maintained in either group due to reductions in training intensity after 10 weeks of training.

Collectively these studies make a strong case for intensity being the most important training variable to maintain training induced adaptations and endurance performance. The physiological adaptations are explored later on in this literature review.

Meta-analysis of all taper studies with "actual performance" criterion suggests that the optimal strategy is a drop in volume by 41-60%, maintenance of training intensity, and to maintain frequency of training. This analysis included 27 studies and involved predominantly aerobic sports such as running, swimming, cycling, and rowing. Approximately 2 weeks was

deemed the optimal duration. Although larger performance gains have been observed in both shorter and longer studies, 14 days and a exponentially reduced volume by 41-60% was determined to give an athlete the highest probability of improved performance. This guideline is made with one the premise of not adapting frequency or intensity of training, and therefore use of the term “optimal” may be inaccurate.

Recently modeling investigations have become major topic of interest in the developing field of sport performance and attempts to quantify the impact of training on the body, and to suggest optimal taper duration, volume and intensity. Original work based on a systems model approach has expanded into complex mathematical modeling of training inputs vs. the balances of fatigue and training adaptations.

Banister et al. (1999) displayed that an exponential decay taper protocol is superior to stepwise reductions in training volume, and that a fast exponential decay is superior to a slow exponential decay. This was calculated based on a systems model, and was subsequently tested on elite triathletes. Both the model and field trials determined that an exponential reduction in training is more beneficial than a stepwise reduction, and that a fast exponential taper is superior to a slow exponential taper.

More recent investigations have refined this model so that it can take into account variations in prior training, which is ecologically more accurate (Thomas et al., 2008). This model suggests that a period of heavy training prior to taper can elicit further performance gains; however, the taper duration in these circumstances must be quite long, approximately 22.4 days.

Physiological Adaptations to taper

It is well accepted that increases in VO_2max lead to proportional increases in power output. To have an increase in performance in a timed aerobic sport such as rowing, power output must be increased through out the performance, therefore, it is assumable that VO_2max would increase as well. This is often not the case, and many studies have shown vast increases in performance, without concomitant increases in VO_2max. This improved performance is often attributed to changes in the sub-maximal oxygen cost at a given power output. Changes in movement economy would likely better explain improvements in performance than increases in VO_2max, because of the sub-maximal and endurance components to a variety of sports (Mujika et al., 2004). An enhanced ability to transfer metabolic energy into mechanical energy would improve the amount of energy one could produce for the same cost, thus enhancing performance. At elite levels, movement economy has been shown to be the best predictor of endurance performance in a variety of sports including running (Paavelainen et al., 1999), cycling (Coyle et al., 1992) cross country skiing (Osteras et al., 2002) swimming(Chatard et al., 1990) and rowing (Ingham et al., 2007).

Improvements in movement economy have been associated with increased muscle temperature (Johnson et al., 1974), muscle fiber type recruitment (Kaneko et al., 1990), increased muscle stiffness (Paavolian et al., 1999), and increased mitochondrial presence (Holloszy et al., 1977). Collectively these adaptations will help reduce the disturbance in muscle homeostasis and improve oxygen utilization per mitochondrial respiratory chain (Saunders et al., 2004)

In sports like rowing in which the contraction cycle is quite slow relative to running or jumping to utilize elastic energy, movement economy adaptations to taper are likely the result of recovery of contractile mechanisms and neuromuscular adaptations which are explored at a later component of this review.

Houmard et al. (1994) found a 6% decrease in sub-maximal energy expenditure and 7% reduction in calculated caloric expenditure in distance runners running at 80% VO_2peak following a 7 day taper in which volume was progressively reduced by 85%. This week of taper also resulted in 3% improvement in self paced 5 km treadmill running performance. These improvements are suggested to be the result of adaptations at the muscular level rather than with oxygen delivery (Houmard et al., 1994). Supporting these findings, Banister et al. (1999) found that anaerobic threshold changed from 71% to 75% of VO_2max, in conjunction with increases from 68.6 ml/kg/min to 69.2 ml/kg/min in VO_2max, in elite triathletes following 2 weeks of taper.

Further evidence for improved energy efficiency is findings of reduced sub-maximal blood lactate values (Mujika et al., 2004). In elite junior rowers Steinacker et al. (2000) found an 8% higher power output post taper at a blood lactate concentration of 4.0 mmol/L. Performance criterion were not established for this investigation, because the training and taper was for Junior World Championships.

Houmard et al., (1994) did not see decreases in sub-maximal blood lactate, despite a 3% improvement in 5km time trail performance. This was coincident with a significant decrease in oxygen consumption when running at 80% VO_2max. Other tapers, in swimmers, (Flynn et al., 1994; Johns et al., 1992), runners (Stone et al., 1996) and cyclists (Rietjens et al., 2001) have also not seen decreases in sub-maximal blood lactates.

Improvements in movement economy have been attributed to changes in neuromuscular adaptations from longer distance training. The journal of applied physiology has recently done an extensive review of through a “highlighted topics” symposium on neuromuscular adaptations to training and it is likely that many of these areas undergo adaptations from taper, however very

few have been specifically investigated with regards to taper. Adaptations to the electro physical properties of motor neurons and dendrite restructuring have been shown to occur with both endurance and strength training (Gardiner 2006). Carson (2006) discussed changes in muscle coordination, particularly with skill acquisition. Three specific areas were highlighted: Adaptations in activity dependent coupling, the composition of muscle synergies, and hebbian plasticity. Hebbian plasticity is a strengthening of connections between motor neurons. Neurons that release action potentials at the same time will increase cortical neurons, and increase the probability that those motor neurons will fire at the same time (Carson 2006).

There can also be adaptations to the behaviour of motor units. Changes in discharge rates, and the incidences of "doublet firing" have been shown with training, resulting in changes in the muscles rate of force development (Dutchateau 2006)

A final component of this comprehensive series of review articles are the neural adaptations that take place in the brain and spinal cord. Adkins discusses the plasticity of the motor cortex, and highlights how skilled movements include a reorganization of neural circuitry, thus allowing the refinement of skills through training (Adkins 2006).

Neary et al. (2005) looked at muscle oxygenation trends in elite cyclists, using analysis of enhanced oxidative capacity. This group utilized 3 taper protocols, reducing volume by 30, 50 and 80% for 1 week following 3 weeks of training. The only taper to elicit statistically significant performance improvements (4.53%) was the 50% reduction in training. Within this taper group, the only significantly lower cardiovascular response was VE/VO_2 ratio, which reduced from 25.3 ± 2.2 to 22.5 ± 1.7. As well, only the 50% reduction taper resulted in increased muscle deoxygenation with a concomitant increase in oxygen uptake. These results

suggest that taper can lead to increased oxygen extraction for a given blood flow (Neary et al. 2005).

In a similarly designed study, (Neary et al., 2003) investigators found that a 50% reduction taper resulted in increases of 6.0% in VO_2max, whereas again, the 30% and 80% tapers had no effect on VO_2max or 20km time trial performance. This finding is consistent with other reports of increased VO_2max following taper. In cyclists, a 2 week stepwise reduction in training load resulted in a 4.5% increase in VO_2max, and 7.2% faster 8.5 km outdoor time trial performance (Bannister et al., 1999).

Evidence for peripheral adaptations can be seen in the aforementioned work by Shepley et al. (1992). They found an increase of 18% in vastus lateralis citrate synthase activity following a week long high intensity taper. Citrate synthase is the enzyme that controls the first aspect of the citric acid cycle and is often used as a marker of intact mitochondria within the cell (Widmaier et al., 2004). The resulting improved mitochondrial capacity would lead to performance improvements through decreased reliance on anaerobic metabolism, decreasing the amount of interference of by-products on the contractile process (Houmard et al., 1994).

Adaptations have been shown to both oxygen delivery, and oxygen utilization as a result of taper. A net increase in red blood cells, presumably due to decreased training induced red blood cell destruction increases the oxygen carrying capacity of the blood. Training induced hemolysis is addressed later in this review.

Basset and Howley (2000) argue that oxygen delivery is the dominant limitation to endurance performance. They present 3 areas of supporting evidence including: when oxygen carrying capacity is altered, through various practices such as blood transfusion, pharmaceutically (rEPO), and others, VO_2max increases or decreases accordingly. The second

theme of evidence is that increased VO_2max with training is primarily due to increased maximal cardiac output. And the final argument presented for oxygen delivery as the primary limiter to VO_2max is that when a muscle is over-perfused with oxygen rich blood, its capacity for consuming oxygen is very high. (Basset and Howley 2000)

Many studies have found that the best predictor of performance is power output, or pace at lactate or ventilatory threshold. In rowing, Ingham et al (2002) found that in elite rowers, the highest correlation with 2000m ergometer performance was power output at VO2max (r = 0.88), followed closely by power output associated with 4 mmol of lactate (r = 0.87). Combined these two variables explained 98% of the variance in rowing performance.

Muscle glycogen has been shown to increase by up to 34% (Neary et al., 1992). The increase in muscle glycogen may also be related to the intensity of the taper. Neary et al., (2003) compared reductions in session duration over a 7 day taper. One group stepped from 60 minutes at 85% VO_2max to 20 minutes of exercise at 85% VO_2max, versus a constant volume and reduced intensity from 85% VO_2max to 55% of maximal heart rate. Both tapers resulted in increases in Muscle glycogen, with the high intensity taper resulting in 34% increase and the low intensity 29%. Ensuing 40 km cycling time trial performance was improved by 4.3% in the high intensity taper, vs. 2.2% in the reduced intensity taper.

Shepley et al., (1992) did not see increases in the same magnitude as Neary et al., (1992), but none the less, found increases of 18% in muscle glycogen as a result of high intensity taper and the rest only taper in this investigation lead to an 8% increase in muscle glycogen content. The high intensity taper improved run time to exhaustion by 22%, whereas the rest only taper had a non-significant decrease (-3%) in performance.

Peak blood lactate values have been shown to increase following taper, and may be related to the aforementioned increase in muscle glycogen content. The law of mass action states that the reaction rate of any simple chemical reaction is proportional to the molecular concentrations of the reacting substances (Widmaier et al., 2004). Therefore at maximal exercise, and increase in muscle glycogen availability could result in a higher concentration of blood lactate.

Mujika et al. (2002) found an increase of 7.6% in post running race peak blood lactate. Others have also found similar increases and have found high correlations with performance (Jeukendrup 1992).

Smith et al. (2000) observed no significant increase in 500m indoor ergometer performance after 1 week of taper following 3 weeks of overload training. In this study, the training volume was only reduced by 25%, which may have played a role in the effectiveness of the taper, which did not induce changes in 500m sprint performance. The reduced training did however result in an increase in recovery blood lactates and a decrease in blood ammonia.

An increase in blood lactate with taper could be the result of multiple explanations. Motivation, possibly due to improved mood and decreased central fatigue may allow an individual to push themselves harder. Also, increased presence of mitochondrial enzymes such as lactate transporters could improve metabolite efflux from the muscle, resulting in less interference with energy metabolism.

High Intensity Exercise

Short term training, and muscle tissue analysis has been able to show that immediately post exercise we see an increase in protein transcription mRNA for a variety of different

proteins, increases in activity and abundance of metabolic enzymes, and tighter metabolic control (Green et al., 1999). This area of recent attention highlights the potency of intensity as a stimulus for powerful adaptation (Burgomaster et al., 2006)

Na^+-K^+-ATPase activity has been shown to be up regulated with training across the spectrum of untrained to well trained participants after a period of intensified training (Iaia et al., 2008; Green et al., 2004). Na^+-K^+-ATPase is an important enzyme for maintaining membrane excitability due to its regulatory role of $Na^+$and K^+ gradients. A higher Na^+-K^+-ATPase content could enable a higher Na^+-K^+-ATPase activity level, which could have important performance effects by protecting contractility during exercise (Iaia et al., 2008; Aughey et al., 2007) During muscular contraction if Na influx and K efflux exceeds the rate of Na^+-K^+-ATPase mediated transport, there could be a decrease in membrane excitability, which would therefore accelerate muscle fatigue. This acute adaptation may thus be important in alleviating the onset of fatigue by maintaining membrane excitability (Aughey et al., 2007).

Increased Na^+-K^+-ATPase content has been shown following longer periods of high intensity training. For example, Evertson et al. (1997) observed a 16% increase after 5 months of increased intensity training with international cross country skiers.

This upregulation of enzymatic activity corresponding with increases in exercise intensity has lead investigators to hypothesize that if the stimulus is powerful enough, these adaptations might occur as an acute reaction to high intensity exercise.

Green et al. (2004) showed an increase in Na^+-K^+-ATPase content of 9% following 3 days of training for 2 hours per session at 65% VO_2max. They also measured after an additional 3 days of training, and found a 41% increase in Na^+-K^+-ATPase activity. Despite the increase in content after 3 days, there was no significant increase in activity until after the additional 3 days.

In this study, the net increase in protein, likely through alterations in synthetic or degradation rates, was determined to be the primary explanation of the change in Na^+-K^+-ATPase content (Green et al., 2004).The participants in this study were untrained university students and therefore the same results cannot be expected in trained populations if given the same stimulus.

Recently Aughey et al. (2007) investigated the effects of eight, 5 minute intervals at 85% VO_2peak, on the up regulation of Na^+-K^+-ATPase. They looked at both the acute effects of a single session as well as the chronic effects, 2-3 times per week for 3 weeks, in well trained cyclists (VO_2peak 65.9 ml/kg/min). The intense interval exercise session immediately increased mRNA expression for alpha 1, 2, and 3 isoforms. After 3 weeks of training, maximal Na^+-K^+-ATPase activity was increased at rest and immediately after exercise by 6%, however there was not an increase in content or isoform abundance.

This contrasts previous findings in untrained subjects (Green et al., 2004) in which content was increased following a high intensity stimulus and may be confounded by the difference between subjects. The highly training athletes may have needed a more potent stimulus to increase enzyme content, and that other mechanisms may be associated with the increased activity such as changes in membrane composition, protein phosphorylation, and changes in other regulatory proteins (Aughey et al., 2007).

After the 3 weeks of higher intensity training, the cyclists experienced a significant 3% improvement in performance, as measured by peak power output, with no change in VO_{2peak}. It is likely the adaptation occurred at a myogenic level and can be partially explained by the adaptations to the Na^+-K^+-ATPase pump. It is suggested that the Na^+-K^+-ATPase pump is affected by the contractile history of the cell and that it is highly adaptable, possibly having important implications for athletes and performance (Aughey et al., 2007).

In a comprehensive look at the effect of short term training on metabolic adaptations, Green et al (1999) employed 6 days of training, with measurements every 3 days, at 3, 15, and 30 minutes of exercise. The participants were untrained, but split into naturally high (51 ml/kg/min) and low (41 ml/kg/min) VO_2max groups. In this study, no between group differences were found for any variable however after training blood and muscle lactate were reduced at 3 minutes and remained depressed throughout the exercise duration providing additional support for the observed increase in monocarboxylate transporter 1 as an early adaptation to exercise (Green et al., 1999; Bonen et al., 1998). Related, findings of reduced concentrations of allosteric activators free phosphate, ADP and AMP, and propose that training resulted in a more protected energy state, dampening the activation of phosphorylase and PFK (Green et al., 1999). Consistent to this hypothesis, free phosphate ions, ADPf and AMPf were all reduced at 3 minutes of exercise after 3 days of training and a reduction in accumulation of glucose-1-phosphate and glucose-6-phosphate after training (Green et al., 1999).

Citrate synthase is commonly selected as a marker of intact mitochondria, and is a marker of oxidative metabolism. After 6 sessions of sprint training it was increased by 11%, as well as a decrease in net muscle glycogenolysis, lactate accumulation, and presence of pyruvate dehydrogenase (Burgomaster et al., 2006). Green et al. (1999) found increases in the metabolic enzymes citrate synthase, pyruvate dehydrogenase, and non-significant trends in hexokinase after 6 days of training, but these enzymes were not increased at 3 days. From these results, it is suggested that other factors must be involved in the regulation of mitochondrial metabolism.

The metabolic adaptations from short term training appear to be less recruitment of high energy phosphates, and a reduction in the breakdown of glycogen (Green et al., 1999) and that a new exercise stimulus can cause a shift to tighter metabolic control over a range of sub maximal

power outputs and has been demonstrated before actual increases in mitochondria are detectable (Aughey et al., 2007).

Surprisingly many studies pertaining to acute exercise do not report the well documented changes in plasma volume that occur. This is something one must be conscious of when interpreting any variables measured as a concentration within the blood.

Plasma Volume Expansion

One of the first adaptations to endurance exercise in humans is an increase in plasma volume, which may assist in thermoregulation and cardiac output. The effect of plasma volume expansion on VO_2max has been investigated predominantly through the infusion of a plasma solution. Investigators have typically injected 500-700 ml of a dextran saline solution resulting in an 8-11% increase in plasma volume. This shift in red cell mass leads to hemodilution and should theoretically decrease, not increase the amount of oxygen being delivered to the working muscle.

An increased venous return at an unchanged afterload will result in a greater stretch on the myocardial fibers (Warburton et al., 1999). This stretch creates a more optimal interaction of the heart contractile proteins, actin and myosin filaments, and allows the heart to empty to the same extent regardless of the increased volume presented to the heart and is referred to as the Frank- Starling Mechanism. (Warburton et al., 2000). The ensuing impact of an altered stroke volume and cardiac output, results in at least equivalent oxygen transport to the working muscle. If the increase in stroke volume and cardiac output were to offset the negative consequences of hemodilution, it could result in increased oxygen transport (Warburton et al., 2000).

To further understand the myocardial adaptations to short term endurance training, Goodman et al. (2005) used cardiac imaging over the left ventricle. They employed a 3 day exercise protocol induced an 11.4% increase in plasma volume, and a 7% improvement in VO_2max. The subjects were active university male students, with a baseline VO_2max of 45.9 ml/kg/min. Although changes in resting diastolic filling failed to reach statistical significance, there was evidence of enhanced rapid filling phase of diastole and increases in left ventricle ejection fraction at the highest exercise intensities. They also noted an elevated cardiac output during high intensity exercise in the hypervolemic condition. The authors suggest that this may reflect the alteration in red blood cell concentration from the plasma volume expansion (Goodman et al., 2005).

Goodman et al. (2005) also demonstrated a lower heart rate, bradycardia, at all levels of sub-maximal exercise coincident with the increases in stroke volume. These findings were previously identified by an inverse relationship showing that for every 1% increase in plasma volume, there is a proportional decrease in heart rate (Convertino et al., 1983).

Improvements in VO_2max and endurance performance from hypervolemia seem to be most pronounced in untrained subjects. In this population many researchers have found improvements from both infused solutions as well as exercise inducement. A critical review done by Warburton et al. (2000) found improvements in subjects with VO_2max less than 55 ml/kg/min in a range of unchanged to 7%. More recent investigations have found similar results for this population (Goodman et al., 2005)

In moderately trained participants (VO_2max between 55 and 64 ml/kg/min), investigators have seen mixed results regarding improvements in VO_2max. Coyle et al. (1986) found a 2% increase in VO_2max, although interestingly, endurance performance was not enhanced.

Conversely, Luetkemeier et al (1994) found an unchanged VO_2max, but an 11% improvement in 90 minute simulated time trials. With this study, the investigators found no difference between an infused dextran solution vs. exercise induced hypervolemia.

There only been 2 investigations targeting highly trained endurance athletes (VO_2max >64ml/kg/min). Neither found a change in VO_2max following the plasma volume expansion. Only one of these investigated plasma volume expansion from an ergogenic perspective. Coyle et al. (1986) found that 3 weeks of detraining resulted in a 9% decrease in blood volume due primarily to a 12% lowering in plasma volume. They then infused dextran saline solution to bring the athletes to within 2% of their trained state blood volumes. Interestingly, this infusion completely reversed the effects of detraining, and indicates that the decline in cardiovascular function after detraining is largely due to a decrease in blood volume, which appears to limit ventricular filling. (Coyle et al., 1986)

This article is often cited as evidence that plasma volume expansion does not enhance VO_2max in trained athletes, and that performance will not be enhanced by acute hypervolemia. This notion does not truly reflect the results of this investigation because plasma volume was not expanded beyond the "trained-state" value, and the results could actually be taken as evidence to support the use of hypervolemia as an ergogenic.

Thus, there is only one investigation of the effects of plasma volume expansion on elite cyclists as an ergogenic. This study was a rare design in that it was able to control for the Hawthorne effect. This mixed model, double blind, cross-over designed study used infused dextran solutions, or a sham expansion conditions on elite cyclists (VO2max 64.4-76.5) (Warburton et al., 1999). They tested VO_2max by an incremental step test, as well as time to exhaustion cycling at 95% VO_2max. There was no significant change in either parameter, as well

as no change in heart rate or perceived exertion during high intensity exercise. Although statistical significance was not reached it is interested to note VO_2max in the plasma volume expansion group was a non-statistically significant 1% higher. Surprisingly time to exhaustion did not follow suit, and was a non-statistically significant 4.6% less (Warburton et al., 1999).

The decrement in performance time to exhaustion, seen statistically significant in investigations has been postulated to be the result of increased local lactate production (Coyle et al., 1990). It is possible that the hemodilution caused a decreased buffering capacity the blood, thereby impairing performance. Warburton et al (1999) found an increase in lactate production at rest as a result of plasma volume expansion. This is not consistent with Nagashima et al. (1999) who found a decreased resting blood lactate level. The amount of lactate produced by working muscles is suggested to be inversely proportional to the amount of oxygen available; therefore, an increased local lactate production should be the result of increased anaerobic glycolysis (Warburton et al., 1999). This is an interesting finding because it suggests less oxygen delivery to the working muscle.

The effects of hypervolemia on blood lactate buffering are rarely reported in the literature, although reported findings suggest no change in end exercise blood lactate level (Green 1989).

Investigations of the hypervolemic response have found powerful evidence to suggest that the primary cause of increased plasma from endurance training is an increase in total protein content of the plasma. Of this increase in total proteins, 85% of it was albumin (Convertino et al., 1980). Gillen et al. (1991) observed the time course of acute adaptations to endurance training, finding that at 1 hr of recovery, plasma albumin was increased by 0.17ml/kg, which fully accounted for the increase in plasma volume of 1.5ml/kg (Gillen et al., 1991). Furthermore, after

a single high intensity exercise session Nagashima et al. (1999) found a 6.4% increase in plasma volume at 22 hrs of recovery. Of this increase, 91% of it could be attributed to the increase in plasma albumin.

The initial increase in plasma volume within the first hour is suggested to be due to a mobilization of lymph flow during and after exercise, because if occurs too rapidly to be accounted for by de novo synthesis, degradation rate, or the transcapillary escape rate of albumin (Gillen et al., 1991).

This hypothesis is supported by evidence that plasma volume increases only occurred in upright exercise and recovery, and not in a matched supine position (Nagashima et al., 1999). Nagashima et al. (1999) measured atrial natriuretic peptide, which is correlated with right atrial pressure, and suggests that elevated atrial natriuretic peptide during supine exercise may reflect suppressed lymphatic return, and consequently, an unchanged plasma protein content and no expansion.

The major regulatory hormones aldosterone, agrinine vasopressin, and atrial natriuretic peptide have been identified as the major fluid regulatory hormones in mammals. Together, these hormones act on the cardiovascular system, sweat glands, and kidneys to defend blood pressure and sodium loss (Grant et al., 1996). These hormones typically are suppressed during exercise, and fluctuate based on the intensity and duration of the session in response to fluid shifts. In a hypervolemic state, Grant et al. (1996) found higher resting levels of atrial natriuretic peptide. Consistent with prior investigations, they concluded that the higher concentrations during exercise that these are carryover from the higher resting levels. (Grant et al., 1996)

In light of the Nagashima et al., investigation, as well as the finding that six degrees head down tilt bed rest induces a decrease in plasma volume within 24-48 hrs (Convertino et al., 1996)

it is suggested that central venous pressure is an important signal stimulus for regulation of body fluids and plasma volume (Goodman et al., 2005). Although there is not currently direct evidence supporting this hypothesis, these observations provide some insight that venous pressure is a necessary stimulus for an increase in plasma albumin content.

Hematology and Exercise

The aforementioned elevated plasma volume has is commonly observed in trained populations. Within an athlete population, a decreased hematocrit because of elevated plasma volume is referred to as athletic or sports pseudoanemia. Exercise may also decrease hematocrit levels due to an increase in red blood cell destruction. This area has received considerable attention, and has been termed athletic or sports anemia (Green et al., 1991).

It is apparent that there is an increased destruction of red blood cells as a result of athletic training. Originally examined in runners, it was thought that the hemolysis was a result of rupturing the red blood cells on foot-strike, however, this condition is prevalent in low impact sports as well, and suggest that other factors contribute to the destruction of RBC's from training (Smith and Roberts 1994).

Other factors that may result in hemolysis are rupture of the cells in blood capillaries during muscle contraction, of destruction due to increased turbulence during blood flow and blood pressure, depressed iron absorption, and increased iron loss through sweat and gastrointestinal processes (Green et al., 1991; Smith and Roberts 1994). Older red blood cells are much stiffer, and it is proposed that this ridigidity makes them much more susceptible to damage (Green et al., 1991)

Evidence that this occurs is primarily based on the higher prevalence of younger erythrocytes in the blood in trained populations compared to controls (Mairbraul et al.,. 1983) Also, there has been an observed increase in hemoglobin in the blood post exercise suggesting an exercise induced hemolysis. This exercise induced hemolysis may cause a decrease in oxygen transport capacity, and stimulate erythropoises, resulting in an increased proportion of erythrocytes in the blood (Green et al., 1991).

A common method of determining age of red blood cells from a complete blood count has been observing changes in mean corpuscular volume. An increased mean corpuscular volume has been determined to be representative of younger red blood cells (Green et al., 1991; Mairbaurl et al., 1983). Other indicators of younger cells are a lowered creatine content, decreased haemoglobin content, higher 2-3 DPG levels, and lower red cell density distribution.

Robertson et al. (1994) measured red cell creatine content, red cell distribution in a density gradient, mean corpuscular volume, and mean cell haemoglobin concentration prior to, immediately following, 1 day, and 16 days after a half marathon.

They found an increase in red cell age without an immediate erythropoiteic response as measured by changes in red cell creatine. In this study, mean corpuscular volume, and mean cell hemoglobin concentration were greatly affected by exercise induced changes in serum osmolality, and are therefore less reliable measures of red cell age (Robertson et al., 1988).

Measurement of many biochemical and hematological variables may vary depending on normal body fluctuations. This may have implications on interpretation of data if unable to control for such variables. Pocock et al., (1989) investigated diurnal changes in 25 serum biochemical and hematological parameters in healthy men over a 2.5 year period. They found a weak trend of all hematological variables to decrease during the daytime. Percentage Variance

due to time of day was as follows: Hematocrit 2.1%, Hemoglobin 1.7%, red cell count 1.6%, and mean cell volume 0.6%.

These values are similar to a recent review by Thirup et al. (2003), who observed an intrasubject variance of 3% in hematocrit levels depending on circadian and seasonal variations in normal adults. Haus (1996) determined that red blood cells, haemoglobin and hematocrit consistently show highly reproducible low amplitude circadian rhythms with peak values occurring at around 11am local time. These changes are suggested to be due to changes in cell distribution, and changes in plasma volume.

White blood cell count had a diurnal variance of 2.2%. White cell count tended to increase throughout the day until around 4-5 pm, and then slightly fall into the evening. This is suggested to be due to increased movement and activity, and its effect on increasing the amount of circulating white blood cells (Pocock et al., 1989).

Measurement procedures such as posture should be standardized. Movement from a supine to an upright posture results in a body water shift into the interstitial space, therefore increasing the concentration of non filterable substances such as red blood cells (Thirup et al., 2003). Hematocrit values have been shown to be 3.5-4.7% higher in upright vs. supine stasis (Lepanin et al., 1988).

Detraining

Detraining has been defined as “the partial or complete loss of training induced adaptations, in response to an insufficient training stimulus.” (Mujuka & Padilla 2000). The decrease occurs across the physiological spectrum at a variant rate, with cardiovascular systems affected first.

The response of highly trained athletes compared to previously sedentary controls who begin an exercise program for research have been shown to have variable responses to exercise, and therefore, previous activity levels must be considered in interpreting findings (Coyle et al., 1984).

VO_2max declines rapidly after the cessation of a training stimulus to anything from 4-20%. A decrease in blood volume, particularly plasma has been shown to be the major factor. This decrease results in lower stroke volumes and cardiac output causing an increase in heart rate.

Accompanying the loss in plasma volume is an increase in sub maximal and maximal heart rate typically in the range of 5-10%. (Mujika & Padilla 2000). Resting heart-rate has been reported to not change after a short period of detraining, however this does not appear to be reported in any studies with highly trained participants and may differ based on these population delimitations.

Left ventricular mass and left ventricular wall thickness have been shown to decrease by 19.5 and 25% after 3 weeks of detraining (Martin et al., 1986). These compliment the data showing a decrease in stroke volume and cardiac output, which have been shown to be reduced by 10-17% and 8% respectively after 3 weeks without training in trained participants.

Substrate utilization is shown to shift RER higher at submaximal and maximal loads following detraining. This is likely the result of a decreased glucose disposal rates as shown by Vukovich et al (1996). This investigation on trained runners found a decreased in GLUT 4 transporter protein by 17.5 ± 5.4% after 6 days of inactivity. Other investigations on GLUT 4 protein reaction to detraining have been less conclusive with Houmard et al (1993) finding no changes. Subsequent investigation convincingly shows whole body insulin mediated glucose

uptake is rapidly decreased following reduced training as well as a down-regulation of lipoproteins that likely contribute to the shift to increased CHO metabolism (Mujika & Padilla 2000).

Conclusion

There appears to be a logical connection between coupling taper and a high intensity exercise stimulus due to the characteristics of each. Investigations on taper have shown increased blood volumes and increase abundance of metabolic enzymes (Shepley et al 1992). High intensity exercise has shown an increase in the activity of metabolic enzymes such as Na+/K+ ATPase, citrate synthase, and lactate transporters. Through proper maintenance of fatigue, and strategic planning of the high intensity stimulus, one could for-see how this stimulus may enhance performance slightly more than a typical high intensity low volume taper.

Appendix B Informed Consent

University of Victoria *Participant Consent Form*
Department of Physical Education

You are being invited to participate in a study entitled "The impact of a single high volume exercise stimulus incorporated into a traditional taper on 2000m ergometer performance" That is being conducted by James Brotherhood (MSc Kinesiology Candidate)

Introduction

I, James Brotherhood am the principle investigator of this research project. I am a graduate student in the Master of Science program in the School of Exercise Science, Physical and Health Education at the University of Victoria and you may contact me if you have any questions by:
Phone: 250-812-5072
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As a Graduate student, I am required to conduct research as part of the requirements for a degree in Kinesiology. It is being conducted under the supervision of Dr. Gordon Sleivert and Dr. Lynneth Wolski. You may contact either of my supervisors at:

Dr. Gordon Sleivert
250-744-5536
gsleivert@pacificsport.com

or

Dr. Lynneth Wolski
250-721-8375
lwolski@uvic.ca

Project Funding

This research is being funded in part by the Pacific Sport Canadian Sports Centre / University of Victoria Support for Graduate Students.

Purpose

The purpose of this study is to examine two different strategies to optimize an athlete's physiology for elite athletic performance through manipulation of training intensity and volume in the days immediately preceding competition.

Research Contributions

The difference between a gold medal, and 4th place, in high performance sport is rarely greater than 1-2%. Therefore, any legal and ethical strategy to improve performance would be worthwhile to the athlete.

The days preceding competition, athletes usually try to minimize the fatigue from prior training by reducing training load. This is commonly referred to as a taper. Traditional taper / peaking strategies have been shown to result in performance gains between 1 and 6%.

Certain taper practices are established, yet the constant search of optimal performance and leads coaches, athletes, and exercise scientists to develop and experiment with new strategies a potential competitive edge. This research will compare the performance effects of 2 taper strategies providing evidence in support for or challenging the scientific basis for the use of these tapers, both of which are currently being used at the highest levels of athletic performance.

Participation

You are being asked to participate in this study because you are a competitive rower between the ages of 18 and 30.

If you agree to voluntarily participate in this research, your participation will include:

- Continuing with your current training during the 28 day training program
- 3 baseline physiological measurement sessions
- 4 thirty minute dependent measure data collection sessions
- 2 all out 2000m ergometer tests

Continuing with current training includes:

- Completion of prescribed training
    - During training your heart rate will be examined using a Polar heart rate monitor (Polar Electro Inc., Port Washington, NY, USA).

Baseline physiological measurement will be done post fall season, pre-study, and post study to determine the effectiveness of the training program employed. These 3 sessions will take approximately 45 minutes each and include:

- Height, Weight
- Body composition measurement through skin fold protocols
- VO_2max test on rowing ergometer (incremental test to exhaustion)

Dependent measurement data collection sessions will take place 4 times throughout the 28 days of the study and take approximately 30 minutes. (Pre-training, Day 20, Day 26, Day 28) The variables collected include:

- 5 ml of Venous blood to be measured for a “Complete Blood Count”
    - Collected under sterile conditions by a certified phlembotomist
- Profile of Mood States Questionnaire
- 3 maximal vertical jumps

A complete blood count provides information on the amount and developmental state of your red and white blood cells. These blood samples will not be analyzed for any other variables.

Participation in a 2000m ergometer test (day 20), and Monster Erg Indoor rowing championships (Day 28)

- Upon completion of the ergometer tests a trained researcher will take a blood lactate sample using the finger prick method under sterile conditions

Inconveniences

The testing in this study will be completed in the time frame of your regular training program. Prescribed training is consistent with which you are currently engaged in and will not likely be viewed as an inconvenience. Reporting to the laboratory prior to the ergometer tests is not likely something one does on a regular basis, and may potentially be viewed as an inconvenience.

Risks

There are some potential risks to you by participating in this research. You will be performing maximal exercise, and will experience high levels of fatigue as a result. Also, as with any puncture of the skin, there is a small risk of minor infection and minor bruising or swelling.

To prevent or deal with these risks the following steps will be taken. Although there are fatiguing components as a result of exercise to this investigation, you are highly trained athletes and are accustom to this type of training. The fatigue in these sessions will not exceed the fatigue you typically undergo during racing and possibly training. Additionally, all researchers and assistants are trained in basic rescuer CPR and fully trained in emergency procedures. In case of emergency EMS will be contacted.

All blood measurements will be taken under sterile conditions and according to the School of Exercise Science, Health and Physical Education departmental exposure control plan, including immediate coverage by a band-aide.

In the event of any emergency or incidence requiring immediate action such as a needle stick, an Emergency Action Plan has been developed and will be implemented.

Benefits

The potential benefits of your participation in this research include expanding the pool of knowledge in the area of taper/ peaking. The results of this study may be used in future competitive settings to provide athletes with enhanced performance.

Voluntary Participation

Your participation in this research must be completely voluntary. If you do decide to participate, you may withdraw at any time without any consequences or any explanation. If you do withdraw from the study your data will be removed from the data base and not used for analysis.

To make sure that you continue to consent to participate in this research, you will be informed before each test session that your participation is voluntary that that you may withdraw at any time without penalty.

Anonymity

Your anonymity will be only partial since the other participants and I will know your participation. Results of this project may be published but any data included will in no way be linked to you specifically. Additionally you will be assigned a personal identification number to ensure anonymity in the analysis and documentation of results. I will be the only one that will access the data collected in this study.

Your confidentiality and the confidentiality of the data will be protected. The data collected and the coded identifications will be securely stored in separate locked cabinets in such a way that I will be the only one able to gain access to it. Data will be stored at the Pacific Sport National Sport Centre Victoria in Dr. Sleivert's office.

Dissemination of Data

It is anticipated that the results of this study will be shared with others in the following ways: The data will be disseminated directly to you, will be used as part of a Master's Thesis and be published in a scientific journal.

Data Disposal

At the end of the project any personal information will be destroyed immediately except that, as required by the University's research policy, any raw data on which the results of the project depend will be retained in secure storage for five years, after which it will be destroyed.

Contact

In addition to being able to contact the researcher and supervisor at the above phone numbers, you may verify the ethical approval of this study, or raise any concerns you might have, by contacting the Human research ethics office at the University of Victoria (250-472-4545).

Your signature below indicates that you understand the above conditions of participation in this study and that you have had the opportunity to have your questions answered by the researchers.

Name of Participant	*Signature*	*Date*

A copy of this consent will be left with you, and a copy will be taken by the researcher.

This project has been reviewed and approved by the Ethics Committee of the University of Victoria

Appendix C Subjective Rowing Intensity Scale

SYSTEM OF TRAINING INTENSITY CATEGORIES FOR ROWING CANADA NATIONAL TEAM

Intensity category	Approximate Heart Rate Change	Duration One Piece (min)	RATIO Work:Recovery	GOALS of the Training Intensity	Practical Examples(SR= Stroke Rate)	Lactate Level (mmol/l)
I	MAX 1 HR ie 180 - 200	0.5 - 1.5	1:4 1:5	Anaerobic Capacity Transportation = Development of Cardiopulmunar System Ability + Feeling of Start/Spurt Aggression	1 - 6 x 500M (with start) Interval Training (Short Pieces) Series of 30 - 60 Strokes Or: Series of 1 -2 min. SR: > Race - SR	>10
II	MAX HR ie 180 - 200	2 - 7	1:2 1:3	Race Endurance Transportation = Development of Cardiopulmunar System Race Speed Feeling Race Attitude/Plan	Race over 1500 - 2000M 6 x 2 min. 3 x 1000M 5 x 750M SR: Race - SR	8-14
III	MAX HR ie 180 - 200	6-10	2:1 1:2	Development of Aerobic Capacity Strength Endurance Tactics Technique	4x7min 3x2.000m Constant Speed 5x5min Strength-Endurance Water	5-8
IV	165-175	10-45	4:1	Anaerobic Threshold Development of Aerobic Capacity Efficiency Strength Endurance	2x20min with SR-Change 3x5km Time Control 10km Head Race 3x12min Strength Endurance on Water SR: 3-6 less than Race SR	~4
V	150-165	30-90	-	Basic Endurance Utilization of Aaerobic Capacity Maintenance Technique	30-90min Steady State SR: 10-12 less than Race SR	~3
VI	135-150	>45		Utilization of Anaerobic Capacity Regeneration Maintenance Technique	45-120min Steady State at Low Intensity SR: 18-24/min	~2

Appendix D Example of Weight Training Program

Date:

DAY 1 WEEK 3	Sets	Reps	Tempo	Rest	Set 1		Set 2		Set 3		Set 4		Set 5	
					Wt	Reps	Wt	Reps	Wt	Reps	Wt	Reps	Wt	Reps
OH Squats	1	10	n/a	30s										
Good Mornings	1	10	n/a	30s										
Pistol Squats	1	5/leg	n/a	30s										
Hurdle work			n/a											
Hang Clean Pull	5	3	x	90s										
Romanian DL	4	5	20x	90s										
Back Squat	4	5	20x	90s										
Bent over Row	4	5	21x	90s										
Bench Press	4	5	20x	90s										
Wood Chop	3	8	212	none										
Incline Sit-up	3	25	add weight											
Back Extensions	3	25	as needed											

Appendix E Profile of Mood States Questionnaire

Profile of Mood States
Subject's Initials _____
Birth date _____
Date _____
Subject Code No. _____
Directions: Describe HOW YOU FEEL RIGHT NOW by checking one space after each of the words listed below:

FEELING	Not at all	A little	Moderately	Quite a bit	Extremely
Friendly	1	2	3	4	5
Tense	1	2	3	4	5
Angry	1	2	3	4	5
Worn Out	1	2	3	4	5
Unhappy	1	2	3	4	5
Clear-headed	1	2	3	4	5
Lively	1	2	3	4	5
Confused	1	2	3	4	5
Sorry for things done	1	2	3	4	5
Shaky	1	2	3	4	5
Listless	1	2	3	4	5
Peeved	1	2	3	4	5
Considerate	1	2	3	4	5
Sad	1	2	3	4	5
Active	1	2	3	4	5
On edge	1	2	3	4	5
Grouchy	1	2	3	4	5

Blue	1	2	3	4	5
Energetic	1	2	3	4	5
Panicky	1	2	3	4	5
Hopeless	1	2	3	4	5
Relaxed	1	2	3	4	5
Unworthy	1	2	3	4	5
Spiteful	1	2	3	4	5
Sympathetic	1	2	3	4	5
Uneasy	1	2	3	4	5
Restless	1	2	3	4	5
Unable to concentrate	1	2	3	4	5
Fatigued	1	2	3	4	5
Helpful	1	2	3	4	5
Annoyed	1	2	3	4	5
Discouraged	1	2	3	4	5
Resentful	1	2	3	4	5
Nervous	1	2	3	4	5
Lonely	1	2	3	4	5
Miserable	1	2	3	4	5
Muddled	1	2	3	4	5
Cheerful	1	2	3	4	5
Bitter	1	2	3	4	5
Exhausted	1	2	3	4	5
Anxious	1	2	3	4	5
Ready to fight	1	2	3	4	5
Good-natured	1	2	3	4	5

Gloomy	1	2	3	4	5
Desperate	1	2	3	4	5
Sluggish	1	2	3	4	5
Rebellious	1	2	3	4	5
Helpless	1	2	3	4	5
Weary	1	2	3	4	5
Bewildered	1	2	3	4	5
Alert	1	2	3	4	5
Deceived	1	2	3	4	5
Furious	1	2	3	4	5
Effacious	1	2	3	4	5
Trusting	1	2	3	4	5
Full of pep	1	2	3	4	5
Bad-tempered	1	2	3	4	5
Worthless	1	2	3	4	5
Forgetful	1	2	3	4	5
Carefree	1	2	3	4	5
Terrified	1	2	3	4	5
Guilty	1	2	3	4	5
Vigorous	1	2	3	4	5
Uncertain about things	1	2	3	4	5
Bushed	1	2	3	4	5

Appendix F Dill and Costill Equation for calculating changes in plasma volume

$BV_a = BV_b (Hb_b / Hb_a)$

$CV_a = BV_a (Hct_a)$
$PV_a = BV_a - CV_a$

$BV\% = 100 (BV_a - BV_b) / BV_b$
$CV\% = 100 (CV_a - CV_b) / CV_b$
$PV\% = 100 (PV_a - PV_b) / PV_b$

Where,

BV = Blood Volume
CV = red cell volume
PV = plasma volume

and

b = before treatment
a = after treatment

Dill, D.B. and Costill, D.L. (1974). Calculation of percentage changes in volumes of blood, plasma and red cells in dehydration. *Journal of Applied Physiology*. 37(2): 247-248

Appendix G Standardized Warm-up for Ergometer testing

A standardized warm up was given to each athlete on paper to be performed as self directed. The warm-up is outlined as follows: 3 minutes light pressure rowing, followed by 3 minutes, 2 minutes, and 1 minute at half, three-quarter, and full pressure respectively at low rates (approximately 21 strokes per minute). 2 minutes of light functional stretches, 15 strokes on full pressure at 28 strokes per minute, 1 minute of light rowing, 15 strokes at 32 strokes per minute, 1 minute of light rowing, 1 minute on race pace (approximately 30-33 strokes per minute), 2 minutes of light rowing, 15 stroke start followed by light rowing for 4-5 minutes before the erg test will begin. This warm-up took approximately 27 minutes