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**THE MONITORING OF TRAINING AND RECOVERY  
IN ELITE CROSS-COUNTRY SKIERS**

*by:*

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A Thesis

Submitted to the School of Graduate Studies & Research

in Partial Fulfillment of the Requirements

for the Degree

**Master of Science**

Lakehead University

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## ABSTRACT

The purpose of this investigation was to monitor and assess the training program of elite cross-country skiers over an eight week period in order to develop a comprehensive profile of elite cross-country skiers. This was performed by: i) observing the training responses of skiers over time to physiological, biochemical and performance variables; ii) examining the relationship between the Overstress Monitoring Inventory to both resting values of heart rate and blood pressure; iii) observing the contrasting effects of physiological, biochemical and performance variables over a four and eight day period of reduced training. Ten (8 male, 2 female) elite cross-country skiers (19-27 yrs.) participated in the study and performed endurance training 10-24 hours per week. Following eight weeks of training, subjects were divided into two groups (N=4, N=6), and reduced training volume by 50% for four and eight days respectively. Physiological measurements consisted of evaluating peak and threshold (at TLac)  $\text{VO}_2$ , resting heart rate and blood pressure, blood lactate, lactate threshold (TLac), maximal heart rate, heart rate at TLac, % $\text{VO}_2$  (at TLac), % $\text{VO}_2$  at OBLA (onset of blood lactate accumulation) and treadmill time to exhaustion. Biochemical measurements consisted of evaluating white and red blood cell count, hematocrit, hemoglobin, serum ferritin, serum urea, creatine phosphokinase (CPK), lactate dehydrogenase (LDH), cortisol and testosterone. Psychological measurements were assessed with the use of a 29 question inventory prepared by Cross-country Canada. Performance evaluations consisted of both a mini timing study and technique analysis. Subjects were required to ski using the free techniques a 5 km course and were videotaped while skiing for one complete cycle (pole plant to pole plant) up a 5% grade for 30 meters. Tapes were then digitized using the peak 2D system and analyzed for kinematic parameters of cycle rate, time and length.

Significant differences were found in maximal heart rate ( $p < 0.05$ ) and hemoglobin ( $p < 0.01$ ) levels following 4 and 8 weeks of endurance training, respectively. Mean values of CPK decreased significantly ( $p < 0.05$ ) following 4 weeks of training. Cortisol concentrations demonstrated an increasing trend throughout 8 weeks of training, however, no significant variations ( $p < 0.05$ ) were observed during training or between groups following 4 and 8 days of reduced training. Testosterone levels profiled a similar response as cortisol, however, marked variations ( $p < 0.05$ ) between groups after 4 and 8 days reduced training were demonstrated. Treadmill time to exhaustion increased significantly ( $p < 0.05$ ) following 8 days of reduced training, although mean race time decreased following 4 days of reduced training. Mean scores of kinematic parameters failed to identify any significant variations throughout the period of the investigation. Similarly, correlational examination of the stress inventory and resting systolic blood pressure and heart rate also failed to illustrate any particular trends throughout the study. It was concluded that the elite skiers examined in the study demonstrated improvements in physiological, biochemical and performance evaluations following 8 weeks of endurance training. Specifically, 8 days of reduced training (a 50% decrease in training volume) illustrated a greater improvement in physiological, biochemical and performance evaluations when compared to 4 days of reduced training.

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## **CHAPTER ONE**

### **INTRODUCTION**

#### **1.1 Purpose**

The purpose of this investigation was to monitor and assess the 1992-93 Cross Country Canada training program of elite cross-country skiers over an eight week period, in order to develop a comprehensive profile of elite cross-country skiers. This was performed via:

- i) an observation of the training responses of skiers over time on physiological, biochemical and performance variables
- ii) an investigation of the relationship between the Overstress Monitoring Inventory to resting heart rate and resting blood pressure
- iii) an observation of the contrasting effects of a four and eight day period of reduced training

#### **1.2 Significance of Study**

Preparation for high performance sport competition involves periods of high volume training (Vos, Fry and Kraemer, 1992). The difficulty in determining the optimal loads for training, along with adequate recovery and rest, is problematic for both coaches and athletes. Training programs must be monitored continually to assess whether the stimulus for adaptation is effective. However, even in the presence of regular monitoring, training loads can become too excessive, resulting in poor performance.

Several terms have been used to identify states of physical exhaustion. Overreaching is the intentional or unintentional inducement of short term overtraining, the symptoms of which can be reversed by allowing a longer than usual regeneration period (Fry, Morton and Keast, 1992; Vos, Fry and Kraemer, 1992). This typically occurs when an athlete attempts to adapt to training loads that

are greater than the body's metabolic processes can deal with. Recovery from overreaching usually involves a few days up to two weeks of rest (Lehmann, Foster and Kuel, 1993). In contrast, Kuipers and Keizer (1988) identified muscular overtraining as transient local fatigue and muscle soreness following exercise that exceeds muscular stress tolerance. Furthermore, Kuipers and Keizer (1988) indicated that this form of overtraining generally occurs after single or repeated bouts of excessive exercise, which promotes structural damage to muscle fibers.

When overloading exceeds recovery and adaptation within a specified time, overtraining or an over-reaching response can occur (Fry, Morton and Keast, 1992; Kuipers and Keizer, 1988). Overtraining syndrome can then be described as the final stage in chronic fatigue states, developing from overreaching (Fry, Morton and Keast, 1992; Vos, Fry and Kraemer, 1992). Recovery typically requires a long-term process, involving biochemical, physiological and psychological parameters returning to normal levels. Budget (1990) indicated that overtraining may be induced by sudden increases in training load such as participating in intensive exercise with short rests and frequent competitions. Some authorities have suggested that weekly increases in training volumes of more than 5 to 10% may induce overtraining (Mackinnon and Hooper, 1991). Furthermore, other contributing factors such as frequency of competition and travel, intense strength training and year round training with no break between sessions can predispose athletes to overtraining (Mackinnon and Hooper, 1991). In addition, other factors such as the accumulation or deficiency in life stressors, diet, rest, occupational and relationship stress also can produce a cumulative effect in predisposing an athlete to overtraining.

Procedures for monitoring training stress should involve a multifaceted approach of evaluating the individual's response on physiological, biochemical, psychological and performance variables



over a period of time. Assessment of training loads should examine all parameters which respond to a training stimulus. This is necessary in order to accurately evaluate effectiveness of the training stimulus, and to make necessary amendments when required. Many authorities have indicated that with a decrease in the volume of training or extended breaks lasting one to two weeks, complete recovery from training overstress and performance improvements may occur (Budget, 1990; Fry et al., 1992; Houmard, 1991; Kuipers and Keizer, 1988). In other words, an inability to observe positive changes after rest, indicates that a state of overtraining may exist. Moreover, this period of recovery is necessary to facilitate effective adaptations to the training stimulus, thus permitting the athlete to advance to a higher load.

It is hoped that the results from this investigation will provide insight into the effectiveness of the current method of monitoring training loads of elite cross-country skiers through the provision a detailed profile of physiological, biochemical, psychological and performance parameters throughout eight weeks of endurance training. Furthermore, the study has examined the contrasting effects of a four and eight day period of reduced training following a period of heavy loading.

### **1.3 Limitations**

1. The analysis of the overstress monitoring inventory was dependent on the accuracy of information recorded by each athlete.
2. The records of resting morning heart rate were self reported and the responsibility of each athlete.
3. The scheduling of the race simulation test was dependent upon adequate weather conditions.
4. The analysis of whole blood for lactate values was restricted to the accuracy of the Yellow

Springs Instrument model 23L analyzer.

5. The analysis of peak  $\text{VO}_2$  was restricted to the accuracy of the Beckman Metabolic Measurement Cart and the subjects ability in reaching their maximal potential.
6. The analysis of serum cortisol and CPK was restricted to A.M. samples only, and as such diurnal variations in concentrations could not be accounted for.
7. The analysis of performance was limited to one complete cycle of the offset technique per skier on a 5% grade uphill section in order to identify cycle characteristics.
8. Commitments to national program events (ie. time trials, training camps) was responsible for the withdrawal from participation in the study.
9. Academic commitments and examination timetables determined which recovery group subjects were assigned to.
10. The training programs were provided and monitored by the training center coach.

#### **1.4 Delimitations**

1. This study was delimited to ten elite cross-country skiers (two females, eight males) who ranged in age from 19 to 27 years of age and all resided in Thunder Bay, Ontario.
2. Blood measures analyzed were hematocrit, hemoglobin, white blood cell count, red blood cell count, serum ferritin, serum urea, creatine phosphokinase, lactate dehydrogenase, testosterone, cortisol and lactate.
3. The test protocol for peak  $\text{VO}_2$  was established by Cross-Country Canada.
4. The performance analysis was delimited to cycle rate, cycle length and cycle velocity.
5. The timing study was delimited to comparison of percent total time of laps between test days.

## **CHAPTER TWO**

### **REVIEW OF LITERATURE**

#### **2.1 INTRODUCTION**

Endurance training promotes adaptation responses in aspects of metabolic and physiological functioning. However, when the stimulus that promotes adaptation is too severe, performance in physical activity and sport may be compromised. Far too often, inadequate preparation time and methodology of training inhibit optimal performance in the endurance athlete. The following literature review examines physiological, biochemical, psychological and performance adaptations to endurance training. In addition, it investigates the parameters which identify states of approaching fatigue and overtraining in endurance athletes when ineffective monitoring is done. Moreover, it profiles adaptations and responses to prolonged endurance training, which coaches, sport scientists, athletes and medical professionals look for when monitoring and assessing any training regime.

#### **2.2 PHYSIOLOGICAL ADAPTATIONS TO PROLONGED ENDURANCE TRAINING**

Endurance training produces a variety of physiological and metabolic adaptations over time resulting in an overall improvement in the efficiency of oxygen and nutrient delivery and utilization by the muscles. There is an increased concentration of oxidative enzymes, and an increased metabolism of free fatty acids. Overall physical performance improves, and the time for recovery from exertion is less. The following will illustrate these adaptations specific to elite skiers and endurance athletes. Particular attention will focus on both optimal adaptive changes and responses to prolonged endurance training.

### 2.2.1 Physiological Profile of Elite Cross-Country Skiers

Endurance can be defined as the individual's capability to exploit as much of their maximal oxygen uptake as possible for a period of time lasting longer than 30 minutes (Pate and Branch, 1992). Cross-country skiers demonstrate a high endurance capacity due to their ability to utilize a high percent of their  $VO_{2max}$  for long periods. It has been suggested that these levels range in value from 80-95%  $VO_{2max}$  (Bergh, 1982). The benefit of operating at a high level of  $VO_{2max}$  is that it increases racing speed, which results in a faster racing time. For example, Bergh (1982) illustrated how increasing from 80% to 85%  $VO_{2max}$  can result in a speed increase from 12 to 18-19 km/hr. In other words, in a 30 minute race, a speed of 18-19 km/hr would produce a reduction in race time of 4 minutes.

Due to the larger number of muscle groups involved, the energy cost of cross-country skiing has been shown to be one of the highest of all endurance sports (Clifford, 1992). The duration of skiing activity for females ranges from a minimum of 16 minutes duration for 3 km races to a maximum of 2.5 to 4 hours for 20 km races. In contrast, the duration of ski activity for male skiers ranges from 30 minutes for 10 km races to a maximum of 2 to 4 hours for 30 and 50 km races (Eisenman, Johnson, Bainbridge and Zupan, 1989). Shorter races have been shown to require a contribution of approximately 85 to 90% energy from aerobic metabolism, while longer races utilize a contribution of at least 98% from aerobic metabolism (Eisenman et al, 1989). Values of maximal oxygen uptake for elite male and female skiers are seldom less than  $80 \text{ ml kg}^{-1} \text{ min}^{-1}$  and  $70\text{-}75 \text{ ml kg}^{-1} \text{ min}^{-1}$  respectively (Eisenman et al, 1989). Measurements obtained from treadmill exercise demonstrates a 2.5-3.0% higher maximal oxygen uptake value for elite ski racers as opposed to elite runners. This is an increase in  $VO_{2max}$  of approximately  $10 \text{ to } 12 \text{ ml kg}^{-1} \text{ min}^{-1}$  higher than running at the same

velocity (MacDougall, Hughson, Sutton and Moroz, 1979). Elite skiers possess high maximal aerobic power values, with the highest ever recorded being  $94 \text{ ml kg}^{-1} \text{ min}^{-1}$  for a male Olympic champion (Bergh, 1982). Several researchers have reported that most elite male skiers have maximal oxygen uptake values in excess of  $80 \text{ ml kg}^{-1} \text{ min}^{-1}$ , and that top competitive skiers are able to utilize at least 85% or more of their individual maximal oxygen uptake value (Astrand and Rodahl, 1986; Bergh, 1982; Eisenman et al, 1989). However, seasonal variation in maximal aerobic power has been shown to occur as a result of changes in training from skiing to running or roller-skiing. Bergh (1982) has reported that the lowest values in maximal aerobic performance in skiers were revealed in the period from mid May to mid June. These seasonal variations in oxygen uptake ranged from a decrement of 5-23%, however, following 5 to 6 weeks of ski training, maximal oxygen uptake values increased. Bergh (1982) has suggested that variations in oxygen uptake of 5-10% occur during the period from May to June, and that this variation is a result of the lower level of training intensity for dry land exercise as opposed to training on snow.

Cross-country skiers have been shown to demonstrate a high percentage of type 1 fibers within the vastus lateralis muscle. It has been well established that endurance training at 70-90%  $\text{VO}_{2\text{max}}$  results in the recruitment of type 1 fibers, as evidenced by the depletion and re-synthesis of muscle glycogen (Astrand and Rodahl, 1986). Furthermore, it has been demonstrated that the type 1 fibre profile of elite Swedish and Finnish ski racers ranges between 63-91% and 45-100% of skeletal muscle respectively (Bergh, 1982). More importantly, Bergh (1982) reported that individuals with the greatest percentage of type 1 fibers have been shown to demonstrate the highest race tempo. An investigation by Sprynarova, Bass, Mackova, Vondra, Vitek, Teisinger and Malkovska (1980) examined changes in muscle enzyme activity in cross-country skiers (n=9) throughout two stages

of the annual training cycle. Subjects increased both training volume and intensity throughout the competitive year. Muscle biopsy samples were taken at the start of the pre-season training period, and at the conclusion of the competitive season. A decrease of 27 to 59% of muscle specific enzymes TPDH (triose phosphate dehydrogenase), LDH (lactate dehydrogenase), HK (hexokinase), CS (citrate synthetase), MDH (malate dehydrogenase) was accompanied by a related decrease in aerobic capacity and treadmill time to exhaustion. It was speculated that this decrement in muscle enzymes may be a result of both seasonal variations in training methods (skiing versus running) and training volume.

Cross-country skiers require several years of training to develop into elite performers. The more successful Scandinavian skiers have been reported to begin their ski training in preparation for competition at a young age. Rusko (1987) studied the long-term development (3 years) of aerobic power in young Finnish cross-country skiers who were successful at regional and national competitions, and reported that youth skiers were successful in increasing their  $VO_{2max}$  by the conclusion of the training year. Rusko (1987) concluded from the data that oxygen uptake and anaerobic threshold levels increase in young, growing athletes, however, oxygen uptake does not increase faster than body weight in young athletes. After age 20,  $VO_{2max}$  appeared to reach an upper limit, although some exceptions may exist where athletes are able to increase both  $VO_{2max}$  and anaerobic threshold past the age of twenty. Heart volume also demonstrated an increase between 15 and 20 years of age, with the most dramatic increase between 16 and 20 years of age. Rusko (1987) reported that some researchers suggest that this may be due to maximal levels of hormonal activity during puberty, which is more influential for development. In summary, Rusko (1987) reported that increased intensity seemed to be more instrumental for increasing  $VO_{2max}$  in young

skiers, while long, slow distance training was more effective in increasing anaerobic threshold. Heart rate values during skiing have been shown to reach maximal levels during uphill skiing, and drop to only 20 beats per minute below maximal values on downhill runs. Furthermore, heart rate values fall 5 to 15 beats per minute below maximum when skiing on flats for a relatively long period of time (Bergh, 1982).

It has been suggested that the longer the duration of the race, the lower the level of blood lactate concentration measured following the race. Some researchers suggest this may be due to a reduced ability to generate energy by glycogenolysis due to reduced glycogen stores and the inhibitory effects on key glycolytic enzymes (Astrand and Rodahl, 1986). The majority of ski races require a sustained output of high aerobic power. High maximal aerobic power can be achieved as a consequence of reduced lactate production, enhanced lactate elimination, and an increased tolerance of lactate and hydrogen ion accumulation (Eisenman et al, 1989). Improvement in these physiological performance values has been shown to be dependent on training at the lactate threshold. Anaerobic threshold has been suggested to be related to the skiers maximal work rate, and consequently several researchers have recommended that endurance athletes should train at or slightly below the anaerobic threshold (Eisenman et al, 1989). Many programs have been designed to train the skier at or below the anaerobic threshold. The lactate profile and heart rate values obtained during a maximal exercise test have then been used to establish the programs for elite skiers (Underwood, 1986; Reed, 1992a).

### **2.2.2 Lactate and Anaerobic Threshold Responses to Endurance Training**

It has been well documented that training increases the level at which the anaerobic threshold occurs, and consequently, also decreases the concentration of blood lactate at a given intensity

(Jacobs, 1986; Holden, MacRae, Dennis, Bosch and Noakes, 1992; Urhausen, Coen, Weiler and Kindermann, 1993). Specifically, endurance training at or just below the anaerobic threshold enables the athlete to operate at a higher percent of their individual maximum oxygen uptake. The recovery time from exercise is of importance, in so far as those individuals with a higher threshold value are able to recover more quickly. The lactate threshold may be defined as the point at which lactate production exceeds its removal, or when lactate accumulation occurs exponentially (Astrand and Rodahl, 1986). Based on trends of exponential increases in concentration, researchers have established a standard lactate threshold value known as the onset of blood lactate accumulation (OBLA) to be set at 4 mmol/L. However, the individual lactate threshold value has demonstrated a wide variation and often can range from 2.0 to 7.5 mmol/L for endurance athletes. As a consequence of this variation, Stegmann, Kindermann and Schnabel (1982) introduced a mathematical method for determining the threshold value based on the creation of a curve of best fit. Several researchers have experimented with this method, and have reported it to be an accurate means of calculating a true value (Mognoni, Sirtori, Lorenzelli and Cerretelli, 1990; Oyono-Enguelle, Heitz, Marbach, Ott, Gartner, Pape, Vollmer and Freund, 1990; McLellan, Cheung and Jacobs, 1991; Urhausen et al., 1993).

It has been observed that elevated levels of epinephrine have been associated with increased glycogen breakdown following exercise, however, the extent of this has still to be determined (Astrand and Rodahl, 1986). In a study by Mazzeo and Marshall (1989), highly trained cross-country runners and cyclists were examined to determine the relationship between plasma catecholamines, blood lactate and the ventilatory threshold. Subjects were tested under two different modalities, both on the treadmill and the cycle ergometer. Results demonstrated that there was a shift in the lactate



threshold, depending on training specificity. Mazzeo and Marshall (1989) reported that catecholamines (epinephrine) are strong activators of muscle glycogenolysis, and that when glycogenolysis is elevated, so is the rate of lactate production. High circulating levels of catecholamines have been reported as a marker of overtraining and may be due to the premature fatigue associated with greater concentrations of lactate in both the muscle and blood. A study by Acevedo and Goldfarb (1989) investigated the effects of increased training intensity on  $VO_{2max}$ , plasma lactate accumulation, ventilatory threshold and performance. Trained fit distance runners trained for 8 weeks at 90 to 95% of their maximal heart rate employing an interval format. Results of the study revealed that performance time to exhaustion increased by 20%. It was speculated that increasing training intensity may improve endurance performance by reducing the amount of plasma lactate accumulation without any alterations in  $VO_{2max}$ . Furthermore, the study concluded that blood lactate concentrations were lower or unaltered following endurance training at similar intensities. Therefore, significant reductions in lactate occurred at 85-90 % of  $VO_{2max}$ , and OBLA occurred at a significantly greater percent of  $VO_{2max}$  following this form of training. It was concluded that significant reductions in blood lactate content can occur at varying intensities with no alterations in  $VO_{2max}$  (Acevedo and Goldfarb, 1989).

Calculating an individual's anaerobic threshold in order to prescribe specific training intensities has been used extensively (Underwood, 1986; Reed, 1992a). The individual anaerobic threshold has been defined as the metabolic rate at which elimination and diffusion of blood lactate is at maximum and equal (McLellan and Jacobs, 1989). It has been suggested that lactate kinetics are strongly related to endurance performance during sub-maximal exercise. The training effect associated with an increase in anaerobic threshold appears to function by increasing the rate of oxidation or clearance

of blood lactate (Astrand and Rodahl, 1986). Therefore this increase in oxidative potential along with increases in capillarization would enhance the availability, uptake and oxidation of lactate within the muscle. McLellan and Jacobs (1989) investigated the influence of training and recovery on the calculation of the individual anaerobic threshold. Untrained subjects performed pre and post sub-maximal exercise tests on a cycle ergometer following an 8 week training program. Blood samples were taken throughout the tests as well as during two forms of exercise recovery, active (at 35%  $VO_{2max}$ ) and passive. Results indicated that power output and  $VO_{2max}$  for both active and passive recovery increased significantly following the program. McLellan and Jacobs (1989) concluded that for untrained subjects, the individual anaerobic threshold occurred at a higher metabolic rate and was related with higher steady state lactate values following active recovery. Furthermore, since the lactate threshold occurred at approximately 45-50%  $VO_{2max}$ , active recovery (35%  $VO_{2max}$ ) may have enhanced lactate clearance by increasing the uptake and utilization by the muscles.

Lactate variables have been correlated with parameters such as the percent of slow twitch fibers, capillary density, respiratory capacity and glycolytic and oxidative enzymes (Jacobs, 1986). Therefore, identification of the lactate variables may serve as an accurate marker of physiological development and/or decrement throughout the training year. Moreover, prescribing training loads based on lactate concentration may be an effective means in attempting to promote physiological changes characteristic of specific sport group populations (Jacobs, 1986). In some cases, it has been suggested that lactate-related variables are more effective indicators of training adaptation than  $VO_{2max}$  (Jacobs, 1986). Determination of the anaerobic threshold via laboratory analysis is not possible in some sports, or proves to be too costly or inconvenient. Droghetti, Borsetto, Casoni,

Cellini, Ferrari, Paolini, Ziglio and Conconi (1985) attempted to determine anaerobic threshold values in endurance athletes through correlating velocity and heart rate response during exercise. Forty two members of the Italian cross-country ski team had their heart rates monitored while roller skiing uphill over a distance. Ski velocity and heart rates were plotted and anaerobic threshold was determined by observing the deflection velocity, beyond which the increase in work intensity exceeded the increase in heart rate. Droghetti et al. (1985) concluded that there existed a linear relationship between velocity and heart rate, and that velocity may be a sufficient tool for determining the anaerobic threshold.

Endurance athletes utilize strength training during their training, however, the extent to which it should be used and its consequent benefits are varied. Some of the suggested benefits of strength training on endurance performance may be in the increases in muscular strength and power (Paavolainen, Hakkinen and Rusko, 1991). Marcinik, Potts, Schblach, Will, Dawson and Hurley (1991) investigated the effects of strength training on lactate threshold and endurance performance. Eighteen untrained male subjects were randomly assigned to either strength training or control groups for a 12 week training program. Evaluations were made via  $VO_{2max}$ , pedalling time to exhaustion, peak torque and lactate threshold. The results of the study revealed substantial increases in strength of 30%, 52% and 20% in strength exercises. Cycle endurance time at 75%  $VO_{2max}$  increased by 33% following training. As well, this improvement in endurance performance was associated with a 12% increase in the lactate threshold. Marcinik et al. (1991) concluded that strength training did enhance endurance time, however, the means by which this takes place is unknown. Possibly, a greater recruitment of slow twitch, and a reduced rate of fast twitch fibers during constant loads may help to explain the influence of strength training on endurance

performance. An alternate explanation may be related to the greater generation of peak force, which may result in less occlusion of blood flow during pedalling. Therefore, this improvement in circulation may affect lactate levels with little effect on  $VO_{2max}$ . Lactate and anaerobic threshold values do improve with endurance training. Threshold levels occur at a higher percent of maximum due to lower levels of production during exercise, and increased rates of clearance.

### **2.2.3 Performance Adaptations to Prolonged Endurance Training**

Certain physiological parameters have been shown to strongly influence endurance performance. Maximal aerobic power, or  $VO_{2max}$ , represents the ceiling of the body's aerobic metabolic system, and is defined as the greatest rate of oxygen uptake and individual can obtain during maximal exercise (Pate and Branch, 1992). Lactate threshold has been expressed as a percentage of individual  $VO_{2max}$  and is defined as the rate at which lactic acid begins to accumulate in the blood (Astrand and Rodahl, 1986). Economy refers to the rate of energy expenditure that is associated with a given rate of power output. The lower the rate of energy expenditure during a given work rate, the greater the economy. Training strategies are then designed to maximize these physiological parameters, which will contribute to the overall performance capabilities of the athlete. Three basic training regimes have been utilized to promote physiological adaptation. Long duration, moderate intensity training at 60-70%  $VO_{2max}$  for 30 minutes up to 2 hours has been demonstrated to promote muscle glycogen depletion and an increase in fat utilization. Furthermore, this form of training has been demonstrated to maintain both cardiac output values and skeletal muscle oxidation capacity (Astrand and Rodahl, 1986). Moderate duration, high intensity training involves exercise in the vicinity of the athletes lactate threshold. Training sessions may range from 30 to 60 minutes at a constant intensity and this

form of training has often been referred to as "pace or tempo" training (Pate and Branch, 1992). The final method of endurance training is short duration, very high intensity training. This format of interval training involves maximal work of 30 seconds to 3 minute durations. Multiple bouts are performed in a single training session and are alternated with rest intervals. This allows performance of high intensity exercise in a larger volume, thus producing mainly anaerobic adaptations of oxygen and nutrient extraction by the skeletal muscles.

### **2.2.3.1 Evaluation of Elite Skiing Performance**

Most of the recent literature on elite ski performance has concentrated on the physiological responses of skiing. Ski performance, however, is a combination of physiological economy, efficiency, technique and technical strategy. In order to adequately assess performance, each area must be examined individually to determine if maximal efforts are attained. The most common difficulty in researching ski performance is the task of illustrating the connection between its key components. Often, the most important scientific parameters must be assessed within a laboratory setting. As a result, transferability of laboratory results to the competitive skiing environment may suffer due to lack of specificity in the testing.

A study which examined the combined effect of physiology and technique was reported by Norman, Ounpuu, Fraser and Mitchell (1987). The study evaluated mechanical power output and estimated metabolic costs of Olympic Nordic skiers. A segmental model was constructed (stride length, stride rate, mechanical power output) in an attempt to estimate oxygen consumption. It was reported that the faster skiers demonstrated an estimated high  $VO_2$  of 80 to 112 ml  $kg^{-1} min^{-1}$  as compared to slower skiers (53 to 77 ml  $kg^{-1} min^{-1}$ ) during Olympic competition. Norman et al.

(1987) concluded that these athletes perhaps overcame the effects of a rather large oxygen debt by compensating with changes in technique, better developed metabolic energy systems, and stronger physiological tolerances to anaerobic work. In comparison, Norman and Komi (1987) examined mechanical cost of diagonal stride skiing in world class skiers. The mechanical cost of skiing was found to be 2.2 times more demanding than on level terrain. Surprisingly, Norman and Komi (1987) estimated that metabolic rate was 76% of a peak  $\text{VO}_2$  of  $80 \text{ ml kg}^{-1} \text{ min}^{-1}$ . As a result, it was suggested that speed during the diagonal stride was the limiting factor rather than physiological capability. The obvious benefit of examining biomechanical parameters in comparison as opposed to just physiological variables is in the transfer of useable information in assessing skiing technique. Unfortunately, research in this combined field is some what limited, and therefore researchers typically examine the streams independently.

Some of the work that has examined cross-country skiers from an inter-disciplinary approach have compared the classic techniques to the skating techniques. It has been well documented that skating, rather than classic style, generates higher lactate levels, is faster, and generates a more efficient use of power (Bilodeau, Roy and Boulay, 1991; Hoffman and Clifford, 1990; Karvonen et al., 1989; Smith et al., 1989; Underwood, 1986). This may be a result of the delay in the onset of anaerobic energy metabolism during skate skiing, consequently prolonging the onset of fatigue. Karvonen, Kubica, Wilk, Wnorowski, Krasicki and Kalli (1989) reported observing lower heart rate values at a specific intensity when comparing skating to classic skiing.

Different skiing styles produce different physiological outcomes. For example, Hoffman, Clifford, Watts, Drobbish, Gibbons, Newbury, Sulentic, Mittlestadt and O'Hagan (1994) reported that during classic skiing, diagonal stride (DS) was more physiologically economical than double

pole (DP) when roller skiing on a 1.7% grade, however, this difference was reversed at higher grades (7.1%). Others, Watts, Hoffman, Sulentic, Drobish, Gibbons, Newbury, Mittlestadt, O'Hagan and Clifford (1993) found that peak values for heart rate and blood lactate did not differ among skiing techniques. However, when compared to DP roller skiing on a flat terrain,  $VO_2$  requirements were reported lower than skate roller skiing (Hoffman, 1992; Hoffman and Clifford, 1992). Diagonal stride involves a greater mechanical power output, greater mechanical task and metabolic cost (Smith, 1992). However, elite skiers tend to demonstrate longer stride lengths while performing the diagonal stride. Moreover, the glide phase of the stride length has been reported to be strongly associated with velocity and race performance (Smith, 1992; Smith and Gregory, 1994). Friction generated between the ski surface and snow can also effect glide. Street and Gregory (1994) reported that successful Olympic skiers tend to have faster glide speeds, which was suggested to be primarily a result of differences in friction. This variation in ski friction effects skiing velocity and overall ski performance.

Another example of the effect of ski style comes from Boulay, Rundell and King (1995). In this study, skate skiing was observed to be more effective for climbing slopes as opposed to classic skiing, primarily because of its ability to generate higher ski velocities and power. The success of skating has been reported to be largely an indication of: lower trunk position, a more efficient use of trunk and arm muscles, and a more efficient horizontal power component (Bilodeau et al., 1991). Hoffman et al. (1994) suggested that this difference between techniques may have been due to the proportion of propulsive forces along the line of travel. In other words, as the grade increases, the proportion and distribution of force generation increases as well. It has been suggested that elite skiers produce significantly higher levels of forefoot force than recreation skiers (Pierce et al., 1987).

The force generated during the thrust phase of skiing must be large enough to: i) overcome the friction between the ski and snow; ii) overcome the resistance of air; iii) elevate the center of mass during each stride on both flat and slope terrain; iv) accelerate the center of mass and mass of various kinematic segments (Bergh and Forsberg, 1992). Bilodeau, Boulay and Roy (1992) reported that the relative propulsive and gliding phases of skating techniques were significantly longer than diagonal stride in elite skiers. As a result, these likely explained the greater speed attained when skating, as opposed to diagonal stride. Smith, McNitt-Gray and Nelson (1988) suggested that elite skiers increased speed by "straightening out" the center of mass path, providing stride length and rate was maintained.

Another component that has been examined in skiing studies is that of force. Hoffman and Clifford (1992) reported that the speed of muscle contraction may be the limiting factor for maximum velocity. As skiing velocity increases, the force generated must be applied over a shorter period of time, thus the time available for force application is reduced. Another possibility may be the use of metabolic substrate. It has been demonstrated that RER (respiratory exchange ratio) values are lower for skating techniques as compared to classic techniques. Consequently, muscle fatigue may be delayed and thus skiers would be able to use more power for a longer duration (Hoffman and Clifford, 1992).

Evaluating force components during skate skiing is difficult due to the constant change in orientation throughout a cycle (Smith, 1992). However, kinetic analysis of a skier's performance may indicate how well the individual can tolerate fatigue and subsequently how well they can race. Fatigue can compromise the efficiency of movement, and has been related to signs of overtraining and staleness (Fry et al., 1992). As a result, mechanical efficiency in technique may also be



compromised as muscular fatigue increases (Bilodeau et al., 1991; Boulay, Rundell and King, 1995).

The effectiveness of generating adequate power while skiing can be verified through the examination of the effect on individual kinematic parameters. Analysis of the V1 (offset) skating technique from the 1988 Olympics has revealed specific data about the techniques of world class skiers. Employing a kinematic analysis, Smith, Nelson, Feldman and Rankinen (1989) reported that the fastest skiers tend to use longer cycle lengths while maintaining the same cycle rates as slower skiers. It was suggested that a smaller weak ski angle and center of mass contributed to longer cycle lengths. Moreover, cycle rate tended to increase with velocity, while cycle length remained constant. Smith et al. (1989) concluded that faster skiers tended to skate with longer cycle lengths, while maintaining a consistent cycle rate. The linear relationship between a skiing velocity (cycle velocity) and cycle length has also been confirmed by Bilodeau et al. (1991) and Aro, Smith and Nelson (1990). Male skiers demonstrated that cycle rate and cycle length were moderately related to cycle velocity. In contrast, female skiers demonstrated that cycle rate is more strongly related to cycle velocity than cycle length (Smith et al., 1989). Consequently, cycle length and cycle velocity have been observed to decrease in both male and females while skiing steeper slopes. Aro et al. (1990) suggested that Olympic male skiers adjusted their technique by increasing cycle rate more than Olympic female skiers on steeper terrain. Smith (1992) observed that elite skiers demonstrated a greater stance width, longer forward steps, with shorter cycle lengths and less lateral movement while skiing steeper terrain. It has been suggested that these adjustment may be the result of a larger muscle mass in males, thus allowing males to produce a greater capacity to generate force up inclines when compared to female skiers (Bilodeau et al., 1991; Boulay, Rundell and King, 1995).

Alterations in kinematic parameters produce changes within the style of the technique. Classic

techniques have been reported to be controlled through the adjustment in cycle rates, as opposed to the adjustment in cycle length during skate skiing. However, both slower and faster skiers are able to increase their relative speeds by increasing their tempo or cycle rates. (Smith, 1992). For example, elite skiers tend to demonstrate a more efficient glide and stride pattern than non-elite skiers, which may explain why elite skiers are able to cover a greater distance in the same cycle than non-elite skiers (Smith et al., 1989). This can result in a total lower energy cost for exercise, thus delaying premature fatigue. The improvement in metabolic efficiency can be demonstrated through the increase or decrease in cycle rate, cycle length, cycle velocity, lower heart rates, higher lactates and increased velocity. Consequently, these changes result in improvements in technique, race time and overall performance. It has been suggested that skiers should attempt to increase the natural skating cycle length through improving glide and propulsive forces (Smith, 1992). This can be produced by increasing both metabolic and mechanical sources of propulsive force through training.

#### **2.2.4 The Physiological Effects of Reduced Training and Detraining**

Several researchers have suggested that performance decrements are often indicative of fatigue associated with overtraining (Fry et al., 1992; Houmard, 1991; Kuipers and Keizer, 1988; Lehmann, Dickhuth, Gendrisch, Lazar, Kaminski, Aramendi, Peterke, Wieland and Keul, 1991; Shepley, MacDougall, Cipriano, Sutton, Tarnopolsky and Coates, 1992). However, some individuals have reported performance improvements with reductions in training loads over a period ranging from 6 to 21 days (Houmard, 1991; Shepley et al., 1992). It has been suggested that athletes who demonstrate overtraining symptoms have a decreased maximal working capacity and plasma lactate levels. This decrease in plasma lactate level may be explained by the decrease in sympathetic

activation during exercise (Kuipers and Keizer, 1988). Physiological changes with detraining and reduced training have been reported to occur within days or weeks after the cessation of training, and results in a loss of both sub-maximal and maximal performance ( $VO_{2max}$ ) of 25% following 15 days of inactivity. Furthermore, it has also been reported to result in an 8% decrease in time to fatigue following 2 to 4 weeks of detraining (Neufer, 1989). It has been suggested that significant reductions in  $VO_{2max}$  occur within 10 to 21 days following the cessation of training. Specifically, reductions in  $VO_{2max}$  following 21 days of bed rest contributed to a 17-28% decrease in performance (Neufer, 1989). Evidence exists which supports the decrease in  $VO_{2max}$  to reductions in cardiac output and a- $vO_2$  (arterial-venous) differences (Astrand and Rodahl, 1986). The reduction in  $VO_{2max}$  during the early stages of detraining (2 to 4 weeks) has been reported to occur as a result of lower stroke volumes, which may in turn cause an increase in maximal heart rate. However, further decreases in  $VO_{2max}$  are largely due to a decline in a- $vO_2$  difference. Blood volume has also been shown to change with declines in plasma volume, primarily due to a loss in intravascular protein content (Neufer, 1989).

In a review by Neufer (1989), it was reported that as much as 3.5% in total hemoglobin can be lost following 2 to 4 weeks of detraining, and may explain the overall 6% decrease in  $VO_{2max}$  associated with detraining. Overall, lower plasma volumes can severely limit cardiac filling, which may reduce the mechanics of blood transport. Detraining can also effect pre-load and after-load myocardial contractility during both rest and exercise conditions. This may explain a lower stroke volume and consequently lower cardiac output, in so far as lower pre-load levels are associated with reductions in cardiac hypertrophy (Neufer, 1989).

Skeletal muscle oxidative enzyme concentration has been reported to decrease following

detraining, however they are more causally linked with changes in sub-maximal exercise. An investigation by Coyle et al. (1985) as reported by Neuffer (1989) indicated that a 40% decrease in mitochondrial enzyme activity and a 21% increase in LDH (lactate dehydrogenase) activity were found following 8 weeks of detraining. Changes in capillarization and blood flow have also been observed following inactivity, however, they may not depreciate as quickly as other parameters. Neuffer (1989) suggested that reductions in capillary density also reduce muscle blood flow, thus limiting the overall availability of oxygen to the muscle and can contribute to reductions in performance.

### **2.2.5 The Effects of Reduced Training on Performance**

Houmard (1991) studied performance following a reduction of training volumes in collegiate runners. Training frequency was reduced by 50% for a 10 day period (a 70% to 80% reduction in training volume). The results of the study indicated that maximal heart rate and  $VO_{2max}$  were not altered with reduced training (66.8 ml kg<sup>-1</sup> min<sup>-1</sup> versus 66.0 ml kg<sup>-1</sup> min<sup>-1</sup> for normal and reduced training, respectively). Several studies, as cited by Houmard (1991), revealed that measurements in  $VO_{2max}$ , maximal heart rate, and maximal speed or workload in elite distance runners were not diminished with reductions of 70% to 80% training volume for 10 to 28 days. Furthermore, Houmard (1991) reported that muscle power performance will be maintained and even improved following a 6 to 14 day taper with sufficient training frequency and intensity.

A study by Shepley et al. (1992) examined the physiological effects of a 7 day taper in middle distance runners following 8 weeks of training. Three tapers were used; a high intensity/low volume taper (HIT), a low intensity/high volume taper (LIT), and a rest only taper (ROT). The results of the

investigation indicated that maximal oxygen consumption was unaffected in all three tapers, and strength increased significantly in all three tapers. Total blood volume, red cell volume and citrate synthetase activity increased significantly with the HIT. Shepley et al. (1992) concluded that performance improvements can take place in highly trained individuals when intensity is maintained, and volume reduced.

Many sources reported an improvement in performance with the implementation of a reduced training load following heavy training (Budget, 1990; Fry et al. 1991; Houmard, 1991; Shepley et al., 1992; Lehmann et al., 1991). Furthermore, several researchers have suggested that overtraining will not occur if athletes return to normal levels within a set time period of approximately 2 to 3 weeks (Fry et al., 1992; Kuipers and Keizer, 1988; Vos et al., 1992). This improvement in performance was shown to be contingent on the variables of training, primarily intensity, duration and frequency. However, it does appear that with the introduction of a taper, an over-compensation effect takes place, and fatigue is drastically reduced. As a result, use of tapers in elite training programs is both fairly commonplace and effective.

## **2.3 THE PHYSIOLOGY OF THE STRESS RESPONSE, METHODOLOGY OF OVERTRAINING, AND THE CROSS-COUNTRY CANADA NATIONAL TRAINING MODEL**

### **2.3.1 The General Adaptation Syndrome**

Hans Selye (1956), examined the living organism's biological response to a stress stimulus. He investigated the organism's success and failure in the biological adaptation to stress. Selye's model of stress adaptation is characterized by three stages: the alarm-reaction stage, the resistance stage,

and the exhaustion stage. Stimuli for stress in the alarm stage (ie. physical training, illness, injury, shock, emotional difficulties etc.) causes corresponding physiological reactions to evolve. Sympathetic nervous stimulation is increased producing elevated levels of circulating cortisol. Catecholamines (adrenaline and noradrenaline) are also elevated in the alarm stage. These functions are necessary to produce the "flight or fight" response in the organism. Selye (1956) observed that with prolonged periods of exposure to stress, the adrenal cortex becomes enlarged, and ulceration may occur in the stomach due to increases in sympathetic innervation, and atrophy of the lymphatic system may result. The resistance or adaptation stage follows this initial stage of arousal. The organism must be able to reduce the effects of the stressor to a manageable level, and thus be able to meet requirements of the stressful situation. Selye (1956) identified this defense mechanism as a decrease in corticoid activity in order to promote adaptation. However, if the corticoid activity remains elevated, other functions may be compromised. In other words, the "fight or flight" response necessary for immediate survival takes precedence over secondary lines of defense such as inflammation or reproduction. In the final stage, exhaustion, the organism is unable to adequately adapt to the stressors. Selye (1956) suggested there is an inability to defend against other external agents, in that adaptive agents may be depleted while the corticoids are elevated. Selye (1956) recognized the importance of establishing an adequate stressor to promote physiological adaptations. However, with endurance training, the stressor must be imposed over a long period of time. Consequently, the endurance athlete may be susceptible to evolving into the "exhaustion stage", and must take proactive steps to avoid reaching this state.

### **2.3.2 The Methodology of Prolonged Training and Overtraining**

Identification of the overtraining state involves a multifaceted approach, which requires an examination of change in the individual's physiological, metabolic, psychological and performance states over a period of time. Lehmann, Foster and Keul (1993) defined overtraining syndrome as the accumulation of fatigue (both exercise and non-exercise), reductions in physical performance, alterations in mood states, presence of muscular stiffness/soreness and a decline in competitive competence over a period of weeks and months. Mackinnon and Hooper (1991) suggested that a 5 to 15% decline in competition performance is not uncommon in overtrained athletes. By far, the most noticeable and overt indicators of overtraining are prolonged fatigue (lasting longer than 1 week), performance decrements, and alterations in mood states (Mackinnon and Hooper, 1991).

Overtrained states have been observed to exist in one of two forms, each of which is a function of the type of exercise that is used in training. Sympathetic overtraining is characterized by an increase in sympathetic innervation at rest, which produces alterations in physiologic functioning such as increased resting heart rate, blood pressure, and weight loss, insomnia and emotional lability. Furthermore, it has been suggested that sympathetic overtraining is more closely associated with explosive, non-endurance anaerobic type physical activity (Kuipers and Keizer, 1988; Vos et al., 1992). In contrast, parasympathetic overtraining is characterized by sympathetic inhibition and parasympathetic dominance, and may be diagnosed after a considerable length of time. Parasympathetic overtraining may be an advanced state of overtraining that is associated with exhaustion of the neuro-endocrine system. Endurance athletes have been shown to be more susceptible to experiencing this form (parasympathetic) of overtraining, which is characterized by early fatigue, low resting pulse rate, higher levels of sleep than normal, and poor endurance

performance (Kuipers and Keizer, 1988; Vos et al., 1992). If not corrected, long-term changes in catecholamine, hematological and endocrine levels will occur. At this stage, recovery is a lengthy process, which may require several weeks to months off from training.

### **2.3.3 The Cross-Country Canada National Training Program**

The national model was designed to provide long-term training objectives for elite skiers so as to promote the necessary adaptations required for elite performance. The model is based on a five zone concept, which matches heart rate values to lactate levels. Athletes are exposed on a regular basis to field tests to determine their lactate levels at different heart rate intensities. The program then requires the athlete to train within zones depending upon what training cycle in the annual training plan the athlete is participating in. Zone one is low intensity, non-specific, general activity. It is characterized by exercising between 60-75% maximal heart rate with lactate levels less than 2.0 mmol/L. Zone two focuses on aerobic improvement with participation in more specific activity. It is characterized by exercising between 75-85% maximal heart rate with lactate levels between 2.0-3.0 mmol/L. The objective of zone three training is to promote lower levels of lactate accumulation. It is characterized by sport specific exercise between 85-90% maximal heart rate with lactate values between 3.0-4.5 mmol/L. Zone four, which aims to improve lactate tolerance, is characterized by exercising at maximum intensity between 90-95% maximal heart rate with lactate levels between 4.5-8.0 mmol/L. Zone five training is solely anaerobic in nature, and involves hill training at 95% maximum heart rate with lactate levels exceeding 8.0 mmol/L.

There are five periods identified in the annual plan (1992-93) devised by Cross-country Canada. The general aerobic period lasts 10-12 weeks in duration from mid May to the end of July. It consists



of both zone one and two training, with a gradual introduction to zone three at the conclusion of this period. The specific aerobic period concentrates on a training intensity of 60-80% of maximal heart rate, and relies heavily on ski specific training. It lasts from early August to the start of the ski racing season. At least one zone three and one zone four session is included each week. The competition period lasts from the start of the racing season to the first taper/peak period. All training is 100% ski specific with an incorporation of two zone four and one zone five session per week. The taper/peak period ranges from 10 days to two weeks in duration. During this period, a reduction takes place with the introduction of primarily zone 1 and zone 2 training. Zone four is maintained 2-3 times per week, with emphasis on short intervals. The length of the regeneration period is based on individual needs and may last from 3 to 6 weeks in duration. Medical assessment is done during this period. Activities are more general in nature, with no participation in skiing. To assist the coach in evaluating the training status of the skier, Cross-country Canada has implemented a monitoring inventory to record the athletes psychological and behavioral state. The objective of this inventory is to screen those athletes who are at risk of developing overstress syndrome. Upon concluding that an athlete is at risk, the coach then intervenes by reducing the number of stressors, or by increasing the athlete's ability to adapt to the stressors. When necessary, the coach is required to obtain the assistance of medical professionals and other resources for assistance.

## **2.4 BIOCHEMICAL ADAPTATIONS TO PROLONGED ENDURANCE TRAINING**

### **2.4.1 Hematological and White Blood Cell Profiles of Endurance Athletes**

Prolonged exposure to training has been demonstrated to produce cardiovascular changes that range from increases in resting blood pressure and heart rate, to a slow return of post-exercise heart rate to normal (Newhouse, 1984; Vos et al., 1992). Symptoms of overtraining have been associated with a decrease in the hemoglobin concentration and hematocrit due to anemia and/or hemolysis (Houmard, 1991). Budget, (1990) has differentiated between true anemia (iron deficiency), and low hemoglobin levels due to increases in plasma volume as a response to training. It has been observed that serum ferritin levels as low as 12 ug/L can be tolerated without any effect on performance (Budget, 1990). A study by Fry et al., (1992) examined hematological changes in response to 10 days of intensive interval training (twice per day), followed by 5 days of recovery. Performance decrements occurred at the conclusion of the training, but returned to baseline levels. Blood analysis indicated an elevated expression of lymphocyte antigens after training and recovery, with serum ferritin concentrations being significantly depressed from pre-training to recovery states. The study revealed that hemoglobin and hematocrit assessments alone may not be very accurate in determining physiological fatigue states of athletes. Most researchers suggested that hematological assessments be used in conjunction with other tests, in order to obtain an effective evaluation of fatigue and adaptations to the training program (Mackinnon and Hooper, 1991; Houmard, 1991; Fry et al., 1992).

Endurance trained athletes have demonstrated a susceptibility to iron deficiency, and low hemoglobin levels (Vos et al., 1992). Deficiencies may be explained on the basis of depressed bone

marrow iron stores from various causes such as low dietary iron intake, sweating, gastrointestinal blood loss during heavy efforts, and increased red blood cell turnover (Pattini and Schena, 1990). Elite cross-country skiers have also exhibited similar iron deficiencies, which could compromise performance through reduced oxygen transport. Pattini and Schena (1990) examined the effects of iron supplementation in iron deficient cross-country skiers over a four month period of regular training. A level of serum ferritin  $<30$  ug/L was used as the criterion for iron deficiency. Results demonstrated that following four months of treatment, serum ferritin levels improved by 67.8% (160 mg/day), and 63.6% (160 mg/day plus 1 gm ascorbic acid). Pattini and Schena (1990) reported that cross-country skiers frequently use running and roller skiing during the off-season as methods of training. During this period of training, skiers may experience iron deficient states, which if left untreated could extend into the competitive season and adversely affect performance. Therefore, skiers should be monitored for low iron levels on a regular basis during off-season training, and receive iron supplementation when necessary.

Exercise stress has been shown to effect levels of white blood cells within the body. Osterud, Olsen and Wilsgard (1989) investigated changes in blood monocytes and coagulation during prolonged strenuous exercise. Thirty one subjects, twelve of which were national cross-country skiers, who trained an average of 3-5 hours per day for 20 hours a week, for 7 to 10 years were examined. It has been well established that physical exercise induces various degrees of hyper-coagulability and increased fibrinolytic activity. Products of blood cell activation can influence micro-circulation, by altering blood flow and oxygen transport. The results revealed a rise in white blood cell (WBC) count after regular training (20 and 50 km races) of 250%, whereas white cell count only increased 50% following a 20 km race. Osterud, Olsen and Wilsgard (1989) reported that

there is a marked increase in WBC counts in relation to the duration of exercise, and consequently, this relationship was more dependent upon intensity than duration of activity. Osterud, Olsen and Wilsgard (1989) observed low levels of WBC counts following ski racing (20 km), in contrast to higher WBC counts in runners. Top level ski racers exhibited a substantial sensitivity to stimulation of monocytes, as expressed by the increased synthesis of thromboplastin which is necessary for the production of blood clotting. It was suggested that this was most likely due to mobilization of both new and more activation-prone monocytes present in circulation. In conclusion, high WBC levels in endurance athletes during exercise may compromise performance due to changes in vascular permeability.

#### **2.4.2 Endocrine Profiles of Endurance Athletes**

Prolonged periods of endurance training produce changes in the endocrine system of exercising humans. Exercise stress has both short and long-term influences on the endocrine profiles of the individual. The most overt changes, however, may be seen in the variations in concentrations following exercise stress. The research literature has indicated that alterations in serum testosterone, cortisol and catecholamine levels are most common with endurance training and overtrained athletes.

##### **2.4.2.1 Catecholamine Concentration**

Prolonged periods of stress will promote hypothalamic dysfunction. The hypothalamus coordinates endocrine, behavioral and autonomic nervous system functions. Stress activates the hypothalamus, which in turn influences an increase in the adrenocorticotrophic hormone (ACTH) releasing factor. This increase activates the anterior pituitary, which releases ACTH to activate the

adrenal cortex. An increase in neuro-endocrine innervation causes activation of the pituitary adrenocortical system. The result is large increases in circulating plasma levels of catecholamines and cortisol (Kuipers and Keizer, 1988; Newhouse, 1984). Several changes in metabolic function have been associated with overtrained states. Catecholamines have been shown to increase during exercise due to a release of noradrenaline from sympathetic nerve endings, and provide the means whereby an increase in heart rate, stroke volume, basal metabolic rate and ventilation can result (Boone, Sherraden, Pierzchala, Berger and VanLoon, 1992; Gaesser, 1994; Kuipers and Keizer, 1988). Furthermore, this can result in an overall increase in blood shunting due to the vasoconstriction and vasodilation of tissues in response to the increase in catecholamines (Newhouse, 1984). Following long term exercise, plasma catecholamine levels have been observed to fall well below initial levels due to a suppressed catecholamine response. Thus it is this stress response that allows catecholamines to be employed as a reliable indicator of stress. (Kuipers and Keizer, 1988). Some researchers have indicated that overtraining may be associated with suppressed catecholamine levels, and may serve as one factor to be examined in the monitoring of overtraining. Moreover, it has been suggested that measurement of nocturnal catecholamine excretion levels are a strong indicator of intrinsic sympathetic activity (Mackinnon and Hooper, 1991).

A study by Lehmann et al., (1991) examined catecholamine excretion levels of middle and long distance runners after periods of high volume endurance training. The runners demonstrated lower levels of catecholamine excretion than baseline after four weeks of overload training (an increase from an average of 85.9 km in week 1, to 174.6 km in week 2). In addition to this, runners were also monitored on a four point complaint index, which demonstrated a relationship between statements of fatigue and excretion levels of catecholamines. Lehmann et al. (1991), concluded that a decrease

in catecholamine levels may indicate a sign of overexertion, as observed in endurance based (parasympathetic overtraining) events.

#### **2.4.2.2 Testosterone Concentration**

Endurance training has been shown to increase the concentration of both serum testosterone and cortisol. Furthermore, it has been observed that during periods of intensive stress lasting several days, serum testosterone falls and serum cortisol increases in endurance trained athletes (Hakkinen, Keskinen, Alen, Komi and Kauhanen, 1989). A study by Hackney (1989) examined testosterone responses in endurance trained males. Normal resting blood testosterone levels in adult males ranged from 3.0 to 11.0 ug/L, the majority of which was produced by the Leydig cells of the testes. Testosterone serves both reproductive and anabolic roles, and regulation is performed via a negative feedback involving the hypothalamic-pituitary-testicular axis. The majority (97%) of circulating testosterone is transported bound to carrier proteins. The remaining 3% is unbound and circulates as "free testosterone". "Free" testosterone is considered the biologically active form, and the combination of both bound and free testosterone are referred to as total testosterone (Hackney, 1989).

Research on the effects of endurance training on testosterone levels has revealed that endurance athletes tend to display lower resting levels of testosterone. Investigators have reported that low levels of free testosterone are characteristic of overtrained individuals (Lehmann, Foster and Keul, 1993; Fry et al., 1992). Testosterone profiles of endurance athletes, as reported by Hackney (1989), demonstrated lower levels of serum testosterone and free testosterone in chronically endurance trained males. The factors which influence testosterone levels are a function of production, secretion

and metabolic clearance. Average testosterone production in adult males has been determined to be 7000 ug/day, and this rate is controlled by production stimulators (LH receptors and substrate availability). Metabolic clearance of testosterone has been estimated at 1100 ug/day, which is a result of both target tissue uptake and hepatic degradation (Hackney, 1989). Testosterone responses to short term, maximal exercise produced an increase in circulating levels, which may be a result of hemo-concentration, reduced clearance and/or a response in elevated catecholamines (Hackney, 1989). The responses to sub-maximal exercise were somewhat varied, with reports ranging from little or no increases, to even decreases in concentration. It has been well established that decreased testosterone production in sub-maximal exercise may be as a result of reduced testicular blood flow during exercise, therefore compromising hepatic blood flow and metabolic clearance (Hackney, 1989). Studies which have reported low levels of circulating testosterone were those which examined endurance trained subjects who had been training for a minimum of 5 years and were competitive at elite levels. Therefore, these results may reflect the long-term effects of training. Although it has been well established that testosterone production and clearance is sensitive to endurance training, there is little evidence that demonstrates prolonged training produces significant effects on testosterone dependent functions in the body. However, it is evident that extremely high or low levels of testosterone over an extended period of time is associated with certain conditions (ie. overtraining), and that monitoring testosterone profiles may be of value in assessing training adaptations.

Hakkinen et al. (1989) investigated serum hormone concentrations during prolonged training in both strength and endurance trained athletes. It is known that exercise intensity and duration promote acute responses in the endocrine system. The corresponding changes in the endocrine

system to endurance training primarily involve adaptations in energy production through oxidative metabolism. Hakkinen et al. (1989) measured weightlifters and swimmers at four month intervals during the course of one year. The results of the study demonstrated that during the most intensive part of the training year, small but insignificant changes of serum testosterone, free testosterone and cortisol did occur in both groups. These results indicated that mean serum cortisol for the endurance trained group significantly increased during the year. It was concluded that testosterone and cortisol levels do fluctuate and are sensitive to training loads throughout the year. Therefore, hormonal profiles may then prove effective in the monitoring of exercise stress and prevent conditions of overtraining.

Fry, Morton, Garcia-Webb and Keast (1991) examined endocrine responses in 14 subjects of varying fitness levels to 2, 4, 8, and 24 hours post-exercise anaerobic interval training. Uric acid, urea, and CPK (creatine phosphokinase) were found to be elevated higher than-pre-exercise levels even after 24 hours post-exercise. Lactate, CPK, testosterone and cortisol concentrations were elevated by 2 hours post-exercise and returned to normal at 24 hours post-exercise. However, testosterone levels eventually fell even lower than pre-exercise levels in recovery. Budget (1990) reinforced this observation by indicating that intense and prolonged exercise will increase cortisol, but decrease levels of testosterone. As a result, the testosterone/cortisol ratio may fall in response to prolonged periods of training. Kuipers and Keizer (1988) suggested that low testosterone levels following exercise might be the result of an inhibition of testicular secretion, due to an increase in luteinizing hormone. In conclusion, Fry et al. (1992) observed that hormonal imbalances due to intensive training were not totally reversed within a 24 hour period. Although this study did present a sound hormonal profile as a result of intensive exercise, it may not be directly applicable to



overtraining in elite athletes. These (elite) individuals have been subjected to a high volume and intensity of training for a prolonged period, unlike that of the varying fitness levels of the subjects in the aforementioned study. Consequently, the degree of response would vary as a consequence of the higher levels of fitness. Nevertheless, these studies do illustrate the varied response of testosterone to physical activity.

#### **2.4.2.3 Cortisol Concentration**

The monitoring of cortisol levels has been reported to be an effective way in assessing stress (Sapsee, 1984). It has been documented that stressful situations increase levels of plasma cortisol. Cortisol is divided into 80% corticosteroid binding globin (CBG), 10% serum albumin, and 10% "free" cortisol (Sapsee, 1984). Furthermore, Sapsee (1984) has reported that a normal resting level of adult total cortisol has been measured at 12 ug/dl, with large increases in total cortisol after surgery (24-48 ug/dl) and infection (63-99 ug/dl). Increments in plasma cortisol have been found after exercise, however it appears that cortisol returns to pre-exercise levels after cessation of exercise (Kuipers and Keizer, 1988). It has been observed that following long lasting intense exercise, cortisol and catecholamine levels may decrease below normal levels for several days due to decreased catecholamine synthesis. Kuipers and Keizer (1988) have suggested that this decrease in catecholamine and cortisol levels results in a decrease in mobilization of the energy reserve, which consequently lowers physical work capacity. This decrease in work capacity may predispose the athlete to injury.

A study by Tsai, Johansson, Pousette, Tegelman, Carlstrom and Hemmingsson (1991) examined the effects of cortisol and androgen concentrations in elite endurance athletes. Nine elite orienteers

and seven cross-country skiers were assessed in the off-season, pre-season and competitive season to determine relative changes in hormonal concentrations throughout one season. Venous blood samples were taken during nocturnal fasting procedures the following morning. The researchers revealed that cortisol values increased significantly from the off to the competitive season, and that female mean concentrations were higher than that of males during the competitive season. Although there was no difference between the skiers or the orienteers, there was a difference in levels of concentration between gender. Furthermore, it was suggested that the ratio of free testosterone to cortisol may be effective in the early detection of overtraining. As a result, Tsai et al. (1991) stated that female endurance athletes adapt differently than male endurance athletes to endurance training. Consequently, female cortisol responses may be more sensitive to increases in training load than males. The endocrine system is highly sensitive to the physiological demands of exercise. The most important hormonal changes are increases in both catecholamines and cortisol. Unlike testosterone, these usually return to baseline levels with adequate rest. Monitoring these hormones throughout the training year provides an effective means in preventing overtraining.

## **2.5 PSYCHOLOGICAL AND BEHAVIORAL RESPONSES TO ENDURANCE TRAINING**

Various sources have identified changes in mood state, behavior, motivation to train, irritability, depression, insomnia, loss of libido, and loss of appetite as signs of overreaching and overtraining (Budget, 1990; Fry et al., 1992; Hooper, Mackinnon, Howard, Gordon and Bachmann, 1995; Morgan, Brown, Raglin, O'Connor and Ellickson, 1987; Sapse, 1984; Vos et al., 1992). Hormonal changes induced by prolonged training cause a variety of behavioral responses. Increased

catecholamines and cortisol affect the central nervous system, which may cause anxiety and depression to occur (Sapsee, 1984). The following examines states of fatigue, alterations in mood states and susceptibility to injury in illness as it relates to training and overtraining.

### **2.5.1 The Relationship of Fatigue to Lactate**

Some researchers have demonstrated the existence of a relationship between participation in intense exercise and self reports of fatigue and exertion. A study by Synder, Jeukendrup, Hesselink, Kuipers and Foster (1992), examined well trained cyclists over 6 weeks of training in normal, overtrained and recovery states. Blood lactate (HLac) analysis was made and compared to Borg's Rating of Perceived Exertion (RPE) scale in regular testing every 2 weeks. The results of the study demonstrated that the HLac:RPE ratio decreased throughout the overtrained period (29% in week 3, and 48% in week 4). Lactate concentration decreased with training, however, RPE values were maintained. In conclusion, Synder et al. (1992), suggested that the decline in the ratio was due to an overall decrease in the utilization of glycogen, although another possible explanation was the improvement in lactate clearance rates. In the recovery state, the HLac:RPE ratio was found to return to normal. This study provided evidence that perceived fatigue can serve as a good indicator of overreached states.

### **2.5.2 Alterations in Mood States**

The most highly recognized work in the monitoring of mood states has been done by Morgan et al. (1987), who examined mood states of varsity swimmers throughout a typical year with high volume training performed during the competitive season. This investigation utilized the Profile of

Mood States (POMS), which is a questionnaire composed of 65 items measuring various emotions. A complete score is determined by adding five negative mood states (tension, depression, anger, fatigue, and confusion), and subtracting the one positive mood state (vigor). Elite athletes and active individuals tend to score lower than the population average on the five negative mood states, and score higher than average on the one positive mood state (Morgan et al., 1987). Several studies were performed on the same population demonstrating a general mood disturbance in the mid-season which included peak training. These changes in mood were due to an increase in fatigue and a decrease in vigor. In summary, Morgan et al. (1987) indicated that alterations in fatigue and vigor generally precede more serious mood disturbances (depression or anxiety). The POMS also demonstrated an increase in anger and depression. Depression also seems to be related to overtraining, as previously cited, however, it is worthwhile to report that mood states did improve after titration of the training load.

A study by Hooper, Mackinnon, Gordon and Bachmann (1993) investigated hormonal changes throughout a 6 month training season in elite swimmers, leading up to peak performance for the World team trials. The aim of the project was to monitor signs and symptoms of overtraining throughout the season via hormonal and mood state changes. The athletes were evaluated via self report training logs and POMS (profile of mood states) questionnaires. Blood samples were collected at early (2-3 weeks), mid (12-14 weeks), late seasons-(5 -6 weeks), a taper period (3-5 days) and post competition (1-3 days). The results demonstrated that 3 of the 14 swimmers exhibited signs of overtraining, and that these three were female athletes. Moreover, fatigue ratings were higher in mid-season, tapering and post-competition periods. Cortisol and epinephrine levels throughout the whole season were not significantly different between the stale and well-trained swimmers.

However, norepinephrine levels were found to be greater for the stale compared to the well-trained swimmers during the taper period. Hooper et al. (1993) reported that the stale swimmers had actually maintained a higher volume of training than the well-trained swimmers. This was most likely due to their poor performance through the season, which motivated them to work harder in an attempt to improve performance. In conclusion, Hooper et al. (1993) reported significant findings in the incidence of negative mood states with corresponding increases in training load. As well, Morgan et al. (1987) also observed that these negative mood states disappeared with tapering and unloading of training volume. The changes in mood state were directly related to the corresponding changes in other physiological parameters (endocrine, cardiovascular etc.). Morgan et al. (1987) concluded that use of the POMS provides an effective means for evaluating individual training loads. In summary, ratings of fatigue and mood states may be associated with increases in training loads. The previous studies have illustrated that monitoring of training stress via psychological and behavioral changes are related to the corresponding physiological changes. Regular use of such measurements may enable the coach to assess training progression, and then implement the appropriate load changes.

### **2.5.3 Susceptibility to Illness and Injury**

Several authorities have indicated that the increase in the occurrence and incidence of injuries is both a sign and result of overtraining (Budget, 1990; Callister, Callister, Fleck and Dudley, 1990; Fry et al., 1992; Kibler, Chandler and Stracener, 1992). Physical activity places demands on the musculoskeletal system, which may result in mechanical trauma or injuries. Kibler et al. (1992) suggested that injuries due to overtraining may range from subclinical (as seen in decrements in

performance), to overt (an injury which inhibits performance). Newhouse (1984) also identified overuse injuries (ie. tendonitis, or mechanical musculoskeletal) as a possible outcome of overtraining. The mechanisms which facilitate these chain of events may lie in the large amounts of circulating cortisone. Cortisol acts as an anti-inflammatory and inhibits growth, therefore protein synthesis will be compromised. Training increases protein catabolism, and recovery promotes protein re-synthesis. Kuipers and Keizer (1988) reported that with incomplete recovery, premature fatigue of the motor unit pool will occur during exercise performance. Therefore, the individual will require an increase in nervous innervation to generate the comparable levels of force. This may place a greater demand on the muscular system than the individual can meet, thus resulting in an injury. In addition, other researchers suggested that the chronically fatigued athlete who has insufficient glycogen levels is more susceptible to injury. The greater energy costs and higher heart rates in fatigued athletes may decrease the economy of movement, and may produce mechanical errors in technique, thus predisposing the individual to injury (Mackinnon and Hooper, 1991).

Other investigators have reported that fatigued and overtrained athletes experience a higher incidence of colds, flues and infections, which take a longer time than usual to recover (Budget, 1990; Fry et al., 1992; Hooper et al., 1995; Newhouse, 1984; Vos et al., 1992). Cortisol has been identified as a powerful immuno-suppressor, thus lowering the immune resistance of the body (Sapse, 1984). It reduces the level of T-helper cells, and suppresses fibroblast growth. T-lymphocytes and fibroblasts are essential for the production of interferon, therefore there may be a causal relation between stress and diminished immune response to viral infections (Sapse, 1984). Selye (1956) demonstrated that during the alarm stage, there is atrophy of the lymphatic system. It is suggested that this may be the result of a diminished number of circulating lymphocytes in the

blood, which will impair resistance to viral infections. Budget (1990) confirmed this by reporting that following exercise, there is a release of white blood cells (WBC), causing a temporary leucocytosis. Following intense exercise, Budget (1990) has also observed a reduced T-helper cell:T-suppressor cell ratio, however, this did not correlate with temporary suppression of lymphocyte function. An alternative explanation for diminished immune response may be due to reduced plasma glutamine levels. Budget (1990) illustrated that plasma glutamine levels provide 35% of the necessary energy for lymphocyte metabolism, therefore if lymphocyte metabolism is inhibited, this may account for the immuno-suppression response. In either case, the exact explanation for the reduction in immune function is not exactly known. However, it does appear that prolonged exposure to exercise training without an adequate recovery period produces specific responses in the endocrine system. These changes influence the contribution of the lymphocytes and T-cells, which in turn alter their effectiveness in deterring the onset of infection or illness.

## 2.6 SUMMARY

Most elite training programs attempt to induce short term cycles of intensive training in order to maximize training adaptations. However, if not monitored regularly, these training sessions can predispose the athlete to states of overtraining. Several parameters can be used in the assessment and evaluation of training progression. It does appear that a multi-faceted approach is optimal in determining whether overtraining has taken place. Studies have demonstrated that several factors often determine subsequent ones. In other words, the biology of overtraining can be observed in physiological, psychological and performance evaluations. Regular testing and monitoring of training loads throughout the season is the only way to assess the success of the training program.

## **CHAPTER THREE**

### **METHODS AND PROCEDURES**

#### **3.1 Purpose**

The purpose of this investigation was to monitor and assess eight weeks of a training program for elite cross-country skiers in order to produce a comprehensive profile of the athletes' response to the training program (1993-94) established by Cross-Country Canada. This involved, firstly, an assessment of the effectiveness of the training program as measured by training responses over time on physiological, biochemical and performance variables. Secondly, the investigation of the relationship between the Overstress Monitoring Inventory to resting heart rate and resting blood pressure. Thirdly, an examination of the contrasting effects of physiological, biochemical and performance variables to a four and eight day period of reduced training.

#### **3.2 Subjects**

Ten (eight male; two female) elite cross-country skiers were examined in the present study. Eight were members of the national ski development team who were training in Thunder Bay, Ontario at the national development center. The remaining two subjects were provincial level racers, and had some experience of competing at the national level. The subjects age levels ranged from 19 to 27 years, and all had volunteered to participate in the study. Each athlete was training a minimum of 10 hours per week, with training volume sometimes reaching a maximum of 24 hours per week. The national development team athletes followed training programs established by Cross-Country Canada and were under the coaching direction of a national development center coach. The remaining two provincial athletes both had personal coaches

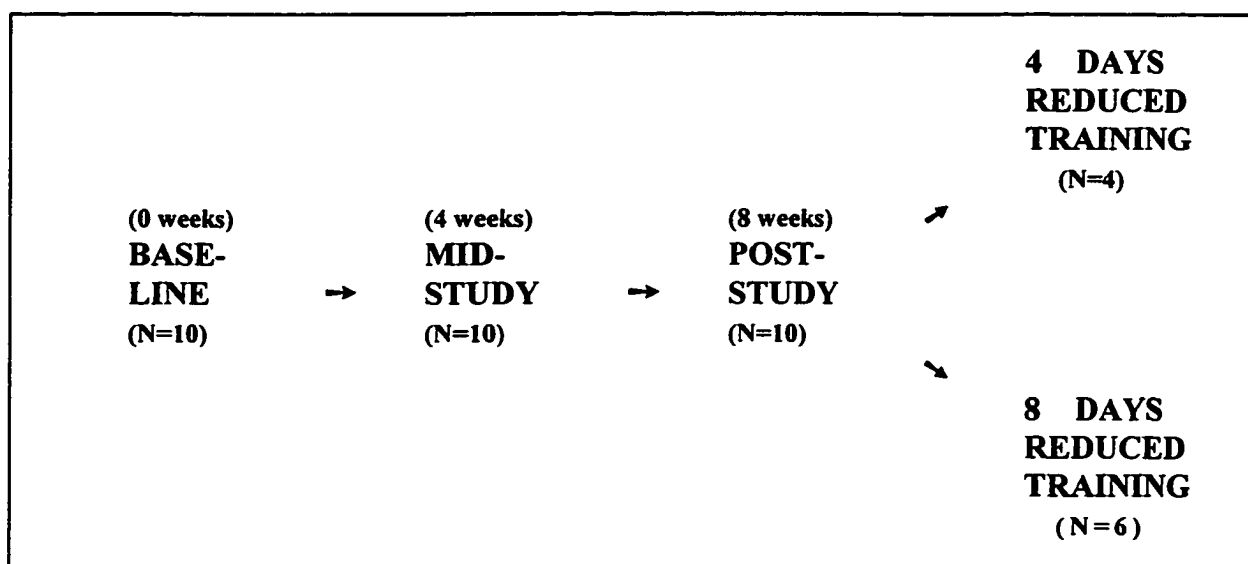


who followed the same program as that designed for the national development team.

### 3.3 Procedures

The present study examined eight weeks of both dry land and on snow training for national development and provincial level-cross-country skiers. Testing was initiated at the end of October 1993 with the final tests occurring in mid December 1993. The period of training under investigation was the "specific aerobic period", which consisted of a training intensity of 60-80% maximal heart rate. Subjects adjusted training intensity by including a minimum of one zone three and one zone four training session in their regime during this period which lasted from August to the start of the competitive season. This period of training was determined to be the phase with the highest volume of training load in the annual training plan. Therefore, this period of the training cycle provided the best profile of the demands required by skiers during training. The testing phase was divided into three cycles, the first two each consisting of four weeks of incremental increases in training volume. These phases were categorized by the volume and intensity of training, and were identified by the following: baseline (0 weeks), mid-study (4 weeks) and post-study (8 weeks). The final cycle consisted of a period of reduced training defined by a 50% reduction in training volume, while training primarily in zone one and two. Subjects were then divided into two groups; a four day and an eight day group, in order to compare the effects of the reduced training. Assignment to groups was determined by the individual subject's examination timetable (December 1993), which was provided by the university registrar. Subjects consented to participate in both laboratory and field tests throughout the nine weeks of the study. With the exception of the reduced training stage, lab tests were performed at four week increments. Lab tests during reduced training were performed

at the completion of either the four or eight days of reduced training. Testing was performed at the same time of day, and day of the week, for each athlete. Laboratory tests consisted of the determination of peak  $\text{VO}_2$ , lactate profiles determined from the incremental exercise protocol for  $\text{VO}_{2\text{max}}$ , heart rate values, blood analysis, and blood pressure. Evaluation of skiing performance consisted of utilizing field tests, which involved a timing and technique analysis. This was performed at both the start and completion of four and eight days of reduced training. Field tests used videotaping and watches to record the ski performance during a race around a 5 km loop.



**Figure 1:** Schematic representation of testing format throughout 8 weeks of training, and eight days of reduced training.

### 3.4 Anthropometric Measurements

Percent body fat was determined via a seven site skinfold analysis using John Bull skinfold calipers (British Indicators Inc., England). Weight, to the nearest 0.1 of a kilogram, was calculated using a Healthometre balance scale (Continental Scale Corp., Brideview, IL.). Height, to the nearest 0.1 of a centimeter, was measured with a tape measure mounted to the wall.

### 3.5 Physiological Measurements

Each athlete was responsible for taking and recording his/her daily morning resting heart rate. Data was collected by both the coach and the researcher at the conclusion of the training week. Resting blood pressure was monitored once weekly by a technician using both a stethoscope and sphygmomanometer. Three trials were used in determining resting blood pressure. Values were taken early morning (8:00 AM) on the same day of the week. All tests were done on the left arm. Data for both tests were collected for a total of six weeks, which alternated with the physiological tests.

Four tests were performed on each athlete at baseline (0 weeks), mid-study (4 weeks), post-study (8 weeks) and following reduced training (4 or 8 days). Maximal aerobic power was determined while subjects exercised to exhaustion on a treadmill (Quinton Instruments, model no. 1860; Seattle, Washington) with increasing grade. Gas analysis was performed with the assistance of a Beckman MMC Horizon system gas analyzer (Beckman Instruments Inc.; Anaheim, California). Expired gas was collected and analyzed for  $VO_2$  (both  $l\ min^{-1}$ , and  $ml\ kg^{-1}\ min^{-1}$ ),  $VECO_2$ ,  $VEO_2$ ,  $FECO_2$ ,  $FEO_2$ , RER and pulmonary mechanics (breathing frequency, ventilation, tidal volume). The testing protocol, for the determination of peak  $VO_2$  was that established by Cross-Country Canada. For the purposes of the study, peak  $VO_2$  is defined as the highest value obtained in the treadmill test. Male subjects were allowed a seven minute warm-up at 0% grade at 6.5 mph. Immediately following the warm-up, treadmill speed was increased to 8 mph for 2 minutes at 0% grade elevation. Workloads were then increased by a 2% grade elevation every 3 minutes thereafter until the subject could not maintain the workload. Female subjects were allowed a seven minute warm-up period at 5.5 mph at 0% grade. Immediately following the warm up, treadmill speed was increased to 7 mph at 0%

elevation. Workloads were then increased 2% elevation every 3 minutes until the subject could no longer continue to maintain running. Heart rates were monitored with the use of a wireless heart watch (Polar Vantage XL), and were recorded every minute for both male and female subjects during the treadmill test. Blood samples for lactate analysis were collected via fingertip aspirations using the Monolet Lancet gun (Ulster Scientific Inc.). Monolet sterile lancets (Sherwood Medical; St. Louis, MO) were mounted on the gun, and samples were taken during treadmill exercise to exhaustion. Blood was then collected with Red-Tips Heparinized micro-hematocrit capillary tubes, and immediately analyzed for lactate concentration with the use of the YSI Model 23L Lactate Analyzer (Yellow Springs Instrument Company; Seattle, Washington). Calibration was made with the use of YSI L-lactate 5.0 mmol/L and 15.0 mmol/L standards. Resting blood samples were collected prior to the exercise test. Samples were then collected at the conclusion of every workload. A final sample was obtained immediately following cessation of running when the subject could no longer maintain exercise. Recovery lactate samples were then taken at 1, 2 and 3 minutes post exercise.

Determination of lactate threshold was achieved using the method reported by Stegmann, Kindermann, and Schnabel (1982). This involved the plotting of lactate values versus workload, and establishing a curve of best fit to obtain a sigmoidal curve. Lactate threshold was then determined by observing at which point on the curve the rate of lactate production equals the rate of lactate elimination. Lactate threshold was expressed as a percent of peak  $\text{VO}_2$  to observe if any relative changes in performance had occurred. Similarly, OBLA (Onset of Blood Lactate Accumulation) was also expressed as a percent of peak  $\text{VO}_2$ .

### **3.6 Biochemical Measurements**

Blood was collected at four different times throughout the study; at baseline (0 weeks), mid test (4 weeks), post test (8 weeks) and following reduced training (4 or 8 days). Sampling was performed at 8:00 AM the same day of the week throughout the duration of the investigation. Furthermore, blood samples were collected by the same technician for each subject. All samples were collected at the Human Performance Laboratory at Lakehead University. Samples were analyzed for white blood cell count, red blood cell count, hematocrit, hemoglobin, serum ferritin, serum urea, creatine phosphokinase, lactate dehydrogenase, testosterone, and cortisol concentrations. Sampling procedures required subjects to remain seated quietly for 10 minutes prior to sampling. A 25 ml sample was then collected from an ante-cubital vein by use of a syringe-vacutainer system. A portion of this blood was collected in 2.5 ml vacutainers containing ethylenediaminetetraacetic acid (EDTA) for analysis of hematocrit, hemoglobin, white blood cell count and red blood cell count using the Cell-Dyne 3000. Serum was obtained by allowing the blood to stand on ice for 2 hours, and was centrifuged at 3000g for 10 minutes in a refrigerated system. A 10 ml sample was collected for the analysis of serum urea, creatine phosphokinase and lactate dehydrogenase using a Kodak Ektachem 700. Serum ferritin was determined via IMX Ferritin Radioimmunoassay (Abbott Laboratories Diagnostics Division, Abbott Park, IL). Use of a local diagnostic lab, Medical Diagnostic Services (MDS), was employed for the determination of cortisol and testosterone concentration. Cortisol serum was prepared by Gammacoat (Clinical Assays; Dade; Cambridge, MA) radioimmunoassay. Serum testosterone was prepared with RSL (Radioassay System Laboratory; Carson, CA) radioimmunoassay. Both serums were then counted on a Gammacounter to determine relative serum levels. Analysis of serum testosterone was restricted to male subjects

only. Due to the combined male and female subjects, cortisol and testosterone were examined separately, and not as a ratio value.

### **3.7 Psychological Measurement**

This involved the use of a self report twenty-nine item questionnaire (see appendix for sample), which is currently used by Cross-country Canada to monitor and predict signs and symptoms of overtraining and fatigue. Coaches use the inventory sheets in combination with self-reports of resting heart rate to monitor athlete levels of fatigue and stress. When scores and/or resting heart rate levels demonstrated an increase without returning to normal levels, the coach would reduce training requirements accordingly in an attempt to return scores and resting heart rate values to normal (baseline) levels. The questionnaire was designed to monitor changes in mood states, sleep and eating patterns, motivation, muscular fatigue and interpersonal relationships. It consisted of three categories (from lowest to highest) asking the recipient to identify with one of the three qualifications, and consists of a total of twenty-nine questions overall. In addition, there is a log section which allows the athlete to provide feedback as to the previous week's training. The athlete was then required to hand in the completed questionnaire along with a training log summary to the coach each week. Coaches have a template to mark and record scores from the questionnaire. Summaries from the completed questionnaire were collected from each athlete and examined by the coach their individual response to training. Evaluation of the inventory consisted of score summaries from four categories; medical, psychological and other. Each category was then summed to produce a total inventory score for that week. Inventory sheets were then collected at the conclusion of weeks 1,2,3,5,6 and 7 and compared to scores of both resting heart rate and resting systolic blood pressure

in an attempt to determine if subjective inventory scores demonstrated any pattern of relationship to objective physiological measurements. The selection of these variables (resting heart rate and resting systolic blood pressure) was based on the premise that coaches regularly use these methods to monitor training levels in cross-country skiers.

### **3.8 Performance Evaluation**

A race simulation test, which consisted of both a mini timing study and technique analysis, was conducted. The purpose of the race simulation test was to contrast and compare the effects of sport performance to a 4 day and 8 day period of reduced training. There were two race simulation tests conducted at the Lappe Nordic Center, in Thunder Bay, Ontario. The first test was at the completion of 8 weeks of training, and the second test was either 4 or 8 days following this period of training. The skiers were required to ski two laps of a 2.5 km loop as fast as possible using cross-country free techniques. Each subject was individually started and timed. All testing was performed at the same time of day, and subjects were required to use the same wax for each test. Subjects were required to use the same equipment on both test days in an attempt to isolate the effect of equipment on the test results. Snow conditions were recorded, and trails were groomed prior to each test.

The timing study involved obtaining lap times in order to assess whether or not time changes between lap 1 and lap 2 could be related to the period of reduced training, and to determine whether a difference existed between groups. The lap times were expressed as a percent of the total time in order to compare times between different days. Timers used digital split second stopwatches. Technique analysis examined skiers using off-set techniques skiing up a 5% grade hill of 30 meters in length. Analysis involved examining one complete cycle from pole plant to pole plant in order

to determine cycle characteristics. Technicians videotaped all athletes performing the off-set technique during each lap. The Peak 2D Analysis System was used to digitize the taped records of each skier on each lap. The X and Y spatial co-ordinates for a kinematic model were generated in order to compute measures for cycle rate, cycle length and cycle velocity. Further kinematic analysis of the off-set technique was not completed for this investigation. Cycle characteristics were then expressed as a percent change between lap one and two. Data was then compared between different days to determine if any differences were observed within groups (either following 4 or 8 days of reduced training) or between groups (contrasting 4 days to 8 days of reduced training).

### 3.9 Statistical Procedures

Descriptive statistics (mean and standard deviation) of physiological, biochemical, psychological, and performance variables are presented.

The Pearson product moment correlation was utilized to examine the relationships between the psychological inventory, resting heart rate and resting blood pressure on data collected during weeks 1,2,3,5,6 and 7 of the training program. A test of significance for each correlation employed an alpha level of  $p < .05$ .

Both physiological [ peak  $VO_2$ ,  $VO_{2max}$  (at TLac), percent  $VO_{2max}$  (at both OBLA and TLac), lactate profiles and lactate threshold, treadmill time to exhaustion, sub-maximal and maximal heart rates ], and biochemical (white and red blood cell count, hematocrit, hemoglobin, serum ferritin, serum urea, creatine phosphokinase, lactate dehydrogenase, cortisol and testosterone) data was subjected to a repeated measures one-way Analysis of Variance (ANOVA) to determine if any variations existed between baseline (0 weeks), mid-study (4 weeks) and post-study (8 weeks). A



Dunn's test (Bonferroni *t*) has been reported to be an acceptable method of controlling the influence of familywise error rate when using linear contrasts by establishing a more conservative alpha level for each comparison (Howell, 1987). As a result, *a-priori* comparisons between group means were performed utilizing Dunn's test (Bonferroni *t*) at an alpha level of  $p < .05$  when appropriate.

A 2 X 2 between groups Analysis of Variance (ANOVA) was used to observe any differences between post-study, 4 days and 8 days of reduced training for physiological, biochemical and performance (percent total time for lap one and two; percent change in cycle rate, cycle length, and cycle velocity between lap one and two; time to complete lap one and lap two) parameters. *A-priori* comparisons between group means were performed utilizing Dunn's test (Bonferroni *t*) at an alpha level of  $p < .05$  when appropriate. The assumption of homogeneity of variance was examined with the use a Greenhouse-Geisser test when appropriate.

All data was analyzed using Statistical Packages for the Social Sciences (SPSS) version 4.0 for personal computers. Calculation of lactate threshold was determined using the method reported by Stegmann, Kindermann and Schnabel (1982). Graphs constructed for the calculation of lactate threshold employed a polynomial curve of best fit available through the Deltagraph software package.

## CHAPTER FOUR

### RESULTS

Ten elite cross-country skiers participated in this study. Their mean age, height and weight is presented in Table 1.

**Table 1** Descriptive statistics of anthropometric measurements on ten elite cross-country skiers.

<b>Variable (N=10)</b>	<b>Mean</b>	<b>± S.D.</b>	<b>Minimum</b>	<b>Maximum</b>
Age (yrs)	21.0	± 2.61	19.0	27.0
Height (cm)	175.1	± 7.46	162.0	187.5
Weight (kg)	70.4	± 8.41	64.0	82.3

The investigation period was eight weeks and 8 days in duration. Physiological and biochemical measurements were taken at four separate stages during the training: baseline (0 weeks), mid-study (4 weeks), post-study (8 weeks), and following 4 and 8 days of reduced training. Resting systolic blood pressure, resting heart rate and scores from the overstress monitoring inventory were collected during weeks 1,2,3,5,6,7. A performance analysis was obtained at post-study and following 4 and 8 days of reduced training. Tables 7, 8 and 9 (see appendices) illustrate the mean score and standard deviation values for: peak  $VO_2$  ( $PVO_2$ ),  $VO_2$  at lactate threshold ( $TVO_2$ ), percent  $VO_2$  at lactate threshold ( $\%VO_2$  TLac), percent  $VO_2$  at OBLA ( $\%VO_2$  OBLA), peak lactate (PLac), lactate threshold (TLac), recovery lactate (post-exercise 1,2 and 3 minutes), maximal heart rate (MHR), heart rate at lactate threshold (THR), and treadmill time to exhaustion (TIME).

#### 4.1 Physiological Measurements

Both mean (N=10)  $VO_2$  (peak and at lactate threshold) values decreased (non-significantly) approximately by 7.2 % and 2.6% respectively throughout eight weeks of training (see figure 2). However, mean values (N=10) of percent  $VO_2$  at lactate threshold and at OBLA demonstrated inverse trends over eight weeks of training. Lactate threshold increased while OBLA decreased (both non-significantly) as a percent of  $VO_2$  following eight weeks of training (see figure 3).

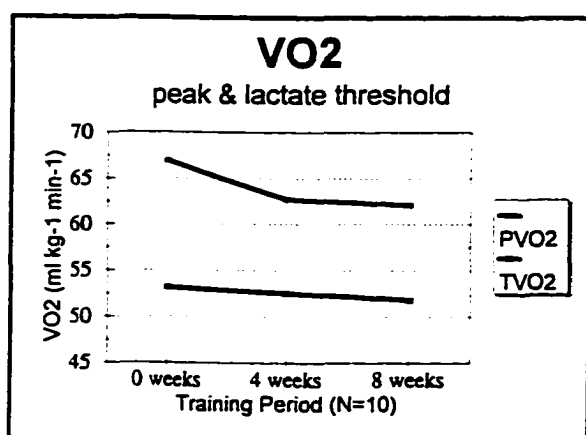


Figure 2: Mean  $VO_2$  (peak & at lactate threshold) values of elite cross-country skiers through-out 8 weeks of training.

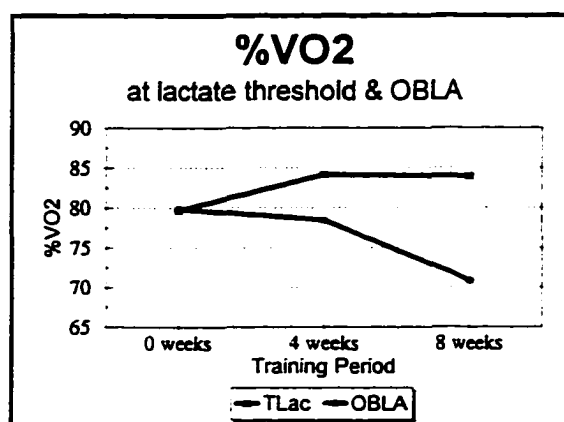
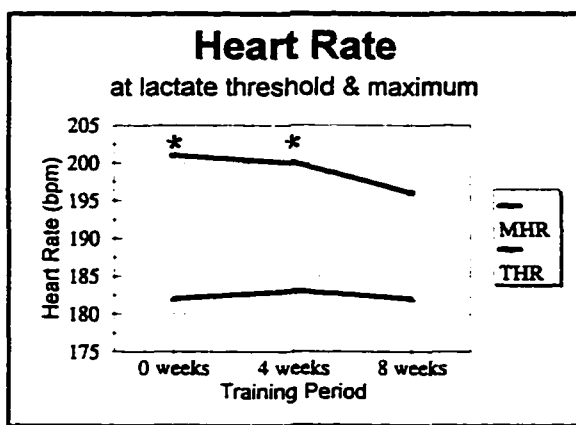


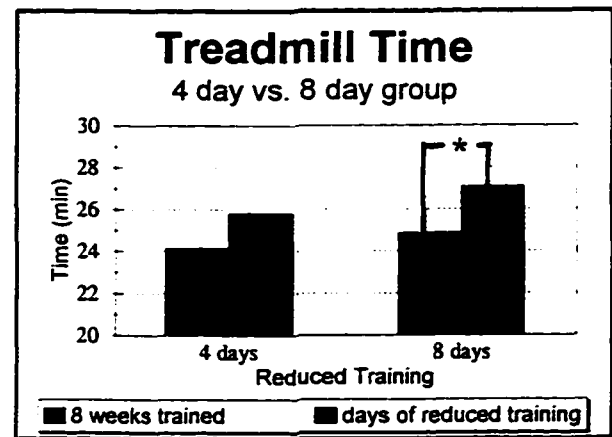
Figure 3: Mean  $VO_2$  (at lactate threshold & OBLA) values of elite cross-country skiers throughout 8 weeks of training.

A one-way repeated measures ANOVA of maximal heart rate demonstrated a main effect for time [  $F(2,18) = 7.01, p = .006$  ] as illustrated in Table 10 (see appendices). Figure 4 demonstrates that *a-priori* comparison (table 39) confirmed that mean maximal heart rate values in both baseline (201 bpm) and mid-study (200 bpm) phases were significantly higher ( $p < .05$ ) when compared to the post-study (196 bpm) phase. However, no significant differences were observed in heart rate at lactate threshold throughout 8 weeks of training. A 2 X 2 between groups ANOVA was performed on treadmill time to exhaustion. Figure 5 illustrates that a main effect for time [  $F(1,8) = 7.39,$

$p = .026$  ] for treadmill time to exhaustion did occur (see table 11 in appendices). *A-priori* comparison (table 39) confirmed that mean treadmill time to exhaustion significantly increased ( $p < .05$ ) from 24:53 (min:sec) to 27:06 (min:sec) following 8 days of reduced training. However, a Greenhouse-Geisser (epsilon=.56354) and corresponding Chi-square [ $\chi^2_{(2)} = 11.91$ ;  $p = .003$ ] test indicated that the assumption of homogeneity of variance was violated.



**Figure 4:** Mean maximal heart rate values of elite cross-country skiers throughout 8 weeks of training (\*  $p < .05$ ).



**Figure 5:** Mean treadmill time to exhaustion in elite cross-country skiers throughout 8 weeks following 4 & 8 days of reduced training (\*  $p < .05$ ).

Heart rate at the lactate threshold (HRT) demonstrated an interaction (group by time effect) [ $F(1,8) = 5.54$ ,  $p = .046$ ] when analyzed by a 2 X 2 between groups ANOVA (see table 11). However, *a-priori* comparison approached but failed to reach significance at the  $p < .05$  criterion in identifying differences between groups (4 day or 8 day), or within subjects (following both 4 and 8 days of training). A Greenhouse-Geisser (epsilon=.80560) and corresponding Chi-square [ $\chi^2_{(2)} = 2.2093$ ;  $p = .331$ ] test demonstrated that the assumption of homogeneity of variance was not violated. Mean values ( $N=4$ ) for peak  $VO_2$ , threshold  $VO_2$ , % $VO_2$  (at lactate threshold) and % $VO_2$  (at OBLA)

following 4 days of reduced training (see table 9.0) were unavailable for analysis due to unrelated technical difficulties with the laboratory equipment.

## 4.2 Biochemical Measurements

Both white blood cell count (WBC) and red blood cell count (RBC) (see table 2.0) demonstrated inverse trends with WBC increasing approximately 5.1% and RBC decreasing approximately 10.4% throughout 8 weeks of training. Both hematocrit (Hct) and hemoglobin (Hgb) increased from baseline (0 weeks) to post-study (8 weeks). Tables 3.0 and 4.0 demonstrate both mean and standard deviation for white blood cell (WBC), red blood cell (RBC), hematocrit (Hct), hemoglobin (Hgb), serum urea, serum ferritin, cortisol and testosterone following both 4 and 8 days of reduced training.

Figure 6 illustrates that a one-way repeated measures ANOVA revealed a main effect for time for mean (N=10) hemoglobin concentration following 8 weeks of training [  $F(2,18) = 8.92, p = .002$  ] (see table 10). *A-priori* comparison demonstrated that mean hemoglobin levels significantly increased ( $p < .01$ ) from baseline (148 g/L) to both mid-study (153 g/L) and post-study (155 g/L) following 4 and 8 weeks of training, respectively. Figure 7 profiles mean (N=10) concentrations of threshold (TLac), peak (Plac) and recovery [ (Rec) at 1, 2 and 3 minutes post-exercise] lactate values throughout 8 weeks of training. Overall, lactate threshold increased while peak lactate decreased from baseline (0 weeks) to post-study (8 weeks). Figure 8 illustrates the trend in mean cortisol concentration throughout 8 weeks of training. A one-way repeated measures ANOVA approached but failed to demonstrate a true main effect for time for cortisol [  $F(2,18) = 3.52, p = .051$  ] (see Table 10). No significant differences were observed at the  $p < .05$  criterion between mean values at baseline (609.1 nmol/L), mid-study (721.1 nmol/L) and post-study (727.2 nmol/L).

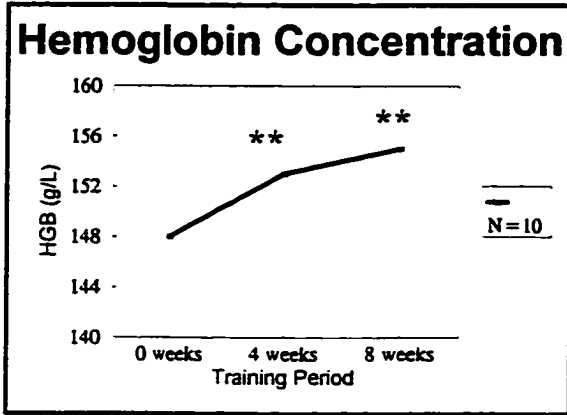


Figure 6: Mean hemoglobin values of elite cross-country skiers throughout 8 weeks of training ( \*\* p < .01).

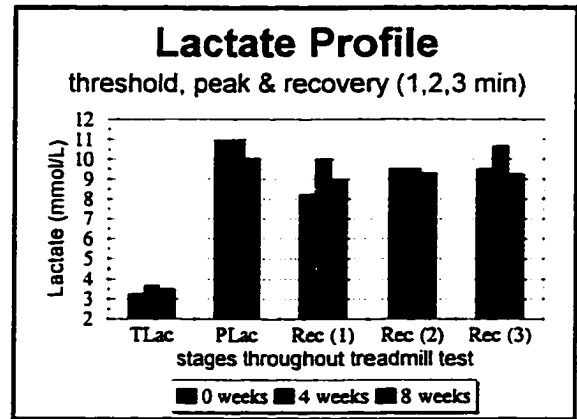


Figure 7: Mean concentrations of threshold, peak & recovery lactate values of elite cross-country skiers throughout 8 weeks of training.

A one-way repeated measures ANOVA demonstrated a main effect for time for CPK during 8 weeks of training [  $F(2,18) = 11.65, p = .001$  ] as revealed in Table 10. Figure 9 illustrates that *a-priori* comparison (table 39) determined that there was a significant decrease ( $p < .05$ ) in mean CPK concentration from baseline (223.0 U/L) to mid-study (159.9 U/L) following 4 weeks of training.

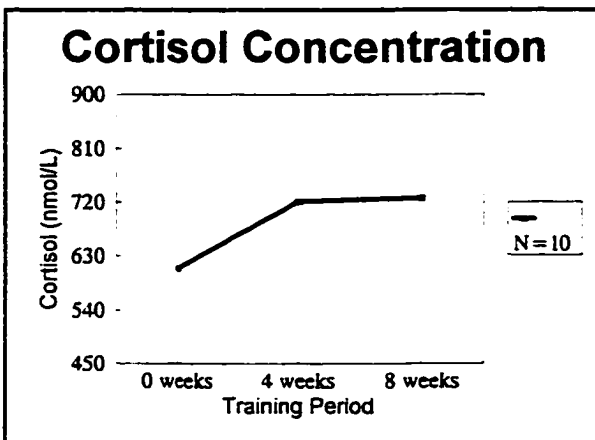


Figure 8: Mean cortisol values of elite cross-country skiers throughout 8 weeks of training.

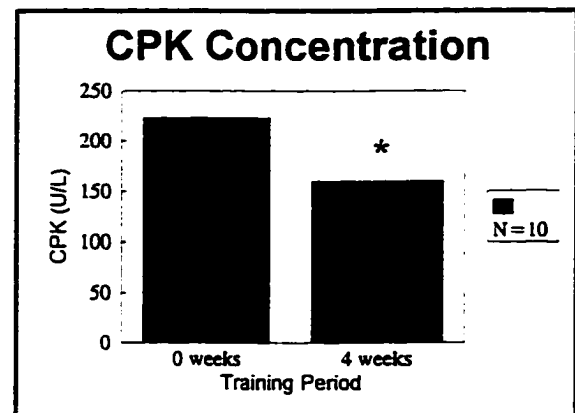
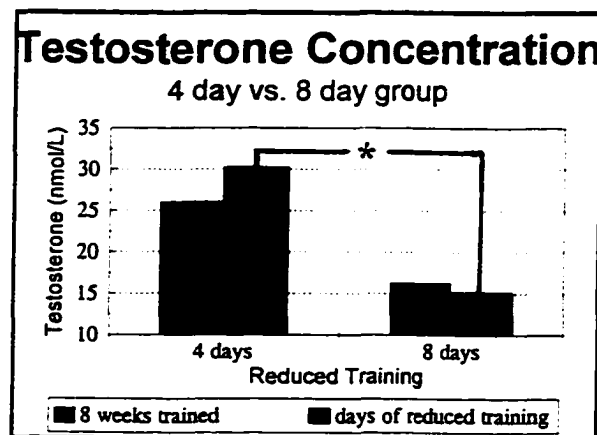


Figure 9: Mean CPK concentration of elite cross-country skiers throughout 4 weeks of training (\* p < .05).

Mean values for creatine phosphokinase (CPK), serum ferritin (Ferritin), serum urea (Urea), and lactate dehydrogenase (LDH) following 8 weeks of training were unavailable for analysis due to unrelated technical difficulties (see tables 2.0. and 4.0). As a result, analyses comparing all 3 periods (baseline, mid-study and post-study) were not completed for these variables.

A 2X2 between groups ANOVA demonstrated that a main effect for group [  $F(1,6) = 16.73$ ,  $p = .006$  ] for testosterone did exist (see table 11). Figure 10 illustrates that *a-priori* comparison (table 39) identified a significant difference ( $p < .05$ ) in mean testosterone values between groups following 4 days (30.3 mmol/L) and 8 days (15.2 mmol/L) of reduced training. However, a Greenhouse-Geisser (epsilon=.58363) and corresponding Chi-square [  $\chi^2_{(2)} = 7.498$ ;  $p = .024$  ] test indicated that the assumption of homogeneity of variance was violated.



**Figure 10:** Mean testosterone values of elite cross-country skiers following 4 days (N=3) and 8 days (N=5) of reduced training ( \*  $p < .05$ ).

**Table 2.0** Descriptive statistics of: white blood cell (WBC), red blood cell (RBC), hematocrit (HCT), hemoglobin (HGB), serum urea, serum ferritin, cortisol and testosterone (males only, N=8).

<b>Variable (N=10)</b>	<b>0 weeks X ± S.D.</b>	<b>4 weeks X ± S.D.</b>	<b>8 weeks X S.D.</b>
WBC (10e9/L)	5.39 ± 0.91	5.24 ± 1.31	5.68 ± 0.99
RBC (10e12/L)	5.12 ± 0.34	5.08 ± 0.29	4.59 ± 1.48
HGB (g/L)	148 ± 8.40	<b>** 153</b> ± 8.92	<b>** 155</b> ± 11.32
HCT (L/L)	0.442 ± 0.02	0.448 ± 0.021	0.447 ± 0.031
Ferritin (ug/L)	38.8 ± 18.4	35.2 ± 17.5	N/A
Urea (mmol/L)	5.99 ± 1.47	6.19 ± 1.45	N/A
CPK (U/L)	223.0 ± 112.2	<b>* 159.9</b> ± 98.9	N/A
LDH (U/L)	479.0 ± 79.9	489.3 ± 83.3	N/A
Cortisol (nmol/L)	609.1 ± 147.4	721.1 ± 284.3	727.2 ± 241.1
Testost. (nmol/L)	19.6 ± 6.48	22.0 ± 8.10	20.0 ± 5.60

n/a: Missing samples for 4 subjects..

\* : p < .05

\*\* p < .01



**Table 3.0** Descriptive statistics of: white blood cell (WBC), red blood cell (RBC), hematocrit (HCT), hemoglobin (HGB), serum urea, serum ferritin, cortisol and testosterone (males only, N=5) in 8 day (N=6) recovery group.

<b>Variable (N=6)</b>	<b>8 WKS: TRAIN X ± S.D.</b>	<b>8D: REDUCED TRAINING X ± S.D.</b>
WBC (10e9/L)	5.45 ± 0.79	6.33 ± 2.33
RBC (10e12/L)	4.99 ± 0.38	5.22 ± 0.37
HGB (g/L)	152 ± 10.85	156 ± 8.31
HCT (L/L)	0.438 ± 0.031	0.453 ± 0.025
Ferritin (ug/L)	35.4 ± 10.5	35.9 ± 15.4
Urea (mmol/L)	5.28 ± 1.41	5.36 ± 1.29
CPK (U/L)	101.3 ± 30.14	121.6 ± 76.6
LDH (U/L)	389.1 ± 49.9	415.8 ± 105.0
Cortisol (nmol/L)	660.3 ± 151.4	511.6 ± 120.5
Testosterone (nmol/L)	16.3 ± 2.87	* 15.2 ± 7.16

\* : p < .05

**Table 4.0** Descriptive statistics of: white blood cell (WBC), red blood cell (RBC), hematocrit (HCT), hemoglobin (HGB), serum urea, serum ferritin, cortisol and testosterone (males only, N=3) in 4 day (N=4) recovery group.

<b>Variable (N=4)</b>	<b>8 WKS: TRAIN X ± S.D.</b>	<b>4D: REDUCED TRAINING X ± S.D.</b>
WBC (10e9/L)	6.02 ± 1.29	5.55 ± 0.89
RBC (10e12/L)	3.99 ± 2.35	5.17 ± 0.34
HGB (g/L)	160 ± 11.97	157 ± 11.14
HCT (L/L)	0.462 ± 0.030	0.470 ± 0.034
Ferritin (ug/L)	n/a	38.9 ± 22.3
Urea (mmol/L)	n/a	5.32 ± 1.37
CPK (U/L)	n/a	171.5 ± 136.2
LDH (U/L)	n/a	415.0 ± 89.5
Cortisol (nmol/L)	827.5 ± 337.5	718.5 ± 102.7
Testosterone (nmol/L)	26.1 ± 1.85	* 30.3 ± 3.60

n/a: Missing samples for 4 subjects.

\* : p < .05

### 4.3 Psychological Measurements

Table 5 presents the mean score and standard deviation for systolic blood pressure (SBP), resting heart rate (RHR), and scores from the Overstress Monitoring Inventory (OMI). Pearson product moment correlation coefficients were performed on OMI, SBP and RHR. However, analyses failed to demonstrate any significant relationships between systolic blood pressure, resting heart rate and inventory scores throughout seven weeks of training.

**Table 5.0** Descriptive statistics of: resting systolic blood pressure (SBP), resting heart rate (RHR) total score from Overstress Monitoring Inventory (OMI) for 7 weeks of training (N=10).

<b>Var.</b>	<b>Week 1 X ± SD</b>	<b>Week 2 X ± SD</b>	<b>Week 3 X ± SD</b>	<b>Week 5 X ± SD</b>	<b>Week 6 X ± SD</b>	<b>Week 7 X ± SD</b>
SBP mm/Hg	113 ± 6.07	117 ± 7.83	118 ± 5.67	115 ± 6.27	115 ± 7.43	112 ± 9.48
RHR bpm	47 ± 7.43	48 ± 6.78	47 ± 6.01	48 ± 7.24	45 ± 4.50	49 ± 5.07
OMI	44.6 ± 22.5	40.0 ± 18.9	38.8 ± 9.32	45.9 ± 17.4	47.5 ± 16.4	45.8 ± 17.1

### 4.4 Performance Measurements

Table 6 illustrates the mean and standard deviation scores for percent total time lap 1 and 2 (%time), percent change for cycle rate (c.rate), percent change for cycle length (c.length), percent change for cycle velocity (c.vel.), time lap 1, time lap 2 and total time for lap 1 and 2. A 2 X 2

between groups ANOVA failed to identify any significant differences at the  $p < .05$  criterion in any of the kinematic parameters following either 4 days or 8 days of reduced training.

**Table 6.0** Descriptive statistics of kinematic variables: percent total time for laps (%Time), mean time for lap 1, mean time for lap 2, total mean time; percent change in time for: cycle rate (%Change C. Rate), cycle length (% Change C. length) and cycle velocity (%Change C. Vel.) comparing post-study ( 8 weeks) to both 4 days and 8 days of reduced training.

Variable	4 DAYS REDUCED TRAINING GROUP (N=4)		8 DAYS REDUCED TRAINING GROUP (N=6)	
	8 weeks X ± S.D.	4 days X ± S.D.	8 weeks X ± S.D.	8 days X ± S.D.
%Time Lap 1	48.32 ± 1.15	49.50 ± 0.46	49.06 ± 0.39	48.85 ± 0.93
%Time Lap 2	51.68 ± 0.46	50.50 ± 0.46	50.94 ± 0.93	51.15 ± 0.93
Total Lap 1	7:01 ± 0.34	6:49 ± 0.21	7:01 ± 0.27	7:35 ± 0.53
Total Lap 2	7:06 ± 0.28	6:54 ± 0.24	7:15 ± 0.26	7:58 ± 0.35
Total Lap Time	14:07 ± 1.13	13:43 ± 1.28	14:16 ± 1.02	15.31 ± 1.21
%Change C.Rate	4.01 ± 0.42	2.51 ± 0.02	7.26 ± 0.89	6.20 ± 0.50
%Change C.Length	5.55 ± 0.34	7.65 ± 0.83	4.00 ± 0.24	20.35 ± 2.02
%Change C.Vel.	3.09 ± 0.63	8.17 ± 1.09	7.86 ± 0.93	21.58 ± 1.93

N.B. : Lap times are represented as min:sec.

## CHAPTER FIVE

### DISCUSSION

This investigation monitored the training of (N=10) elite cross-country skiers over an eight week period, and compared the effects of a four and eight day period of reduced training at the completion of the training period.

#### 5.1 Hemoglobin and Hematocrit Concentration

Blood measurements demonstrated changes from baseline values throughout the course of training and during the reduced training phase. Mean hemoglobin (Hgb) levels increased significantly ( $p < .01$ ) 3.5% and 4.5% from 148 (0 weeks) to 153 (4 weeks) and 155 (8 weeks) g/L throughout the training period respectively. However, Hgb values were well within the normal ranges for adult males (135-165 g/L) and females (115-160 g/L), and are similar to values observed in triathlon athletes, cross-country skiers, and middle and long distance runners (Davidson, Robertson and Maughan, 1986; Haymes and Dickinson, 1982; Lehmann et al., 1991; Pattini and Schena, 1990). As Hgb levels directly affect performance through oxygen binding to the red blood cells for transport within the body, it has been shown that levels decline in response to both excessive training and detraining. Ross and Attwood (1984) reported a 13% decrease in Hgb levels from  $15.9 \pm 0.7$  to  $13.5 \pm 0.7$  g/dl following 2 weeks of intensive mountain climbing. Meanwhile, Neuffer (1989), reported a decrease of approximately 3.5% (30 g/L) following 2-4 weeks of detraining. A decrease in Hgb of this magnitude can produce a 6.0% reduction in  $VO_{2max}$ , and as a result can effect performance in endurance activities (Neuffer, 1989).

Decrements in Hgb concentration following exercise are attributed to an increase in plasma

volume as a result of hemo-dilution (Mackinnon and Hopper, 1991). Pattini and Schena (1990) have suggested that endurance athletes and cross-country skiers are more prone to lower levels of stored iron and may develop anemia more often as a consequence of intensive training. An increase in Hgb concentration during training, however, would indicate that iron stores are being replenished.

In the present investigation, Hct remained fairly consistent (44%) throughout eight weeks of training. As a result, the likelihood of Hgb increasing as a result of plasma volume shifts alone is unwarranted. Subjects demonstrated a significant increase in Hgb concentration over time in conjunction with fairly stable Hct levels, and consequently demonstrated a positive response to the training load. As such, using hemoglobin and hematocrit profiles are only effective in monitoring training adaptation when combined with other relevant variables. Moreover, the response of these blood parameters may also suggest that the subjects were able to adequately recover from the training sessions.

## **5.2 Creatine phosphokinase Concentration**

Creatine phosphokinase (CPK) demonstrated significant ( $p < .05$ ) decreases of 28.3% from 223.0 to 159.9 U/L following 4 weeks of training (refer to figure 9) and a subsequent 16.7% increase following 8 days of reduced training (see table 3.0). The values are well within typical values (35-230 U/L) for adult males and females, and are similar to those reported by Koutedakis, Raafat, Sharp, Rosmarin, Beard and Robbins (1993) for untrained and Olympic rowers; and for middle and long distance runners (Lehmann et al., 1991).

It has been documented that enzymes CPK and LDH (lactate dehydrogenase) both increase in concentration following a period of endurance training (Astrand and Rodahl, 1986). These enzymes,

located within the muscle cell, may leak into the blood as a result of damage to the muscle cell membrane following a bout of intense exercise (Koutedakis et al., 1993). Mackinnon and Hooper (1991) indicated that measuring blood levels of CPK and LDH may provide an indirect means of monitoring the response to training as the molecular structural damage will precede other chronic more debilitating effects of prolonged training. Thus, monitoring the CPK profile in response to intense exercise may indicate that overtraining is imminent, and intervention (rest) will be required until levels return to normal.

Athletes in the present investigation revealed that the mean CPK values decreased significantly ( $p < .05$ ) following 4 weeks of training, and supports a positive response to the training stimulus as evidenced by the likelihood of reduced cellular damage in response to the stress of exercise. The increase in Hgb and stable Hct levels observed following 4 weeks of training further supports an improvement in physical fitness. In contrast, it has been suggested that detraining results in a non-linear decrease in mitochondrial and glycolytic enzyme activity. This decrease has been shown to be more closely associated with a decrease in sub-maximal performance rather than being causally linked to decrements in  $VO_{2max}$  (Neufer, 1989). CPK levels have been demonstrated to be sensitive to both the short term (< 24 hours) and long term (over several weeks) effects of training. Moreover, post-exercise levels have been reported to remain elevated anywhere from 2 to 6 days following prolonged exercise (Koutedakis et al., 1993). However, Fry et al. (1992) reported a decrease in mean CPK levels following a period of reduced training.

In the present study, the subjects did not experience a drop in mean CPK concentration following an 8 day period of reduced training. The 8 days of reduced training should have been sufficient in reducing CPK levels. Perhaps the intensity and/or volume of exercise performed during the period

of reduced training was too stressful to allow complete recovery. Furthermore, the 8 days of reduced training could have resulted in some detraining subsequently causing an increase in CPK concentration. However, Houmard (1991) reported that detraining only starts to occur following 7 to 10 days of complete inactivity, which was not the case in the present investigation. Although unlikely, a potential explanation for the present findings is that the samples (CPK) may have been elevated as a consequence of the previous days training session. Fry et al. (1992) and Koutedakis et al. (1993) have reported that CPK levels can remain elevated 24 hours post-exercise. This may partially explain the increase in enzyme concentration following recovery as the samples in the present study were collected the morning following a bout of low intensity exercise.

### **5.3 Cortisol Concentration**

Mean cortisol levels demonstrated a 15.5 % and 16.3% increase following 4 and 8 weeks of training, respectively (refer to figure 8). The 4 day group (reduced training) demonstrated higher mean levels than the 8 day group (reduced training) following 8 weeks of training. However, during the 4 and 8 days of reduced training, mean cortisol levels declined 13.17% (from 827.5 to 718.5 nmol/L) and 22.52% (from 660.3 to 511.6 nmol/L), respectively. These values are higher than those reported by Hooper et al. (1993) for elite swimmers (489.2 nmol/L) following 3 to 5 days of reduced training, but fall within normal ranges for adult males and females (170-720 nmol/L).

Cortisol is a hormone which is extremely sensitive to stress, illness and injury. Moreover, elevated cortisol measurements obtained during training have been associated with periods of overtraining and prolonged fatigue (Hooper et al., 1995; Stray-Gundersen, 1990). As a consequence, cortisol measurements have been used as a means to monitor training effects in endurance athletes



(Kuipers and Keizer, 1988; Sapse, 1984). The difference in cortisol concentration between groups in the present study may have been due to the duration of the reduced period of training. Kuipers and Keizer (1988) reported that high cortisol levels return to normal (baseline) values following a period rest ranging from 2 to 7 days. Consequently, in the present study it may have been possible that 8 days of reduced training was more effective in lowering mean cortisol levels than the 4 days of reduced training.

Cortisol measurements have been observed to demonstrate a diurnal variation (Hooper et al., 1993). However, in the present investigation, all samples were taken early morning prior to training or everyday activity. It is possible, however, that these values could have been elevated as a consequence of the training done 12 to 24 hours prior to sampling. This may explain why cortisol levels increased in concentration throughout the investigation, as subjects may have not been completely recovered from the previous days training session.

Some researchers have not been able to observe differences in mean cortisol concentration between stale (overly fatigued) and rested athletes following a training season (Hooper et al., 1993). It has been observed that increased cortisol levels negatively effect physical performance through catabolic actions, and higher than normal cortisol levels have been associated with a higher incidence of injury, illness and decrements in physical performance (Kuipers and Keizer, 1988). In the present study, it was demonstrated that the 8 day group performed for a longer duration on the treadmill following the period of reduced training. Thus, the lower mean levels of cortisol following 8 days of reduced training may have contributed to the improved treadmill performance. Tsai et al. (1991) reported that cortisol concentration was higher in elite female orienteers and cross-country skiers than male counter parts over a one year period. It was suggested that the difference between gender

was due to the differing adaptive responses to exercise between males and females. In other words, female endurance athletes may be more sensitive to increases in intensity of exercise than males. In the present investigation, only two female subjects were studied. Both females demonstrated higher individual cortisol values when compared to the mean values of the reduced training groups. It is possible that these higher individual values might have influenced mean cortisol. Nevertheless, individual values for both females did decrease (as well as mean values) as a consequence of reduced training, and therefore suggests that the time off from training may have influenced the overall reduction in cortisol concentration for both males and females.

#### **5.4 Testosterone Concentration**

In male athletes, mean testosterone concentration demonstrated changes following the reduced training periods. During the initial 8 weeks of training, mean testosterone levels remained consistent and ranged between 19.6 and 20.0 nmol/L, and were within normal (10.5-34.5 nmol/L) values as reported for adult males (Hackney, 1989). During the reduced training phase, mean levels increased 13.86% and decreased 6.75% following 4 and 8 days of reduced training, respectively (refer to figure 10). Consequently, this resulted in a significant mean difference ( $p < .05$ ) of 49.84% between the 4 and 8 day groups.

It has been suggested that monitoring testosterone profiles is an effective means in evaluating the athletes response to training (Lehmann, Foster and Keul, 1993; Fry et al., 1992). Banfi, Marinelli, Roi and Agape (1993) suggested that using testosterone profiles for females is not an effective means of monitoring training due to the lower levels (0.7-2.8 nmol/L) of concentration. Consequently, the present study monitored testosterone profiles for male subjects only. Lower levels of both serum and

free testosterone levels have been observed in chronically trained endurance males (Hackney, 1989).

The present investigation demonstrated that mean testosterone levels increased following 4 days of reduced training. This is consistent with findings reported by Hakkinen et al. (1989) and Banfi et al. (1993), who observed increases in mean serum testosterone levels of elite endurance swimmers and speed skaters following a decrease in training volume. Acute increases in serum testosterone have been observed following a period of endurance training, however, these increases have been shown to decline in concentration after prolonged exercise exposure (Hackney, 1989; Hakkinen et al., 1989). It has been suggested that the increase in concentration may be due to hemo-concentration, reduced metabolic clearance and/or a catecholamine mediated increase in production (Hackney, 1989). Others have indicated that inhibitions in testicular secretions occurs as a consequence of increases in concentration of luteinizing hormone (Kuipers and Keizer, 1988).

The investigation did not examine the mechanism(s) (previously cited) responsible for increases in testosterone concentration. However, the present study demonstrated that mean testosterone concentration increased concurrently with a decrease in cortisol concentration following 4 days of reduced training. This inverse relationship may have been due to the short term effects of exercise and possibly hemo-concentration, rather than the effects of reduced training. This is confirmed when examining the responses of the 8 day group, which also demonstrated a decrease in cortisol levels with minimal changes in testosterone levels. Therefore, it is less likely that reduced training aided in changing testosterone levels due to the minimal differences in concentration within the 8 day group.

However, significant ( $p < .05$ ) differences were found for testosterone values between the 4 day and 8 day reduced training group. Although, when examining the effect of homogeneity of variance,

a Greenhouse-Geisser and corresponding Chi-square significance test demonstrated that this assumption may have been violated. More than likely, the difference between groups was due to different baseline levels initially as a result of the relatively small sample sizes. In spite of this confound, it is commonly reported that the analysis of variance is relatively robust against reasonable violations of this assumption (Howell, 1987; Keppel, 1982). Moreover, sizeable differences in variances do not appear to seriously distort the F-distribution in Monte Carlo simulations. As a result, most researchers do not even test these assumptions (Keppel, 1982). Consequently, the findings of the present study should examine testosterone concentration in comparison with concurrent changes in cortisol levels. In other words, despite group differences, the benefit of monitoring testosterone profiles is in the observation of the extent of relative changes throughout a training period.

### **5.5 Lactate Profile, Lactate Threshold and Performance Measures**

Mean lactates remained relatively constant throughout 8 weeks of training (refer to figure 7) with the greatest changes occurring in the recovery period (from peak to 3 minutes post-exercise). However, no significant differences were observed in lactate variables between the 4 and 8 day groups. Non-significant increases in peak lactate of 7.88% (9.35 to 10.15 mmol/L) and 14.64% (10.5 to 12.3 mmol/L) were observed following a 50% reduction in training volume for 4 days and 8 days, respectively.

It has been suggested that one of the mechanisms responsible for improvement in lactate kinetics with training may be related to both extra-cellular changes (plasma volume expansion), or intra-cellular changes (mitochondrial density) (Holden et al., 1992). In the present study, the 4 day

reduced training group demonstrated a 20.69% decrease in lactate concentration from peak (10.15 mmol/L) to 3 minutes post-exercise (8.05 mmol/L). In comparison, the 8 day group revealed a decrease of 8.54% from peak (12 mmol/L) to 3 minutes post-exercise (11.25 mmol/L). The improvement in lactate kinetics during recovery from exercise could have been due to an enhanced clearance rate. This may be partially explained by the increase in plasma volume and the increased ability of the subjects to metabolize lactate. As a result, the larger plasma volume may provide an increase in the distribution space to dilute the effects of increased lactate levels (Holden et al., 1992). However, the present investigation revealed that mean Hct only slightly increased (0.462 to 0.470 L/L), while mean Hgb levels decreased (160 to 157 g/L) concurrently following 4 days of reduced training. This decrease in Hgb with only minimal changes in Hct may have been due to a slight increase in plasma volume, however, it is not likely. Ideally, Hct should remain constant and/or decrease with corresponding increases in Hgb concentration for plasma volume to increase in concentration.

Adaptation to endurance training is associated with an improvement in performance times and recovery from maximal exercise (Gilman and Wells, 1993). Enhanced recovery has been demonstrated to be related to improvements in lactate kinetics. This is primarily due to an increase in lactate elimination or clearance and a decrease in lactate production (Holden et al., 1992). The present investigation demonstrated that treadmill time to exhaustion increased significantly ( $p < .05$ ) from 24:53 to 27:06 (min:sec) following 8 days of reduced training (refer to figure 5). Both recovery (3 minutes post-exercise) lactates (9.55 to 11.25 mmol/L), and TLac increased (3.21 to 3.83 mmol/L) non-significantly following 8 days of reduced training. The present findings are similar to observations reported by Shepley et al. (1992) and Houmard, Scott, Justice and Chenier (1994) who

found that a 7 day period of reduced training with a high intensity format improved running time to fatigue by 22% and 4%, respectively. Others (Koutedakis, Budgett and Faulmann, 1990), have observed that mean treadmill time to exhaustion in under-performed (stale) elite athletes significantly increased approximately 4.2% following 3 to 5 weeks of cessation of training.

The results in treadmill performance may have been influenced due to different baseline performance levels. A test for homogeneity of variance indicated that this assumption may have been violated. Although this may have occurred, it has been reported that violations of such assumptions has little effect on the statistical outcome of an F-distribution (Howell, 1987; Keppel, 1982). As a result, treadmill time should be examined concurrently with other variables in order to demonstrate a trend in response to the training program. Therefore, the concurrent increase in both treadmill time to exhaustion and TLac in the present investigation demonstrated that subjects were able to exercise longer and with a greater intensity at the same relative workload. This increase in performance may have been due to lower lactate levels in combination with an improvement in running economy and metabolic efficiency. Subsequently, subjects may have been able to delay oncoming fatigue and run longer on the treadmill, which may be indicative of an adaptation response to training.

Lower levels of lactate production have been associated with an increase in mitochondrial density in skeletal muscle. It has been reported that lactate clearance rate may be a function of arterial concentration, circulation to the tissues capable of metabolizing lactic acid, and the fractional removal per unit flow. Consequently, as lactic acid concentration increases, the fractional removal rate decreases in a non-linear fashion (McLellan, Cheung and Jacobs, 1991). This may effect performance by encouraging a greater use of fatty acids as an energy substrate, thus using less

anaerobic work and delaying the onset of fatigue (Holden et al., 1992; Oja, Laukkanen, Kukkonen-Harjula, Vuori, Pasanen, Nittymaki and Solakivi, 1991). Although this was not investigated in the present study, it may have a contribution to the overall improvement in lactate kinetics. The evidence for this may reside in performance, in that subjects were able to still perform longer on the treadmill, in spite of higher peak lactates following both 4 and 8 days of reduced training.

Another reported explanation for lower lactate production may be related to catecholamine levels. Mazzeo and Marshall (1989) reported that catecholamine production following endurance training may also decrease lactate production by lowering the effect of epinephrine on muscle glycogen. Muscle glycogenolysis increases linearly with increases in catecholamine levels, consequently the production of lactate increases as well. Therefore, increased levels of catecholamines may effect performance by promoting a greater production of lactate, and consequently, causing fatigue to occur sooner. Hooper et al. (1993) observed that subjects who demonstrated performance improvements following 3-5 days of reduced training also had lower levels of plasma nor-epinephrine, which seems to support the findings of Mazzeo and Marshall (1989). Although the present investigation did not measure plasma nor-epinephrine, it was observed that mean levels of peak lactate increased following both 4 and 8 days of reduced training. However, the increase in lactate levels following the final test period were more than likely due to the individual achieving a true state of maximal fatigue as a result of pushing themselves harder.

Improvements in lactate function may have been due to the muscle fibre type profile of the subjects. Endurance athletes have been shown to exhibit a higher percent of slow oxidative muscle fibers, which would be more efficient in utilizing lactate. Eisenman et al. (1989) suggested that lower lactate levels observed in cross-country skiers while working at a set intensity may be due to

the greater percentage of slow oxidative muscle fibers. Furthermore, Rusko (1992) suggested that improvements in both  $VO_{2max}$  and AnT (anaerobic threshold) following intensive and distance training may be due to the increasing the potential of both slow and fast motor units in metabolizing lactate. Although both catecholamine and muscle fibre type was not examined in the present investigation, it is likely that the type of training adhered to by the athletes in the training program contributed to the improvement in the metabolism of lactate in both type I and type IIA muscle fibers. Moreover, the literature demonstrates that improvements in lactate kinetics involve many processes. The major evidence for this appears to be the relative improvements in physical performance following a specified period of training and rest.

### **5.6 OBLA and TLac as a percent of peak $VO_2$ .**

The present study utilized both OBLA (onset of blood lactate accumulation) and the TLac (individual lactate threshold) as a percent of peak  $VO_2$  to compare changes in response to training over time. Mean values of % $VO_2$  (TLac) increased non-significantly 5.35% from baseline (79.62%) to mid-study (84.12%) following 4 weeks of training. However, mean values of % $VO_2$  (OBLA) decreased 11.47% from 79.93% to 70.82% following 8 weeks of training.

Endurance training has been demonstrated to increase lactate threshold (Astrand and Rodahl, 1986; Urhausen et al., 1993). Cross-country skiers have been reported to show a high TLac, which increases following the ski training season (Rusko, 1992). Others (Mazzeo and Marshall, 1989), have reported cross-country runners and cyclists to have a % $VO_{2max}$  (TLac) of 75% and 65.3%, respectively following a period of endurance training. During the competitive season, skiers are required to train at a higher  $VO_{2max}$ . It has been suggested that training at this intensity is ideal for



promoting adaptations to TLac (Rusko, 1992). At the start of the investigation, endurance training primarily consisted of long slow distance running. Towards the end of the study, as subjects approached the competitive season, training intensity increased as skiing replaced running. The larger physiological demand for skiing as opposed to running may have assisted in the development of a higher %VO<sub>2</sub> (TLac) demonstrated in the first four weeks of the study. However, mean values of %VO<sub>2</sub> (OBLA) decreased from mid-study to post-study, while values of %VO<sub>2</sub> (TLac) remained relatively constant. Subjects may have reached OBLA sooner, and as a result, OBLA would occur at a lower VO<sub>2</sub>. The earlier onset of OBLA may have been caused by a higher resting level of lactate due to confounding factors such as fatigue and/or excitement prior to the test. Rusko (1992) reported that significant increases in VO<sub>2max</sub> are associated with large increases in oxidative enzymes. However, lower intensity ski training has been shown to increase the oxidative capacity of muscle by 40-90% without a significant increase in VO<sub>2max</sub> (Rusko, 1992). This may have occurred in the present study, such that peak VO<sub>2</sub> declined throughout 8 weeks of training despite increases in %VO<sub>2</sub> (TLac). Although, the investigation measured peak values of VO<sub>2</sub> during a maximal test to determine lactate threshold, rather than a calculation of VO<sub>2max</sub>. Rusko (1992) observed that oxidative capacity may increase 30-40% during the training season, when training at 4-8 mmol/L for 3 sessions per week. The Cross-Country Canada training model (1993-94) used for this investigation utilized zone training, which employs working at or slightly below the individual anaerobic threshold when training zone 3 (Reed, 1992). Each zone is individually set at the start of the training season, and monitored as training improvements develop. Subjects were performing two to three zone 3 and zone 4 training sessions per week. Adaptations to training at this intensity would assist in delaying the early onset of fatigue, thus allowing the skier to generate a higher power output for a longer

period of time. Consequently, it would appear that the changes in %VO<sub>2</sub> (TLac) may be due to the variation in the training program (increases in zone 3 and zone 4 workouts), and thus may have resulted in an increase in the oxidative capacity of the muscle.

### 5.7 Peak and Threshold VO<sub>2</sub> Measures

Mean peak VO<sub>2</sub> demonstrated a decrease of 6.25% from 66.89 to 62.71 ml kg<sup>-1</sup> min<sup>-1</sup> following 4 weeks of training. However, mean values of peak VO<sub>2</sub> increased non-significantly by 9.62% from 64.08 ml kg<sup>-1</sup> min<sup>-1</sup> to 70.90 ml kg<sup>-1</sup> min<sup>-1</sup> following 8 days of reduced training.

Injer (1991) reported mean VO<sub>2max</sub> values of male ski racers to range from 85.6, 81.5 and 79.4 ml kg<sup>-1</sup> min<sup>-1</sup> respectively for world, medium and less successful racers. Female skiers were somewhat lower at 70.1, 70.6 and 64.2 ml kg<sup>-1</sup> min<sup>-1</sup> for world, medium and less successful racers respectively. In comparison, the present findings for peak VO<sub>2</sub> were similar to VO<sub>2max</sub> values of world class elite female racers (approximately 70 ml kg min<sup>-1</sup>). However, one particular male subject demonstrated a peak value following 8 days of reduced training of 84.1 ml kg<sup>-1</sup> min<sup>-1</sup>, which is close to world class males. Injer, (1991) reported seasonal variations in VO<sub>2max</sub> of both world class and less successful elite ski racers to be approximately 8-10% and 3-5% respectively. Similarly, Rusko (1992) observed seasonal changes of VO<sub>2max</sub> in elite skiers to vary between 5 to 10%, and explained that these changes occurred as a result of an increased recruitment and adaptation of fast twitch muscle fibers. Although, the present study only examined 9 weeks of a ski season, improvements in peak VO<sub>2</sub> of approximately 6% and 9% were observed. It is unlikely, however, that the significant increase in mean peak VO<sub>2</sub> following recovery to be a result of alterations in fibre type recruitment. This is primarily a long term adaptation, and would require several seasons or years increasing

training volume to promote the necessary cellular adaptations. However, it is more likely that the increases in peak  $\text{VO}_2$  following reduced training are a result of increases in TLac. Increases in both  $\text{VO}_{2\text{max}}$  and performance have been associated with increases in TLac following endurance training (Rusko, 1992; Urhausen et al., 1993). Furthermore, it has been suggested that world class skiers must be able to ski at a high  $\% \text{VO}_{2\text{max}}$  in order to be competitive (Eisenman et al., 1989). Droghetti et al. (1985) has observed that the anaerobic threshold is an important factor in limiting the utilization of  $\text{VO}_{2\text{max}}$  in cross-country skiers. The current study appears to have confirmed these observations such that the subjects examined demonstrated the ability to work at a high  $\% \text{VO}_2$ . The evidence for this exists in the concurrent increase in the  $\% \text{VO}_2$  (TLac) by 5.76% following 8 days of reduced training. In addition, treadmill time to exhaustion and peak  $\text{VO}_2$  also increased following 8 days of reduced training. Thus, the training loads used appeared to be sufficient in yielding adaptational responses necessary for endurance performance.

### **5.8 Threshold and Maximal Heart Rate Measures**

Mean maximal heart rate (MHR) demonstrated a significant decrease ( $p < .05$ ) of 2.49% from baseline (201 bpm) to post-study (196 bpm). However, mean values increased non-significantly 0.5% (from 201 to 202 bpm) and 2.53% (193 to 198 bpm) following 4 and 8 days reduced training, respectively. This is in contrast to Koutedakis, Budget and Faulmann (1990), who reported mean MHR values in under-performing elite athletes to decrease from 192.9 to 190.3 bpm following 3 to 5 weeks of cessation of training. In comparison, Lehmann et al. (1991) observed mean MHR values of middle and long distance runners to be 184, 179 and 178 bpm following 0, 2 and 4 weeks of increased training volume.

Mean heart rate at lactate threshold (THR) demonstrated conflicting trends, as THR decreased 1.6% (188 to 185 bpm) and increased 4.33% (177 to 185 bpm) following 4 and 8 days reduced training, respectively. This increase is similar to findings reported by Koutedakis, Budget and Faulmann (1990), who found heart rate at anaerobic threshold to increase from 171.3 to 176.1 bpm following 3 to 5 weeks of cessation of training in under-performing elite athletes.

It is possible that the subjects examined may not have reached their individual maximum heart rate value at the conclusion of 8 weeks of training. This may be confirmed when examining the lower values of peak  $\text{VO}_2$  and lactate levels measured during the same time (post-study) following 8 weeks of training. Mean values of peak  $\text{VO}_2$ , time to exhaustion and lactate recovery were also lower during post-study evaluation. Furthermore, cortisol levels were highest following 8 weeks of training. High cortisol levels may suggest that the subjects were fatigued, and if so, would explain why they were unable to perform to their true potential. It was anticipated that the OMI (Overstress Monitoring Inventory) would identify fatigue states and that the various physiological measures obtained during the eight weeks of training would support the OMI findings. However, when examining scores from the OMI, the findings suggest that the athletes were not in a state of fatigue. Subjects did not present any unusual patterns of behavior or indicate that they were unable to complete training sessions. Moreover, when resting heart rate (RHR) and systolic blood pressure (SBP) were correlated with scores from the OMI, no significant relationships were observed.

### **5.9 Resting Heart Rate, Systolic Blood Pressure and the OMI**

Mean values for RHR and SBP ranged from 45 to 47 bpm and 112 to 118 mmHg, respectively throughout weeks one to seven. Mean RHR was lower (48 to 51 bpm) than those reported by

Lehmann et al. (1991) following 4 weeks of heavy training in middle and long distance runners. OMI, RHR and SBP did not identify any significant relationships between each variable over seven weeks of training. The normal findings from OMI, RHR and SBP would seem to suggest that the subjects in question did not experience any excessive state of fatigue during the investigation despite high cortisol levels. More than likely, the training regime was adequate, and did not appear to be overly demanding. As a consequence, the low MHR, peak  $VO_2$  and lactate responses encountered at the same time were probably limited to an inability to reach maximum, rather than a result of excessive fatigue. Or, it may suggest that subjects could have been fatigued but not overtrained during the examination period. In fact, MHR, peak  $VO_2$  and lactate variables (ie. peak and recovery values) all improved following a period of reduced training, suggesting that subjects were more successful at reaching a true maximal value.

The use of mood state questionnaires for the monitoring of training has been widely practiced and reported in the literature. Several researchers have observed the relationship between specific statements relating to fatigue, and the presence of abnormally high or low concentrations of catecholamines and hormones (Budget, 1990; Fry et al., 1992; Morgan et al., 1987; Hooper et al., 1993, Vos et al., 1992). As a consequence, some researchers have identified specific mood states to be an external cue for fatigue, thus predicting a potential case for overtraining. The questionnaire used in the current investigation was designed with this purpose. However, the instrument was limited to the accuracy of the subject's response, and as a result, could be misleading if used to monitor training independently of other objective measures. Although the subjective statements from the inventory were not compared with some biochemical data, the current findings would seem to suggest that the inventory was successful in revealing that no conflicts existed between mood

states and physiological observations.

### **5.10 Performance Parameters**

The present investigation examined changes in cycle and temporal characteristics of skiing technique following 4 and 8 days of reduced training. Subjects were required to race a 2.5 km track twice using free-style techniques. Two specific areas were examined: first, the time to complete both laps were investigated as a percent of the total race time; and second, as a percent difference between laps. There were no significant differences observed between mean values of percent total time for lap 1 and percent total time for lap 2 following either 4 or 8 days of reduced training. However, both groups did demonstrate a individual relative increase in the percent time for lap 2 only of 1.98% and 4.5% following both 4 and 8 days reduced training respectively. Mean total lap time for the 4 day group demonstrated a non-significant increase of 3.92% between lap 1 (7:01) and lap 2 (7:06) following 8 weeks of training. This trend was also observed following 4 days of reduced training as a 1.65% increase was observed in mean time for lap 1 (6:49) and lap 2 (6:54).

Although mean total race time was lower following 4 days of reduced training (13:43) as opposed to 8 weeks of training (14:07), differences between times may not be attributed to technique alone. Snow conditions may partly explain why faster times were observed following 4 days of reduced training. Warmer temperatures cause snow to melt and soften producing a "suction" effect between ski and snow. This then increases the friction and hydrodynamic drag force on the ski, which increases overall external resistance and slows down the ski racer (Frederick, 1992; Saibene, Cortili, Rio and Colombini, 1989). As a result of changing weather conditions, the choice of wax would also effect the glide and resulting velocity of skiing. Although in the present study subjects used the

same wax on both test days to control for individual differences, the variation in climate and snow conditions may have been more ideal for the wax used on warmer days. As a result, race times may have been inflated due to better waxing rather than technique and physiological development.

Increases both during and between each lap could also be possibly due to the accumulation of fatigue, thus negatively effecting performance of the ski technique. Fatigue would compromise performance by decreasing the amount of muscular force necessary in the propulsion of the skier while racing. Performance decrements would be most noticeable when the physiological demand is greatest (ie. incline racing). In an attempt to examine if this may have been a contributing factor, subjects were videotaped skiing (offset technique) uphill a 5% grade for 15 meters. The tapes were analyzed for variations in cycle rate, cycle length and cycle velocity. In order to compare the relative differences in mean time between race test days, three comparisons were made. These were a percent change for cycle rate, cycle length and cycle velocity from lap one to lap two.

Mean cycle rate decreased 37.4% and 14.6% from lap 1 to lap 2 following 4 and 8 days of reduced training respectively. However, both mean cycle length increased 27.45% and 80.34%, and mean cycle velocity increased 62.18% and 63.58% from lap 1 to lap 2 following 4 and 8 days reduced training respectively. The difference between laps in cycle rate may have been caused by the corresponding increase in cycle length. Subjects increased cycle length (m) by a large amount for approximately the same cycle velocity (m/sec). As a result, cycle rate (sec) did not decrease as much in lap 2 when compared to lap 1. These findings were consistent with those reported by Boulay et al. (1995), who found that elite skiers can gain a 4% increase in cycle velocity when increasing cycle length by 24%, and decreasing cycle rate by 16%. In contrast, however, Smith et al. (1989) reported that elite skiers tend to demonstrate longer cycle lengths and increases in cycle

rate with faster velocities on flat terrain. When examining kinematic profiles of Olympic skiers, they (Olympic skiers) tend to increase cycle rate when adjusting technique to ski on steeper terrain (Aro et al., 1990; Smith, 1992; Smith and Heagy, 1994).

Changes in cycle characteristics may demonstrate alterations in skiing economy. Specifically, a greater cycle length and greater cycle rate will allow ski racers to cover a greater distance from pole plant to pole plant in a shorter period of time. As a result, as skiing velocity increases, force must be applied over a distance in a shorter period of time. The present study demonstrated that following both 4 and 8 days of reduced training, cycle length increased and cycle rate decreased between lap 1 and lap 2. Differences appear to be greatest between laps for cycle length following 8 days (20.35%) when compared to 4 days (7.65%) of reduced training. However, caution must be observed when attempting to interpret these results. Although it may appear that fatigue did not affect technique as much following 8 days of reduced training, other potential confounding variables may exist. As a result, further analyses are necessary.

### **5.11 Summary and Recommendations**

Training resulted in increases of maximal heart rate and hemoglobin levels following 4 and 8 weeks of endurance training respectively. Both the %VO<sub>2</sub> (at TLac) and CPK levels demonstrated inverse differences with %VO<sub>2</sub> increasing and CPK decreasing following 4 weeks of training. Lactate threshold increased while peak lactate decreased throughout 8 weeks of training. Cortisol concentrations demonstrated an increasing trend throughout 8 weeks of training, and decreased in concentration following both 4 and 8 days of reduced training. Testosterone levels demonstrated marked variations between groups after 4 and 8 days of reduced training. Both treadmill time to



exhaustion and peak  $\dot{V}O_2$  values increased after 8 days of reduced training. Mean scores of performance parameters failed to identify any significant variations throughout the period of the investigation, despite improvements in both cycle length and cycle velocity following both 4 and 8 days of reduced training. Similarly, examination of the Overstress Monitoring Inventory (OMI) and resting systolic blood pressure and resting heart rate remained fairly consistent and did not illustrate any significant variations throughout the examination period. These findings may be interpreted as the following:

- i) Eight days of reduced training (a 50% decrease in training volume) resulted in greater improvements in physiological and biochemical measurements when compared to four days of reduced training. These factors (ie. peak  $\dot{V}O_2$ , cortisol levels, treadmill performance time) have been identified as important physiological and biochemical traits necessary in performing skiing at an elite level.
- ii) Results from the OMI did not reveal any specific relationship (ie. positive/negative correlation) when compared with RSP and RHR. Although the instrument did not specify any subjective states of stress, it was still effective in monitoring the feelings of the subjects throughout the investigation period. However, the instrument could be re-assessed comparing data to more stringent markers of stress (ie. cortisol and testosterone levels).
- iii) The ski training program used appeared to be effective in yielding the physiological returns necessary for elite ski performance. Moreover, it was demonstrated that cycle characteristics improved (cycle length) despite slower race times following a period of reduced training. These improvements occurred concurrently with positive adaptations in both physiological and biochemical variables. Although the period investigated demonstrated improvements in performance, the transfer of these adaptations to ski performance was not adequately assessed. To effectively analyze the applicability of training to ski performance, a comparison to a ski race would have been more appropriate. Furthermore, the training period examined should have extended longer in order to observe the effects of training on the ski season and the training year overall.

The benefit of determining individual lactate kinetics appears to be a major contributing factor in the success of the present training program. It provided markers for short and long term improvements in training progression. Furthermore, the training program that was investigated

appeared to yield the appropriate physiological and biochemical adaptations necessary for ski performance. Overall, the subjects investigated demonstrated similarities in these parameters when compared to other elite and world class skiers. Although the OMI did not yield any specific direction, the necessity for a subjective tool to monitor training is of importance. As a result, the instrument used may need to be re-assessed in order to streamline some psychological and behavioral markers that are more indicative of stress.

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## **APPENDIX**

**Table 7** Descriptive statistics of peak and threshold data of:  $\text{VO}_2$ , percent  $\text{VO}_2$  peak and recovery lactate and heart rates; and treadmill running time to exhaustion.

<b>Variable (N=10)</b>	<b>0 weeks X <math>\pm</math> S.D.</b>	<b>4 weeks X <math>\pm</math> S.D.</b>	<b>8 weeks X <math>\pm</math> S.D.</b>
$\text{PVO}_2$ (ml $\text{kg}^{-1}$ $\text{min}^{-1}$ )	66.89 $\pm$ 7.25	62.71 $\pm$ 7.06	62.08 $\pm$ 7.37
$\text{TVO}_2$ (ml $\text{kg}^{-1}$ $\text{min}^{-1}$ )	53.17 $\pm$ 5.90	52.51 $\pm$ 4.64	51.80 $\pm$ 5.68
% $\text{VO}_2$ (TLac)	79.62 $\pm$ 5.71	84.12 $\pm$ 7.05	83.91 $\pm$ 8.04
% $\text{VO}_2$ (OBLA)	79.83 $\pm$ 5.88	78.40 $\pm$ 8.31	70.82 $\pm$ 5.94
PLac (mmol/L)	10.95 $\pm$ 3.03	10.97 $\pm$ 4.66	10.04 $\pm$ 2.65
TLac (mmol/L)	3.30 $\pm$ 0.34	3.70 $\pm$ 0.57	3.53 $\pm$ 1.25
Lac: 1min (mmol/L)	8.26 $\pm$ 6.56	10.02 $\pm$ 6.10	8.98 $\pm$ 4.09
Lac: 2min (mmol/L)	9.53 $\pm$ 5.32	9.52 $\pm$ 5.70	9.31 $\pm$ 2.84
Lac: 3min (mmol/L)	9.53 $\pm$ 4.44	10.68 $\pm$ 4.62	9.28 $\pm$ 2.67
MHR (bpm)	* 201 $\pm$ 7.58	* 200 $\pm$ 7.68	196 $\pm$ 10.49
THR (bpm)	182 $\pm$ 9.48	183 $\pm$ 14.50	182 $\pm$ 13.23
TIME (min:sec)	26:15 $\pm$ 3.21	26:29 $\pm$ 2.84	24:59 $\pm$ 4.06

\*:  $p < .05$

**Table 8** Descriptive statistics of peak and threshold data of:  $\text{VO}_2$ , percent  $\text{VO}_2$  at TLac and OBLA; peak and recovery lactate and heart rates; and treadmill running time to exhaustion for the 8 day recovery group.

VARIABLE (N=6)	8 WKS: TRAIN X $\pm$ S.D.	8 D: REDUCED TRAINING X $\pm$ S.D.
$\text{PVO}_2$ (ml kg <sup>-1</sup> min <sup>-1</sup> )	64.08 $\pm$ 3.94	70.90 $\pm$ 9.11
$\text{TVO}_2$ (ml kg <sup>-1</sup> min <sup>-1</sup> )	51.46 $\pm$ 5.62	50.50 $\pm$ 23.54
% $\text{VO}_2$ (TLac)	80.21 $\pm$ 6.53	85.11 $\pm$ 9.82
% $\text{VO}_2$ (OBLA)	79.16 $\pm$ 6.10	80.91 $\pm$ 8.48
PLac (mmol/L)	10.50 $\pm$ 1.88	12.30 $\pm$ 2.76
TLac (mmol/L)	3.21 $\pm$ 0.79	3.83 $\pm$ 0.68
Lac: 1min (mmol/L)	9.03 $\pm$ 4.66	9.78 $\pm$ 5.44
Lac: 2min (mmol/L)	9.88 $\pm$ 2.38	10.63 $\pm$ 5.91
Lac: 3min (mmol/L)	9.55 $\pm$ 1.86	11.25 $\pm$ 3.75
MHR (bpm)	193 $\pm$ 9.46	198 $\pm$ 7.42
THR (bpm)	177 $\pm$ 9.37	185 $\pm$ 9.97
TIME (min:sec)	24:53 $\pm$ 2.35	* 27:06 $\pm$ 3.10

\*: p < .05

**Table 9** Descriptive statistics of peak and threshold data of:  $\text{VO}_2$ , percent  $\text{VO}_2$  at TLac and OBLA; peak and recovery lactate and heart rates; and treadmill running time to exhaustion for the 4 day recovery group.

VARIABLE (N=4)	8 WKS: TRAIN X $\pm$ S.D.	4 D: REDUCED TRAINING X $\pm$ S.D.
P $\text{VO}_2$ (ml kg <sup>-1</sup> min <sup>-1</sup> )	59.07 $\pm$ 10.83	n/a
T $\text{VO}_2$ (ml kg <sup>-1</sup> min <sup>-1</sup> )	52.30 $\pm$ 6.59	n/a
% $\text{VO}_2$ (TLac)	89.45 $\pm$ 7.34	n/a
% $\text{VO}_2$ (OBLA)	58.30 $\pm$ 4.10	n/a
PLac (mmol/L)	9.35 $\pm$ 3.76	10.15 $\pm$ 3.26
TLac (mmol/L)	4.00 $\pm$ 1.78	3.97 $\pm$ 1.17
Lac: 1min (mmol/L)	8.90 $\pm$ 3.73	9.35 $\pm$ 3.08
Lac: 2min (mmol/L)	8.45 $\pm$ 3.64	8.45 $\pm$ 2.31
Lac: 3min (mmol/L)	8.87 $\pm$ 3.91	8.05 $\pm$ 2.49
MHR (bpm)	201 $\pm$ 11.52	202 $\pm$ 7.32
THR (bpm)	188 $\pm$ 16.76	185 $\pm$ 11.46
TIME (min:sec)	24:10 $\pm$ 6.32	25:48 $\pm$ 4.22

n/a: Technical problems experienced with equipment; unable to collect gases.

**Table 10:** One-way repeated measures ANOVA expressing a main effect of time between dependent variables and training volume and intensity for 8 weeks duration.

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
MHR	Time	133.07	2, 18	66.53	7.01	.006
Hgb	Time	236.60	2, 18	118.30	8.92	.002
CPK	Time	133704	2, 18	66852	11.65	.001
Cort.	Time	88429	2, 18	44214	3.52	.051

**Table 11** A 2 X 2 between groups ANOVA expressing a main effect of time, group and/or group by time between dependent variables and training volume and intensity for 4 and 8 days of reduced training.

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
TIME	Time	17.83	1, 8	17.83	7.39	.026
Test.	Group	577.22	1, 6	577.22	16.73	.006
HRT	Grp by Time	151.87	1, 8	151.87	5.54	.046



**Table 12: One way repeated measures ANOVA summary table for peak and threshold data of: VO<sub>2</sub>, percent VO<sub>2</sub> peak and recovery lactate and heart rates; and treadmill running time to exhaustion throughout 8 weeks of training. Note: Refer to table 10 for statistics of MHR.**

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
PVO <sub>2</sub>	Time	136.68	2, 18	68.34	3.18	0.066
TVO <sub>2</sub>	Time	9.39	2, 18	4.69	0.49	0.619
%VO <sub>2</sub> (TLac)	Time	128.99	2, 18	64.5	2.02	0.161
%VO <sub>2</sub> (OBLA)	Time	468.94	2, 18	234.47	1.29	0.300
PLac	Time	5.64	2, 18	2.82	0.32	0.732
TLac	Time	0.81	2, 18	0.40	0.75	0.487
Lac (R) (1 min)	Time	15.66	2, 18	7.83	0.38	0.687
Lac (R) (2 min)	Time	10.38	2, 18	5.09	0.37	0.685
Lac (R) (3 min)	Time	11.15	2, 18	5.57	0.39	0.684
HRT	Time	12.20	2, 18	6.10	0.30	0.748
TIME	Time	15.00	2, 18	7.50	2.17	0.144

**Table 13:** One way repeated measures ANOVA summary table of: white blood cell (WBC), red blood cell (RBC), hematocrit (HCT), testosterone (males only, N=8) throughout 8 weeks of training. Note: Refer to table10 for statistics for Hgb, CPK and cortisol.

Depen. Var.	Source	SS	DF	MS	F-ratio	Sig of F
WBC	Time	1.00	2, 18	0.50	0.94	0.409
RBC	Time	1.70	2, 18	0.85	1.18	0.329
HCT	Time	0	2, 18	0	1.21	0.320
Test.	Time	25.08	2, 18	12.54	0.97	0.401

N.B. Summary statistics for ferritin, urea and LDH following 8 weeks of training were not available due to missing sample during the data collection. As a result, ANOVA analysis was not performed.

**Note:** A 2 X 2 between groups ANOVA following 4 and 8 days of reduced training was not performed on the following variables due to missing data at time of collection. Tables 2, 4, and 9 specify which variables had missing data during this period: PVO<sub>2</sub>, TVO<sub>2</sub>, %VO<sub>2</sub> (TLac), %VO<sub>2</sub> (OBLA), ferritin, urea, CPK, LDH.

**Table 14:** A 2 X 2 between groups ANOVA summary table for Peak Lactate.

Depen. Var.	Source	SS	DF	MS	F-ratio	Sig of F
PLac	Group	13.07	1, 8	13.07	0.97	0.353
PLac	Time	8.11	1, 8	8.11	2.84	0.130
PLac	Grp by Time	1.20	1, 8	1.20	0.42	0.535

**Table 15: A 2 X 2 between groups ANOVA summary table for Lactate Threshold.**

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
TLac	Group	1.03	1, 8	1.03	0.55	0.480
TLac	Time	0.42	1, 8	0.42	0.81	0.394
TLac	Grp by Time	0.49	1, 8	0.49	0.96	0.357

**Table 16: A 2 X 2 between groups ANOVA summary for Recovery Lac. (1 min).**

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
RLac (1)	Group	0.39	1, 8	0.39	0.02	0.903
RLac (1)	Time	1.73	1, 8	1.73	0.10	0.756
RLac (1)	Grp by Time	0.11	1, 8	0.11	0.01	0.938

**Table 17: A 2 X 2 between groups ANOVA summary for Recovery Lac. (2 min).**

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
RLac (2)	Group	15.70	1, 8	15.70	0.71	0.423
RLac (2)	Time	0.67	1, 8	0.67	0.07	0.805
RLac (2)	Grp by Time	0.67	1, 8	0.67	0.07	0.805

**Table 18:** A 2 X 2 between groups ANOVA summary for Recovery Lac. (3 min).

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
RLac (3)	Time	18.02	1, 8	18.02	1.47	0.260
RLac (3)	Group	0.92	1, 8	0.92	0.14	0.723
RLac (3)	Grp by Time	7.65	1, 8	7.65	1.13	0.320

**Table 19:** A 2 X 2 between groups ANOVA summary table for peak MHR.

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
MHR	Group	165.68	1, 8	165.68	1.15	0.315
MHR	Time	46.87	1, 8	46.87	2.92	0.126
MHR	Grp by Time	16.88	1, 8	16.88	1.05	0.335

**Table 20:** A 2 X 2 between groups ANOVA summary table for THR.

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
THR	Group	143.01	1, 8	143.01	0.59	0.466
THR	Time	27.07	1, 8	27.07	0.99	0.349
THR	Grp by Time	151.87	1, 8	151.87	5.54	<b>0.046</b>

**Table 21:** A 2 X 2 between groups ANOVA summary table for treadmill time.

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
TIME	Group	4.80	1, 8	4.80	0.17	0.694
TIME	Time	17.83	1, 8	17.83	7.39	<b>0.026</b>
TIME	Grp by Time	0.40	1, 8	0.40	0.16	0.696

**Table 22:** A 2 X 2 between groups ANOVA summary table for WBC.

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
WBC	Group	0.05	1, 8	0.05	0.02	0.903
WBC	Time	0.20	1, 8	0.20	0.14	0.720
WBC	Grp by Time	2.21	1, 8	2.21	1.53	0.251

**Table 23 :** A 2 X 2 between groups ANOVA summary table for RBC.

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
RBC	Group	1.32	1, 8	1.32	1.10	0.325
RBC	Time	2.34	1, 8	2.34	2.11	0.185
RBC	Grp by Time	1.09	1, 8	1.09	0.98	0.351

**Table 24 :** A 2 X 2 between groups ANOVA summary table for HGB.

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
HGB	Group	102.67	1, 8	102.67	0.49	0.502
HGB	Time	1.87	1, 8	1.87	0.19	0.673
HGB	Grp by Time	39.67	1, 8	39.67	4.06	0.079

**Table 25 :** A 2 X 2 between groups ANOVA summary table for HCT.

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
HCT	Group	0	1, 8	0	1.30	0.287
HCT	Time	0	1, 8	0	4.72	0.062
HCT	Grp by Time	0	1, 8	0	0.56	0.478

**Table 26 :** A 2 X 2 between groups ANOVA summary table for Cortisol.

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
Cortisol	Group	167851	1, 8	167851	3.37	0.104
Cortisol	Time	79670.5	1, 8	79670.5	3.93	0.083
Cortisol	Grp by Time	1888.13	1, 8	1888.13	0.09	0.768

**Table 27 : A 2 X 2 between groups ANOVA summary table for Testosterone.**

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
Test.	Group	577.22	1, 6	577.22	16.72	<b>0.006</b>
Test.	Time	9.05	1, 6	9.05	0.85	0.392
Test.	Grp by Time	25.61	1, 6	25.61	2.40	0.172

Note: Subjects used were male only: 4 day group (N=3); 8 day group (N=5).

**Table 28 : A 2 X 2 between groups ANOVA summary for % Total Time Lap 1.**

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
%TT (1)	Group	0.01	1, 8	0.01	0.02	0.897
%TT (1)	Time	1.10	1, 8	1.10	1.68	0.231
%TT (1)	Grp by Time	2.32	1, 8	2.32	3.55	0.096

**Table 29 : A 2 X 2 between groups ANOVA summary for % Total Time Lap 2.**

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
%TT (2)	Group	0	1, 8	0	-	-
%TT (2)	Time	4.98	1, 8	4.98	4.02	0.078
%TT (2)	Grp by Time	2.03	1, 8	2.03	1.62	0.239

**Table 30 : A 2 X 2 between groups ANOVA summary for % Change Cycle Rate.**

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
% C.R.	Group	57.74	1, 8	57.74	5.73	0.054
% C.R.	Time	7.87	1, 8	7.87	0.13	0.730
% C.R.	Grp by Time	0.24	1, 8	0.24	0	0.952

**Table 31: A 2 X 2 between groups ANOVA summary for %Change Cycle Length.**

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
% C.L.	Group	149.08	1, 8	149.08	0.54	0.482
% C.L.	Time	408.00	1, 8	408.00	2.03	0.192
% C.L.	Grp by Time	243.82	1, 8	243.82	1.21	0.303

**Table 32: A 2 X 2 B-G ANOVA summary for %Change Cycle Velocity.**

<b>Depen. Var.</b>	<b>Source</b>	<b>SS</b>	<b>DF</b>	<b>MS</b>	<b>F-ratio</b>	<b>Sig of F</b>
% C.V.	Group	396.91	1, 8	396.91	1.29	0.289
% C.V.	Time	424.20	1, 8	424.20	1.54	0.250
% C.V.	Grp by Time	89.61	1, 8	89.61	0.33	0.584



**WEEK ONE:****Table 33 :** Correlation matrices for resting systolic blood pressure (RSP), resting heart rate (RHR) & scores from the Overstress Monitoring Inventory (OMI) at week one.

<b>VAR.</b>	<b>RSP</b>	<b>RHR</b>	<b>OMI</b>
RSP 1	1.000	-.3947	.0936
RHR 1	-.3947	1.000	.4011
OMI 1	.0936	.4011	1.000

**WEEK TWO:****Table 34 :** Correlation matrices for resting systolic blood pressure (RSP), resting heart rate (RHR) & scores from the Overstress Monitoring Inventory (OMI) at week two.

<b>VAR.</b>	<b>RSP</b>	<b>RHR</b>	<b>OMI</b>
RSP 2	1.000	.5482	.3544
RHR 2	.5482	1.000	.5778
OMI 2	.3544	.5778	1.000

**WEEK THREE:****Table 35 :** Correlation matrices for resting systolic blood pressure (RSP), resting heart rate (RHR) & scores from the Overstress Monitoring Inventory (OMI) at week three.

<b>VAR.</b>	<b>RSP</b>	<b>RHR</b>	<b>OMI</b>
RSP 3	1.000	.0952	.0672
RHR 3	.0952	1.000	-.1141
OMI 3	.0672	-.1141	1.000

**WEEK FIVE:****Table 36** : Correlation matrices for resting systolic blood pressure (RSP), resting heart rate (RHR) & scores from the Overstress Monitoring Inventory (OMI) at week five.

<b>VAR.</b>	<b>RSP</b>	<b>RHR</b>	<b>OMI</b>
RSP 5	1.000	.1272	.3298
RHR 5	.1272	1.000	.2754
OMI 5	.3298	.2754	1.000

**WEEK SIX:****Table 37** : Correlation matrices for resting systolic blood pressure (RSP), resting heart rate (RHR) & scores from the Overstress Monitoring Inventory (OMI) at week six.

<b>VAR.</b>	<b>RSP</b>	<b>RHR</b>	<b>OMI</b>
RSP 6	1.000	.2654	.4201
RHR 6	.2654	1.000	-.0806
OMI 6	.4201	-.0806	1.000

**WEEK SEVEN:****Table 38** : Correlation matrices for resting systolic blood pressure (RSP), resting heart rate (RHR) & scores from the Overstress Monitoring Inventory (OMI) at week seven.

<b>VAR.</b>	<b>RSP</b>	<b>RHR</b>	<b>OMI</b>
RSP 7	1.000	.0748	.3350
RHR 7	.0748	1.000	-.0083
OMI 7	.3350	-.0083	1.000

**Table 39: T-tests of Significant Variables**

T-value, degrees of freedom & probability value of peak heart rate (HRP), treadmill time to exhaustion (TIME), creatine phosphokinase (CPK), testosterone (TES) and hemoglobin (HGB). [ 1=baseline, 2=mid-study, 3=post-study, 4=reduced training (either 4 or 8 days) ].

<b>VAR's.</b>	<b>t-value</b>	<b>dF</b>	<b>2-tail prob.</b>
HRP1/ HRP3	2.80	9	.021
HRP2/ HRP3	2.66	9	.026
TIME3/ TIME4	-3.59	5	.016
CPK1/ CPK2	2.91	9	.017
TES4	-3.93	5.96	.008
HGB1/ HGB2	-3.74	9	.005
HGB2/ HGB3	-4.34	9	.002

**PARTICIPANT CONSENT FORM**

SUBJECT.....

DATE.....

TIME.....

(i) I agree to participate in an investigation by Dr. Bob Thayer and his research assistant Wayland Pulkkinen and/or any other assistants as may be selected to perform the following procedure (s):

- a) VO<sub>2</sub>max test
- b) Threshold Performance Test
- c) Race Simulation Test
- d) Blood Analysis (both fingertip and forearm aspirations)
- e) Resting Blood Pressure
- f) Overstress Monitoring Questionnaire

(ii) I also understand the voluntary nature to my consent to participate, and understand that I may withdraw at any time from further participation if I so choose. I am fully aware and informed of the test procedures, and understand that physical discomfort may occur as a result of testing. Furthermore, I understand that all of my results are confidential, as that subjects will only be identified by number. Following completion of the study, subjects will be able to meet with the researcher to discuss their individual results.

.....  
witness

.....  
signature of subject

.....  
signature of researcher(s)

**TRAINING STUDY ON NATIONAL CROSS-COUNTRY SKIERS**  
**Letter to Participants**

contacts: Wayland Pulkkinen w: 343-8187 r: 346-8478      Dr. Bob Thayer      w: 343-8544  
(School of Kinesiology)

Letter of Introduction and Design of Experiment

Some of Canada's top elite cross-country skiers will be training here in Thunder Bay in preparation for the 1995 Nordic World Ski Championships. Consequently, Thunder Bay has been recently titled a national training center for cross-country skiing. These elite skiers train on a year round basis combining both dry land and on snow training. National team members follow a plan set forward by Cross Country Canada, which involves training based on heart rate intensities called "Zone Training". These athletes are monitored regularly via performance time trials, physiological tests and fatigue questionnaires to establish progression and improvement. A frequent problem in highly trained endurance athletes is overtraining, which exists in various forms. It usually occurs in individuals who have trained for several years at a high volume year round, and can drastically hinder performance. In order to prevent such a situation from occurring, it is imperative to monitor the athlete during their current training. In order to maximize the current training regime, each athlete should be monitored individually in order to modify the program to their individual needs.

This study will examine 9 weeks of the skier's heaviest training period which involves October through to December. Testing will follow the same format as stated above, with some specific tests to determine to what degree training has improved or decreased. This 9 week period will be divided into three cycles, with cycle 1 and 2 composed each of 4 weeks training, and cycle 3 being either a 4 or 8 day taper. Testing will involve a combination of one of three formats.

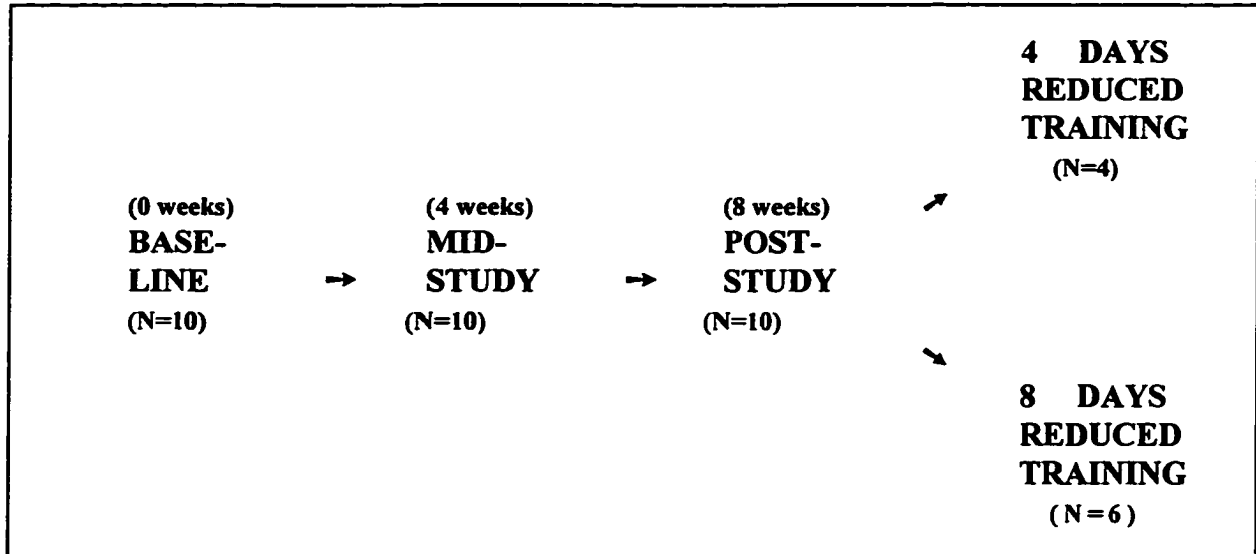
**Format A:**

- i) systolic blood pressure
- ii) monitoring questionnaire
- iii) peakVO<sub>2</sub> test
- iv) threshold performance test
- v) race simulation test
- vi) resting blood sample

**Format B:**

- i) systolic blood pressure
- ii) monitoring inventory
- iii) morning heart rate

### Schematic of Testing Procedure:



### Description of Tests

The following describes the nature and procedure of the testing that will be involved in all three testing formats. It is important to note that the subjects involved in this experiment are highly trained athletes who are familiar with the tests used, and are aware of any complications and/or side effects of the procedures described.

#### a) Resting Blood Pressure:

Both diastolic and systolic blood pressure will be taken with the use of a sphygmomanometre and stethoscope. Subjects will be required to sit quietly for 20 minutes prior to the test.

#### b) Resting Blood Sample:

Blood samples will be taken before exercise. The process involves the insertion of an indwelling catheter in a forearm vein. This simple procedure is sterile, and will be performed by a qualified technician. Subjects will be required to rest quietly in an upright position for 20 minutes duration. Immediately following, a 25 ml sample of blood will be extracted and analyzed for hemoglobin, hematocrit, and white blood cell count. Some slight bruising and discomfort may occur as a result of the procedure.

c) peak  $\text{VO}_2$ :

Subjects will perform an incremental workload test on a treadmill until voluntary exhaustion. Expired gases will be collected during the exercise test to aid in the determination of  $\text{VO}_2$  (maximal amount of oxygen uptake consumed). The length of this test is approximately 10 to 15 minutes in duration, depending on the fitness level of the individual. Subjects may experience slight discomfort due to fatigue. Blood samples may be extracted via fingertip aspiration in order to determine blood lactate levels during the testing procedure. Aspirations will be made with the use of a disposable sterile lancet, or with a medical lancet gun designed for small fingertip blood samples. The amount of samples necessary for an accurate determination of blood lactate may range from 5 to 6. Slight discomfort may follow as a result of sampling.

d) Overstress Monitoring Inventory:

This is a self report questionnaire which evaluates how the athlete feels following training and throughout the week. It is in likert scale form rating feelings of fatigue, depression, irritability etc. from low (not present at all) to high (present daily). Subjects will submit weekly reports of this questionnaire, thus providing a summary of how physical activity was experienced during that week. All reports will be kept confidential, and will only be visible to the experimenter and the coach of the athlete.

e) Threshold Performance Test:

Each subject will perform a Zone 3 check protocol (race pace test) over a predetermined distance at a prescribed heart rate. This test procedure is regularly done during the subjects training program, as outlined by Cross-Country Canada training guidelines. This test will only be implemented 3 times, at the start of cycle 1, at the end of cycle 2, and at the end of cycle 3.

f) Race Simulation Test:

There will be five race simulation tests conducted at the Lappe Nordic Center. Prior to the tests all skiers will complete a half hour warm-up. The skiers will then be instructed to ski 3 laps of a 2.5 km loop as fast as they are able to using the classic style ski technique. Each subject will be individually started on the test and timed. Pre-lactate and recovery lactate levels may be measured using the same format as described during the  $\text{VO}_2$  test. Furthermore, each athlete will be required to wear a Vantage XL Heart Rate Monitor during each test. These will then be downloaded onto a computer following each test.

**THIS WILL BEGIN WHEN SNOW IS AVAILABLE**

### Testing Dates and Sign Up Times

The final times for testing still yet have to be determined as that times are contingent upon individual work and academic schedules (ie. exam time tables). Subjects will set up times with the researcher and coach. Test times **must** be on the same day, and preferably at the same time of day. It is therefore advised that when test times are being determined, use a day when the individual schedule is most open. Please call Wayland or Bob a minimum of 24 hours in advance if there are any conflicts in testing times.

Once again, thank-you for your co-operation and commitment to this endeavor. I trust the data that you will have access to will be of great significance in tailoring your individual training plan. All results from your tests will be kept confidential and will only be accessible to the coach and the researchers involved.

I will produce a summary of all your tests complete with analysis of the data upon request.

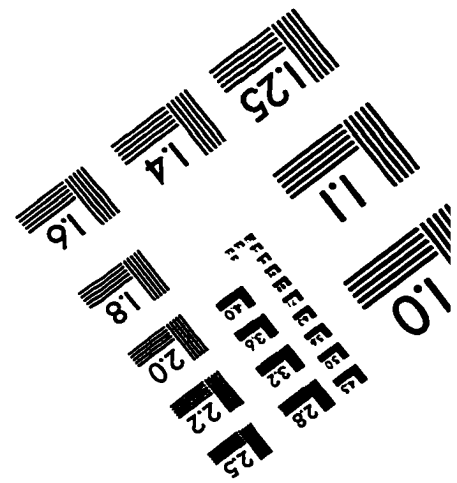
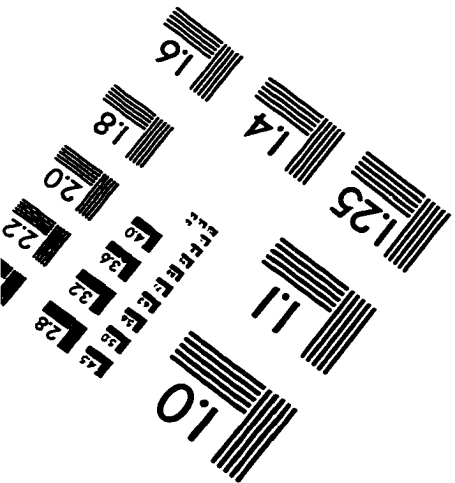
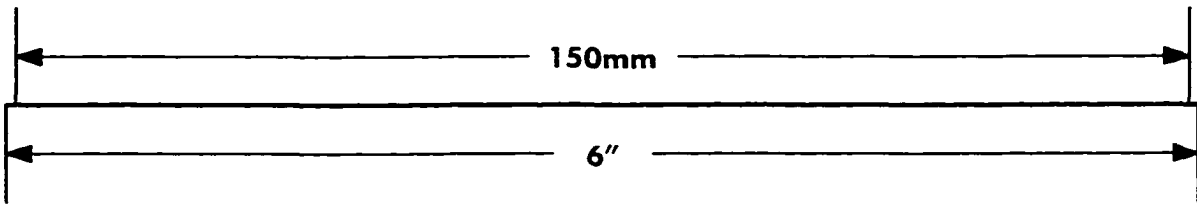
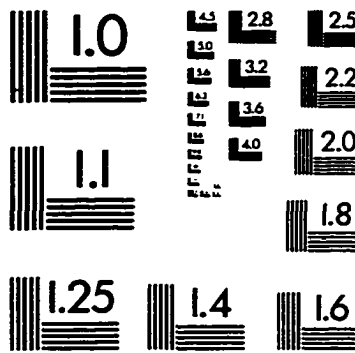
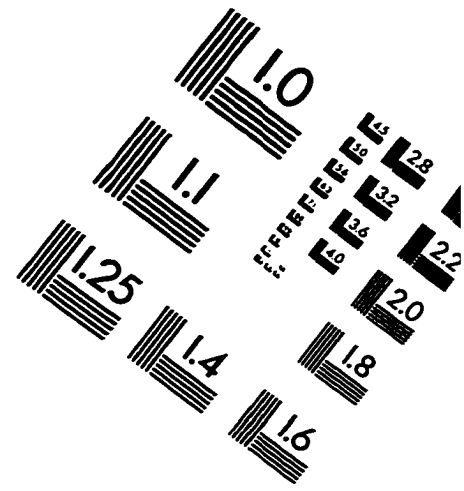
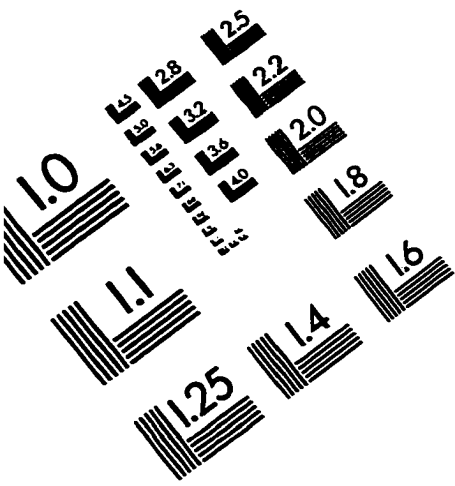
Sincerely,

Wayland Pulkkinen





# IMAGE EVALUATION TEST TARGET (QA-3)



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