DIFFERENCES IN FIVE KILOMETER TIME TRIAL PERFORMANCE DURING THE RECOVERY PERIOD AFTER AN ULTRAMARATHON RACE

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Declaration

I, Tamlyn Jane Benney, hereby declare that the work on which this dissertation is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university.

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(Date)

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List of abbreviations

BMI	Body mass index
cm	Centimetre
CNS	Central nervous system
DOMS	Delayed onset muscle soreness
E-C coupling	Excitation-contraction coupling
EIMD	Exercise-induced muscle damage
EMG	Electromyography
HR	Heart rate
km	Kilometre
$m.s^{-1}$	Minutes per second
min	Minute
MVC	Maximal voluntary contraction
Na^+ - K^+ ATPase	Sodium - potassium adenosine triphosphatase
RER	Respiratory exchange ratio
RPE	Rate of perceived exertion
s	Second
SSC	Stretch shortening cycle
TT	Time trial
VAS	Visual analogue scale
VE	Respiratory minute volume
VE/VO_2	Ventilatory equivalent ratio for oxygen
VO_{2max}	Maximum oxygen consumption

Glossary of terms

Comrades Marathon	The Comrades Marathon is a 90 km ultramarathon run annually between Pietermaritzburg and Durban. In 2011, athletes took part in the "*up*" run, which starts in Durban and finishes in Pietermaritzburg [(1)].
Fatigue	Fatigue is an acute impairment of exercise performance which includes both an increased perceived level of effort to maintain a certain force output and the eventual inability to produce that force output [(2)].
Rate of perceived exertion	Perception of effort is derived from sensory input arriving from a variety of different biological systems such as the musculoskeletal, cardiovascular and respiratory systems during exercise. The rate of perceived exertion is a subjective measure often used as an index of effort during exercise [(3)]. The conscious perception of fatigue has traditionally been measured on the rate of perceived exertion (RPE) scale described by Borg [(4)], to measure the degree of heaviness and strain experienced in physical work [(5)].

Teloanticipation	This is a feed forward process which allows performance beliefs to influence perceptions of exertion, pacing decisions and subconscious efferent muscle control [6].
Visual analogue scale (VAS)	This is a subjective measure used to quantify pain. The participants rate their pain by drawing a vertical line on a 100 mm pain rating scale, where 0 mm represents "*no pain*", and 100mm represents "*maximal pain*". The distance along the pain rating scale to the vertical line drawn is measured in millimetres (mm), and the pain score is given [7].

Abstract

Background

Athletes require an appropriate balance between training, competition stress and recovery to achieve maximal performance. Previous studies have shown that exercise induced muscle damage has a negative influence on endurance running performance, and that these negative effects may be mediated by an increased perception of effort. There is a lack of evidence regarding the effects of ultramarathon distance races on running performance, and the optimal duration of the recovery period before returning to competitive running.

Aim

The aim of this study was to determine the changes in running performance during the recovery period after an ultramarathon race.

Specific objectives

The specific objectives were: (a) to determine differences in running performance between the experimental group (runners participating in the 2011 Comrades Marathon) and a control group (distance runners not taking part in the 2011 Comrades Marathon) during the recovery period after an ultramarathon race; (b) to determine the differences in muscle pain, heart rate and perception of effort during a 5 km time trial between groups and over time before, and during the recovery period after an ultramarathon race; and (c) to determine if there were any relationships between prior experience, training history, and running performance during the recovery period following an ultramarathon race.

Methods

This study had a quasi-experimental design. The experimental group consisted of 16 runners participating in the 2011 Comrades Marathon. Fifteen distance runners who were not taking part in the 2011 Comrades Marathon formed the control group. All participants were required to have run a marathon (42.2 km) in under five hours in the past four months. Participants in the experimental group were required to have run a minimum of one previous Comrades Marathon prior to the 2011 race. Participants in both groups were required to have a minimum average training mileage of 40 km per week for the four months preceding the study. Participants attended a familiarisation session 10 days prior to the Comrades Marathon. Participants completed an informed consent form, the Modified Physical Activity Readiness questionnaire, and a medical and training questionnaire. Body composition measurements were performed and the participants were familiarised with all testing procedures. Running performance was assessed with 5 km time trial that were performed seven days before the Comrades Marathon; and at six, 13 and 20 days after the race. Muscle pain was measured in both groups before each time trial. Heart rate (HR), the relative perception of effort (RPE) and running performance were recorded during the 5 km time trials.

Participants in the experimental group were required to complete the Comrades Marathon. Muscle pain was also recorded daily one day before, and for seven days following the Comrades Marathon in the experimental group.

Results

There were no significant differences in the total 5 km time trial performance between the groups; however there was a significant main effect of time ($p = 0.0001$). Time trial performance was significantly improved at post-race time trial three (TT3) compared to post-race TT1 ($p = 0.013$). There were no significant differences in heart rate between groups or over time. There were no significant differences in RPE between groups, although there was a significant main effect of time ($p = 0.01$), with RPE increasing over time. Quadriceps pain scores were significantly higher in the experimental group at time trial 1 (TT1) ($p = 0.006$) and TT2 ($p = 0.002$), compared to the control group. Hamstrings pain scores were significantly increased in the experimental group at TT2, compared to the control group ($p = 0.0004$). Gastrocnemius pain scores were significantly higher in the control group compared to the experimental group at TT2 ($p = 0.001$). In the experimental group there were significant positive relationships between early delta performance and Comrades finishing time as a percentage of the 10 km personal best time ($r = 0.55$; $p = 0.03$); and early delta performance and Comrades finishing time as a percentage of the 42 km personal best time ($r = 0.53$; $p = 0.04$).

Discussion and conclusion

There were no significant differences between the experimental and control groups during the recovery period after an ultramarathon although there was a significant improvement in performance for both groups over time. Early changes in performance following the ultramarathon had a positive relationship to previous pacing strategies used for personal best times in 10 km and 42 km races, which may support the role of central regulation and prior experience affecting performance in the presence of fatigue and muscle damage during the recovery period.

The change of the rate of perceived exertion throughout the testing procedure may have initially been low as a result of the tapering process; or as a protective mechanism to avoid injury prior to the race. Future research should investigate the effects of different training volume and intensities on the performance following an ultramarathon as well as investigating the differences in performance following an ultramarathon using different outcome measures for performance such as maximum treadmill running speed or a 20m sprint test. Functional performance tests could also be incorporated such as vertical jump height or maximal isometric force measurements. .

CHAPTER 1: INTRODUCTION AND SCOPE OF THESIS

1.1 INTRODUCTION

Recovery is an important factor in determining when an athlete may return to competition [8]. It has been defined as the point at which the athlete is able to train without an increased risk of injury [8]. An insufficient recovery time does not allow for adaptation to occur and the athlete may develop symptoms of overtraining due to excessive exposure to training stress [9]. There are multiple factors that may affect the recovery of an athlete after an endurance event, and there also appears to be large inter-individual variability in recovery times [8][10][11]. The recovery period after an endurance event may be influenced by a complex interplay between neuromuscular fatigue, exercise-induced muscle damage and endurance training history [10].

Studies investigating the effects of an acute endurance running bout have demonstrated damage to the muscle structure [13][14][15][16], as well as numerous metabolic [17][18][19][20], and biomechanical [21][22][23][24] changes. These changes have been associated with a decrease in endurance running performance following the initial exercise bout [25][26]. Regular endurance training has been shown to result in adaptations that reduce the extent of cellular disturbances during subsequent training bouts [27][28]. Chronic adaptations of skeletal muscle, including increased mitochondrial enzyme density, increased capillary density, and an increased reliance on fat as a fuel, have been linked to the cumulative effects of regular exercise training [28][29]. These adaptations are associated with an improvement in endurance performance [29][30].

Previous studies have provided evidence that most, but not all physiological responses to endurance exercise are heightened when muscle damage is present, [31][32] and that performance is at a greater risk of being affected in athletes with exercise induced muscle damage than those without [33][34].

There is limited literature evaluating the effects of exercise induced muscle damage on endurance running performance, as well as the recovery of running performance following an endurance event. It is important to understand the effect exercise induced muscle damage has on performance during the sub-acute and long term recovery phase when the athlete is asymptomatic.

1.2 AIMS AND OBJECTIVES

1.2.1 Aim

The aim of this study was to determine the changes in running performance during the recovery period after an ultramarathon race.

1.2.2 Specific objectives

- To determine differences in five kilometre time trial performance between an experimental group (runners participating in the 2011 Comrades Marathon) and a control group (distance runners not taking part in the 2011 Comrades Marathon) during the recovery period after an ultramarathon race.

- To determine differences in indirect markers of muscle damage namely: muscle pain, heart rate and perception of effort during a 5 km time trial between groups and over time before, and during the recovery period after an ultramarathon race.

- To determine whether there were any relationships between prior experience, age, training history, and running performance during the recovery period after an ultramarathon.

1.3 SIGNIFICANCE OF THE DISSERTATION

There is a lack of evidence for the optimal recovery period after marathon and ultramarathon events. The findings of this study will provide information regarding the recovery process after an endurance event, and will attempt to assess when running performance returns to pre-event levels. This study may also provide insight into the amount of time needed to safely recover from ultramarathon events, thereby potentially reducing the risk of overtraining or injury. Athletes of all ability levels were included in this study to ensure that a wide variety of the athletic community were observed. The findings of this study may also be of practical relevance to health professionals and coaches, and may contribute to the development of evidence-based guidelines regarding return to training and competition after an ultramarathon event.

1.4 PLAN OF DEVELOPMENT

In preparation for the quasi-experimental study of this dissertation, a comprehensive review of the literature examining the interaction of neuromuscular and mechanical factors on endurance running performance and recovery will be presented (Chapter 2). This will be followed by a description of the study designed to investigate the performance of athletes during the recovery period after an ultramarathon race (Chapter 3). A summary and conclusion section, including recommendations for future research (Chapter 4) will complete this dissertation.

CHAPTER 2: LITERATURE REVIEW

2.1 INTRODUCTION

Endurance running performance was traditionally thought to be determined by maximal oxygen consumption (VO_{2max}), running economy and lactate threshold [35]. However, Paavolainen et al [36] showed that a combination of strength and endurance training improved muscle strength, running economy and performance with no changes in VO_{2max} [36]. This indicates the possibility that neuromuscular factors may affect the performance of endurance athletes [37]. The limitations in performance may be explained by central nervous system integration of the input [27], and adjustments in the recruitment of skeletal muscle according to the exercise conditions to prevent damage to the body's vital organs [38]. Thus changes in recruitment patterns of skeletal muscle during exercise may provide an explanation for individual differences in athletic performance [39].

Recovery after endurance running has not been studied extensively [11]. It is an important factor in determining when a runner may return to competition [40]. There appears to be large inter-individual variability in recovery times [8]. The recovery period after an endurance event may be influenced by the interplay of neuromuscular fatigue, exercise-induced muscle damage (EIMD), and endurance training history. A recent study demonstrated a positive relationship between running performance during the recovery period after an ultramarathon race, and prior endurance training and racing experience [12].

This review will discuss the literature on the effects of endurance training, exercise induced muscle damage, neuromuscular fatigue, prior experience, and how these variables may influence endurance running performance. The main focus of the review will be to examine the interaction of neuromuscular and mechanical factors on endurance running performance, and how these factors might influence recovery after an endurance event.

Data were sourced from sports medicine and physiology literature utilising searches on Medline and PubMed. Keywords used in the search included: *"endurance performance," "endurance training," "recovery following endurance activities," "marathon performance," "physiology of endurance sports," "fatigue," "neuromuscular fatigue," "stretch shortening cycle," "exercise induced muscle damage," "prior endurance running experience," "performance markers in endurance sports," "teloanticipation," "running economy," "rate of perceived exertion,"* and *"5 km time trial reliability".*

2.2 ENDURANCE TRAINING

Primary factors affecting distance running performance are maximum oxygen consumption (VO_{2max}), lactate threshold and running economy [35]. However, elite distance runners exhibit similar levels of $VO_{2max,}$ lactate threshold and running economy, which indicates that other factors may contribute to performance [37]. Noakes [27] suggested that muscle power factors, such as the rate and force of cross-bridge activity, may limit endurance performance.

Key components of a training programme are the volume, intensity and frequency of exercise sessions [29]. These three inputs may collectively be called the training stimulus. The training stimulus may either enhance the fitness or reduce the performance capacity of an athlete [29]. Endurance training leads to increased aerobic capacity [30][28]. Improvements in peak oxygen uptake (VO_{2max}) after endurance training are associated with changes in cardiovascular, metabolic and muscular responses to exercise [28]. Cardiovascular changes include increased working muscle capillary density, increased blood volume and thus a resultant decrease in heart rate at similar exercise intensities [29]. Muscular changes associated with endurance training include greater muscle glycogen storage and an increase in the sodium - potassium adenosine triphosphatase (Na^+ - K^+ ATPase) activity, as well as an increase in mitochondrial enzymes [28]. Endurance training has also been shown to lower plasma lactate concentrations at similar work rates due to a greater mitochondrial capacity to oxidise fat [41][28]. The higher fat oxidation rate may help extend moderate endurance exercise by preserving carbohydrate stores [41].

These training adaptations may be associated with a reduction in the reliance on anaerobic energy systems, and increased time to fatigue [42]. Coggan and Williams [41] suggested that training status has an important role in endurance running, as highly trained athletes oxidise less carbohydrate and have higher glycogen stores when compared to sedentary individuals for the same work load. This was thought to be due to a lower rate of glycogenolysis during the exercise bout [41].

Hauwsswirth and Lehanaff [43] found that a gradual progression in training results in a more stable adaptive response and allows a greater chance to overcome strenuous race conditions without compromising the athletes physiological integrity [43]. In the following sections the physiology of endurance training will be discussed in more depth and the effect of endurance training on running performance will be presented.

2.2.1 Physiology of endurance training

The chronic responses of skeletal muscle to endurance exercise involve morphological, acid-base and substrate metabolic changes [44]. These chronic adaptations in skeletal muscle are the result of the cumulative effect of repeated bouts of exercise, while the initial signalling responses that lead to these chronic adaptations occur after each training session [45]. Training responses have been found to be directly proportional to the volume of work performed, although there is a maximal duration beyond which additional training does not cause any further increase in functional capacity (that is, an increase in capillary and mitochondrial density or enzyme function) [29]. This means that the adaptive responses are eventually balanced by exercise duration and a plateau is reached [46].

Muscle composition has been seen to differ between elite sprint athletes and endurance trained athletes, with the sprinters having a marked predominance of type II fibres in their leg muscles, and the endurance trained athletes having a high proportion of type I fibres [46].

Type I fibres have a higher capillary density and oxidative potential than type II fibres, and the energy cost per unit force per cross-sectional area is greater in type II than type I fibres [46]. Coyle, Feltner and Kautz [47] found in a cross-sectional study that the number of type I fibres in trained athletes is related to the number of years of prior endurance training.

There is also evidence that endurance training increases the capillary supply to skeletal muscle and thus in highly trained muscle the diffusion distances for substrates and gases are reduced [29].

The acid-base status of an individual is another chronic response to endurance training. An athlete's maximal sustainable power output is highly related to their lactate threshold. The rate of lactate disposal must be greater than or equal to its rate of production for a steady state plasma lactate concentrations to occur [29]. In endurance trained athletes the capacity to transport lactate across the sarcolemma is significantly higher than in untrained individuals [29]. In the trained athlete one of the most consistent adaptations is an increase in the number of muscle mitochondria. The increase in the number of mitochondria is reflected by the changes in the maximal activity or protein content of enzymes in the in tricarboxylic acid cycle and electron transport chain [44][29]. This adaptation of skeletal muscle metabolism occurs as a result of a tighter regulation in the coupling between adenosine-triphosphate (ATP) supply and demand [48][44]. The tighter regulation of ATP supply and demand is associated with less stimulation of glycolysis, resulting in a decrease in lactate production and glucose utilisation [48]. Endurance training also results in an improved utilisation of fats [41]. This is due to an increased oxidation of muscle triglyceride, as extra muscular fat oxidation and turnover are reduced with training [29]. These adaptations favour the oxidation of fat-based fuels over carbohydrate-based fuels during exercise [46]. Collectively, these adaptations may be associated with improved exercise performance in endurance runners due to maximal energy production from anaerobic and aerobic pathways, increased economy of the movement, and increased resistance to fatigue [29].

2.2.2 The effect of endurance training on running performance

Athletic performance is known to be related to genetic and training factors. The genetic factors are fixed; however in contrast, physical training may have profound effects on physiological adaptations and athletic performance [49]. The most important physiological determinants of endurance running performance are $VO_{2max,}$ lactate threshold and running economy, thus effective training programmes should focus on the enhancement of these factors [49].

Distance runners have typically used long slow distance (LSD) training, which is relatively high mileage of moderately paced running, and little emphasis has been placed on high intensity training [50]. Increasing the volume of the LSD training has been shown to be ineffective for enhancing the VO_{2max} of already well trained athletes [50]. Training intensity has been shown to be an important variable that can be manipulated to increase $VO_{2max.}$ It has been suggested that to enhance $VO_{2max,}$ runners should train at 90% to 100% of VO_{2max}. Training at this intensity should place maximal stress on the physiological processes and structures that limit $VO_{2max,}$ thus providing an optimal stimulus for adaptation [49].

Improved endurance performance has been attributed to an increased lactate threshold [32]. Lactate threshold refers to the point at which blood lactate accumulates above resting values during increased exercise intensity [51]. Accordingly, the rate of lactate disappearance must be greater than or equal to its rate of appearance or production for steady state plasma lactate concentrations to exist [51]. The capacity to transport lactate across the sarcolemma is significantly higher in trained athletes [29]. A runner with a high lactate threshold is able to run at a high percentage of VO_{2max} before the lactate production rate exceeds lactate removal rate [37]. High intensity anaerobic workouts have been shown to increase lactate transporter levels.

These workouts undertaken twice a week for as little as three weeks have been shown to increase muscle buffering capacity in already well-trained athletes [52]. These results show that a large volume of endurance training alone may not be sufficient to improve the ability to transport lactate. Table 2.1 provides a summary of relevant experimental studies that assessed various training methods on endurance running performance [53][54][55][56][36][57][58][59][38][52].

Table 2.1: Summary of studies assessing the effects of different training methods on endurance running performance.

Article	Study objective	Participants	Training intervention	Outcome measures	Results	Conclusions
Acevedo and Goldfarb(53)	Determine the effects of increased training intensity in trained distance runners.	7 competitive long distance runners. 20-35 years old.	8 weeks of high intensity training at 90-95% of peak heart rate.	VO_2 max, plasma lactate accumulation ventilatory threshold , performance.	Increased 10 km running performance by 3% ($p < 0.05$) increased supramaximal running endurance by 20% ($p < 0.01$).	Previously trained runners can increase training intensity to improve endurance performance by lowering lactate at the intensity at which they trained despite no changes in VO_2 max and ventilatory threshold.
Hickson et al(54)	To obtain information regarding the time course and magnitude of the increase in VO_2 max and endurance that occurs in response to strenuous exercise when the training stimulus is kept approximately constant relative to maximum aerobic capacity.	8 sedentary and recreationally active participants aged between 18 and 35 years.	10 weeks of high intensity training (alternating intervals of 40 mins cycling intervals VO_2max and 40 min high intensity running).	Endurance performance, VO_2 max, and time to attainment of peak heart rate.	Average VO_2 max increased 5% ($p < 0.05$) during the 1st wk. Endurance, Vo2 max, and time to attainment of peak heart rate all increased linearly during the 10 wk. The average weekly increase in VO2 max was 0.12 l/min. The total increase in Vo2 max averaged 16.8 ml/kg per min (44%).	High intensity training can elicit a rapid improvement in aerobic work capacity.

Table 2.1: Summary of studies assessing the effects of different training methods on endurance running performance (continued)

Article	Study objective	Participants	Training intervention	Outcome measures	Results	Conclusions
Billat et al(55)	Determine an appropriate training stimulus between inefficient training and overtraining.	8 male middle and long distance runners aged between 20-40.	8 week trial, 4weeks of normal training then 4 weeks of overtraining.	VO2max, running economy, time to exhaustion, heart rate, lactate threshold.	Normal training significantly improved velocity as a result of improved running economy ($p = 0.02$). Heart rate decreased significantly after Normal Training ($p < 0.01$). Lactate threshold remained the same after normal training. Overload training changed neither the performance nor the factors concerning performance. Submaximal heart rate decreased after overload training maximal heart rate was not significantly different after NT and OT ($p = 0.1$).	Performance and aerobic factors associated with the performance were not altered by the 4 wk of intensive training at VO2max.
Lehman et al(56)	Investigating overtraining as a cause of stagnation or decrease in performance capacity of athletes.	8 middle and long distance runners aged between 20-45.	Induce an overtraining syndrome over 4weeks based on an increase in training volume from an average 85.9 km (week 1) to 115.1 km (week 2) and 143.1 km (week 3) to 174.6 km per week (week 4).	cardiovascular, metabolic and hormonal parameters.	Stagnation in endurance performance capacity and a decrease in maximum working capacity were observed. Glucose, lactate, ammonia, glycerol, free fatty acids, albumin, cholesterol, haemoglobin level, leukocytes, and heart rate decreased significantly ($p=0.01$). Urea, creatinine, uric acid, serum electrolytes (except phosphate and calcium) remained constant.	The sportsmen could neither improve nor could they even approximately reach their personal records during the subsequent competitive season due to the overtraining.
Paavolainen et al(36)	To investigate the effects of simultaneous explosive strength and endurance training on performance.	18 endurance trained athletes, 10 experimental and 8 control.	9week testing, total training was kept similar but 32% of training in the experimental group was replaced by explosive type strength training.	Performance of 5 km time trial, running economy, maximal 20m speed, jump tests. Maximal velocity in anaerobic and aerobic running tests. VO_2max.	5 km time, running economy and maximal velocity in anaerobic and aerobic running tests improved ($p < 0.05$) in the experimental group. There were no changes seen in the control group. Maximal 20m speed and jump test increase ($p < 0.01$) in the experimental group and decreased ($p < 0.05$) in the control.	Simultaneous explosive strength and endurance training improved the 5km time in well trained athletes without changes in VO_2max.

Table 2.1: Summary of studies assessing the effects of different training methods on endurance running performance (continued)

Article	Study objective	Participants	Training intervention	Outcome measures	Results	Conclusions
Hakkinen et al(57)	Effects of concurrent strength and endurance training versus strength training only.	11 men in the strength and endurance group (SE) 16 men in the strength group (S) aged from 23-24years of age.	training period of 21 weeks.	EMG, Maximal isometric force, rate of force production, cross-sectional area, muscle fibre proportion, VO_2max, one repetition max.	No changes occurred in the first week. After 21weeksof training: increases in one rep max load (21%) ($p<0.001$) max isometric force (22%) ($p<0.001$) occurred in the S and increases in one rep max load (22%) ($p<0.001$) max isometric force (21%) ($p<0.001$) occurred in SE. There was a 26% ($p<0.05$) increase in EMG for S and 29% ($p<0.001$) in SE. VO_2max increase in SE ($p<0.001$). Rapid force development increased in S.	Concurrent strength and endurance training leads to interference in explosive strength development due to limitations of rapid voluntary neural activation in trained muscles.
Hennessey and Watson(58)	Effects of three preseason training programs on endurance, strength, power, and speed.	56 athletes were divided into four groups: Endurance (E), strength (S), S+E group combined S and E training, control (C) group.	Endurance (E) group completed a running endurance program 4 days week1; the strength (S) group trained 3 days week1; the S+E group combined S and E training programs 5 days week-1; the control (C) group did not train. They were followed up after 8 weeks.	Performance, Power (vertical jump) speed (20m sprint time) strength.	After 8 weeks, the E and S+E groups had similar gains in endurance running performance ($p<0.05$) , the S group had no change, while the C group showed a decline ($p<0.01$). No strength gains were noted in the C or E groups, but strength gains were made in the S+E and S groups. Power (vertical jump performance) and speed (20-m sprint time) Gains were noted only for the S group ($p<0.05$).	These findings show that training for strength alone results in gains in strength, power, and speed while maintaining endurance. S+E training, while producing gains in endurance and upper body strength, compromises gains in lower body strength and does not improve power or speed.

Table 2.1: Summary of studies assessing the effects of different training methods on endurance running performance (continued)

Article	Study objective	Participants	Training intervention	Outcome measures	Results	Conclusions
Iaia et al(59)	The effect of an alteration from regular endurance to speed endurance training.	Seventeen endurance-trained runners were assigned to either a speed endurance training (SET) group (9) or a control (8) group (Con).	4-wk intervention period, SET replaced the ordinary training (45 km/wk) with frequent high-intensity sprint sessions with additional 10 km/wk at low running speed, whereas controls continued the endurance training	Oxygen uptake, blood lactate, energy expenditure during submaximal exercise and its relationship to mitochondrial uncoupling protein 3 (UCP 3) capillarization.	After the IT period, oxygen uptake was 6.6, 7.6, 5.7, and 6.4% ($p < 0.05$) lower at running speeds of 11, 13, 14.5, and 16 km/h, respectively in SET whereas remained the same ($p = 0.06$) in Con. No changes in blood lactate during submaximal running were observed. The protein expression of skeletal muscle UCP3 tended to be higher in SET maximal oxygen uptake and 10-km. Performance time, remained unaltered in both groups. In SET, the capillary-to-fibre ratio was the same before and after the intervention period.	Speed endurance training can maintain muscle oxidative capacity, capillarization, and endurance performance in already trained individuals despite significant reduction in the amount of training. And results in lowered energy expenditure.
Nummela et al(38)	The effects of the neuromuscular and force-velocity characteristics in distance running performance and running economy.	Eighteen well-trained male distance runners with a mean age of 34 years	Runners performed five different tests: 20 m maximal sprint, running economy, 5 km time trial, maximal anaerobic running test (MART), and a treadmill test to determine VO2max.	Average EMG ratio, max 20m speed, running economy, MART, VO_2max.	A significant relationship also existed between running economy and MART VO2gain ($p<0.001$). A significant correlation existed between 5km speed and Average EMG ratio($p<0.05$) during the ground contact phase at the 3 km suggesting that neural input may affect distance running performance.	The results support the idea that distance running performance and running economy are related to neuromuscular capacity to produce force and that the maximal velocity in the MART can be used as a determinant of distance-running performance.

Table 2.1: Summary of studies assessing the effects of different training methods on endurance running performance (continued)

Article	Study objective	Participants	Training intervention	Outcome measures	Results	Conclusions
Pilegaard et al(52)	The study examined the effect of high intensity exercise training on muscle sarcolemmal lactate transport and the monocarboxylate transporters as well as lactate release during intense exercise.	Seven male participants, they were habitually physically active aged between 20-24 years.	Participants maintained their normal activity pattern during the training period. The training lasted for 8 weeks, and the frequency of training increased from 3 days/wk in the first week to 5 days/wk in the fifth week. The training consisted of dynamic one-legged knee extensor exercise in the supine position on a modified Krogh bicycle.	One-Legged Performance Test, Maximal kicking. Endurance test, Maximal isometric contraction. Muscle sampling.	The rate of lactate transport was 12% ($p<0.05$) higher in the trained than in untrained muscle. The content of monocarboxylate transporters was also higher (76 %) in trained muscle. Release of lactate from the quadriceps muscle at the end of intense exhaustive knee-extensor exercise was similar in the trained and untrained leg, although the estimated muscle intracellular-to-interstitial gradients of lactate was lower ($p < 0.05$)in the trained than in the untrained muscle. .	The main findings were that high-intensity training induced an increase in the sarcolemmal lactate transport capacity as well as an enhanced content of monocarboxylate transporters. These result suggest that these transporters can be altered by intense training and that they play an important role in the regulation of pH in human skeletal muscle.

2.2.2.1 Changes in running economy as a result of endurance training

Running economy is defined as the energy demand for a given velocity of submaximal running and is an important predictor of aerobic running performance, particularly elite runners who have a similar aerobic power (i.e. maximal oxygen uptake, VO_{2max}) [60]. Athletes with a high running economy have a lower energetic cost at submaximal velocity and consequently tend to run faster at a given distance or are able to run longer at a constant velocity [60]. There is a close affiliation between running economy and aerobic running performance with running economy being a better predictor of performance than VO_{2max} [9]. Training volumes and the number of years of running experience may be important variables for improved running economy [49]. Mayhew [61] found that the number of years of training significantly correlated with running economy. Therefore, a critical factor in enhancement of running economy may be the cumulative distance the runner has covered over the years of training and not volume at a specific time period [61]. Midgley et al [49] explained that this may be related to continued long term adaptations in skeletal muscle or a slow, progressive improvement in mechanical efficiency.

Other factors that have been proposed to have an influence on the running economy of trained athletes include the oxidative muscle capacity and muscle stiffness [11]. The oxygen cost may be modified by interventions such as training, environment and muscle damage while the muscle stiffness refers to the ability of the muscle to store and release elastic energy [9]. There are significant associations between running economy and muscle stiffness, and the ability to develop muscular force [62]. The stiffer muscle-tendon complexes increase elastic energy storage by reducing the ground contact time and thus reducing the running oxygen cost [23]. Similarly a higher rate of force development of a muscle is associated with a shorter time to generate a contraction thus also reducing the ground reaction time and running oxygen cost [21]. Dalleau, Belli, Bourdin and Lacour [63] concluded that the energy cost of running is significantly related to the stiffness of the propulsive leg.

Byrne et al [64] observed that reduced neural activation in the eccentric phase of the stretch shortening cycle resulted in impaired muscle-tendon elastic properties and thus caused the mechanical efficiency to decrease. These findings indicate a potential link between neuromuscular and biomechanical efficiency during the running stride.

In summary, for an endurance athlete the goal of training is to increase the ability to sustain a level of power or speed for a certain distance or time. This means that training to improve the endurance capacity of an athlete should aim to cause physiological and metabolic adaptations that will allow the athlete to maximise energy production from anaerobic and aerobic pathways, increase economy of the movement involved, and increase the working muscles resistance to fatigue [29]. It is evident that endurance training has predominantly positive effects on endurance running performance. The next section will review the physiology of exercise induced muscle damage, and the effects of muscle damage on running performance.

2.3 EXERCISE INDUCED MUSCLE DAMAGE

Exercise induced muscle damage occurs following unaccustomed or intense exercise, and is commonly associated with lengthening muscle actions as seen in marathon running [60][64]. During a lengthening muscle action, the muscle lengthens while simultaneously contracting against the force acting to lengthen it [65]. Lengthening muscle actions induce damage to the sarcomeres, sarcoplasmic reticulum and sarcolemma; these structural changes in turn affect the strength-generating ability of the affected muscles [66][67][68]. In addition, muscle damage has been observed to increase the physiological demand of endurance exercise and to increase thermal strain during exercise in the heat [69][66][64].

Furthermore, exercise induced muscle damage may be associated with a decreased range of movement and leakage of myofibrillar proteins into the blood [64].

The most frequently reported symptoms of exercise induced muscle damage are stiffness, soreness and swelling; and these symptoms are collectively referred to as delayed onset muscle soreness [70][65].

2.3.1 Delayed onset muscle soreness

Delayed onset muscle soreness (DOMS) is a common occurrence in both trained and untrained athletes following unaccustomed or intense exercise [67][71][72]. It is used to describe a combination of clinical signs and symptoms that occur after muscle damage. These symptoms include muscle soreness, weakness, tenderness, loss of range of motion, stiffness and swelling [72][73]. It is characterised by the perception of discomfort starting approximately eight hours post exercise, peaking at 24 to 48 hours and then reducing after seven to ten days [74][73][75]. The sensation of soreness has been thought to arise from damage and inflammation of the muscle and the connective tissue [45][73]. Delayed onset muscle soreness is frequently used as an indirect marker of muscle damage [76][77]. However Nosaka et al [78] showed poor correlations among indirect indicators of muscle damage such as soreness and swelling, thus suggesting that DOMS does not accurately reflect the magnitude of muscle damage [78]. Muscle function may also remain impaired when soreness has dissipated [74][73].

2.3.2 Physiology of exercise induced muscle damage

The exact mechanisms responsible for damage, repair and adaptation from lengthening muscle actions remain inconclusive despite substantial research into exercise induced muscle damage [67]. It has been theorised that the initial events that occur as a direct outcome of eccentric exercise may be divided into mechanical and metabolic factors [79][80].

2.3.2.1 The mechanical theory

The mechanical theory relates to the damage that occurs as a direct consequence of the mechanical loading on the microfibers and includes adaptations of the whole muscle as well as at the level of the myofibrils specifically the cytoskeleton [79]. The lengthening of the sarcomeres is not uniform under lengthening muscle loading [80]. Some sarcomeres resist stretch more than others, possibly due to their myofilament overlap being closer to the optimum value [78][79][64]. As a result of this the weaker sarcomeres take up most of the stretch [67]. These sarcomeres get progressively weaker until there is no overlap between myofilaments [67]. Due to this non uniformity some passive structures assume more tension than others and undergo "popping", which results in Z-band streaming [67]. This Z-band streaming is the disorganisation of the area that joins the repeating contractile components of the myofibrils together and is a direct indication that muscle damage has occurred [73].

Histological examinations have shown direct damage to the sarcolemma, T-tubules and the cytoskeleton following eccentric exercise [73]. The cytoskeleton is comprised of structural proteins such as titin, desmin, nabulin and function to maintain the structural integrity of the myofibrillar lattice [79]. Changes in the cytoskeletal organisation are major contributing factors to the cellular adaptations [79][80].

It has been theorised that high tension can cause breakage of the intermediate filaments of the cytoskeletal Z-bridges and thus result in stretching out of the space between the pairs of intermediate filaments that surround the Z-bands of single sarcomeres causing the destruction and streaming of the Z-bands [80]. Damage to the sarcolemma and T-tubules results in an increase in the intracellular calcium concentration that results in a cascade of metabolic events which eventually lead to myofibre degeneration [81]. Thus repeated lengthening muscular contractions cause failure of these passive structures and are evident as the reduction in the muscles ability to generate force [64].

2.3.2.2 The metabolic theory

The metabolic theory proposes that the initial events in exercise induced muscle damage are caused by metabolic deficiencies within the working muscle, or that these deficiencies increase the vulnerability of the muscle fibre to mechanical stress [81]. During physical activity there is a metabolic flux through oxidative and glycolytic pathways to match the rate of adenosine triphosphate (ATP) synthesis to the rate of ATP hydrolysis [81][82]. However there is consistently a reduced concentration of ATP during muscular activity. This theory suggests that it is possible for the level of ATP to reduce to a concentration that is sufficiently low to induce muscle damage, particularly in the presence of severe glycogen depletion [82]. Warhol et al [26] found that there was focal and confined damage to muscle fibres where there was almost complete glycogen depletion in their histological study on marathon runners [26].

Metabolic muscle injury may also occur due to the disruption of the intracellular calcium homeostasis which leads to the damage seen in exercise induced muscle damage [71]. An increased intracellular calcium level is thought to be caused by the loss of the sarcoplasmic reticulum membrane integrity [80][67].

The influx of calcium into the cytosol initiates a cascade of events which further damage the muscle cell by causing damage to the cytoskeleton, sarcoplasmic reticulum, mitochondria and myofilaments [67]. Loss of membrane integrity causes leakage of intracellular proteins, which may be present in the blood for many days post exercise [67][83].

Lengthening muscle actions that lead to symptoms of exercise induced muscle damage demonstrate an immediate and prolonged reduction in power, impaired neuromuscular control, selective fast twitch fibre damage and reflex inhibition [83].

Endurance exercise in the presence of muscle damage results in an elevated physiological response and an increase in subjective effort, and would likely impair athletic performance [84][64].

The magnitude of muscle damage and loss of muscle function has been found to be attenuated after one bout of eccentric exercise [85]. This is phenomenon called the repeated bout effect, and has a protective effect that is exhibited by reduced decrements and faster recovery of muscle strength and delayed onset muscle soreness [60]. The repeated bout effect has been demonstrated after a few days following eccentric exercise and the protective effects may last up to six months [86]. McHugh [87] hypothesised that the repeated bout effect may be mediated by neural, cellular and mechanical mechanisms. The neural changes which contribute repeated bout effect to the repeated bout effect are increased type I muscle fibre recruitment and synchronising of motor unit firing, improved distribution of the workload among muscle fibres and, improved synergist muscle force production [71].

Mechanical adaptations associated with repeated bout effect are increased muscle stiffness, due to increased intramuscular connective tissue and changes in the intermediate filament system [87]. Cellular adaptations include an increase in the number of sarcomeres in the myofibrils, which is thought to decrease myofibrillar disruption in the next exercise bout, an increase in protein synthesis, strengthened plasma membranes and remodelling of the cytoskeleton [79].

Another adaptation is a reduced inflammatory response due to the decreased mechanical disruption after the first eccentric exercise bout [85].

2.3.3 The effect of exercise induced muscle damage on running performance

Studies have shown that exercise induced muscle damage has a negative impact on endurance running performance [60]. The effect of exercise induced muscle damage on endurance running performance appears to be mediated by an increased perception of effort [33]. It is possible that a higher central motor command may be necessary to produce similar running speeds within injured or sore muscles [88].

Other studies have shown that the brain's response to the inflammation in exercise induced muscle damage may result in central fatigue during endurance events [33]. In this section the effect of exercise induced muscle damage on endurance exercise performance [33][31][32], neuromuscular control [64][32], perception of effort [33][89] and selective fibre damage [33][90][91] will be discussed.

2.3.3.1 The effect of exercise induced muscle damage on endurance exercise performance

It has been established that exercise induced muscle damage causes significant changes within the sarcomere of the exercising muscle [90][79]. It has also been found that these structural changes result in prolonged reduction in muscle strength as well as changes in athletic performance requiring muscle power [65][64][33][34]. However the effect of exercise induced muscle damage on endurance running is unclear [33].

Marcora and Bosio [33] investigated the effect of exercise induced muscle damage on the endurance performance in moderately trained endurance runners. One hundred drop jumps were performed to induce exercise induced muscle damage.

Four direct markers of muscle damage were measured, namely: delayed-onset muscle soreness (DOMS), creatine kinase, mid-thigh circumference and knee extensor strength. The participants performed a standardised constant speed run at submaximal intensity, ten minutes later the participants performed a 30 minute time trial. Forty-eight hours later the participants returned for a post test visit. The results showed a mean four percent difference in endurance running performance between the exercise induced muscle damage group and the control group [33]. It was concluded that participants with muscle damage were at a greater risk of a significant decrease in performance, compared to participants that were not exposed to a bout of exercise that induced muscle damage [33].

A study evaluating the effects of exercise induced muscle damage on perceived exertion and cycle endurance performance was carried out by Twist and Eston [34]. Seven athletes performed two submaximal fixed-load exercise bouts followed by a 5-min time-trial before completing 100 counter-movement jumps. These submaximal exercise bouts and the time trial were repeated at 48 hours and 168 hours following the 100 counter-movement jumps. $VO_{2max,}$ heart rate, respiratory exchange ratio (RER) and blood lactate concentration remained unchanged during the fixed-load exercise bouts following the 100 jumps. However, the rate of ventilation and the ventilatory equivalent ratio for oxygen increased at 48 hours following the counter-movement jumps. RPE values were higher at 48 hours following the jumps. In the time-trial the mean VO_2 peak power output, mean power output, distance covered and post exercise blood lactate were lower at 48 hours following the jumps. These findings indicate that the ventilatory equivalent for oxygen and perceived exertion at submaximal work rates are increased 48 hours following lengthening muscular contractions.

Furthermore it was concluded that exercise induced muscle damage increases the level of perceived exertion and impairs performance during a five minute time trial [34].

A study investigating the cardiorespiratory, hormonal and haematological responses to submaximal cycling performed two days after exercise bouts involving either lengthening muscle actions or shortening muscle actions [31] showed an elevated physiological response to endurance exercise after muscle damaging exercise. In this study six untrained participants performed 15 minutes of submaximal exercise on the cycle ergometer at 80% VO_{2max}. Minute ventilation, breathing frequency, heart rate and perceived exertion were measured. All measurements were significantly higher two days after eccentric exercise when compared the corresponding values two days after concentric exercise, except for VO_{2max}. In another study investigating the effect of exercise induced muscle damage on the blood lactate response to incremental exercise [32], elevated blood lactate concentrations were observed in response to incremental cycle ergometer exercise performed two days after eccentric exercise, although there were no differences for the VO_{2max} or endurance time. In both studies there was no difference observed for VO_{2max} after eccentric exercise [32].

These studies demonstrate that most, but not all physiological responses to endurance exercise are heightened when muscle damage is present, [31][32] and that performance has a greater chance of being affected in athletes with exercise induced muscle damage than those without [33][34]. There is however very limited literature addressing the effect of exercise induced muscle damage on endurance running performance, thus further research is needed.

2.3.3.2 The effect of exercise induced muscle damage on neuromuscular control

Exercise induced muscle damage is commonly associated with a reduction of muscular strength [92][64]. It has been suggested that this reduction in force is attributable to "peripheral" and "central" mechanisms within the neuromuscular system [93]. It is assumed that the reduced neural input to the muscle is a protective adaptation of the neuromuscular system to prevent further damage [93].

This impairment in neuromuscular efficiency also has been shown to outlast the other symptoms of muscle damage such as soreness and loss of strength [64]. Damage to the muscle or connective tissue during eccentric exercise may lead to changes in recruitment patterns or changes in the temporal sequencing of muscle activation patterns [94][64].

Proprioception is also impaired after eccentric exercise [64][94][95]. Weerakkody et al [89] investigated proprioception after eccentric exercise. Participants were asked to generate a given level of force in elbow extensors of one arm and match it with their other arm. Participants achieved matched accuracy to within a few percentage points. Elbow flexors of one arm were then exercised eccentrically. Participants made large errors; the direction of the errors was consistently with matching efforts and not forces [89]. This indicates that participants with exercise induced muscle damage perceive higher effort when producing the same force and produce less force for the same effort [33][89]. In a different study investigating proprioception and motor control following lengthening contractions, with the exercised arm acting as its own control [94]. The misjudgements and disturbed motor outputs which occurred were thought be as a result of a mismatch between the central motor command and an impaired motor control after muscle damage [94].

In a study investigating the effects of exercise induced muscle damage on the neuromuscular functioning of the quadriceps muscle [93] the quadriceps muscle of 15 participants were damaged.

This was done by performing four bouts of 25 maximal voluntary lengthening-shortening contractions. Over a period of seven days, the contribution of agonist muscle activation and contractile properties to the change in isometric maximum voluntary torque was investigated. The results showed the isometric maximum voluntary torque was impaired at 24 hours post exercise and 48 hours post exercise, while rate of torque development and voluntary activation were only decreased immediately after the intervention. The contractile properties were impaired immediately after exercise and at 24 hours following the damaging exercise. Sensation of muscle soreness was present at 24 hours, 48 hours and 72 hours post exercise. The conclusion was that the reduced voluntary action and altered contractile properties contributed to the force loss immediately after the lengthening-shortening muscle action exercise. Thereafter, the impairment of the contractile properties were responsible for the reduced torque [93].

Saxton et al [95] examined the effects of exercise induced muscle damage on proprioception and muscle tremor components of neuromuscular function. Twelve participants performed 50 lengthening contractions using the forearm flexors of the non-dominant arm. The muscle tremor, perception of force and joint position were monitored to assess changes in neuromuscular function. Serum creatine kinase activity increased after exercise. This was accompanied by prolonged impaired joint range of motion and reduced maximum strength. Muscle soreness peaked three days post exercise. Muscle tremor amplitude was increased until 48 hours after exercise, whereas the muscle power was unaffected. Perception of joint position and perception of force were significantly impaired when the control arm acted as the reference. Joint positions were more accurately reproduced when the experimental arm acted as its own reference. Saxton et al [95] concluded that the increased muscle tremor amplitude and loss of proprioceptive function in the days after the lengthening contractions suggest a significant impairment of neuromuscular function [95].

These studies indicate that exercise induced muscle damage, impairs proprioception [64][89][94][95], as well as the force production ability of the muscle due to impaired motor control [93][94]. Both force production and proprioception are important factors in endurance running and thus a deficiency in these will impact negatively on endurance performance [96][97].

2.3.3.3 The effect of exercise induced muscle damage on selective fibre damage

Investigations have reported selective damage to type II muscle fibres following eccentric exercise [90][91]. Friden and Lieber [90] found myofibrillar disruption in type II fibres to be three times more prevalent when compared to type I fibres three days after eccentric cycle ergometer exercise. This mechanism may occur during the initial stages of eccentric exercise as type II fibres are instantaneously fatigued. As a result of their inability to regenerate ATP; these fibres enter a state of rigor and result in mechanical disruption of the muscle.

In muscle with a mixed muscle fibre composition, motor units differ in their vulnerability to active lengthening because of differences in optimal length-tension characteristics [91]. Brockett et al [91] suggested that it is possible that as type II muscle units exert a given force at a shorter optimal length a resulting stretch of the muscle fibre will lead to greater disruption of the type II fibres. Structurally there are differences between the two fibre types which may predispose these fibres to selective damage. Type II fibres have narrower Z-lines that reflect a lower thick and thin filament attachment and therefore a weaker sarcomere connection [90].

Magal et al [98] investigated the relationship between serum creatine kinase activity after exercise induced muscle damage and muscle fibre composition. Seventeen untrained males underwent a VO_{2max} test, Wingate test, and a protocol to cause exercise induced muscle damage.

Muscle soreness and blood samples were recorded before, immediately after, and 24, 48, 72, and 96 hours after exercise. Biopsy samples were collected one week after the exercise induced muscle damage protocol and were assessed for muscle fibre composition. No significant relationship was found between muscle fibre composition and creatine kinase activity. A significant positive correlation was observed between soreness 48 hours after exercise and type II and IIb fibres, and a significant negative correlation was found between soreness 48 hours after exercise and type I muscle fibres [(98)].

Significant positive correlations were observed between soreness 48 hours after exercise and fatigue, relative average power, and relative anaerobic capacity. It was concluded that creatine kinase activity following exercise induced muscle damage may not be related to muscle fibre proportions, and that high post-exercise muscular pain may be related to a predominance of type II muscle fibres and a higher anaerobic capability [(98)].There is very limited literature examining the effects of exercise induced muscle damage on different muscle fibre types. It has also been suggested by Falvo and Bloomer [(99)] that the available literature may not be applicable to the athletic population. This is due to the more recent studies using a laboratory based approach (using exercises such as unilateral single joint actions) [(85)(86)(95)] rather than sports specific exercises and the participants in the studies often have no formal exercise experience [(99)]. It is therefore evident that further research is needed for specific exercise populations into the effect of muscle type on exercise induced muscle damage.

To summarise, exercise induced muscle damage not only affects muscle strength and power but also has a negative impact on endurance running performance [(97)]. Exercise induced muscle damage affects the perception of effort, and Marcora and Bosio [(33)] suggested that these factors may be responsible for a reduced endurance running performance [(33)].

Highly trained endurance runners may be less likely to suffer from exercise induced muscle damage when compared to moderately trained athletes due to the repeated bout effect [100][33]. The neural changes which contribute to the repeated bout effect are increased type I muscle fibre recruitment [101][91][90]. It has been discussed above that eccentric exercise results in selective damage to type II muscle fibres and therefore the repeated bout effect is an adaptation that greatly reduces the signs and symptoms of exercise induced muscle damage [83][100][101]. Table 2.2 provides a summary of relevant experimental studies that assessed the effects of exercise induced muscle damage on running performance. [65][33][102][103][51][104][105]
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Table 2.2: Summary of studies assessing the effects of exercise induced muscle damage on running performance.

Article	Study objective	Participants	Training intervention	Outcome measures	Results	Conclusions
Highton et al (103)	To study the effects of exercise induced muscle damage on sprint running and agility performance.	12 healthy adults were randomly divided into treatment (n=7) and control (n=5) group they were aged between 20-24 years.	The treatment group completed 100 plyometric jumps, while the control group did not.	Isokinetic peak torque of the knee extensors at 60 and 270 degrees.. Sprint running time over 5 and 10 m. Perceived muscle soreness .Timed agility test and ground contact time during the agility turn point. These measurements were recorded at baseline and then 24, 48 and 168 hours following the plyometric protocol.	Perceived muscle soreness was significantly increased (p 0.05) at each testing day with soreness peaking at 48 hours following EIMD.	The results show that agility and sprint performance is reduced following EIMD. This provides further evidence that performance of activities requiring rapid force generation of is impaired following EIMD.
Gleeson et al (51)	To investigate whether DOMS inducing exercise affects blood lactate responses to subsequent incremental dynamic exercise.	Ten recreationally active subjects (9 male, 1 female), were randomised into two groups (eccentric exercise or control). They had a mean age of 20.	Physiological and metabolic responses to a standardised incremental exercise task were measured two days after the performance of an eccentric exercise bout (Experimental group) or in a control condition of no prior exercise.	Muscle soreness, peak VO2, plasma creatine kinase levels, minute volume, respiratory exchange ratio, heart rate and blood lactate concentration.	Two days following the eccentric exercise protocol, all participants in the experimental group reported leg muscle soreness and showed increased levels of plasma creatine kinase activity ($p < 0.05$). Endurance time and peak VO2 during exercise were unaffected by the eccentric exercise. Venous blood lactate concentration was higher ($p < 0.05$) during exercise after eccentric exercise compared with the control condition. Peak blood lactate concentration, observed at 2 min post-exercise was higher in the experimental group ($p < 0.01$). The increased blood lactate concentration during exercise after eccentric exercise was attributable to an increased rate of glycogenolysis arising from an increased recruitment of Type II muscle fibres.	The increased blood lactate concentration during exercise after eccentric exercise may be attributable to an increased rate of glycogenolysis, arising from an increased recruitment of Type II muscle fibres.

Table 2.2: Summary of studies assessing the effects of exercise induced muscle damage on running performance (continued)

Article	Study objective	Participants	Training intervention	Outcome measures	Results	Conclusions
Tsatalas et al(104)	Effects of localised knee muscle damage on running kinematics at varying speeds.	Nineteen women with a mean age of 23.	The group performed an eccentric muscle damage protocol of the knee extensors and flexors of both legs. Lower body kinematics were assessed during treadmill running at three speeds before and 48 hours after the eccentric exercise.	Isometric torque, muscle soreness and serum creatine kinase activity were measured.	Results showed all indices changed significantly after exercise (p=0.05), indicating muscle injury. Step length reduced and stride frequency significantly increased (p=0.05) 48 hours post exercise, only at the fastest running speed (3m/s). Support time and knee flexion at toe-off increased only at the preferred transition speed and 2.5m/s. Knee flexion at foot contact, pelvic tilt and obliquity significantly increased (p=0.001).Hip extension during stance-phase, knee flexion during swing-phase, as well as knee and ankle joints range of motion significantly decreased (p=0.001) 48hours post exercise at all speeds.	The effects of eccentric exercise on particular tempo-spatial parameters and knee kinematics of running are speed dependent. Pelvic and lower joint kinematics indicate similar behaviour at the examined running speeds. The results provide information on running kinematics at different speeds and how the kinematics are compensated for following muscle damage.
Marcora and Bosio(33)	The effects of EIMD on endurance performance.	Thirty adult runners (24 men and six women) with a mean age of 31 were randomly assigned to EIMD or control.	The EIMD group jumped 100 times from a 35 cm bench, while controls did not perform any muscle-damaging exercise. Participants were tested before and 48 h after treatment. Tests were performed during a constant speed submaximal run for 30 min on a treadmill.	Time trial performance, muscle soreness, perceived exertion, creatine kinase, and knee extensors strength, were measured.	Significant changes in muscle soreness, creatine kinase, and knee extensors strength ($p<0.01$) were seen. EIMD significantly reduced self-paced time trial performance by 4% ($P<0.01$) and participants reduced running speed ($p=0.02$), with no change in perceived exertion ($p=0.31$). No significant alterations in running economy and other physiological responses to submaximal running were found. There was a trend ($p=0.08$) for increased perceived exertion, which was correlated with decreased time trial performance ($p<0.01$).	EIMD had a significant impact on endurance running performance. This was found to be mediated by alterations in perceived effort.

Table 2.2: Summary of studies assessing the effects of exercise induced muscle damage on running performance (continued)

Article	Study objective	Participants	Training intervention	Outcome measures	Results	Conclusions
Twist and Eston(65)	Effects of exercise induced muscle damage on fixed-load cycling and 5-min time-trial performance.	Seven athletes with a mean age of 22years.	The perticipants performed two submaximal fixed-load exercise bouts followed by a 5-min time-trial before, 48 and 168 hours following 100 counter movement jumps.	VO_2 heart rate, RER and blood lactate concentration, RPE and peak power output.	Blood lactate concentration , VO_2, heart rate, RER were unchanged during the fixed-load bouts following jumping exercise. VE and VE/VO_2 increased ($p < 0.05$) at 48 hours after the jumps. RPE increased at 48 hours, as did the ratio of RPE:HR and RPE:VO_2 ($p < 0.05$). In the time-trial, mean VO_2 peak power output, mean power output, distance covered and post exercise blood lactate concentrations were lower at 48 hours after the jumping protocol ($p < 0.05$). RPE did not change between trials.	Ventilatory equivalent for oxygen and perceived exertion at submaximal work rates are increased 48 hours following eccentric exercise. EIMD increased perceived exertion and impaired performance during a 5-minute sprint.
Burt and Twist(105)	To investigate whether a single bout of plyometric exercise, affected cycling endurance performance.	Seventeen participants were randomly assigned to a muscle damage (n = 7) or non muscle damage (n =8) group aged from 20-30 years.	Before and at 48 hours, participants were measured for peak isokinetic strength, muscle soreness, and physiological, metabolic, and perceptual responses during 5-minute submaximal cycling at ventilatory threshold (VT) and a 15-minute time trial.	Perceived muscle soreness, peak isokinetic strength. Physiological, metabolic, and perceptual responses during a 5-minute cycle and 15-minute time trial.	Perceived muscle soreness and isokinetic strength were significantly altered in the muscle damage group after EIMD ($p<0.05$). No changes in heart rate or blood lactate were measured during submaximal exercise ($p >0.05$). VO2,VE, and rating of perceived exertion values were increased at VT in the muscle damage group at 48 hours after EIMD ($p<0.05$). During the time trial, mean power output, distance covered, and VO2 were lower in the muscle damage group at 48 hours after EIMD ($p >0.05$). There were no changes in RPE ($p > 0.05$).	Individuals using concurrent plyometric and endurance training programs should be aware of the acute impact of muscle damaging exercise on subsequent cycling performance.

2.3.4 Measurement of exercise induced muscle damage

Muscle soreness is commonly used as an indirect marker of exercise induced muscle damage and delayed onset muscle soreness [33]. It has also been shown to be the most well recognised indicator of muscle damage among athletes [64]. Subjective measures of muscle pain include numerical scales, questionnaires and visual analogue scales (VAS). The visual analogue scale is a commonly used tool to determine pain intensity. It has been found to be sensitive, have good test-retest reliability and has been validated as accurate, reliable and appropriate in evaluating pain intensity following delayed muscle soreness when repeated measurement is involved [106].The visual analogue scale has been used in studies to establish the time course of muscle pain associated with DOMS and to assess the effects of various interventions on muscle pain [106]. The use of a multidimensional VAS provides a clear description of muscle pain associated with DOMS [107]. There is evidence that a multidimensional pain scale may be more effective than a unidimensonal scale in detecting small differences in pain [108]. A multidimensional VAS was used in the study investigating the differences in running performance during the recovery period after an ultramarathon race, as seen in Chapter Three (Section 3.4.4, page 65).

Objective measures of exercise induced muscle damage have been acquired by using a "myometer" to measure applied force to a muscle group at the pain threshold [64]. Direct methods of assessment of exercise induced muscle damage involve muscle biopsy and magnetic resonance imaging (MRI). Muscle biopsy is invasive and the muscle damage is not uniformly distributed throughout the muscle, therefore the biopsy may not be a true representation of what is happening throughout the damaged muscle [109]. Magnetic resonance imaging is expensive and often inaccessible. The interpretation of MRI findings, in relation to exercise induced muscle damage has not been clearly established [102].Damage to the myofibrils following eccentric exercise results in a leakage of myofibre proteins into the blood plasma.

The most common protein which is used as an indirect marker of the muscle damage is creatine kinase (CK) [102]. Studies have demonstrated that the biochemical indices of inflammatory mediators (for example, platelet activating factor), and collagen degradation (for example, hydroxylysine) may also be used as markers of exercise induced muscle damage [110].

Endurance performance is significantly affected by symptoms of exercise induced muscle damage [33][64][65][77]. Nummela et al [38] found that the fatigue during the 5 km time trial was related to endurance performance and factors affecting the pacing strategy. In this dissertation, 5 km time trials were used to measure the endurance performance after exercise induced muscle damage occurred.

Fatigue is another factor which is thought to affect endurance running performance. In the next section neuromuscular fatigue is discussed as well as the effects of fatigue on running performance.

2.4 NEUROMUSCULAR FATIGUE

Fatigue is defined as an acute impairment of exercise performance including both an increased perceived level of effort to exert a certain force output and the eventual inability to produce that force output [2]. The decline in force or performance potentially involves the processes at all levels of the motor pathway from the brain to the skeletal muscle [111]. To study fatigue the typical strategy has been to determine whether the mechanism responsible for the fatigue is located in the exercising muscle or in the nervous system. This has resulted in the differentiation between central and peripheral fatigue [112]. Peripheral fatigue refers to the muscle contractile properties and the transmission and conduction velocity of the action potential from the nerve endings across the sarcolemma; whereas central fatigue includes both spinal and supraspinal factors [113]. Neuromuscular fatigue is used to describe the combined effects of both central and peripheral fatigue [114][113][2].

2.4.1. Physiology of neuromuscular fatigue

It is generally accepted that fatigue during short term exercise is mainly as a result of metabolic factors and muscle damage, particularly if lengthening muscle actions are involved, and thus fatigue is predominantly due to peripheral mechanisms [115]. Muscle damage results in an immediate and prolonged reduction in muscle force-generating capacity [64]. It has been found that the initial manifestations of muscle damage are due to the physical disruption of the sarcomeres and the components of the excitation-contraction (E-C) coupling system [115]. Excitation-contraction coupling is the sequence of events which allows the passage of an action potential along the sarcolemma resulting in the release of calcium from the sarcoplasmic reticulum [64]. Thus failure of the E-C coupling impairs maximal and submaximal force generation. Peripheral fatigue may also result from alterations in nerve conduction between the nerve ending and the motor endplate [11]. The peripheral fatigue model assumes exercise terminates only after there has been a catastrophic failure in homeostasis [116], and that exercise can only continue until all the available motor units in the active muscle have been activated [117].

Schillings, Hoefsloot, Stegeman and Zwarts [118] investigated the relative contribution of central and peripheral fatiguing factors during a course of fatiguing contractions on 20 healthy participants. To quantify the amount of peripheral fatigue, the force development following a pulse of electrical stimuli on the endplate were compared before and after the contraction. To measure the force loss as a result of central factors, superimposed electrical stimulation was used during the muscle contraction. The influence of peripheral fatigue on the superimposed force responses was taken into account in two ways. The first method compared the force response with the actual voluntary force; the second used an estimation of peripheral fatigue based on linear interpolation between force responses during rest before and after the sustained contraction [118].

The authors demonstrated that during a two minute sustained maximal voluntary contraction of the biceps brachii, the voluntary force decreased to 38%. Peripheral fatigue was responsible for 89% of this decline, and it increased predominantly during the first half of the contraction. In contrast the central fatigue was calculated as 12% and mainly induced a force decrease in the second half of the contraction [118].

Central fatigue is associated with alterations in the function of the central nervous system (CNS). A reduction in CNS output to the muscle may be influenced by afferent feedback from the muscle or by reduction in the corticospinal impulses reaching the motor neurons [2]. Afferent feedback may be affected by both fatigue of the intrafusal fibres of the muscle and changes in the viscoelestic property of the muscle. Local inflammation may also induce motor neuronal inhibition [119]. Reduced central activation may be necessary to protect the body under specific stressful conditions and to protect the neuromuscular system, because maintaining the drive to the muscle would result in a state from which the recovery would be prolonged [115].

In a study examining the time course of contractile and neural alterations of knee extensor muscles during long duration running exercise [120], nine participants were tested at 55% of their maximal aerobic velocity on a treadmill for five hours. Maximal voluntary contraction, maximal voluntary activation level, and electrically evoked contractions (single and tetanic stimulations) of the knee extensor muscles were evaluated before, after each hour of exercise (during 10 minute interruptions), and at the end of the five hour period. Oxygen uptake was also measured at regular intervals during the exercise. The results showed a significant reduction in maximal voluntary contraction and maximal voluntary activation level after four hours of exercise. The reduction in maximal voluntary contraction was highly correlated with the decline of the maximal voluntary activation level. The muscle action potential was also altered after the fourth hour of exercise in the knee extensors. Peak twitch was more powerful at the end of the exercise and tetanic forces were not altered by the exercise [120].

Oxygen uptake increased linearly during the running period. It was concluded that the knee extensor maximal voluntary force generating capability was depressed in the final stages of a five hour run. This impairment of neuromuscular functioning was suggested to be as a result of central activation failure and alterations in muscle action potential transmission [120].

The aetiology of muscle fatigue during prolonged exercise is multifaceted. The relative contribution of central and peripheral fatigue has not been defined [115]. Millet et al [119] characterised neuromuscular fatigue of the knee extensor and plantar flexor muscles after a 65 km ultramarathon race. Nine participants were included in this study. One week before and immediately after the ultramarathon, maximal twitches were elicited by stimulating the femoral and tibial nerves, respectively from the relaxed knee extensor and plantar flexor muscles. Electrically evoked superimposed twitches of the knee extensors were also elicited during maximal voluntary contractions to determine maximal voluntary activation. Maximal voluntary contraction and maximal voluntary activation decreased significantly after the ultramarathon. Peak twitch however increased after the ultramarathon. The compound muscle action potentials were not significantly altered by the ultramarathon. The results revealed a depressed maximal voluntary force capacity because of a decreased maximal voluntary activation, thus highlighting the role of central fatigue after an ultramarathon [119].

Petersen et al [11] investigated muscle mechanical characteristics before and after a marathon race. Eight elite runners underwent a test one week before the marathon, tests were carried out 30 min, two days and five days following the marathon. Countermovement jump power, knee extensor and plantar flexor maximal voluntary contraction as well as muscular contractile properties were assessed. Countermovement jump power decreased 13% post-marathon and remained depressed at day two (18%) and five (12%) post-marathon. Knee extensor and plantar flexor maximal voluntary contraction decreased by 22% on day two and 17% on day five post-marathon [11].

There were no significant changes in evoked contractile parameters of the muscles. It was concluded that the muscle power decreased acutely post-marathon race and recovered very slowly. The decrease in maximal voluntary contraction occurred in the absence of any worsening of the contractile properties of the muscles. It was furthermore concluded that the fatigue following a marathon was of central rather than peripheral origin [(11)].

Martin et al [(111)] examined the contributions of central and peripheral factors to neuromuscular fatigue induced by a 24-hour treadmill run. Neuromuscular function evaluation tests were conducted. These consisted of the determination of the maximal voluntary contractions of the knee extensors and plantarflexors, the maximal voluntary activation of the knee extensors and plantarflexors, and the maximal compound muscle action potential amplitude of these muscles. Tetanic stimulations were delivered to evaluate the presence of low-frequency fatigue and the knee extensor maximal muscle force production ability. Strength loss occurred throughout the exercise, with changes observed in maximal voluntary contractions for both muscle groups, together with reductions in maximal voluntary activation. A reduction of maximal compound muscle action potential amplitude was only seen in the plantar flexors. The knee extensor maximal force production ability was reduced moderately at the end of the 24 hour run. It was theorised that central fatigue was the principal explanation for the neuromuscular fatigue experienced after a 24 hour running bout, and that this limited the extent of peripheral fatigue [(111)].

Although the relative contributions of central and peripheral fatigue on endurance running performance have not yet been established, [(115)(119)(118)] there is an interplay of both factors during prolonged exercise [(111)(119)]. Another type of fatigue needs to be considered during endurance exercise. The repetitive lengthening and shortening muscle actions which occur in normal movement is called the stretch shortening cycle and thus the fatigue resulting from this is referred to as stretch shortening cycle fatigue [(121)]. This will be discussed in the following section.

2.4.2 Stretch shortening cycle fatigue

The stretch shortening cycle of muscle function comes from the observation that body segments are subjected periodically to impact and stretch forces. The combination of lengthening and shortening muscle actions forms a natural type of muscle function called the stretch shortening cycle [121]. In stretch shortening cycle exercise, the action on the muscle in the lengthening phase influences the subsequent shortening phase. These influences take place in both the elastic components of the muscle and the neural input [122]. In a stretch shortening cycle activity such as running the muscle activation peaks before the lengthening phase ends, and thus is reliant on the effective interaction from stretch reflexes [121]. Stretch shortening cycle fatigue is a relevant model to examine fatigue during endurance running as this exercise comprises of repetitive stretch shortening cycle muscle actions [123]. It is also is a useful model to study the processes of reversible muscle damage and the interaction with muscle mechanics, joint and muscle stiffness [121][124][23]. The stretch reflex contributes significantly to force generation during the transition from the stretch phase to the shortening phase [121]. The amplitude of the stretch reflex component varies according to the stretch-load placed on the muscle and the level of fatigue of the muscle. Moderate stretch shortening cycle fatigue may result in slight potentiation of the reflex contribution while exhaustive stretch shortening cycle fatigue may dramatically reduce the same reflex contribution [124][23]. Two primary mechanisms have been proposed to explain the reduction in muscle function following stretch shortening cycle exercise. Firstly the failure of maximal muscle functioning may be as a result of impaired peripheral mechanisms; and secondly as a result of impaired neural input to the muscle which may indicate an impaired central mechanism [125][23].

There is evidence that both short and long duration fatiguing exercise results in the deterioration of neuromuscular performance. Stretch shortening cycle fatigue usually leads to a reversible muscle damage, a process which has consequences on the joint and muscle stiffness and reflex intervention [121].

The acute deterioration in performance following a prolonged stretch shortening cycle exercise has been associated with a decrease in neural input to the muscle, these changes occur together with a reduced reflex sensitivity [23]. Asmussen and Mazin [126] suggested that this decline may originate from the fatiguing muscle itself through reflex pathways. This decline has been thought to be advantageous as it helps protect peripheral neuromuscular structures from excessive exhaustion [127]. The mechanical effects of fatiguing stretch shortening cycle exercise also show long lasting consequences similar to that of lengthening muscle contractions [121]. Noakes [27] suggested that repetitive maximal loading of the skeletal muscle and tendon structure may result in damage to these shock absorbing structures, and that there may be a limit to the number of times that these structures may be damaged, after which the central nervous system will alter the recruitment pattern in an attempt to avoid any further damage [27].

Recovery from stretch shortening cycle fatigue is a delayed process and demonstrates a reduction in both maximal muscle power and maximal muscle strength, which in turn affects the running performance [11]. Recovery after stretch shortening cycle exercise has been shown in numerous studies to have a bimodal pattern [121][128][23]. This bimodal trend involves a dramatic decline in both active and passive stretch reflexes immediately after the intensive exercise, followed by a short recovery and then a subsequent secondary decline in performance peaking either around the second or third day post-exercise [128]. The initial decline in performance may be related to metabolic disturbances, whereas the secondary decline may be associated with an inflammatory process related to the muscle damage [121].

Avela et al [23] investigated further mechanisms relating to the acute and secondary impairment of the stretch reflex function, and the possible interactions between reflex sensitivity and the compliance of the muscle-tendon complex. It was concluded that the acute reduction in stretch reflex sensitivity is of reflex origin in the fatigued muscle, thus implicating the regulation of the γ-loop activity to be important [23][10].

There was also the possibility of mechanical or metabolic fatigue of the intrafusal fibres themselves. The secondary decline in the sensitivity of the reflex markers was thought to be attributable to the secondary injury, because of the inflammatory response to the muscle damage. In addition, impaired contractile properties of the muscle could affect the mechanoreceptors and result in presynaptic inhibition [23]. Dousset et al [128] concluded that muscle force-recovery processes result from different combinations between activation of afferent fibres by fatigued induced factors, and with parallel muscle damage and mechanical factors.

Stretch shortening cycle fatigue occurs during endurance running and may explain the effect of fatigue on the athlete's performance [129][123]. This description of fatigue encompasses both the peripheral and central models of fatigue although the relative contributions of the two are unclear [128]. Deterioration in performance following a prolonged stretch shortening cycle exercise has been associated with a decrease in neural input to the muscle and followed by muscle stiffness [74][76][77]. Recovery following prolonged stretch shortening cycle exercise has not been adequately determined [121][128][23], thus further research is needed to assess the effect stretch shortening cycle fatigue has on running performance after endurance events such as an ultramarathon.

2.4.3 The sensation of and measurement of fatigue

Fatigue is not purely a physical event that occurs during exercise but may also be viewed as a conscious sensation that results from the interpretation of subconscious regulatory processes in the brain [130]. The conscious perception of effort is thus a link between the behavioural changes that may be required to maintain homeostasis and the physiological parameters affected by exercise [5]. The sensation of fatigue is a component of cognitive decision making and may be based on prior experience. However, the areas of the brain are involved in the processing of this sensation are still unknown [5]. Subjective feelings of fatigue are influenced by the expectation about task duration. It has been found that subjects describe higher levels of exertion at the same absolute time point during a shorter duration exercise bout, compared to a longer duration exercise bout, despite performing the different duration exercise bouts at the same intensity [131]. Thus a particular level of perceived exertion results from the interpretation of afferent sensations against an expected outcome, in a teloanticipatory process [131].

The conscious perception of fatigue has traditionally been measured on the rate of perceived exertion (RPE) scale described by Borg [4], to measure the degree of heaviness and strain experienced in physical work [5]. It is believed that the perception of effort is derived from sensory input arriving from a variety of different biological systems such as the musculoskeletal, cardiovascular and respiratory systems, and that the brain monitors these systems during exercise. Depending on the circumstances the exercise is regulated to prevent any damage or harm to the body [5]. The Borg scale has been the main tool for measuring physical stress since the 1950's and has been combined with heart rate to describe intensity level during any given exercise session [132]. Borg claimed a high correlation existed between his scale and heart rate, suggesting that a perceived RPE value, multiplied by 10, would equal an actual heart rate at that moment [133].

This scale is an affordable, practical and valid tool for monitoring exercise intensity, independent of gender, age, exercise modality, and physical activity level [134][135]. The Borg scale was used in this study to determine perceived levels of exertion. Nethery [136] argued that the rating of perceived exertion is not purely the result of afferent sensory feedback but may be set at the beginning of the exercise bout as a feed forward control mechanism; thus causing the RPE to rise linearly to reach maximum levels at exercise completion. Noakes [39] suggested that the brain uses scalar rather than absolute time to set perceived exertion at any time point during an exercise bout, and that the perceived exertion for the exercise bout is set in an anticipatory manner at the start of the exercise.

The OMNI scale is a perceived exertion scale which is used for a variety of physical activity settings. OMNI is short for omnibus which refers to the broadly generalised measurement properties of the scale [137]. This scale has been shown to have concurrent and construct validity to estimate rate of perceived exertion during graded exercise testing on a treadmill [138]. The OMNI scale makes use of pictures of an individual exercising at different levels, the pictures are combined with short verbal cues and arranged along a numerical scale from zero to ten [138][137][139]. This scale was however not used in the study as it was thought to be unfamiliar to the exercising population and time consuming to use [139][140].

2.4.4 The effect of neuromuscular fatigue on running performance

Noakes [117] described a neural control system in the brain and spinal cord that controls the number of motor units activated during exercise to ensure the maintenance of homeostasis. This model is called the Central Governor Model, and it describes the integration of sensory information from the periphery and the appropriate exercise response to maintain homeostasis.

Thus this model predicts that exercise performance is regulated by the recruitment of exactly the appropriate amount of muscle fibres to ensure the exercise bout is completed safely. Impaired performance that develops during exercise, and the differences in intra-individual performance, may be explained by the changes in the number of skeletal motor units recruited during exercise [39].Weir et al [141] argue that the Central Governor Model cannot explain all observations of fatigue, and that the arguments to support this model rely on disproving the "catastrophe models" or lactic acid models of fatigue. Weir et al [141] propose a concept of task dependency, which is that the mechanisms of fatigue vary depending on the task being performed. This view attempts to explain fatigue through a common mechanism in which both central and peripheral factors contribute to muscle fatigue.

Nicol and Komi [142] investigated the influence of marathon fatigue on running kinematics and economy. Eight participants were used in the study, treadmill tests were performed at three steady submaximal speeds before and after the marathon. The contact time, flight time as well as the displacements and angular velocities of the left hip and knee were analysed. Electromyographic measurements of the gastrocnemuis were taken during the treadmill tests. The results showed that the energy expenditure was not related to the kinematic changes and that there were large variations between individuals as fatigue progresses. It was hypothesised that the observed kinematic changes may reflect an adaptation to fatigue rather than failure to compensate for it [142].

In another study by Nicol et al [143], repeated stretch shortening cycles of muscle activity during marathon running was associated with reduced force production of the participants. This was found to be as a result of reduced neural input to the muscles that caused deterioration of the contractile efficiency, thus implicating central fatigue mechanisms for the reduction in strength [143]. Further, Nicol et al [144] tested nine experienced endurance runners to investigate effect of marathon running on neuromuscular performance.

The participants performed individual marathon runs that involved several tests of neuromuscular performance before, during and after the marathon. The results showed an overall decrease in performance from the marathon. Sprint velocity decreased during the marathon. Similarly, the maximal isometric knee extension torque and drop jump performance were reduced following the marathon. These reductions were accompanied by alteration in the ground reaction forces, suggesting a reduced tolerance to stretch load as well as loss in the recoil characteristics of the muscles. The integrated electromyographic activity of both vastus medialis and vastis lateralis decreased after the marathon. These results indicate a modification in the neural input to the muscle following the marathon and thus explain the alteration in performance of the participants from before to after the race [144].

The findings of these studies support the theories stating that the central nervous system is mainly responsible for exercise limitation in humans [117] and that there may be a continuous feedback loop between central and peripheral fatiguing factors during prolonged exercise [145][117][118]. Table 2.3 provides a summary of relevant experimental studies that have investigated the effects of neuromuscular fatigue on running performance[122][142][143][144][22][146][147].

Table 2.3: Summary of studies assessing the effects of neuromuscular fatigue on damage running performance.

Article	Study objective	Participants	Training intervention	Outcome measures	Results	Conclusions
Nicol and Komi (142)	To establish the influence of marathon fatigue on running kinematics and economy.	Eight participants with a mean age of 24 years.	Treadmill test at 3 steady submaximal speeds performed before and after the marathon. One complete left leg cycle was videotaped at 100 Hz from the left side at each speed. The electromyographic activity of the gastrocnemius muscle at each running velocity, and energy expenditure (Ė) of the last 30 seconds at the 2 slowest speeds complimented the video measurements.	Submaximal treadmill test , analysis of foot contact time and flight time were measured. Displacements and angular velocities of the left hip and knee. EMG activity of the gastrocnemius muscle and energy expenditure.	The results indicated significant ($p=0.05$) increases of energy expenditure at these the last 30 seconds at the 2 slowest speed. The relative duration of the push-off phase was significantly increased at the 2 slowest speeds ($p=0.001$), and the gastrocnemius muscle presented higher integrated EMG values in the respective braking (NS) and push-off phases at the 2 fastest speeds . This was thought to indicate a loss of tolerance to impact during the belt contact.	Running kinematics and running economy are not interrelated as fatigue progresses. The results indicated that it is possible that the kinematic changes may reflect some adaptation to fatigue.
Kyröläinen et al(22)	Investigating interactions between running economy and mechanics before, during, and after an individually run marathon.	Seven triathletes with a mean age of 29.	Participants performed a 5 minute submaximal running test on a treadmill at an individual constant marathon speed one week before the marathon.	Heart rate and expired respiratory gas were measured during the run. Serum creatine kinase activity (S-CK), skeletal troponin I (sTnI), and blood lactate (B-La). A video analysis to investigate running mechanics. External work of each subject.	The results demonstrate that after the marathon, a standardized 5-min submaximal running test resulted in an increase in oxygen consumption, ventilation, and heart rate ($p< 0.05$). A simultaneous decrease in oxygen difference between inspired and expired air, and respiratory exchange ratio ($p < 0.05$). B-La did not change during the marathon, while sTnI and S-CK values increased ($p < 0.05$), peaking 2 hours and 2 days after the marathon, respectively. Running kinematics showed minor increase in stride frequency and a similar decrease in stride length ($p < 0.01$).	These results demonstrate that running economy cannot be explained by changes in running mechanics. It is suggested that the increased physiological loading is due to increased utilization of fat as an energy substrate, possible muscle damage and increased demands of body temperature regulation.

Table 2.3: Summary of studies assessing the effects of neuromuscular fatigue on damage running performance (continued)

Article	Study objective	Participants	Training intervention	Outcome measures	Results	Conclusions
Nicol et al(143)	Investigate the fatigue effects of marathon running on neuromuscular performance.	Seven participants mean age of 24 years.	Participants performed 2 unilateral isometric strength tests before and after the marathon race.	Maximal isometric voluntary actions of the knee extensor muscles and isometric endurance of 60% of pre-marathon MVA.	Results showed dramatic reductions of the maximal isometric torque (26 14%) and endurance time (39 9%). Maximal integrated electromyographic activity (IEMG) of the vastus medialis muscle decreased by 36 26% and that of the vastus lateralis muscle by 42 25%. In the submaximal endurance test, the post-marathon isometric knee extension started at a higher level of IEMG and lower mean power frequency for 6 of 7 subjects.	These results suggest that the repeated stretch-shortening cycles during the marathon race affected the force production by reducing neural input to the muscles and deteriorating the efficiency of the contractile mechanism.
Nicol el at(144)	The fatigue effect of marathon running on neuromuscular performance. And the changes in muscle force and stiffness characteristics.	Nine endurance runners. Aged 20-30 years old	Participants performed individual marathon runs. Several tests of neuromuscular performance were conducted before, during and after the marathon.	Sprint velocity, maximal isometric knee extension torque, drop jump and 5 jump performance, ground reaction force.	Rresults showed an overall decrease in performance following the marathon. The maximal sprint velocity decreased during the marathon, reaching the final value of 84 % of the pre-marathon one. Other test results after marathon indicate that maximal isometric knee extension torque was 78%, the performance in a special rebound test drop jump 84% and the 5-jump performance 92% of the pre-marathon values. The reductions were also accompanied by changes in the ground reaction force curves in the sprint and jump tests.	These results suggest reduced tolerance to stretch load as well as loss in the recoil characteristics of the muscles following a marathon.

Table 2.3: Summary of studies assessing the effects of neuromuscular fatigue on damage running performance (continued)

Article	Study objective	Participants	Training intervention	Outcome measures	Results	Conclusions
Ross et al (146)	This study investigated peripheral and central fatigue after a marathon on a treadmill.	Nine runners with a mean age of 32 years.	Isometric ankle dorsiflexion force and electromyographic responses of the tibialis anterior in response to magnetic stimulation of the peroneal nerve (PNMS) and the motor cortex (TMS) were measured before, immediately after, 4 and 24 hours post-marathon.	Maximal voluntary contraction, evoked response to magnetic stimulation of the peroneal nerve (PNMS) and the motor cortex (TMS).	Maximal voluntary contraction decreased by 18 7% immediately after the marathon ($p = 0.009$) and remained significantly decreased after 4 hours. The amplitude of the evoked response to TMS, but not to PNMS, was depressed immediately post-marathon by 25% ($p = 0.04$). Resting twitch force was reduced in response to both TMS and PNMS post marathon ($p = 0.035$ and 0.037, respectively), and voluntary activation was reduced to 61.9 18% immediately post-marathon($p < 0.05$). All measures had returned to baseline values after 24 hours.	These results suggest that fatigue was attributable to both a disturbance of the contractile apparatus within the muscle and submaximal output from the motor cortex.
Kyröläinen and Komi (122)	To investigate differences in neuromuscular function between power athletes and endurance athletes.	Participants comprised of ten power athletes and ten endurance athletes with the mean age of 24 years.	Participants performed 10 maximal drop jumps from the optimum dropping height, and sledge jumps. The five to six best jumps were taken for further analysis.	Average angular velocities of the knee and the ankle joints in the drop jumps (DJ) and in the sledge jumps were measured. The mean of the average reaction forces and muscle activity (IEMG) were recorded.	Average angular velocities of the knee and ankle joints did not differ between the subject groups in the braking phase of the take-off either in the drop jumps or in sledge jumps. The contact times were shorter ($p<0.001$) and take-off velocities were higher ($p<0.001$) in the power athletes compared to the endurance-trained athletes in every condition and thus had better physical performance compared to their endurance counterparts.	High and fast pre-activation prepares the muscles to resist the high impact loads during the eccentric phase of SSC. The resulting stiffness further enhances the subsequent efficient recoil characteristics during the push-off phase.

Table 2.3: Summary of studies assessing the effects of neuromuscular fatigue on damage running performance (continued)

Article	Study objective	Participants	Training intervention	Outcome measures	Results	Conclusions
Hobara et al (147)	To examine the determinants of the difference in the leg stiffness between endurance and power trained athletes.	Seven distance runners and seven power-trained athletes with the mean age of 26 years.	The participants performed in-place hopping, matching metronome beats at 3.0 and 1.5Hz. Leg and joint stiffness were calculated from kinetic and kinematics data.	Electromyographic activity (EMG) was recorded from six leg muscles. Hip, knee, and ankle joints were measured for stiffness and touchdown angles.	Tthe power-trained athletes demonstrated significantly higher leg stiffness than the distance runners at both hopping frequencies. Ankle stiffness was significantly greater in the power-trained athletes than the distance runners at 3.0H z ($p<0.01$) as was knee stiffness at 1.5Hz. ($p<0.05$) There was no significant difference was seen in touchdown angle between the groups. Significant differences in EMG activity existed between two groups; it was always greater in the distance runners than the power-trained athletes.	These results suggest that the difference in leg stiffness between endurance-trained and power-trained athletes is best attributed to increased joint stiffness. The difference in joint stiffness between the two groups may be attributed to a lack of similarity in the intrinsic stiffness of the muscle-tendon complex rather than in altered neural activity.

2.5 PRIOR EXPERIENCE

The Central Governor Model [130] explains that exercise pace or intensity is regulated by the brain so that the physiological systems can always be maintained within homeostatic limits. The sensation of fatigue is a protective mechanism which over-rides the desire to increase exercise intensity if homeostasis cannot be maintained at a higher intensity level [148]. During exercise there is constant interpretation of sensations of exertion, which are interpreted by comparing mental representations and beliefs which have been developed through exposure to similar experiences in the past [148]. Ulmer [6] described this feed forward process as teloanticipation. This allows performance beliefs to influence perceptions of exertion, pacing decisions and subconscious efferent muscle control [148][6].

The previous memory of a fatiguing exercise will allow athletes to estimate their energy reserves and tolerance levels, as well as to allow decision making as to whether to continue the activity, reduce the activity, or discontinue the activity. Accordingly, memory formation and decision making will involve representations of sequences of events, within a framework of general semantic knowledge linked by common places and events [130].

Mickelwright et al [148] investigated the effects of experience and feedback on pacing and performance during cycling time trials. Cyclists performed three rating of perceived exertion 20 km time trials. The first time trial was performed without any performance feedback. The second time trial was performed with accurate performance feedback or with false feedback by showing speed to be 5% greater than the actual speed. For the third trial all cyclists received accurate feedback. The results showed that pacing was influenced by the interaction of feedback and previous experience. Cognitive processes such as the interpretation of sensory afferent signals and environmental cues were influenced by mental representations developed during similar experiences that in turn led to a certain pacing and rate of perceived exertion [148].

Furthermore Burgess [12] showed a positive relationship between running performance and previous endurance training and racing experience. The aim of the study was to investigate the effects of exercise induced muscle damage and fatigue induced by an ultramarathon on muscle preactivation and running performance in experienced ultramarathon runners. The participants consisted of an experimental group of 11 male runners competing in a 90 km ultramarathon and a control group of 12 male runners who did not participate in the ultramarathon. Peak treadmill running speed and maximum oxygen consumption were measured two weeks prior to the race. Muscle pain and plasma creatine kinase activity were recorded daily for seven days following the ultramarathon. Track tests consisting of a 5 km time trial, 20 m sprint tests, and a 1.4 km submaximal run were performed seven days before and ten days after the ultramarathon. The results indicated a significant correlation between the change in 5 km time trial time and the total number of years of running, number of marathons and ultramarathons completed for the experimental group. This finding was thought to support the role of teloanticipation and central regulation following fatigue and exercise induced muscle damage [12]. There is a lack of literature investigating the effects of prior experience on endurance exercise and an even more limited field investigating the effects on endurance running performance.

2.6 ENDURANCE RUNNING PERFORMANCE

Endurance running performance has been thought to be determined by running economy, maximal oxygen consumption (VO_{2max}), and lactate threshold [57][58]. However endurance athletes must also be able to maintain relatively high velocities during endurance races. This emphasises the importance of neuromuscular characteristics related to voluntary and reflex neural activation, muscle force and elasticity, and running mechanics, as well as the role of anaerobic characteristics of an endurance athlete [57].

The biomechanical and physiological effects of prolonged running have been shown to cause a worsening in running economy [11]. A reduction in running economy following a marathon has been related to decreased shock absorption capacity of muscles [144]. Impaired muscle-tendon elastic properties and thus reduced mechanical efficiency may be related to decreased neural activation in the eccentric phase of the stretch shortening cycle [64]. Therefore, there may be a direct link between the neuromuscular and biomechanical efficiency during the running stride [11].

2.6.1 Recovery from endurance running

Recovery following endurance running has not been studied extensively. Different recovery patterns have been observed in the parameters that have been measured following endurance running [149][11], these include plasma CK activity, plasma myoglobin [150][151][152], C-reactive protein [151], interleukin-6 [152] and lactate dehydrogenase [152]. Recovery is an important factor in determining when an athlete may return to competition [8], and has been defined as the point at which the athlete is able to train without an increased risk of injury [153]. There are multiple factors that may affect the recovery of an athlete after an endurance event, these include exercise-associated collapse [154], nutrition [155][156], muscle fatigue and muscle damage [92][157], In addition, there is large inter-individual variability in recovery times [8]. There also appears to be an association to the type of fatigue; either central or peripheral, and the speed of recovery [11][10].

Kuitenen et al [10] observed the acute and long-term fatigue effects of stretch shortening cycle (SSC) exercise on the stiffness of the ankle and knee joints. Five participants were fatigued by performing 100 maximal rebound jumps on a sledge apparatus followed by continuous submaximal jumping until exhaustion. The investigators measured neuromuscular fatigue effects in submaximal hopping and maximal drop jumps on a force plate. Force and reflex measures were made using an ankle ergometer. Tests were performed before, immediately after, two hours, two days and seven days after the SSC exercise. The SSC exercise induced an acute and prolonged reduction in knee and ankle joint ranges, and joint stiffness was more noticeable in the knee than the ankle. Recovery of all the EMG parameters occurred within two hours, whereas the force recovery was still incomplete at two hours following the exercise protocol. The recovery of maximal voluntary contraction was prolonged and had not returned to pre test levels by the seventh day. The investigators suggested that the rapid recovery of the joint stiffness occurred as an effect of both central and peripheral fatigue, whereas the prolonged recovery in the force production was probably due to peripheral fatigue, such as contraction failure due to muscle damage [10].

Avela et al [23] investigated the possible interactions between reflex sensitivity and the compliance characteristics of the muscle-tendon complex after marathon running. Marathon running resulted in reductions in maximal isometric force and the maximal rate of force production. The plantar flexor maximal voluntary contraction had recovered two days post marathon, while counter movement jump which tested the landing impact forces, had not recovered at six days post-marathon [23].

Petersen et al [11] investigated the recovery of the neuromuscular capacity and energy expenditure in elite athletes after a marathon race. Eight runners were tested one week before the marathon; and post-tests were conducted at 30 minutes, and two and five days post-marathon. Participants were tested for isometric strength parameters, which was measured by assessing the maximal voluntary contractions of the plantar flexors and knee extensors.

Muscle contractile properties were measured by electrical stimulation of the vastis lateralis muscle, and strength and power was measured by a countermovement jump on a force platform. The results showed increased energy expenditure after the marathon, a 25% reduction in knee extensor MVC, and no changes in electrically evoked muscle contractions. A difference was found in the strength recovery between the plantar flexor and the knee extensor muscles. And a reduction in jumping power was measured. It was concluded that the fatigue experienced by the elite runners was of central rather than peripheral origin, as there was a decreased maximal voluntary contraction of the knee extensors in the absence of any intrinsic muscular damage [11].

Endurance running affects multiple components of exercise performance. There seems to be interplay between central and peripheral fatigue, which may be associated with different recovery times for various components of exercise performance. This is seen in the rapid recovery in joint stiffness [10], compared to the slower recovery of maximal voluntary contraction [11].

2.6.2 Measurement of endurance running performance

A time trial test is defined as an endurance performance test with a known endpoint [158]. In these tests the participants are required to complete a set distance in as fast a time as possible, or complete as much work as they can in a given time period. The subjects are aware of the trial distance or duration in a time trial so that they are able to adjust their work output to pace themselves towards this known endpoint [159]. The 5 km time trial is a reliable measure of endurance performance [160]. Self-selected speed running time-trial tests have been shown to have high levels of reliability [159]. Constant power tests are another method of quantifying changes in endurance performance, this testing makes use of time to exhaustion to measure performance [160].

Time to exhaustion has similar sensitivity to factors affecting endurance performance to that of a time-trial [160]. Laursen et al [159] investigated the reliability of time to exhaustion tests and time trial treadmill running tests of high and moderately high exercise intensity in endurance-trained male distance runners. The results indicated greater variability of the time to exhaustion tests than that of the time trial tests.

Time trials have greater validity than time to exhaustion as they provide a similar physiological simulation of actual performance and correlates with actual performance [161]. Five kilometre time trials were used to measure the endurance performance in this study. These tests helped assess the recovery of the participants after the ultramarathon by monitoring changes in running performance.

2.7 SUMMARY OF LITERATURE

A variety of training methods have been investigated for their effect on endurance running performance [53][54][55][56][36][57][58][59][38][52]. The common goal of all endurance raining is to increase the ability to sustain a level of power or speed for a certain distance or time [29], and thus allow for physiological and metabolic adaptations for maximum energy production [29]. The evidence presented for the various endurance training methods has shown positive effects on endurance running performance.

Marcora and Bosio [33] showed that exercise induced muscle damage may affect the perception of effort. This elevated perceived exertion is thought to be responsible for reduced endurance running performance [65][33][102][103][51][104][105]. The repeated bout effect has been seen to reduce effects of exercise induced muscle damage [100][33]. The repeated bout effect results in increased type I muscle fibre recruitment [101][91][90].

The neural changes that result in the change in muscle fibre recruitment are adaptations that reduce the signs and symptoms of exercise induced muscle damage [83][100][101]. Exercise induced muscle damage has a negative impact on endurance running performance due to the effects on muscle strength and power [97].

The cause of muscle fatigue during prolonged exercise is multifaceted. The contribution of central and peripheral fatigue has not been defined [115]. Studies have shown that fatigue experienced following a marathon is of central rather than peripheral origin, and it was found that central fatigue was the principal explanation for the neuromuscular fatigue experienced after an extreme endurance run and this limited the extent of peripheral fatigue [11]. The description of the Central Governor Model demonstrates the integration of sensory information from the periphery and the appropriate exercise response to maintain homeostasis. Thus this model predicts that exercise performance is regulated by the recruitment of exactly the appropriate amount of muscle fibres to ensure the exercise bout is completed safely [117]. Teloanticipation has been demonstrated to affect pacing strategies and levels of perceived effort during exercise [148].

Recovery after endurance running has not been studied extensively [11]. Current research shows a variety of recovery patterns for a number of parameters (namely, serum creatine kinese activity, skeletal troponin and reflex sensitivity) following an endurance race [23]. There appears to be large inter-individual variability in recovery times [8]. There is little evidence regarding the effects of an ultramarathon on exercise performance during the recovery period after the race, and the optimal duration of the recovery period before returning to competitive running [8]. The length of an appropriate “regeneration” period has been difficult to define as a result of different training backgrounds of athletes and the nature of the preceding training [9].

It is evident from the literature that running performance is influenced by the complex relationship of training, [53][54][55][56][36][57][58][59][38][52] previous experience, [12][148][130] EIMD [33][31][32][34] and fatigue [39][142][143][144]. Thus more research is required to understand changes in running performance during the recovery period after an ultramarathon race. .

CHAPTER 3: DIFFERENCES IN RUNNING PERFORMANCE DURING THE RECOVERY PERIOD AFTER AN ULTRAMARATHON RACE

3.1 INTRODUCTION

Athletes require an appropriate balance between training, competition stress and recovery to achieve maximal performance [162]. Running performance is influenced by a complex interaction between training, previous experience, exercise induced muscle damage and fatigue. Intense training and competition may result in exercise induced muscle damage and delayed onset muscle soreness [163][164]. Exercise induced muscle damage is associated with changes in muscle function and joint mechanics, which may also lead to a reduction in exercise performance [162][77]. The recovery period is an important factor to consider in training regimens, as it has significant effects on athletes' performance, and reduces the risk of developing overtraining syndromes and overuse injuries [165]. However, there is a lack of evidence regarding both the effect of ultramarathon distance races on running performance, and the optimal duration of the recovery period before returning to competitive running [8].

3.2 AIM

The aim of this study was to determine the changes in running performance during the recovery period after an ultramarathon race. The specific objectives have been described in Section 1.2.2 (page 2).

3.3 METHODS

3.3.1 Research design and recruitment

Thirty-five healthy male runners were recruited for this study, which had a quasi-experimental design. This study was submitted and approved by the Faculty of Health Sciences Human Research Ethics Committee, University of Cape Town (HREC REF: 164/2011) (Appendix I). The participants were recruited through advertisements (Appendix II) placed at local running clubs, sports shops, running websites, and through word of mouth. Participants were allocated to an experimental group or a control group. The experimental group consisted of 20 runners participating in the 2011 Comrades Marathon. Fifteen distance runners who were not taking part in the 2011 Comrades Marathon formed the control group.

3.3.2 Sample size calculation

Data from a previous study that measured exercise performance during the recovery period after an ultramarathon race [12] were used to ensure the sample size would provide a significant statistical power. Five km time trial performance was selected to determine the required sample size, as it was the main outcome measures of this study. The required sample size was calculated using a smallest meaningful difference of 30 s, and a standard deviation of 15 s. With the statistical significance accepted as $p < 0.05$, groups of 9, 12, 14 participants provided 80%, 90% and 95% statistical power for 5 km time trial performance respectively. Therefore a total of 35 participants were recruited for the study.

This was to ensure sufficient statistical power if some participants were unable to complete the study, particularly due to the prevalence of musculoskeletal injuries associated with distance running [166][167] and the risk of upper respiratory tract infections following endurance running competition [168][169].

3.3.2.1 Inclusion criteria

Participants were healthy male runners between 20 and 60 years of age. All participants in both the experimental and control groups were required to have run a marathon (42.2 km) in under five hours in the past four months [170], and were required to have a minimum average training mileage of 40 km per week for the four months preceding the study. Participants in the experimental group must have entered the 2011 Comrades Marathon, and needed to have run a minimum of one previous Comrades Marathon, at any stage prior to the 2011 race.

Participants in the control group did not take part in the 2011 Comrades Marathon. Female runners were not included in this study, as one of the main outcome measures was exercise performance, and the menstrual cycle has been shown to influence exercise performance in female athletes [27].

3.3.2.2 Exclusion criteria

Participants were excluded from the study if they developed any flu-like symptoms in the two weeks prior to the Comrades Marathon, and if they became ill during the testing period after the Comrades Marathon. Participants that reported any relevant medical or surgical history, including a history of lumbar spine or lower limb injury or pathology; or the use of any intervention or medication (for example ice, compression garments,

massage, analgesics and non-steroidal anti-inflammatory drugs) to relieve muscle pain or facilitate recovery during the course of the study were excluded. Participants in the experimental group were excluded if they did not complete the Comrades Marathon.

3.4 TESTING PROCEDURE

All participants were requested to attend a familiarisation session 10 days prior to the Comrades Marathon. This familiarisation process was conducted to reduce error associated with participants performing unaccustomed exercise, and using unfamiliar tests. Participants completed an informed consent form (Appendix III), a Physical Activity Readiness Questionnaire (PAR-Q) (Appendix IV), and a medical history and training questionnaire (Appendix V). Body composition measurements were performed and the participants were familiarised with all testing procedures (Appendix VI). The participants were requested to avoid any medication, and strenuous training, other than the Comrades Marathon, for the duration of the study (± 30 days). Participants were also instructed to maintain the same eating habits and training regimen for 24 hours prior to the testing sessions. To facilitate adherence with instructions the participants were required to complete a logbook for the duration of the study. The logbook included the day of the race to assess compliance during the race. (Appendix VII). In addition, participants completed a compliance questionnaire prior to each test session (Appendix VIII). Muscle pain was measured before, and for seven days after the Comrades Marathon. The running performance of the participants was measured using times achieved in the 5 km time trials.

The 5 km baseline time trial was conducted seven days before the Comrades Marathon, and the post-Comrades time trials were performed at six, 13 and 20 days after the race. Participants in the experimental group were required to record their muscle pain in a specially designed logbook (Appendix VII) from the day before the Comrades Marathon to seven days after the race.

Muscle pain was also measured in both groups before each time trial. Heart rate (HR), the relative perception of effort (RPE) and running performance were recorded during the 5 km time trials.

The time trials were conducted at a similar time on each testing day. All participants in the experimental group were required to complete the ultramarathon, and official race times were recorded. The testing procedure is summarised in Figure 3.1.

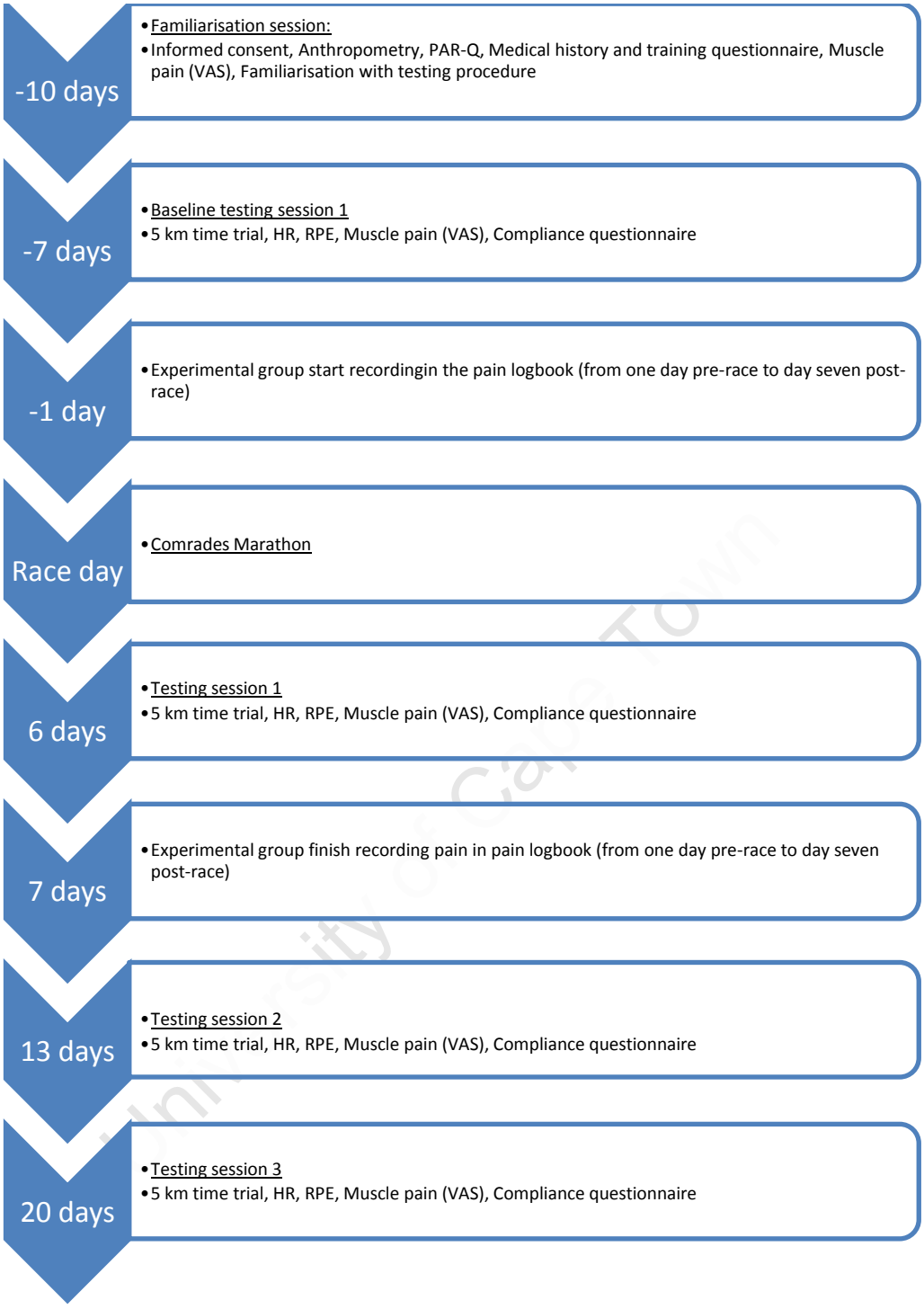

Figure 3.1: Testing procedure.

3.4.1 Informed consent

All participants were required to complete an informed consent form prior to their involvement in the study (Appendix III). This form contained all the relevant information regarding the testing procedure, the potential risks and benefits of the study, and that all data would be treated with confidentiality. The informed consent form also stated that the participants would be allowed to withdraw from the study at any stage. The consent form was discussed at the familiarisation session and the participants were given an opportunity to raise any questions or concerns they had.

3.4.2 Questionnaires

Participants were required to complete the Modified Physical Activity Readiness Questionnaire (PAR-Q) during the familiarisation session (Appendix IV). This is a valid self-guided questionnaire that is used to screen participants for safe participation in physical activity [171][172]. The participants were also required to complete a medical history and training questionnaire, detailing demographics, general medical and surgical history, training, competition and tapering histories (Appendix V). This questionnaire was based on a similar questionnaire used by UCT students to gather data for a study examining the recovery of athletes after the Two Oceans ultramarathon [173]. The questionnaire had been validated as part of the students' study thus this questionnaire was modified minimally to make it more specific for information regarding the Comrades Marathon. All the questions were the same but were specific to the Comrades Marathon and not the Two Oceans. A logbook was issued to each participant at the familiarisation session in which all dietary and training information was documented on a daily basis from the date of this session (Appendix VII).

In addition, a brief compliance questionnaire was completed on each testing day to determine the compliance to the instructions regarding training and recovery treatments during the study (Appendix VIII).

3.4.3 Anthropometry

Body mass (kg) and stature (cm) were recorded using a calibrated scale and stadiometer respectively (Detecto model 6437DHR, Missouri, USA). Body fat was calculated using the sum of seven skinfold measurements namely, biceps, triceps, supscapular, suprailiac, calf, thigh and abdomen [174][175]. Body fat was also expressed as a percentage of body mass [164].

3.4.4 Muscle pain

Muscle pain was recorded before each time trial test. In addition, daily measurements of muscle pain in the experimental group were recorded for one day before, and for seven days after the Comrades Marathon using a logbook (Appendix VII). Muscle pain was measured using a multi-dimensional visual analogue scale (VAS). Participants were required to rate the pain in the quadriceps, hamstrings, and gastrocnemius muscles according to "*general pain at rest*", "*pain during activities of daily living*", "*pain during a passive stretch*", and "*pressure pain*". For pressure pain, digital pressure was applied manually using the pad of the thumb, to the mid-belly of each muscle until moderate tissue resistance was felt. Participants were required to rate the pain in the aforementioned categories for each muscle by drawing a vertical line on a 100 mm pain rating scale, where 0 mm represents "*no pain*", and 100 mm represents "*maximal pain*". The distance along the pain rating scale to the vertical line drawn by the participant was measured in millimetres (mm), and the pain score for each condition was recorded [7].

Cleather and Guthrie [107] established that the multi-dimensional pain scale is a valid and reliable method of assessing pain associated with EIMD.

The average post race pain for the three different muscle groups was calculated using the total muscle pain scores for the four different situations for which pain was described (as mentioned above) and an average pain score for the day was established. The average pain scores for the two groups of participants were calculated in a similar manner on each of the testing days.

3.4.5 Five kilometre time trial performance

Participants performed a 5 km time trial on an indoor track to assess their running performance. Five kilometre time trials have been used by Nummela et al [176][38] to determine factors related to endurance performance and pacing strategy respectively. Pre-race time trials were conducted seven days before the race. Post-race time trials were conducted on days 6, 13 and 20 after the race. Participants were required to perform an individualised ten minute warm-up before each time trial. During the 5 km time trial, participants were instructed to run "*as fast as possible*", and were provided with standardised verbal encouragement during the run. Participants were provided with the split time and distance covered at every kilometre split. Participants were also required to indicate their RPE, using a modified Borg scale [4][133] at every kilometre split (Appendix IX). Heart rate was recorded (Suunto Memory belt, SS013444000, Vantaa, Finland) at five-second intervals throughout the 5 km time trial.

3.4.6 Comrades Marathon

The participants in the experimental group were required to complete the Comrades Marathon on the 29th May 2011. The Comrades Marathon is a 90 km ultramarathon run annually between Pietermaritzburg and Durban. In 2011, the race was an "*up*" run, which starts in Durban and finishes in Pietermaritzburg.

Race finishing times were retrieved off the official race website [(1)] and the average running speeds were expressed as a percentage of the average running speed of the participants' 10 km and 42 km personal best times to provide an approximation of exercise intensity during the race. All participants were required to complete a compliance questionnaire following the Comrades Marathon to assess compliance with regards to using recovery modalities, anti-inflammatory medication or analgesics. Non compliance would result in exclusion from the study.

3.4.7 Statistical analyses

Statistical analyses were performed using Statistica, a data analysis software package (Statsoft, Inc. 2011, version 10), (www.statsoft.com) and GraphPad Prism (version 5.00 for Windows, GraphPad Software, San Diego California USA, www.graphpad.com). An independent t-test was used to determine differences in descriptive variables, and training and racing history between groups. Levene's test was used to assess if the variances of each variable in the two groups were equal. An analysis of variance (ANOVA) with repeated measures, was used to determine the main effects of group (experimental vs. control), pre-post (changes between the pre-race and post-race time trials), and time (within time trial changes between kilometre one to five), and the interactions between group, pre-post, and time for performance variables (heart rate, RPE, time trial performance).

Unequal HSD *post-hoc* analyses were performed where necessary. A Mann-Whitney U test was used to assess differences in the pre-time trial pain scores between groups. Dunn's multiple comparison test was used to assess differences in the pain scores within groups over time for the experimental group post-race pain scores, and the pre-time trial pain scores in both groups. A Pearson's product-moment correlation coefficient determined the relationships between variables (change in 5 km time trial performance, age and running and competition history). All data are presented as the mean ± standard deviation. Statistical significance was accepted as $p < 0.05$.

3.4.8 Ethical considerations

This study was performed in accordance with the principles of the Declaration of Helsinki (Seoul version, 2008). This study was approved by the University of Cape Town, Faculty of Health Sciences Human Research Ethics Committee (HREC REF: 164/2011) (Appendix I). All participants were required to complete an informed consent form (Appendix III), as described in Section 3.3.1 (page 51).

3.4.8.1 Risks to participants

3.4.8.1.1 Anthropometry

There were no risks associated with the measurements of mass, stature and skin fold thickness. There may however have been some temporary discomfort in the measurement of skinfold thickness, due to the tips of the skinfold calliper.

3.4.8.1.2 5 km Time trial

The time trial is a maximal performance test during which the participants were vulnerable to musculoskeletal injuries. However the participants were all moderately trained athletes who were familiar with running 5 km time trials. Each athlete performed a warm up prior to performing the time trial to minimise the risk of injury.

3.4.8.1.3 Comrades Marathon

All participants in the experimental group completing the 2011 Comrades Marathon were exposed to the inherent risks of participating in an endurance event. The participants were required to complete a PAR-Q (Appendix IV) which served to screen for safe participation in exercise. The medical history and training questionnaire (Appendix V) also served as a screening tool for individuals at risk.

3.4.8.2 Benefits to participants

The participants were given their individual results (anthropometric measurements, time trial results, heart rate and perceived exertion measures) in an information pack once the study was completed. This would help the athlete understand their recovery and how their performance may change in the recovery period after an ultramarathon event. Participants were also provided with a summary of the final results of the study (Appendix X).

3.5 RESULTS

3.5.1 Participants

Thirty-one healthy male runners participated in this study. Sixteen Comrades runners, who completed the race, formed the experimental group and fifteen distance runners who were not participating in the 2011 Comrades Marathon formed the control group. The study sample is summarised in Figure 3.2.

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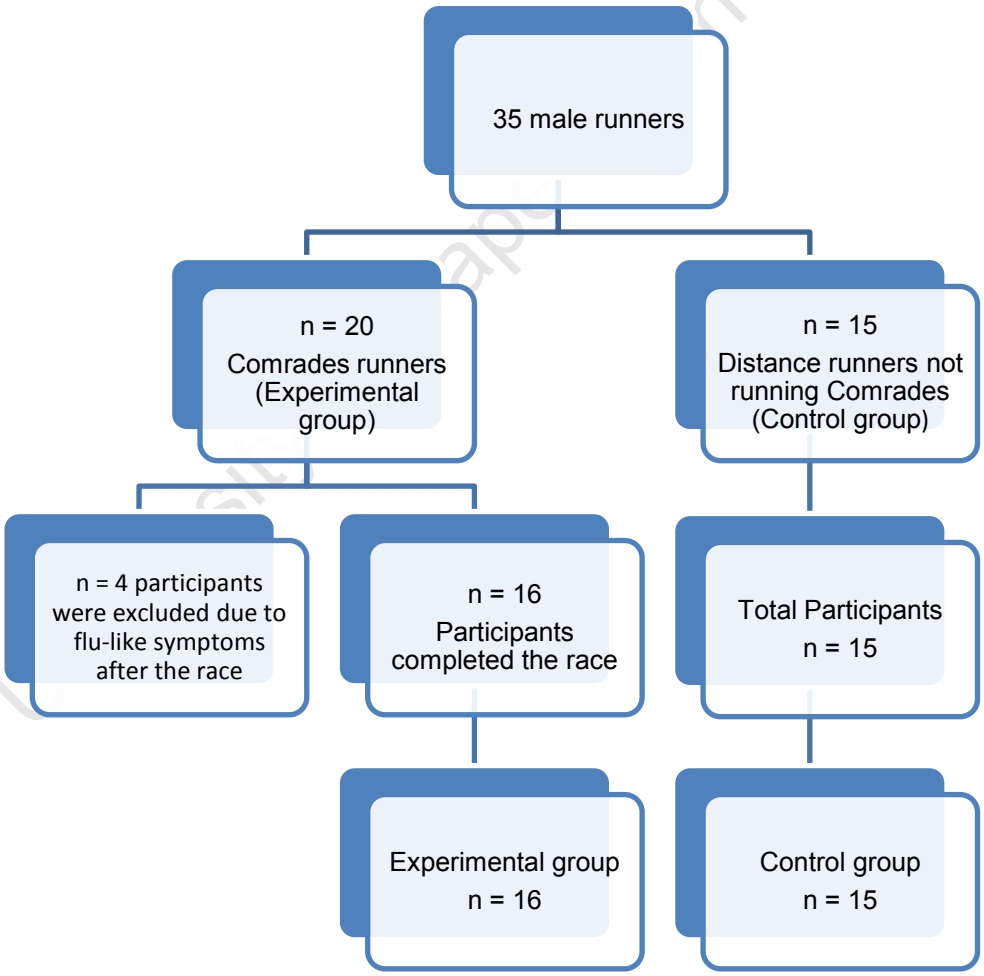

Figure 3.2: Summary of study sample.

All participants completed compliance questionnaires prior to the testing sessions. These questionnaires indicated that there was 100% compliance with instructions during the recovery period after the race.

The descriptive characteristics of the participants are shown in Table 3.1. There was a significant difference between groups in percentage body fat, with the participants in the control group having a lower percentage body fat compared to the experimental group ($p = 0.0008$).

There was also a significant difference between the two groups when comparing the sum of the skinfolds, with the participants in the control group having a smaller sum of seven skinfolds than those in the experimental group ($p = 0.0002$). There were no significant differences between the groups for any other descriptive variables.

Table 3.1: Descriptive characteristics of participants in the experimental (n = 16) and control (n = 15) groups. Data are expressed as mean ± standard deviation.

Variable	Experimental	Control
Age (years)	36.6 ± 8.5	38.9 ± 12.5
Height (cm)	176.1 ± 6.0	178.7 ± 7.7
Body mass (kg)	74.4 ± 6.8	76.4 ± 9.2
% Body fat **	20.3 ± 4.0	15.5 ± 3.0
LBM (kg)	59.3 ± 6.1	62.5 ± 8.2
Sum skinfolds (mm) **	91.4 ± 20.1	63.5 ± 16.3

** $p < 0.01$

3.5.2 Training and racing history

The training and racing history of the participants are shown in Tables 3.2 and 3.3 respectively. There were significant differences between the two groups with regards to the average training distance per week in the three months leading up to the Comrades Marathon, the maximum training distance per week in the three months leading up to the Comrades Marathon, and the average training days per week. The experimental group had a significantly higher average training distance per week in the three months prior to the race, compared to the control group ($p = 0.0008$). The experimental group also had a significantly higher maximum training distance per week in the three months prior to the race, compared to the control group ($p = 0.0006$).

The experimental group had a significantly lower number of training days per week in the three months prior to the race compared to the control group ($p = 0.012$).

Table 3. 2: Training history of participants in the experimental (n = 16) and control (n = 15) groups. Data are expressed as mean ± standard deviation.

Variable	Experimental	Control
Average weekly training distance three months prior to the (km) **	66.9 ± 10.1	52.7 ± 11.0
Maximum weekly training distance three months prior to the (km) **	93.4 ± 18.5	71.3 ± 13.0
Average training intensity three months prior to the race ($min.km^{-1}$)	5.5 ± 0.6	5.6 ± 0.7
Maximum training intensity three months prior to the race ($min.km^{-1}$)	5.3 ± 0.4	5.5 ± 0.8
Average training frequency three months prior to the race (days per week) **	4.1 ± 0.8	4.9 ± 0.7
Maximum training frequency three months prior to the race (days per week)	5.6 ± 1.0	5.7 ± 0.6

** $p < 0.01$

Table 3.3: Racing history of participants in the experimental (n = 16) and control (n = 15) groups. Data are expressed as mean ± standard deviation.

Variable	Experimental	Control
Personal best 10 km (min)	46.9 ± 5.9	47.7 ± 10.0
Number of Comrades Marathons	3.0 ± 2.9	1.9 ± 2.7
Personal best 21.1 km (min)	104.1 ± 15.2	107.9 ± 25.0
Personal best 42.2 km (min)	234.3 ± 28.1	223.7 ± 29.0

3.5.3 Comrades Marathon

The mean Comrades finishing time for the experimental group was 631 ± 48.9 minutes (Table 3.4). The participants' average running speed during Comrades Marathon was more similar to their marathon speed (79.7% ± 11.4) than to their 10 km speed (67.3% ± 10.4).

Table 3.4*:* Comrades race data of the experimental group (n = 16). Data are expressed as mean ± standard deviation.

Variable	Experimental
Predicted Comrades time 2011 (min)	627.5 ± 55.9
2011 Comrades time 2011 (min)	631.0 ± 48.9
Comrades 2011 speed as % of personal best (PB) 10 km running speed	67.3 ± 10.4
Comrades 2011 speed as % of PB 42.2 km running speed	79.7 ± 11.4

3.5.3.1 Post-race pain scores

Figure 3.3 shows the average daily pain scores for the quadriceps, hamstrings and gastrocnemius muscles of participants in the experimental group before, and for seven days after the Comrades Marathon. Quadriceps pain scores were significantly higher at 24, 48 and 72 hours after the race, compared to pre-race scores ($p = 0.001$). Quadriceps pain scores were also significantly increased at 24 hours after the race, compared to 120, 144, and 168 hour post-race scores ($p = 0.001$); and at 48 and 72 hours after the race, compared to 168 hours post-race scores ($p = 0.001$). Lastly quadriceps pain scores significantly increased at 72 hours after the race, compared to 168 hours after the race ($p = 0.001$).

Hamstrings pain scores were significantly higher at 24 and 48 hours after the race, compared to pre-race scores ($p = 0.001$); and at 24 hours after the race, compared to race day (0 hours) scores ($p = 0.001$). Hamstrings pain scores were also significantly increased at 24 hours after the race, compared to 120, 144, and 168 hour post-race scores ($p = 0.001$); and at 48 hours after the race, compared to 168 hour post-race scores ($p = 0.001$).

Gastrocnemius pain scores were significantly higher at 24 hours after the race, compared to pre-race scores ($p = 0.001$). Gastrocnemius pain scores were also significantly increased at 24 hours after the race, compared to 120, 144, and 168 hour post-race scores ($p = 0.001$); and at 48 and 72 hours after the race, compared to 168 hour post-race scores ($p = 0.001$).

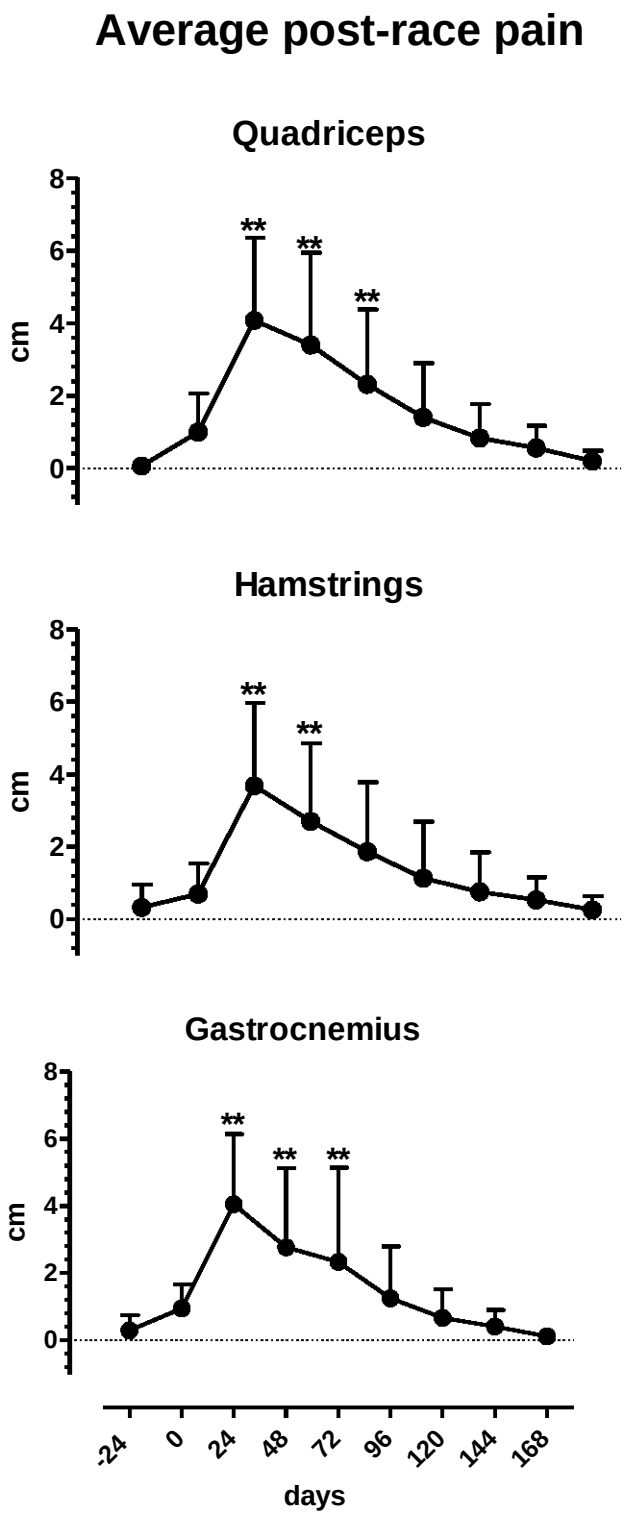

Figure 3.3 Average subjective pain scores (cm) for the quadriceps, hamstrings and gastrocnemius muscles of the experimental (n = 16) group. Daily pain scores were assessed one day before the race, immediately after the race, and for seven days after the Comrades marathon. Data are expressed as mean ± standard deviation.

The legend for this table is continued on the following page.

Significant differences:

Quadriceps:
** 24, 48, and 72 hours vs. -24 hours (p = 0.001)
** 24 hours vs. 120, 144, and 168 hours (p = 0.001)
** 48 and 72 hours vs. 168 hours (p = 0.001)
** 72 hours vs. 168 hours (p = 0.001)

Hamstrings:
** 24 and 48 hours vs. -24 hours (p = 0.001)
** 24 hours vs. 0 hours (p = 0.001)
** 24 hours vs. 120, 144 and 168 hours (p = 0.001)
** 48 hours vs. 168 hours (p = 0.001)

Gastrocnemius:
** 24 hours vs. -24 hours (p = 0.001)
** 24 hours vs. 120, 144 and 168 hours (p = 0.001)
** 48 and 72 hours vs. 168 hours (p = 0.001)

3.5.4 Five kilometre time trial performance

There were no significant differences in the total 5 km time trial performance between groups. However, there was a significant main effect of time ($F_{(4,116)} = 12.04$; $p = 0.0001$). Time trial performance was significantly improved at post-race TT3 compared to post-race TT1 ($p = 0.013$). Figure 3.4 shows the running speeds ($m.s^{-1}$) for both groups during the 5 km time trials. The average pre-race 5 km time trial times were 21.3 ± 1.8 minutes and 21.4 ± 3.7 minutes for the experimental and control groups respectively. Average post-race TT1 5 km time trial times were 21.4 ± 1.9 minutes for the experimental group and 21.7 ± 3.9 minutes for the control group. Average post-race TT2 5 km time trial times were 20.7 ± 1.9 minutes for the experimental group and 21.4 ± 3.6 minutes for the control group, and average post-race TT3 5 km time trial times were 20.4 ± 1.8 minutes and 21.4 ± 3.2 minutes for the experimental and control groups respectively.

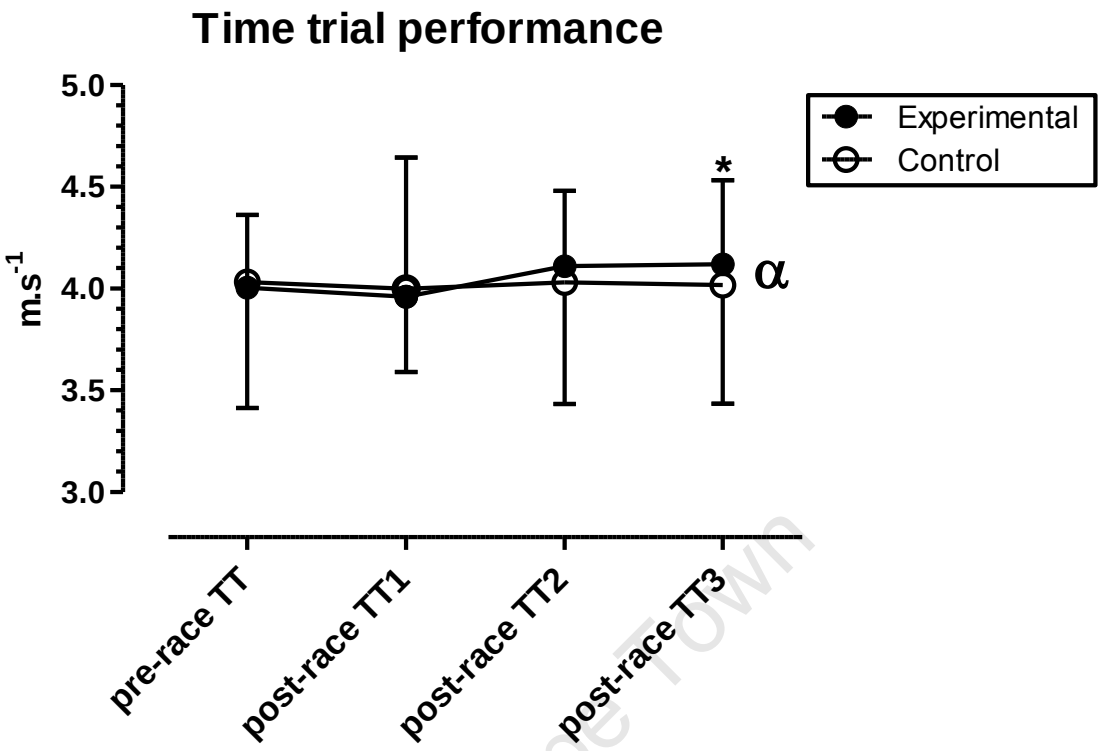

Figure 3.4 5 km time trial running speed (m.s $^{-1}$) of the experimental (n = 16) and control (n = 15) groups. Time trial tests were conducted 10 days before (pre-race), and at days six (post-race TT1), 13 (post-race TT2), and 20 (post-race TT3) after the Comrades marathon. Data are expressed as mean ± standard deviation.

Significant differences:
α main effect of time (p = 0.0001)
* post-race TT3 vs. post-race TT1 (p = 0.013)

The differences in running speed (m.s^{-1}) during each kilometre of the 5 km time trials for the experimental and control groups are shown in Table 3.5. There were significant differences in running speeds between the groups during the time trials. In the pre-race TT, there was a significant difference in running speed between groups at kilometre four (p = 0.002) with the experimental group having a faster running speed than the control group. In the post-race TT1 there were significant differences in running speed between groups at kilometres two (p = 0.007), and four (p = 0.015), at both of these points the experimental group had faster running speeds than the control group.

During the post-race TT2 there were significant differences in running speeds between groups at kilometres two ($p = 0.008$), three ($p = 0.038$), and four ($p = 0.034$) with the control group having faster running speeds at kilometre two and three and the experimental group running faster at kilometre four. In the post-race TT3 there was a significant differences in running speed between groups at kilometre two ($p = 0.045$) with the control group running at a faster speed than the experimental group.

Table 3.5: Time trial running speed ($m.s^{-1}$) of participants for each kilometre in the experimental (n = 16) and control (n = 15) groups. Data are expressed as mean ± standard deviation.

	Experimental				**Control**			
	Pre	Post TT1	Post TT2	Post TT3	Pre	Post TT1	Post TT2	Post TT3
km 1 ($m.s^{-1}$)	4.2 ± 0.5	4.0 ± 0.5	4.5 ± 0.4	4.4 ± 0.5	4.1 ± 0.7	4.2 ± 0.6	4.3 ± 0.7	4.2 ± 0.6
km 2 ($m.s^{-1}$)	4.1 ± 0.4	4.0 ± 0.4	4.0 ± 0.4	4.1 ± 0.4	3.9 ± 0.6	4.0 ± 0.6	3.9 ± 0.7	4.0 ± 0.6
km 3 ($m.s^{-1}$)	4.0 ± 0.4	3.9 ± 0.4	4.1 ± 0.5	4.1 ± 0.5	4.0 ± 0.7	3.9 ± 0.8	4.0 ± 0.8	4.0 ± 0.7
km 4 ($m.s^{-1}$)	3.9 ± 0.3	4.0 ± 0.3	3.9 ± 0.4	3.9 ± 0.5	4.0 ± 0.7	4.0 ± 0.7	3.9 ± 0.7	3.9 ± 0.5
km 5 ($m.s^{-1}$)	3.9 ± 0.5	3.9 ± 0.6	4.0 ± 0.5	4.1 ± 0.5	4.2 ± 0.7	4.0 ± 0.7	4.0 ± 0.7	4.0 ± 0.7

Significant differences:

Experimental group vs. control group:

** Pre TT km 4 p = 0.002
** Post TT1 km 2 p = 0.007
* Post TT1 km 4 p = 0.015
** Post TT2 km 2 p = 0.008
* Post TT2 km 3 p = 0.038
* Post TT2 km 4 p = 0.034
* Post TT3 km 2 p = 0.045

3.5.5 Heart rate

There were no significant differences in heart rate between groups or over time (Figure 3.5).

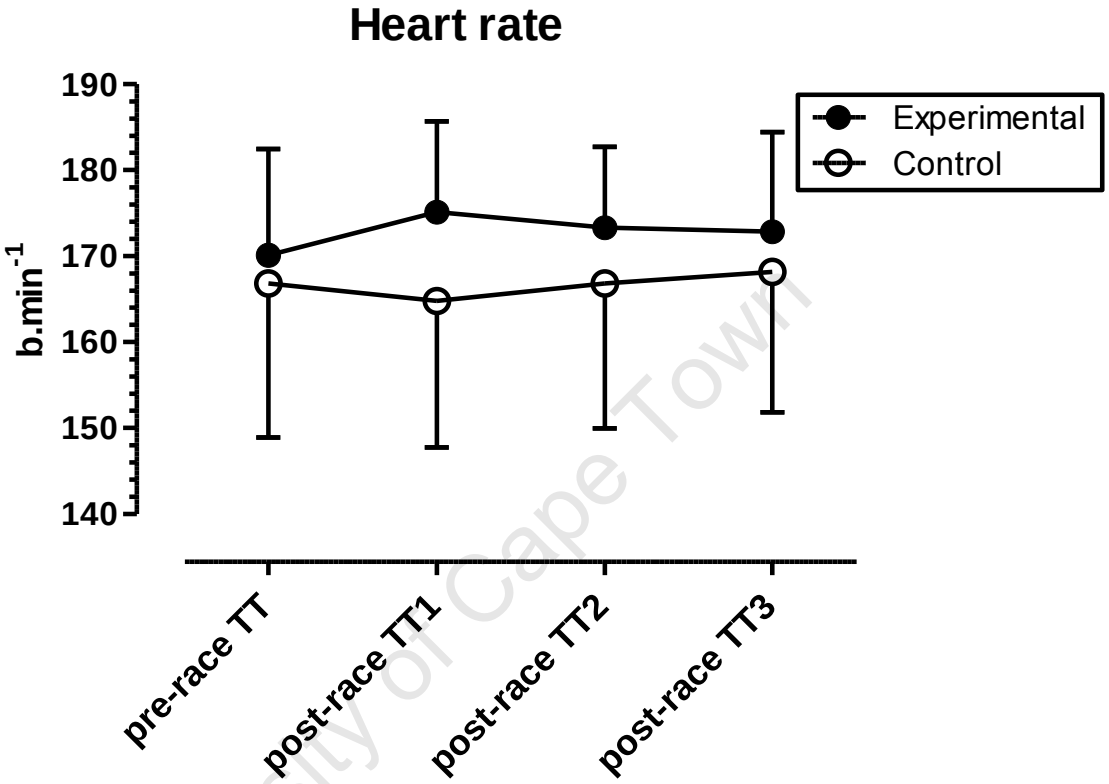

Figure 3.5: Heart rate ($b.min^{-1}$) of participants in the experimental (n = 16) and control (n = 15) groups. Time trial tests were conducted 10 days before (pre-race), and at days six (post-race TT1), 13 (post-race TT2), and 20 (post-race TT3) after the Comrades marathon. Data are expressed as mean ± standard deviation.

3.5.6 Rate of perceived exertion (RPE)

The differences in rate of perceived exertion (RPE) during the 5 km time trials for the experimental and control groups are shown in Figure 3.3. There were no significant differences in RPE between groups. However, there was a significant main effect of time ($F_{(3, 87)} = 4.1$; $p = 0.01$). The rate of perceived exertion was significantly increased at post-race TT2 ($p = 0.02$) and post-race TT3 ($p = 0.02$) in both groups, compared to pre-race values.

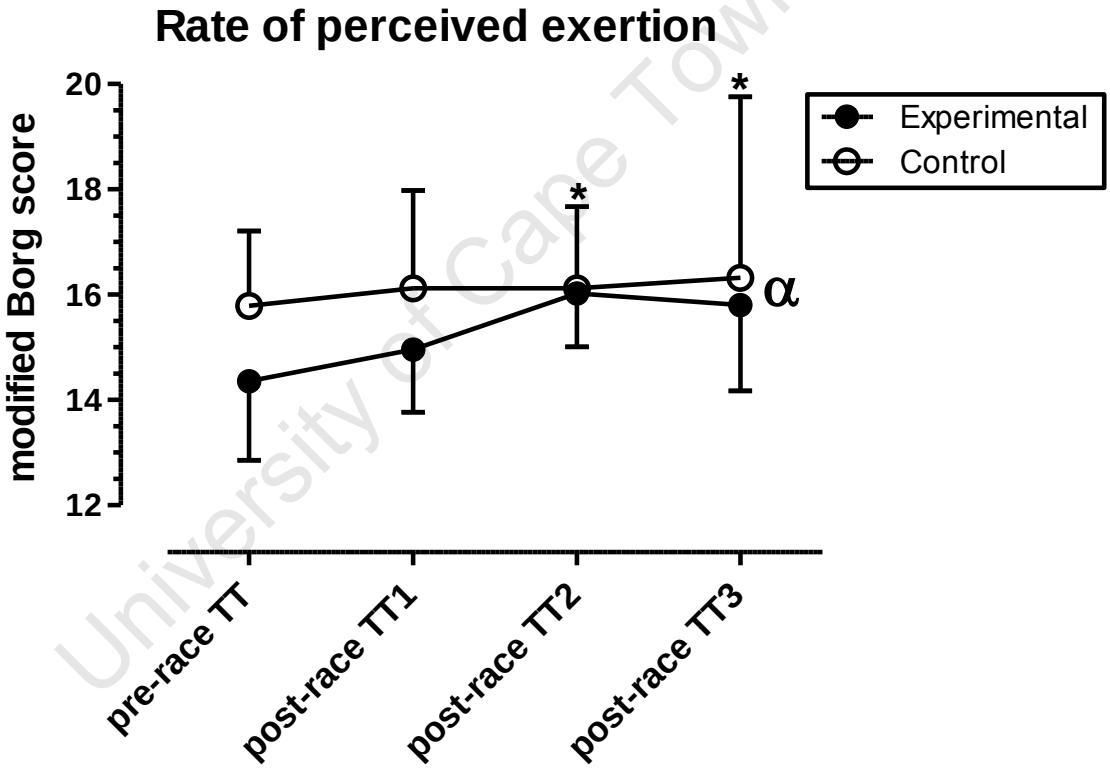

Figure 3.6: Rate of perceived exertion (Borg Scale) of participants in the experimental (n = 16) and control (n = 15) groups Time trial tests were conducted 10 days before (pre-race), and at days six (post-race TT1), 13 (post-race TT2), and 20 (post-race TT3) after the Comrades marathon. Data are expressed as mean± standard deviation.

Significant differences:

α main effect of time ($p = 0.01$)

* post-race TT2 and post-race TT3 vs. pre-race TT ($p = 0.02$)

3.5.7 Average time trial pain

Average pain scores for the quadriceps, hamstrings and gastrocnemius muscles of participants in the experimental and control groups are shown in Figure 3.7. Quadriceps pain scores were significantly higher in the experimental group at TT1 ($p = 0.006$) and TT2 ($p = 0.002$), compared to the control group. Hamstrings pain scores were significantly increased in the experimental group at TT2, compared to the control group ($p = 0.0004$). Gastrocnemius pain scores were significantly higher in the control group compared to the experimental group at TT2 ($p = 0.001$).

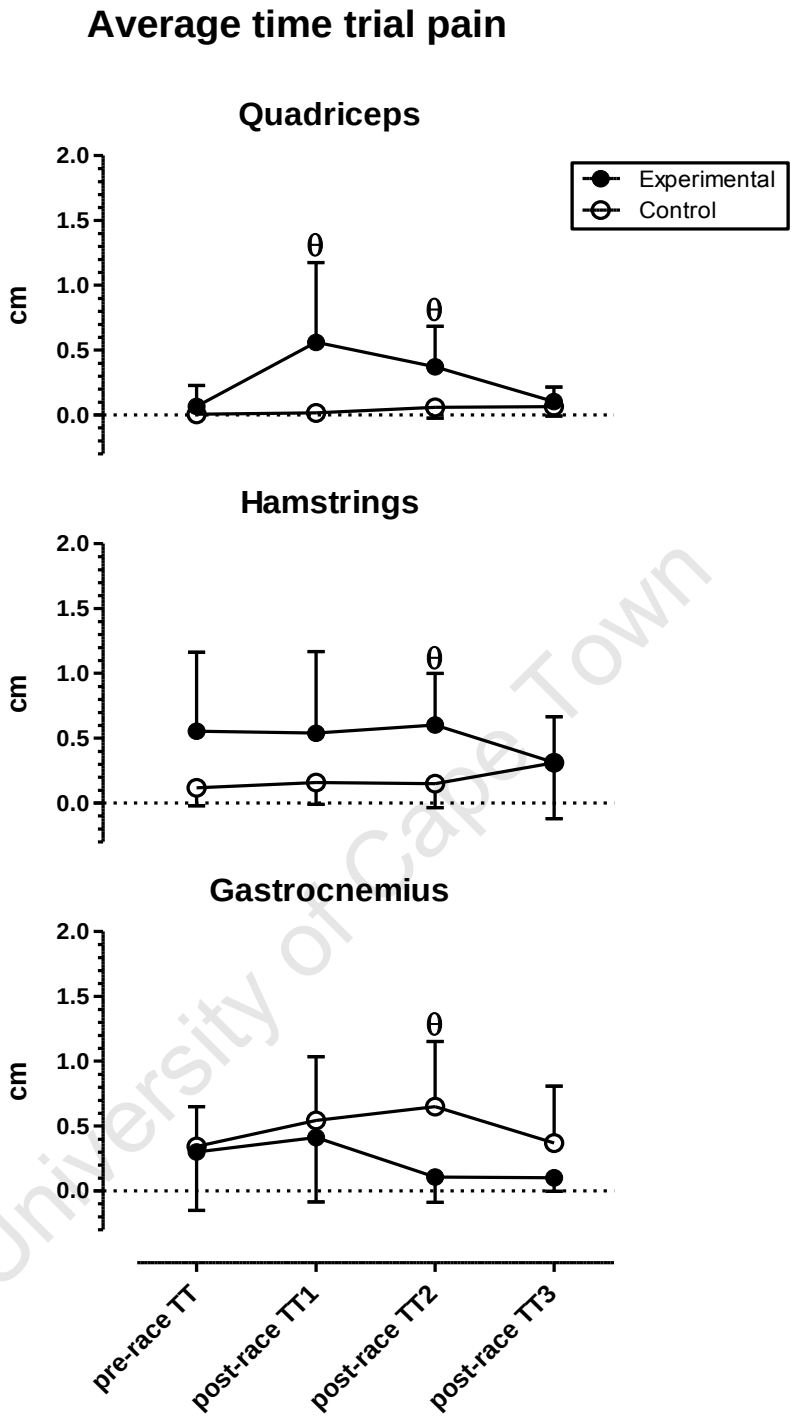

Figure 3.7 Average subjective pain scores (cm) for the quadriceps, hamstrings and gastrocnemius muscles of the experimental (n = 16) and control (n = 15) groups. Time trial tests were conducted 10 days before (pre-race), and at days six (post-race TT1), 13 (post-race TT2), and 20 (post-race TT3) after the Comrades marathon. Data are expressed as mean ± standard deviation.

The legend for this table is continued on the following page.

Significant differences:
Quadriceps: Θ experimental group TT1 vs. control group TT1 (p = 0.006)
Θ experimental group TT2 vs. control group TT2 (p = 0.002)
Hamstrings: Θ experimental group TT2 vs. control group TT2 (p = 0.0004)
Gastrocnemius: Θ control group TT2 vs. experimental group TT2 (p = 0.001)

3.6. Correlational analyses

In this section the relationships between running performance and age will be investigated, as well as the relationships between running performance and previous training and competition history. The relationships between the rate of perceived exertion and running performance, as well as heart rate and running performance will be presented. The differences of post-race TT1 performance and pre-race TT performance are described as early delta (∆) performance. The difference between post-race TT2 performance and pre-race TT performance, and the difference between post-race TT3 performance and pre-race TT performance are described as mid delta (∆) performance and late delta (∆) performance respectively.

3.6.1 Running performance and age

There were no significant relationships between early delta performance and age for the total group (r = -0.18; p = 0.33), the experimental group (r = -0.09; p = 0.74) or for the control group (r = -0.23; p = 0.41). There were also no significant relationships between mid delta performance and age for the total group (r = -0.12; p = 0.52), the experimental group (r = 0.06; p = 0.82) or for the control group (r = -0.24; p = 0.39). In addition, there were no significant relationships between late delta performance and age for the total group (r = -0.01; p = 0.97), the experimental group (r = 0.23; p = 0.40) or for the control group (r = -0.16; p = 0.56). A summary of the relationships between delta performance and age is provided in Table 3.6.

Table 3.6: Relationships between early-, mid-, and late- Δ performance and age for the total group, experimental group and control group.

Correlation	Total		Experimental		Control	
	r	p	r	p	r	p
Early Δ performance	-0.18	0.33	-0.09	0.74	-0.23	0.41
Mid Δ performance	-0.12	0.52	0.06	0.82	-0.24	0.39
Late Δ performance	-0.01	0.97	0.23	0.40	-0.16	0.56

3.6.2 Running performance and training and competition history

3.6.2.1 Running performance and training history

There were no significant relationships between early delta performance and the average training distance for the total group (r = -0.16; p = 0.40), the experimental group (r = 0.02; p = 0.94) or for the control group (r = -0.30; p = 0.28). There were also no significant relationships between mid delta performance and the average training distance for the total group (r = -0.24; p = 0.18), the experimental group (r = -0.16; p = 0.55) or for the control group (r = -0.18; p = 0.51). In addition, there were no significant relationships between late delta performance and the average training distance for the total group (r = -0.22; p = 0.24), the experimental group (r = 0.01; p = 0.98) or for the control group (r = -0.12; p = 0.67). A summary of the relationships between delta performance and the average training distance is provided in Table 3.7.

Table 3.7: Relationships between early-, mid-, and late- Δ performance and average training distance for the total group, experimental group and control group.

Correlation	Total		Experimental		Control	
	r	p	r	p	r	p
Early Δ performance	-0.16	0.40	0.02	0.94	-0.30	0.28
Mid Δ performance	-0.24	0.18	-0.16	0.55	-0.18	0.51
Late Δ performance	-0.22	0.24	0.01	0.98	-0.12	0.67

There were no significant relationships between early delta performance and the average training frequency for the total group (r = 0.02; p = 0.92), the experimental group (r = 0.31; p = 0.24) or for the control group (r = -0.21; p = 0.48). There also were no significant relationships between mid delta performance and the average training frequency for the total group (r = 0.09; p = 0.61), the experimental group (r = 0.05; p = 0.85) or for the control group (r = -0.01; p = 0.97). In addition, there were no significant relationships between late delta performance and the average training frequency for the total group (r = 0.25; p = 0.18), the experimental group (r = 0.19; p = 0.48) or for the control group (r = 0.11; p = 0.69). A summary of the relationships between delta performance and the average training frequency is provided in Table 3.8.

Table 3.8: Relationships between early-, mid-, and late- Δ performance and average training frequency for the total group, experimental group and control group.

Correlation	Total		Experimental		Control	
	r	p	r	p	r	p
Early Δ performance	0.02	0.92	0.31	0.24	-0.21	0.45
Mid Δ performance	0.09	0.61	0.05	0.85	-0.01	0.97
Late Δ performance	0.25	0.18	0.19	0.48	0.11	0.69

The correlational analyses for maximum training distance and maximum training frequency, and delta performance are presented in Appendix XI.

3.6.2.2 Running performance and competition history

There were no significant relationships between early delta performance and the number of previous Comrades Marathons completed for the total group (r = -0.13; p = 0.48), the experimental group (r = -0.25; p = 0.35) or for the control group (r = -0.05; p = 0.86). There were also no significant relationships between mid delta performance and the number of previous Comrades Marathons completed for the total group (r = 0.07; p = 0.73), the experimental group (r = 0.27; p = 0.30) or for the control group (r = -0.01; p = 0.97). In addition, there were no significant relationships between late delta performance and the number of previous Comrades Marathons completed for the total group (r = 0.09; p = 0.62), the experimental group (r = 0.39; p = 0.13) or for the control group (r = -0.01; p = 0.99). A summary of the relationships between performance and the number of previous Comrades Marathons completed is provided in Table 3.9.

Table 3.9: Relationships between early-, mid-, and late- Δ performance and number of previous Comrades Marathons completed for the total group, experimental group and control group.

Correlation	Total		Experimental		Control	
	r	p	r	p	r	p
Early Δ performance	-0.13	0.48	-0.25	0.35	-0.05	0.86
Mid Δ performance	0.07	0.73	0.27	0.30	-0.01	0.97
Late Δ performance	0.09	0.62	0.39	0.13	-0.01	0.99

3.6.2.3 Running performance and Comrades Marathon performance

There was a significant positive relationship between early delta performance and Comrades finishing time as a percentage of the 10 km personal best time for the experimental group (r = 0.55; p = 0.03). However, there were no significant correlations between mid delta performance and Comrades finishing time as a percentage of the 10 km personal best time for the experimental group (r = 0.21; p = 0.66); or between late delta performance

and Comrades finishing time as a percentage of the 10 km personal best time for the experimental group (r = -0.08; p = 0.76).

There was a significant positive relationship between early delta performance and Comrades finishing time as a percentage of the 42 km personal best time for the experimental group (r = 0.53; p = 0.04). However, there were no significant correlations between mid delta performance and Comrades finishing time as a percentage of the 42 km personal best time for the experimental group (r = -0.07; p = 0.80); or between late delta performance and Comrades finishing time as a percentage of the 42 km personal best time for the experimental group (r = -0.20; p = 0.45).

To summarise, a negative correlation indicates that as Comrades finishing times as a percentage of personal best (PB) 10 km and 42 km times increases, delta performance decreases. Similarly a positive correlation indicates that as Comrades finishing time as a percentage of personal best (PB) 10 km and 42 km times increases, delta performance increases. A summary of the relationships between delta performance and Comrades finishing times as a percentage of personal best (PB) 10 km and 42 km times is provided in Table 3.10.

Table 3.10: Relationships between early-, mid-, and late- Δ performance and Comrades finishing times as a percentage of 10 km and 42 km personal best times for the experimental group.

Correlation	**Experimental group**	
Delta performance and Comrades speed % of 10 km personal best	**r**	**p**
Early Δ performance	0.55	**0.03***
Mid Δ performance	0.21	0.66
Late Δ performance	-0.08	0.76
Delta performance and Comrades speed % of 42 km personal best	**r**	**p**
Early Δ performance	0.53	**0.04***
Mid Δ performance	- 0.07	0.80
Late Δ performance	- 0.20	0.45

The correlational analyses for delta performance and Comrades finishing times as a percentage of recent 10 km and 42 km times are presented in Appendix XI.

3.6.3 Running performance and rate of perceived exertion (RPE)

There were no significant relationships between early delta performance and the rate of perceived exertion for the total group (r = -0.17; p = 0.36), the experimental group (r = -0.50; p = 0.05) or for the control group (r = 0.14; p = 0.63). There were also no significant relationships between mid delta performance and the rate of perceived exertion for the total group (r = -0.16; p = 0.38), the experimental group (r = -0.15; p = 0.57) or for the control group (r = -0.03; p = 0.90). In addition, there were no significant relationships between late delta performance and the rate of perceived exertion for the total group (r = -0.25; p = 0.17), the experimental group (r = 0.17; p = 0.53) or for the control group (r = -0.35; p = 0.20). A summary of the relationships between delta performance and the rate of perceived exertion is provided in Table 3.11.

Table 3.11: Relationships between early-, mid-, and late- Δ performance and rate of perceived exertion for the total group, experimental group and control group.

Correlation	Total		Experimental		Control	
	r	p	r	p	r	p
Early Δ performance	-0.17	0.36	-0.50	0.05	0.14	0.63
Mid Δ performance	-0.16	0.38	-0.15	0.57	-0.03	0.90
Late Δ performance	-0.25	0.17	0.17	0.53	-0.35	0.20

3.6.4 Running performance and heart rate (HR)

There were no significant relationships between early delta performance and heart rate for the total group (r = -0.01; p = 0.95), the experimental group (r = -0.31; p = 0.24) or for the control group (r = 0.16; p = 0.57).

There were also no significant relationships between mid delta performance and heart rate for the total group (r = -0.27; p = 0.14), the experimental group (r = -0.50; p = 0.05) or for the control group (r = -0.16; p = 0.75). In addition, there were no significant relationships between late delta performance and heart rate for the total group (r = -0.27; p = 0.14), the experimental group (r = -0.34; p = 0.19) or for the control group (r = -0.23; p = 0.40). A summary of the relationships between delta performance and heart rate is provided in Table 3.12.

Table 3.12: Relationships between early-, mid-, and late- Δ performance and heart rate for the total group, experimental group and control group.

Correlation	Total		Experimental		Control	
	r	p	r	p	r	p
Early Δ performance	-0.01	0.95	-0.31	0.24	0.16	0.57
Mid Δ performance	-0.27	0.14	-0.50	0.05	-0.16	0.57
Late Δ performance	-0.27	0.14	-0.34	0.19	-0.23	0.40

3.7 Summary of results

In summary, the main findings of this study show there were no significant differences in the total 5 km time trial performance between the groups, however there was a significant main effect of time for time trial performance. There was a significant increase in total 5 km time trial running speeds for both groups at post-race TT3, compared to post-race TT. There were no significant differences in heart rate between groups or over time. There were no significant differences in RPE between groups, although there was a significant main effect of time. The rate of perceived exertion was significantly increased at post-race TT2 and post-race TT3 in both groups, compared to pre-race values. Quadriceps pain scores were significantly higher in the experimental group at post-race TT1 and post-race TT2, compared to the control group.

Hamstrings pain scores were significantly increased in the experimental group at post-race TT2, compared to the control group. Gastrocnemius pain scores were significantly higher in the control group at post-race TT2, compared to the experimental group. In the experimental group, there were significant positive relationships between early delta performance and Comrades finishing time as a percentage of the 10 km personal best time; and between early delta performance and Comrades finishing time as a percentage of the 42 km personal best time.

3.8 Discussion

The Comrades Marathon induced symptoms in the experimental group consistent with delayed onset muscle soreness. These results are similar to other studies which have investigated the onset of muscle soreness following exercise induced muscle damage [12][38][176][75]. There were changes in 5 km time trial performance during the recovery period after the ultramarathon; however no differences in performance were found between groups (Section 3.5.4, page 76). Running performance during the recovery period has not been widely researched, but has been thought to be influenced by the relationship of previous experience, exercise induced muscle damage and fatigue [29]. One of the main findings of this study showed that the early change in performance following the Comrades Marathon had a positive relationship to previous pacing strategies used for personal best times in 10 km and 42 km races (Section 3.6.2.3 page 87), which may support the role of central regulation and prior experience affecting performance in the presence of fatigue and muscle damage [148][49][177][116][178].

3.8.1 Descriptive characteristics

The sample size of this study consisted of 16 experimental and 15 control participants, which provided a statistical power of 95%. Other studies investigating the effects of ultramarathons on various physiological processes, and functional outcomes have had sample sizes varying between 15 and 30 participants [179][180][29]. The descriptive characteristics of the participants in this study showed the two groups were similar in age, height, body mass, lean body mass and lean thigh volume. However a significant difference between the groups was seen in percentage body fat (Section 3.5.1, page 70) with the participants in the control group having a lower percentage body fat than the experimental group.

The participants in the control group also showed a significantly a smaller sum of the seven skinfolds than those in the experimental group (Section 3.5.1, page 70).These findings are unlikely to have resulted in any significant change in the two group's performance. This has been demonstrated in a study investigating the influence of anthropometric and training parameters on race performance in ultra-endurance runners during a 24 hour race [181]. In this study, there were no significant associations between endurance performance and anthropometric measurements [181]. A similar study examined the effect of anthropometry, personal best marathon times and training volume on male 100 km ultrarunners [182]. The results indicated that the athletes performance was influenced significantly by the personal best marathon times and training volumes and not by any of the anthropometric variables [182]. Thus the differences in the anthropometric measurements found in the current study should have had no effect on performance.

3.8.2 Training and racing history

The experimental group had a significantly higher average training distance per week and a significantly higher maximum training distance per week in the three months prior to the race compared to the control group (Section 3.5.2, page 72). Despite this the experimental group was found to have a significantly lower number of training days per week in the three months prior to the race compared to the control group (Section 3.5.2, page 72). This suggests that the experimental group underwent a higher intensity endurance training programme compared to the control group. The reason for the higher intensity training may be explained by the study conducted by Midgley et al [49]. This study found that by manipulating the training intensity the athlete's VO_{2max} could be increased, and that training at a high intensity places maximal stress on the physiological processes that limit VO_{2max} resulting in an optimal stimulus for adaptation. Thus by allowing adaptations for an increased VO_{2max} to occur endurance performance may be enhanced, this result would be favourable for the runners in the experimental group.

Midgley et al [49] also found that the training volume was an important factor resulting in improved running economy due to the progressive adaptation of skeletal muscle and improved mechanical efficiency. This may explain why the training programmes which the experimental group followed favoured high volume training. Knechtle et al [182] showed that a high weekly training volume correlated significantly with a fast total race time for a 100 km race, which further supports the training routine of the experimental group in this study.

3.8.3 Comrades Marathon

The Comrades Marathon induced muscle pain in the experimental group consistent with delayed onset muscle soreness. Delayed onset muscle soreness is characterised by the perception of discomfort starting approximately eight hours post exercise, peaking at 24 to 48 hours and then reducing after seven to ten days [75]. The muscle pain in the gastrocnemius, quadriceps and hamstring muscles of the experimental group occurred within the first 24 hours after the race, and returned to a pain free state after seven days (Figure 3.3, page 75).

These findings are supported by other studies investigating the delayed muscle soreness as a result of exercise induced muscle damage [64][65][60]. Peak levels of muscle pain in this study occurred at 24 hours following the Comrades Marathon, which is consistent with the peak pain developed by the athletes who completed the Comrades in the study by Burgess [12]. Similar findings showing peak pain occurring 24 hours following the Comrades Marathon were seen in the study investigating the differences in muscle pain and plasma creatine kinase activity after 'up' and 'down' Comrades Marathon [183]. Delayed onset muscle soreness does not accurately reflect the magnitude of muscle damage [85] as muscle function may remain impaired even after the muscle soreness has dissipated [73]. Thus delayed onset muscle soreness may have affected the participants' performance in the experimental group even though they were not experiencing any perceived discomfort. This is supported by Marcora and Bosio [33] who concluded that the risk of having a significant decrease in performance is higher in participants suffering from exercise induced muscle damage than those who are not.

3.8.4 Five kilometre time trial performance

There were no significant differences in the total 5 km time trial performance between the experimental and control groups. This may be explained by the process of delayed onset muscle soreness which has been found to reduce after seven days following the onset of pain [75]. The time trials were carried out in weekly intervals after the Comrades Marathon, the first post-race time trial (post-race TT1) was conducted six days following the marathon, thus allowing symptoms of pain to return to pre-race levels. If the first time trial following the Comrades Marathon was conducted earlier a larger decrease in the experimental groups' performance may have been seen as the participants were still reporting pain in the muscles up to six days after the race (Figure 3.3, page 75).

There was a significant main effect of time and time trial performance was improved at post-race TT3 compared to post-race TT1 (Section 3.5.4, page 76). Although there were no differences between groups, it may be theorised that the improvement in the experimental groups' time trial performance may be due to the recovery of muscle tissue following the lengthening muscle contractions that occurred during the race. This is supported by Gleeson [32] et al, who observed that the neuromuscular efficiency of muscles were impaired following lengthening contractions. The impairment in neuromuscular efficiency was seen to outlast the other symptoms of muscle damage such as soreness and loss of strength. Another consideration is that the Comrades Marathon may have been a positive stimulus to induce adaptation which resulted in an improved performance once the pain had subsided [12][179]. In this study it was not possible to establish differences between groups in 5 km time trial performance. Further studies should be done using a larger sample size which may allow for the differences between the groups to be more accurately determined.

Another factor which may have affected the participants' 5 km performance during the testing period is teloanticipation. Ulmar [6] described the interpretation of exertion which is compared to mental representations that have been developed through exposure to similar experiences in the past as teloanticipation. This is an integrative process, which culminates in the selection of a specific workload or exercise intensity [158]. In a study investigating the importance of distance knowledge, distance feedback, and prior experience on the setting of a pacing strategy [184], it was found that distance feedback is not essential in developing an appropriate pacing strategy. Rather the prior experience of an unknown distance appeared to allow the creation of an internal, relative distance that was used to establish a pacing strategy. This highlights the effect of teloanticipation in the formation of a pacing strategy. In this study a familiarisation session was included to enable the participants to become familiar with the track running. One session may not have been adequate to eliminate any learning effects. Future studies should consider increasing the number of familiarisation sessions to further reduce the potential for a learning effect over time.

Mickelwright et al [148] concluded that a previous memory of a fatiguing exercise allowed the athlete the ability to estimate their tolerance levels and energy reserves. It was also theorised that the pacing strategies utilised in the study were influenced by an interaction between feedback given during the time trials and previous experience. This study provides additional support for the role of prior experience and its influence on pacing. Teloanticipation may have allowed the experimental group in the current study to perform at a similar level at the post-race TT1 compared to the baseline level (Section 3.5.4, page 76).

During the 5 km time trial of the current study, participants were provided with the split time and distance covered at every kilometre split, they were also given standardised verbal encouragement. In a study investigating the effects of experience and feedback on pacing and performance during cycling time trials [148], it was found that pacing was influenced by the interaction of feedback and previous experience.

These environmental cues were influenced by mental representations developed during similar experiences that in turn resulted in a pacing strategy and influenced their rate of perceived exertion [148]. Thus it can be argued that the cues given to the participants of the current study may have allowed them to develop pacing strategies and in turn gauge their performance.

In summary, 5 km time trial performance may have been affected by the effects of delayed onset muscle soreness initially, prior experience and teloanticipation, as well as the feedback participants received during the time trials. In previous studies investigating running performance investigators have made use of a combination of outcome measures to evaluate the change in performance [12][36][56].

Paavolainen et al [36] investigated the effects of different training protocols on endurance performance and measured performance using a 5 km time trial performance test together with a 20 m sprint and maximal velocity aerobic and anaerobic running tests. Hennessy et al [58] incorporated power tests as well as speed tests to establish change in performance. Marcora and Bosio [68] studied the effects of EIMD on endurance performance using time trial tests and knee extensor strength as outcome measures for performance. Thus the use of the 5 km time trial alone may not provide sufficient sensitivity regarding change in performance. Future studies should make use of more than on outcome measure to establish the change in running performance following an ultramarathon.

3.8.5 Heart rate

There were no significant differences in heart rate between the experimental and control groups or over time (Section 3.5.5, page 79). This is supported by the results of the study conducted by Burgess [12], that investigated the effects of exercise induced muscle damage caused by a 90 km ultramarathon on running economy and stride length in experienced ultramarathon runners. However these findings are contradictory to a previous study [179] which investigated the time course of recovery of muscle function and the heart rate response to steady-state exercise following the Comrades Marathon [179]. These investigators found the participants had a delayed exaggerated heart rate response to steady-state running at moderate to high intensities for 25 days after the race. A reason for the discrepancy in results between the studies may be due to the variation in sample size of the experimental group.

In the study by Chambers et al [179] the sample size was relatively small and consisted of seven athletes who completed the Comrades Marathon as opposed to the larger sample sizes in the studies conducted by Burgess [12] who had eleven Comrades runners, and the experimental group in this study of 16 Comrades runners. In addition Chambers et al [185] suggested the heart rate results should be interpreted with caution, as there was a large inter-individual variation for these measurements and coupled with the small sample group, there was an increased possibility of a type II error of analysis. Additional research is required in investigating the effects of an ultramarathon race on heart rate during exercise in the recovery period.

3.8.6 Rate of perceived exertion (RPE)

There were no significant differences in RPE between the two groups (Section 3.5.6, page 80). However, there was a significant main effect of time. The rate of perceived exertion was significantly increased at post-race TT2 and post-race TT3 compared to pre-race values (Figure 3.6, page 80). Although there were no differences between groups, it may be speculated that the reduction in the pre-race rate of perceived exertion in the experimental group may be due to the pre-race taper. Tapering is a systematic reduction in training load which has been shown to have physiological and psychological benefits which together improve performance [186].

The experimental group may also have been apprehensive to run at a higher rate of perceived exertion prior to the Comrades marathon to avoid possible fatigue and injury. A similar finding was seen in a study investigating the effects of exercise-induced muscle damage caused by a 90 km ultramarathon on submaximal oxygen consumption and running kinematics in experienced ultramarathon runners [12], where the pre-race rate of perceived exertion for the experimental group was lower than the control group. This was thought to be as a result of the combined effects of training, tapering and psychological preparation for the race.

Swart et al [187] investigated exercise regulation by perceived exertion in relation to the duration of exercise and knowledge of an endpoint, showed that when participants were initially unfamiliar with the exercise bout they chose a conservative perceived exertion strategy that maintained a larger metabolic reserve. This strategy was associated with a non-linear growth in RPE over time. As participants became more familiar with the required task the RPE strategy became more aggressive and linear, with a smaller metabolic and cardiorespiratory reserve. These results support the change of RPE found in the current study.

The RPE of the participants was initially low and as the testing continued the RPE increased (Section 3.5.6, page 80). Therefore the changes in RPE seen in the participants could be explained as a result of the initial pre-race taper and a conservative RPE strategy, which then became modified to a more aggressive RPE strategy allowing a smaller metabolic reserve [12][185]. In this study it was not possible to establish the differences between groups for the RPE. Further studies should be done with a larger sample size which will allow for differences to be accurately investigated.

3.8.7 Average time trial pain

Quadriceps pain scores were significantly higher in the experimental group at TT1 and TT2 compared to the control group. Hamstrings pain scores were significantly increased in the experimental group at TT2, compared to the control group However, Gastrocnemius pain scores for the control group were higher than the experimental group at TT2 (Section 3.5.7, page 81). These findings seem unrelated to delayed onset muscle soreness as these symptoms are usually resolved by day ten, following an eccentric exercise bout [75].

Although both experimental and control groups reported pain during the testing procedure, and there were significant differences between the groups for pain in the different muscle groups, these were relatively low measures of pain i.e. less than ten millimetres on the visual analogue scale which measured from zero to 100 mm (Figure 3.7, page 82). This mild muscle pain may have been as a result of the participants not being allowed to partake in any recovery routines or treatments during the length of this study. This may have resulted in a mild pain that they may not have been accustomed to, and that their regular post exercise stretching or icing routines would have eased.

3.8.8 Correlational analyses

The early change in performance (difference between post-race TT1 and pre-race TT performance) was found to be positively associated to the Comrades finishing time as a percentage of the 10 km personal best time, and to the Comrades finishing time as a percentage of the 42 km personal best time. This relationship may be as a result of participants pacing strategy in the presence of neuromuscular fatigue, as this relationship was not seen in the mid and late changes in performance when the fatigue associated with the race should have dissipated. The feed forward process of teloanticipation allows experience of previous performance to influence perceptions of effort, pacing strategies and subconscious muscle control [148]. This process may have regulated the pacing in the pre-race TT and the post-race TT1 more closely than the mid and late time trials as way of protecting the participants from further damage in the presence of exercise-induced muscle damage [65][64].

A similar finding for the relationship between the change of performance following the Comrades Marathon and personal best times for 10 km and 42 km was seen in the study conducted by Burgess [12]. Where there was a positive association between personal best performances over 10 km and 42 km and the change in time trial performance. This relationship was thought to be as a result of the number years of training experience and racing experience.

In a study investigating the relationships between training volume, marathon personal best times and anthropometric variables on 100 km runners performance [182] a correlation was found between personal best marathon time and the total 100 km time. A possible explanation for the personal best 42 km times being correlated to the performance in the 100 km race and Comrades Marathon may due be the high training volume that is necessary to complete these races. The chronic responses of skeletal muscle to endurance exercise are the results of cumulative effect of the repeated exercise.

Training responses have been found to be directly proportional to the volume of work performed [45][29]. Muscle composition has been seen to differ between athletes with the endurance trained athletes having a high proportion of type I fibres [29]. The number of type I fibres in trained athletes was found to be related to the number of years of prior endurance training [47]. Thus it can be postulated that endurance training required for a 42 km race induces the necessary changes over time which assist in the performance of ultramarathons.

In this study the relationship between change in performance and previous personal best times was not constant throughout the recovery process and thus it is suggested that further investigations are made into the factors affecting the relationship of the change in time trial performance and previous endurance running performance.

3.8.9 Limitations of the study

A limitation of this study was that the sample group for both the experimental and control groups were composed of endurance runners who had a high level of endurance running experience, although they ran at a relatively average running pace i.e. personal best marathon times for experimental and control group in minutes are 234.3 ± 28.1 and 223.7 ± 29.0 respectively. These results cannot be generalised for athletes who are of a more elite standard. Future studies should investigate the performance of elite athletes during the recovery period after a marathon or ultramarathon race.

Furthermore, this study investigated the effects of performance following the "up run" of the Comrades Marathon. These results may not be true for the "down run" due to the excessive lengthening contractions which occur during downhill running and thus one would expect a higher level of exercise induced muscle damage which could in turn affect the performance in the recovery period.

Five kilometre time trials may not have provided the sensitivity in order to measure the change in performance following the Comrades Marathon. The results did not provide adequate information required to observe the specific stage the runners had returned to their pre-Comrades performance levels. Additional tests such as a maximal treadmill running test measuring maximum running speed [12][38] or a 20m sprint test [35][58] could be incorporated. Functional performance could also measured using tests such as vertical jump height [58] or maximal isometric force measurements [57][36].

Future studies should be conducted using a larger sample size. This will allow for more detailed analysis to be carried out and differences between groups may be determined for the 5 km performance following an ultra marathon and for the RPE during the recovery period. It is also important for future studies to try and quantify the state of "stress" following the race. Different athletes have varying levels of training and exercise at different effort levels during the Comrades Marathon.

These permutations are likely to have a physiological consequence on the performance during the recovery period. The study design being a quasi-experimental design has its own limitations as it is not a randomized controlled study; this may introduce various confounders limiting the influences on internal and external validity. This study showed a large inter-subject variability when the coefficient of variation was calculated for delta performance and thus is a confounding factor.

3.9 Summary

The results of this study showed no significant difference between the groups for the 5 km time trial performance and that both groups' performance improved during the testing period from post-race TT1 to post-race TT3. In this study it was not possible to establish differences between groups in 5 km time trial performance. Further studies using larger sample sizes may allow for the differences between the groups to be determined. No alterations in heart rate were noticed despite this being found to be delayed and exaggerated in a previous study for up to 25 days following an ultramarathon [(179)]. There was a change of rate of perceived exertion throughout the testing procedure which may have initially been low in the experimental group as a result of the tapering process.

The Comrades Marathon induced muscle pain in the experimental group consistent with delayed onset muscle soreness. The muscle pain in the gastrocnemius, quadriceps and hamstring muscles of the experimental group occurred within the first 24 hours after the race, and returned to a pain free state after seven days. These findings are supported by other studies investigating the delayed muscle soreness as a result of exercise induced muscle damage [(64)(65)(60)].

An interesting finding of this study is that the changes in early performance following the Comrades showed a positive relationship to previous pacing strategies used in 10 km and 42 km races which may support the role of central regulation and prior experience affecting performance in the presence of fatigue and muscle damage.

This is supported by Burgess's [12] findings that there may be an interaction between protective adaptations and central mechanisms during the recovery period after an ultramarathon race. .

CHAPTER 4: SUMMARY AND CONCLUSION

Previous studies have shown that exercise induced muscle damage has a negative effect on endurance running performance [49]. Marcora and Bosio [33] proposed that the effect of exercise induced muscle damage on endurance running performance may be mediated by an increased perception of effort. Muscle damage has also been shown to result in an immediate and prolonged reduction in muscle force-generating capacity which in turn affects performance [65][64][33][34]. Muscle fatigue during prolonged exercise is multifaceted and the relative contribution of central and peripheral fatigue has not been clearly defined [115]. There seems to be a continuous feedback loop between central and peripheral fatiguing factors during prolonged exercise [145][117][118]. Burgess [12] observed a positive relationship between running performance during the recovery period after an ultramarathon, and prior endurance training and racing experience. The overall aim of this study was to determine the changes in running performance during the recovery period after an ultramarathon race. Based on the evidence provided in this dissertation the study objectives described in Section 1.2.2 (page 2) may be answered as follows:

To determine differences in running performance between an experimental group (runners participating in the 2011 Comrades Marathon) and a control group (distance runners not taking part in the 2011 Comrades Marathon) during the recovery period after an ultramarathon race.

There were no significant differences between the two groups during the recovery period after an ultramarathon. The 5 km performance of both groups showed an improvement over time during the recovery period. In this study it was not possible to establish differences between groups in 5 km time trial performance.

Although it may be postulated that the change in performance of the experimental group may to be associated with the recovery from the reduced neuromuscular efficiency due to exercise induced muscle damage, it is not possible to conclude if these changes were due to changes in the experimental group.

To determine differences in muscle pain, heart rate and perception of effort during a 5 km time trial between groups and over time before, and during the recovery period after an ultramarathon race.

The experimental group showed increased quadriceps and hamstring pain at the first and second week following the ultramarathon when compared to the control group. However the control group showed increased gastrocnemius muscle pain at the second week following the ultramarathon. This pain in both groups was relatively mild when measured on the visual analogue scale and may have been as a result of the participants not being able to partake in any recovery routines or treatments following the time trials which they may have been accustomed to, thus allowing the participants to experience mild pain. The experimental groups' pain which occurred as a result of delayed onset muscle soreness was shown to return to pre-race levels by day six. There were no significant differences in heart rate between the experimental and control groups prior to the ultramarathon or over time in the recovery period. There were no significant differences in the rate of perceived effort between the two groups, although there was a change over time for the groups, showing the rate of perceived exertion being significantly higher at week two and week three post-race compared to pre-race values. This change in rate of perceived exertion has been attributed to the pre-race taper, combined effects of training, and psychological preparation for the race. Thus in summary the ultramarathon did not induce significant changes in the heart rate and rate of perceived exertion, there was however a change in muscle pain following the Comrades Marathon.

To determine whether there were any relationships between prior experience, training history, and running performance during the recovery period after an ultramarathon.

The study showed that the early change in performance following the ultramarathon had a positive relationship to previous pacing strategies used for personal best times in 10 km and 42 km races, which may support the role of central regulation and training experience affecting performance in the presence of fatigue and muscle damage during the recovery period.

The experimental group had a higher average and maximum training distance per week when compared to the control group, but the significance of this on the performance during the recovery period has not been clearly established. Future studies should investigate the effects of different training volume and intensities on the performance following an ultramarathon.

Based on the findings of this study it can be seen that the muscle pain induced by the ultramarathon race was resolved in seven days. Heart rate was unchanged. The change of the rate of perceived exertion increased over time this may have initially been low as a result of the tapering process; or as a protective mechanism to avoid injury prior to the race. The 5 km performance of both groups improved throughout the recovery period, this may have been affected by the effects of delayed onset muscle soreness initially and the recovery of the muscles structurally, teloanticipation as well as the feedback participants received during the time trials. This study may provide insight into the amount of time needed to safely recover from ultramarathon events, thereby potentially reducing the risk of overtraining or injury. The findings of this study may also be of practical relevance to health professional and coaches, and may contribute to the development of evidence-based guidelines regarding return to training and competition after an ultramarathon event.

Future research should investigate the effects of different training volume and intensities on the performance following an ultramarathon as well as investigating the differences performance following an ultramarathon using a combination of outcome measures for performance such a maximal treadmill speed test measuring or functional performance measurements.

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Appendix I:

UNIVERSITY OF CAPE TOWN

Health Sciences Faculty
Human Research Ethics Committee
Room E52-24 Groote Schuur Hospital Old Main Building
Observatory 7925
Telephone [021] 406 6338 • Facsimile [021] 406 6411
e-mail: shuretta.thomas@uct.ac.za

13 May 2011

HREC REF: 164/2011

Ms T Guest
c/o Dr T Burgess
Health & Rehab
F45, OMB

Dear Ms Guest

PROJECT TITLE: DIFFERENCES IN RUNNING PERFORMANCE IN THE RECOVERY PERIOD AFTER AN ULTRAMARATHON RACE.

Thank you for responding to the issues raised by the Faculty of Health Sciences Human Research Ethics Committee in your letter dated 12th May 2011.

It is a pleasure to inform you that the HREC has **formally approved** the above-mentioned study.

Approval is granted for one year till the 30 May 2012.

Please submit a progress form, using the standardised Annual Report Form (FHS016), if the study continues beyond the approval period. Please submit a Standard Closure form (FHS010) if the study is completed within the approval period.

Please note that the ongoing ethical conduct of the study remains the responsibility of the principal investigator.

Please quote the HREC. REF in all your correspondence.

Yours sincerely

PROFESSOR M BLOCKMAN
CHAIRPERSON, HSF HUMAN ETHICS
Federal Wide Assurance Number: FWA00001637.

sAricfdien

Annual Progress Report

REC REF Number	164/2011
Title	Differences in Running Performance in the Recovery Period after an Ultramarathon Race
Principal Investigator	Ms Tamlyn Guest

List of documentation

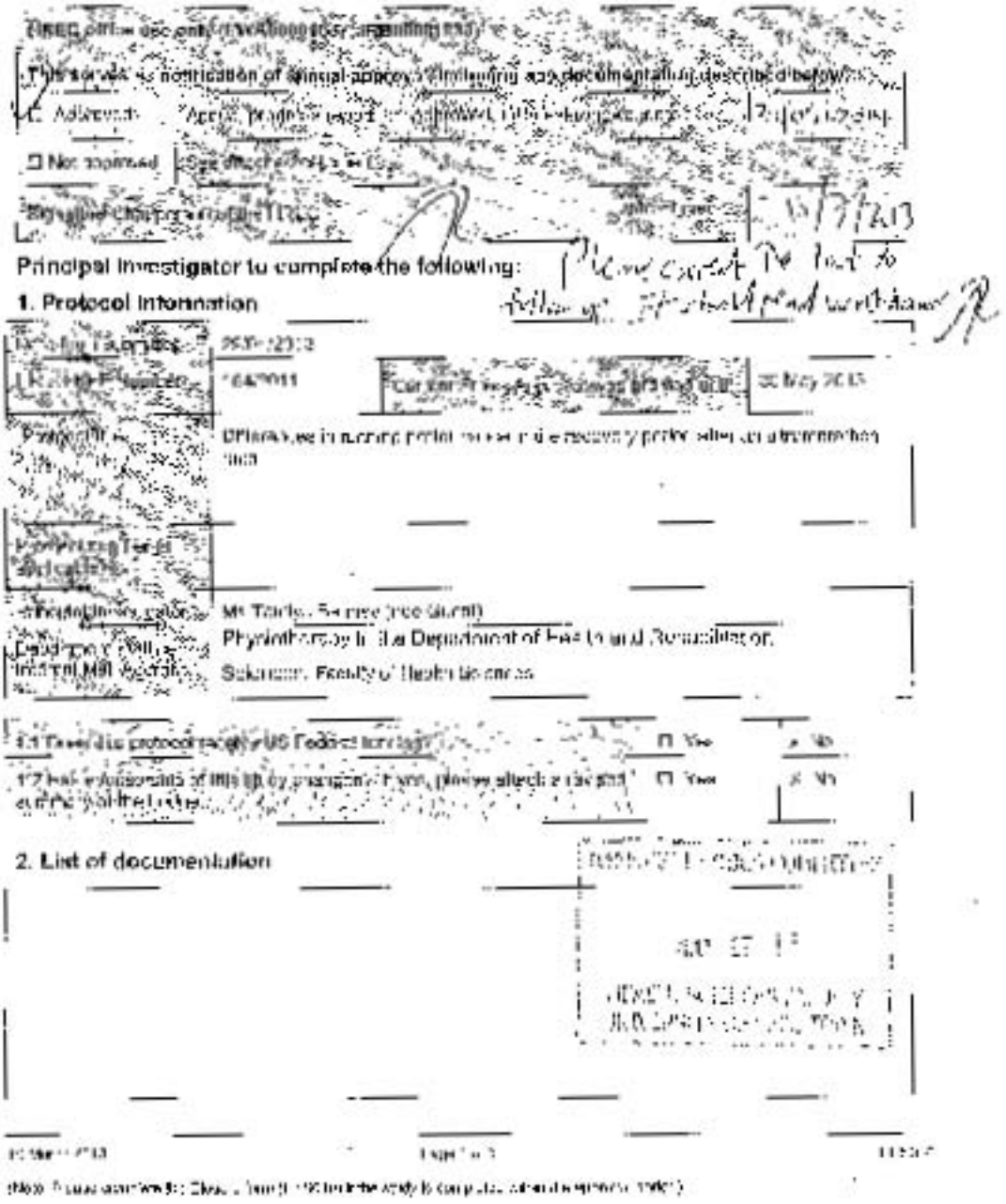
RESEARCH ETHICS COMMITTEE
2012 -04- 19
HEALTH SCIENCES FACULTY
UNIVERSITY OF CAPE TOWN

HREC office use only (FWA00001637; IRB00001938)

☑ Approved	This serves as notification of annual approval, including all documentation described above.		
☐ Not approved	See attached comments.		
Type of review	☑ Expedited	☐ Full committee	
Expiry date	30 MAY 2013		
Signature Chairperson of the HREC		Date	19/4/2012

FACULTY OF HEALTH SCIENCES

FHS016: Annual Progress Report / Renewal



Principal Investigator to complete the following:

1. Protocol Information

2. List of documentation

Appendix II:

UCT/MRC Research Unit for Exercise Science and Sports Medicine
Department of Human Biology
Division of Physiotherapy, Department of Health & Rehabilitation Sciences
Faculty of Health Sciences
University of Cape Town, South Africa

MALE COMRADES MARATHON RUNNERS WANTED FOR UCT RESEARCH

For study investigating the effects of running an ultramarathon on exercise performance during the recovery period

Study outline

I am a Masters student at UCT, investigating running performance during the recovery period after an Ultramarathon. The study aims to provide information regarding optimal recovery time before returning to competitive training and competition.

The study requires participants to complete a pre-race and two post-race 5 km time trials. Muscle pain, heart rate and perception of effort will be measured during the time trials. It will be requested of you to keep a detailed training and general pain and stiffness diary over the testing period.

Those interested in participating should ..

- be male, between the ages of 20 and 60 years

- have a current marathon (42.2 km) time of less than five hours

- have run a minimum of one previous Comrades marathon

Benefits of participating in the study include

- Individual anthropometric measurements (Height, weight, BMI, body fat %)
- 5 km time trial results – this will help you to see when you have recovered after the race
- Feedback regarding the results of the study

DEADLINE FOR APPLICATIONS: 30 May 2011

If you are interested in taking part in the study and would like additional information, please contact:

Tamlyn Guest:
Cell: 083 703 2143
Email: tamlynjane@mweb.co.za

SPORTS SCIENCE INSTITUTE OF SOUTH AFRICA

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UCT/MRC Research Unit for Exercise Science and Sports Medicine
Department of Human Biology
Division of Physiotherapy, Department of Health & Rehabilitation Sciences
Faculty of Health Sciences
University of Cape Town, South Africa

MALE DISTANCE RUNNERS WANTED FOR UCT RESEARCH

For study investigating the effects of running an ultramarathon on exercise performance during the recovery period

Study outline

I am a Masters student at UCT, investigating running performance during the recovery period after an Ultramarathon. The study aims to provide information regarding optimal recovery time before returning to competitive training and competition.

The study requires participants to complete a three 5 km time trials which will be compared to a group of runners who are participating in the Comrades Marathon. Muscle pain, heart rate and perception of effort will be measured during the time trials. It will be requested of you to keep a detailed training and general pain and stiffness diary over the testing period.

Those interested in participating should be

- male, between the ages of 21 and 60 years

- running a minimum average of 40 km per week for the past 4 months

- not be taking part in the 2011 Comrades marathon.

Benefits of participating in the study include

- Individual anthropometric measurements (Height, weight, BMI, body fat %)
- 5 km time trial results
- Feedback regarding the results of the study

DEADLINE FOR APPLICATIONS: 30 May 2011

If you are interested in taking part in the study and would like additional information, please contact:

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Appendix III:

Consent form:

Differences in running performance during the recovery period after an ultramarathon race

Dear Participant

I am a Masters student in the Division of Physiotherapy, University of Cape Town. I will be conducting a study to determine the changes of running performance during the recovery period after an ultramarathon race. There are conflicting ideas regarding the optimal recovery time before returning to pre-race performance levels, and a frequently asked question is ”when can runners return to competitive running following the completion of an ultramarathon race”? This study aims to help coaches and athletes answer this question and identify the recovery period necessary for the return to pre-race performance. You have been selected to participate in this study as you are either preparing to participate in the Comrades Marathon on the 29 May 2011, and will form part of the Experimental group, or you are a distance runner who is not running this year’s Comrades Marathon and will thus form part of the Control group. I will be using the Comrades Marathon 2011 as the ultramarathon from which the recovery will be examined. The information obtained in this study will be used for the completion of a mini-dissertation as required for the partial fulfilment of the Masters in Philosophy in Sports Physiotherapy (MPhil Sports Physiotherapy) from the University of Cape Town. This study has been given ethical approval by the University of Cape Town, Faculty of Health Sciences Human Research Ethics Committee (HREC).

You will be asked to attend a total of five appointments lasting for approximately one hour. These will be conducted at the Wanderers Planet fitness gym as it has an indoor track. You will be required to travel to the gym at your own cost as there is no funding for this study.
This study will be supervised by Dr Theresa Burgess, Senior lecturer in Physiotherapy at the University of Cape Town, and Professor Mike Lambert of the MRC/UCT Research Unit for Exercise Science and Sports Medicine.
Please take time to read through this form thoroughly and carefully before signing. If you have any questions regarding this form please feel free to ask.

The study has the following sessions:

1) Familiarisation session:

This will be run 10 days prior to the Comrades Marathon. During this session you will be required to complete a questionnaire detailing your medical history, previous injuries, training and competition details. Your weight, height and skin fold thicknesses will be measured in order to calculate your body fat percentage. A Base line level of your muscle pain will be taken. All the tests will be explained to you on the day and time will be allocated for any questions you may have. You will also be allowed to run a simulation time trial to familiarise yourself with the track and the testing procedure.

I do request that you refrain from using any medication, recovery treatments, partaking in any strenuous training or racing, other than competing in the Comrades Marathon, for the duration of the study. It will also be required of you to maintain the same diet and training regime for 24 hours prior to a testing day. Compliance to these instructions will be facilitated by completing a logbook for the duration of the study; this will be given to you at this session.

Test session1:

This session will be carried out 7 days before the Comrades Marathon. You will be required to complete a compliance questionnaire, giving information about any exercise you have been doing, and any other factors that may affect your running performance in the 5km time trial. The time trial will be performed at the Wanderers Planet fitness gym. Time will be allocated for you to perform a 10 minute warm up. During the run you will wear a heart rate monitor. At every 1km split your time will be given to you as well as the distance covered. You will be required to rate your level of perceived exertion at every kilometre. A cool down period will be performed at the end of the test.

The Comrades Marathon:

The Experimental group of Comrades runners will be required to run and finish the marathon. The official time will be found on the race website. From the day after the race the participants in the experimental group will be required to report their levels of muscle pain in the logbook provided at the familiarisation session, the muscle pain needs to be recorded for the seven days following the race.

Test session 2:

This will be conducted 6 days after the Comrades Marathon, at the Wanderers Planet fitness gym as before. You will be required to complete a Compliance questionnaire and perform a 5km time trial as you did in test session 1.

Test session 3:

This will be conducted 13 days after the Comrades marathon, and the same procedure will be followed as test session 1 and 2.

Test session 4:

This will be conducted 20 days after the Comrades marathon, and the same procedure will be followed as before.

Potential risks:

During the skin fold thickness test you may feel slight and short-lived discomfort due to the use of the callipers. The 5km time trial is a maximal performance test which requires you to exert yourself, during this type of test you are at risk of injuring yourself, but time will be allocated for you to carry out a warm up so that this risk is minimised. The participation in the Comrades Marathon has its own inherent risks associated with the performance of an endurance event. In an attempt to minimise risks associated with this you will complete screening questionnaires. If any risks are identified you will be referred to your medical practitioner for a medical assessment.

Benefits to participating in this study:

You will receive all your data (anthropometric measurements, time trial results, heart rate and perceived exertion measures) in an information pack once the study is complete. This may help you understand your recovery better and how your performance is affected in the recovery period. The final results of the study will also be given to you. You will be required to travel at your own cost as there is no funding for the study. You will also not receive any payment for taking part in this study.

You are under no obligation to participate in this study, and you do have the right to withdraw from the study at anytime if you do consider participating in it. All personal information which you provide us will be kept confidential. The test scores and measurements are also confidential, no names will be disclosed.

Concerns:

If you have any concerns or questions at any time during the study please feel free to contact myself, Tamlyn Guest or one of my supervisors.

Tamlyn Guest
Cell: 0837032143
Email: tamlynjane@mweb.co.za

Should you have any further queries please contact:

Dr Theresa Burgess

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Observatory 7725

Tel number: 021 406 6171

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Associate Professor Mike Lambert

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Professor Marc Blockman

Chairperson: Faculty of Health Sciences Research and Ethics Committee

Tel: 021 406 6492

E-mail: marc.blockman@uct.ac.za

Please note that UCT does offer a no-fault insurance that will cover all participants in the event that something may go wrong. This insurance will provide prompt payment of compensation for any trial-related injury in accordance with the Association of the British Pharmaceutical Industry (ABPI) guidelines (1991). These guidelines recommend that UCT, without any legal commitment, should compensate you without you having to prove that UCT is at fault. An injury is considered trial-related if, and to the extent that, it is caused by study activities. You must notify the study investigators immediately of any injuries during the trial, whether they are research-related or other related complications. UCT reserves the right not to provide compensation if, and to the extent that, your injury came about because you chose not to follow the instructions that you were given while taking part in the study. Your right in law to claim compensation for injury where you prove negligence is not affected.

By placing your signature below, it serves as confirmation that you have had adequate time to read through, have understood the consent form and that you are willing to participate in this study. You have the right to withdraw at any time. You may ask questions at any time during the study. All the information recorded will be confidential. Your signature is further confirmation that you are aware of the possible risks involved in this study.

_____	_____	_____
Signature of Volunteer	Name (Please Print)	Date
_____	_____	_____
Signature of Investigator	Name (Please Print)	Date

Appendix IV:

PAR-Q

Physical Activity Readiness Questionnaire

Regular exercise is growing in popularity. Being more active is very safe for most people, and for most should not pose any problem or hazard. However, some people should check with their doctor before they start becoming much more physically active. The following list of questions should be completed by anyone who is between the ages of 15 and 69, looking to increase their current activity level, or partake in a fitness testing assessment. The questionnaire helps to determine how safe it is for you.

Common sense is your best guide in answering these questions. Read the questions carefully and answer each one honestly.

Yes	No	
☐	☐	Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?
☐	☐	Do you feel pain in your chest when you do physical activity?
☐	☐	In the past month, have you had chest pain when you were not doing physical activity?
☐	☐	Do you lose your balance because of dizziness or do you ever lose consciousness?
☐	☐	Do you have a bone or joint problem that could be made worse by a change in your physical activity?
☐	☐	Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
☐	☐	Do you know of any other reason why you should not do physical activity?

If you answered YES

If you answered "yes" to one or more questions, talk with your doctor before you start becoming much more active or before you have a fitness test. Tell your doctor about the PAR-Q and which questions you answered "yes" to.

If you answered NO

If you answered "no" honestly to all of the questions, you can be reasonably sure that you can start becoming much more physically active or take part in a physical fitness appraisal – begin slowly and build up gradually. This is the safest and easiest way to go.

Things Change

Even if you answered "no" to all questions, you should delay becoming more active if you are temporarily ill with a cold or a fever, or if you are or may be pregnant. If your health changes so that you then answer "yes" to any of the above questions, tell your fitness or health professional and ask whether you should change your physical activity plan.

Appendix V:

2011 COMRADES MARATHON

Medical and Training Questionnaire

Thank you for taking the time to complete this questionnaire, which will take 30-45 minutes of your valuable time to complete. The completion of the questionnaire is voluntary and all the information will be kept confidential. The information collected will only be used for research purposes.

Instructions

Please complete Sections A, B, C, D, E, F

Section A Personal Details

Section B Racing and Training History

Section C Tapering History

Section D Flexibility Training History

Section F General Personal Medical History

Please complete only the relevant questions in the following section

Section G Additional Detailed Medical History

Section A: Personal details			
2011 Comrades Race Number			
Surname			
First Name			
Postal Address		Postal/ Zip Code	
E-mail address		Phone (day time)	code number
Date of birth	y y y y - m m - d d	Cell	
Height	cm	Gender	Male ☐ Female ☐
Weight	kg	Age	
Occupation			
What **percentage** of your **working** day is spent in the following activities?	Sitting: _____ % Standing: _____ % Walking (Lower body activity) _____ % Manual Labour (upper and body activity) _____ %		

Section B: Racing and training history					
What is your predicted time for the 2011 Comrades Marathon?	_____ hrs:min				
Type of running event	**5 km**	**10 km**	**21.1 km**	**42.2 km**	**Ultra**
Which races have you participated in?	Yes ☐ No☐	Yes ☐ No☐	Yes ☐ No☐	Yes ☐ No☐	Yes ☐ No☐
Year of first event					
How many events have you participated in?					
Personal best time	_____ hrs:min	_____ hrs:min	_____ hrs:min	_____ hrs:min	_____ hrs:min
What is your best time, in a running race, in the **last 4 months**?	_____ hrs:min	_____ hrs:min	_____ hrs:min	_____ hrs:min	_____ hrs:min
Type of event	**Two Oceans Marathon**	**Comrades Marathon**			
Which races have you participated in?	Yes ☐ No☐	Yes ☐ No☐			
Year of first event					
How many events have you participated in?					
Personal best time	_____ hrs:min	_____ hrs:min			
Pacing					
Do you use a pacing strategy when you run marathons?	Yes	No			
If yes please describe your pacing strategy.					

Section C: Tapering history							
Time until race day	16 – 13 weeks	12 - 9 weeks	8 – 5 weeks	4th week	3rd week	2nd week	Last week before the race
How many days a week did you train **during your taper period**?	days/week	days/week	days/week	days/week	days/week	days/week	days/week
How many **hours** did you train **during your taper period**?	_____ hours	_____ hours	_____ hours	_____ hours	_____ hours	_____ hours	_____ hours
What was the **duration** of your training **sessions**?	_____ min _____ max _____ average	_____ min _____ max _____ average	_____ min _____ max _____ average	_____ min _____ max _____ average	_____ min _____ max _____ average	_____ min _____ max _____ average	_____ min _____ max _____ average
What was the **distance** and **pace** of your **LSD runs**?	_____km _____min per km	_____km _____min per km	_____km _____min per km	_____km _____min per km	_____km _____min per km	_____km _____min per km	_____km _____min per km
What was your **slowest, average** and **fastest training pace**?	Slowest _____min per km Average	Slowest _____min per km Average	Slowest _____min per km Average	Slowest _____min per km Average	Slowest _____min per km Average	Slowest _____min per km Average	Slowest _____min per km Average

	_____min per km Fastest _____min per km	_____min per km Fastest _____min per km	_____min per km Fastest _____min per km	_____min per km Fastest _____min per km	_____min per km Fastest _____min per km	_____min per km Fastest _____min per km	_____min per km Fastest _____min per km
What was your **average training pace** in the last **three months**?	_____min per km						
What was your **average race pace** in the last **three months**?	_____min per km						

Section D: Flexibility training history	
Do you perform flexibility training (stretching exercises)?	Yes ☐ No ☐
If YES, please complete the rest of the flexibility training history section below:- If NO, continue completing the questionnaire from the top of page 5 (Fluid intake).	
On average, how many days a week do you perform a stretching session?	days/week
On average, how many times a day do you perform a stretching session?	times/day
Please tick which muscle groups do you include in your stretching session?	☐ Hamstrings ☐ Quadriceps ☐ Calf (gastrocnemius) ☐ Calf (soleus) ☐ Groin (inner thigh) ☐ Upper body limbs ☐ Other: _____
Please tick when you stretch? (before, during and/or after exercising. You can tick more than one box)	☐ Before Exercise ☐ During Exercise ☐ After Exercise
When you stretch an individual muscle group, on average, **how long do you hold the stretch** for?	seconds
When you stretch an individual muscle group, on average, **how many times do you stretch the muscle for**?	☐ Once ☐ Twice ☐ 3 times ☐ 4 times ☐ 5 times ☐ 6 or more times

Section E: Fluid intake	
How do you best describe your fluid intake during a race?	(a) I drink to thirst ☐ (b) I drink as much as tolerable ☐ (c) I drink according to a predetermined fluid intake schedule ☐ (d) I drink to prevent any weight loss during exercise ☐ (e) I combine (a) with (c) ☐ (f) I combine (b) with (c) ☐ (g) Other: _____ ☐
What percentage of your fluid intake will consist of these beverages?	Water: ☐ 0-25% ☐ 26-50% ☐ 51-75% ☐ 76-100% Sports drink: ☐ 0-25% ☐ 26-50% ☐ 51-75% ☐ 76-100% Coke: ☐ 0-25% ☐ 26-51% ☐ 51-75% ☐ 76-100% Other: ☐ 0-25% ☐ 26-50% ☐ 51-75% ☐ 76-100% Specify other: _____
What will be your estimated **total** fluid intake be during the **run**?	ml
Rank the following sources of information on their importance in formulating your drinking strategy. (1 being most influential and the lowest number being least influential)	_____ Fellow triathletes _____ Coach / trainer _____ Magazines / books _____ Website (please specify: _____) _____ Drinking guidelines from sports associations _____ Adverts _____ Self-experimentation _____ Other: _____

Section F: Personal general medical history

In this section, you are asked to read through 14 questions about your personal general medical history. If you answer "yes" to any of questions 1 to 12, please complete the additional questions at the end of the section (Section F).

1. In the **6 weeks before this race** (from 1st February) did you suffer from any **symptoms of flu** (fever, sore throat, blocked or runny nose, cough, wheeze, muscle aches and pains)? **If you answer "yes", please complete the additional questions in Section G.**	Yes ☐ No ☐
2. Have you **ever** in your marathon career suffered from **muscle cramping** during or immediately (within 6 hours) after exercise (in training or competition)?	Yes ☐ No ☐
3. Have you **ever** in your marathon career suffered from **a tendon or ligament injury** (pain, swelling, stiffness) in any tendon (including Achilles tendon, knee tendons, and shoulder tendons) or ligaments (partial or complete tear)?	Yes ☐ No ☐
4. Have you **ever** in your marathon career **used medicines to treat injuries** in the week **before or during a race** – including anti-inflammatory drugs, cortisone (pills, or injection), or pain killers? **If you answer "yes", please complete the additional questions in Section G.**	Yes ☐ No ☐
5. Do you **currently** suffer from any **symptoms of injury** in the muscles, tendons, bones, ligaments or joints? **If you answer "yes", please complete the additional questions in Section G.**	Yes ☐ No ☐
6. Please tick in which anatomical area you ever had **surgery** performed.	☐ Head ☐ Finger ☐ Neck ☐ Lower back ☐ Face ☐ Hip ☐ Front chest ☐ Thigh ☐ Back chest ☐ Knee ☐ Shoulder ☐ Lower leg ☐ Upper arm ☐ Achilles ☐ Elbow ☐ Ankle ☐ Forearm ☐ Foot ☐ Wrist ☐ Abdomen ☐ Other (Specify: _____)

THANK YOU FOR COMPLETING THIS QUESTIONNAIRE

If you have answered **YES** to questions 1, 4, or 6 of the Personal General Medical History questionnaire (Section F) please complete the relevant additional questions that follow in Section G.

Section G: Additional detailed medical history

(Please complete all the sections to which you answered "Yes" in the Personal general medical history)

1. Flu symptoms in the last 6 weeks

If you answered **YES** to **question 1** in section E, please complete the following two questions related to flu symptoms in the last 6 weeks.

(1a) Please tick which of these flu symptoms you suffered from **in the last 6 weeks**.	☐ Fever ☐ Cough ☐ Joint pains ☐ Blocked nose ☐ Wheezing ☐ Runny nose ☐ Muscle aches ☐ Any other flu symptoms (Specify: _____)
(1b) Please tick which of these flu symptoms you suffered from **in the last 7 days**.	☐ Fever ☐ Cough ☐ Joint pains ☐ Blocked nose ☐ Wheezing ☐ Runny nose ☐ Muscle aches ☐ Any other flu symptoms (Specify: _____)

2. Use of medicines to treat an injury before or during participation If you answered **YES** to **question 4** in section E, please complete the following two questions related to medicine use for injuries before or during races.	
(2a) Which of the following medicines have you used in the past to treat an injury **in the week just before** a race?	☐ Paracetamol (e.g. Panado, Tylenol) ☐ Non-steroidal anti-inflammatories (e.g. Voltaren, Cataflam) ☐ Cortisone (pills) ☐ Cortisone injection ☐ Codeine ☐ Anti-inflammatory gels/creams/patches ☐ Any other pain killers (Specify: _____)
(2b) Which of the following medicines have you used in the past to treat an injury **during a race**?	☐ Paracetamol (e.g. Panado, Tylenol) ☐ Non-steroidal anti-inflammatories (e.g. Voltaren, Cataflam) ☐ Cortisone (pills) ☐ Cortisone injection ☐ Codeine ☐ Anti-inflammatory gels/creams/patches ☐ Any other pain killers (Specify: _____)

3. History of any current injury that you suffer from

If you answered **YES** to **question 11** in section E, please complete the following questions (11a. to 11g.) related to each of your current injury/ies (Space is provided for two injuries)

Injury 1	
(3a) What was the approximate date when you first became aware of the injury?	Month Year
(3b) Please indicate which side of your body is injured (if applicable)	☐ Right ☐ Left
(3c) Please indicate which anatomical area is currently injured	☐ Head ☐ Neck ☐ Face ☐ Front chest ☐ Back chest ☐ Shoulder ☐ Upper arm ☐ Elbow ☐ Forearm ☐ Wrist ☐ Finger ☐ Lower back ☐ Hip ☐ Thigh ☐ Hamstring ☐ Quadriceps ☐ Knee ☐ Shin ☐ Achilles ☐ Ankle ☐ Foot Other (Specify: _____)
(3d) Please indicate the type of structure that was injured	☐ Muscle ☐ Tendon ☐ Bone ☐ Ligament ☐ Joint Other (Specify: _____)
(3f) Please indicate the severity of the injury (tick one box please)	☐ I only experience symptoms after exercise - Grade 1 ☐ I experience symptoms during exercise, but it does not interfere with exercise - Grade 2 ☐ I experience symptoms during exercise that may interfere with my training/ competition - Grade 3 ☐ I am so painful that I may not be able to train or compete - Grade 4
(3g) Please indicate how your injury was treated to date (you can tick more than one)?	☐ Rest ☐ Stretches ☐ Physiotherapy ☐ Surgery ☐ Strengthening exercises ☐ Equipment change ☐ Tablets ☐ Cortisone injection ☐ Other injection ☐ Orthotics Other (Specify: _____)

Injury 2		
(3a) What was the approximate date when you first became aware of the injury?		Month Year
(3b) Please indicate which side of your body is injured (if applicable)		☐ Right ☐ Left
(3c) Please indicate which anatomical area is currently injured		☐ Head ☐ Neck ☐ Face ☐ Front chest ☐ Back chest ☐ Shoulder ☐ Upper arm ☐ Elbow ☐ Forearm ☐ Wrist ☐ Finger ☐ Lower back ☐ Hip ☐ Thigh ☐ Hamstring ☐ Quadriceps ☐ Knee ☐ Shin ☐ Achilles ☐ Ankle ☐ Foot Other (Specify: _____)
(3d) Please indicate the type of structure that was injured		☐ Muscle ☐ Tendon ☐ Bone ☐ Ligament ☐ Joint Other (Specify: _____)
(3f) Please indicate the severity of the injury (tick one box please)		☐ I only experience symptoms after exercise - Grade 1 ☐ I experience symptoms during exercise, but it does not interfere with exercise - Grade 2 ☐ I experience symptoms during exercise that may interfere with my training/ competition - Grade 3 ☐ I am so painful that I may not be able to train or compete - Grade 4
(3g) Please indicate how your injury was treated to date (you can tick more than one)?		☐ Rest ☐ Stretches ☐ Physiotherapy ☐ Surgery ☐ Strengthening exercises ☐ Equipment change ☐ Tablets ☐ Cortisone injection ☐ Other injection ☐ Orthotics Other (Specify: _____)

Appendix VI:

Anthropometry

Participants Name:_____

Body mass _____

Stature _____

BMI _____

Dominant leg _____

Skinfold measurements (mm)	
Triceps	
Biceps	
Sub-scapular	
Supra-iliac	
Thigh	
Calf	
Abdominal	
Girth measurements (cm)	
Relaxed arm	
Contracted arm	
Forearm	
Wrist	
Chest	
Abdominal	
Bi-trochanteric	
Sub-gluteal	
Mid-thigh	
Above-knee	
Calf	
Ankle	
Diameter measurements (mm)	

Humerus	
Bi-acromial	
Transverse chest	
AP chest	
Bi-iliac	
Femur	
Length measurement (cm)	
Sub-gluteal to above- knee	

Sum of 7 skinfolds _____

Predicted % body fat _____

Lean body mass _____

Test session data sheet:

Name:_____

5km Time Trail Results: (Kilometre splits)

	1km	2km	3km	4km	5km
Test 1					
Test 2					
Test 3					
Test 4					

Rate of Perceived exertion at 1km splits

	1km	2km	3km	4km	5km
Test 1					
Test 2					
Test 3					
Test 4					

Heart rates during the time trials at 1km splits:

	1km	2km	3km	4km	5km
Test 1					
Test 2					
Test3					
Test 4					

Total Time Trial Times:

	Total time for 5km
Test 1	
Test 2	
Test3	
Test 4	

Appendix VII:

Logbook of muscle pain measurement scores, daily training and dietary information

Multi-Dimensional Pain Scale

Quadriceps

No pain Maximal pain

Pain at rest

No pain Maximal pain

Pain during normal daily activities

No pain Maximal pain

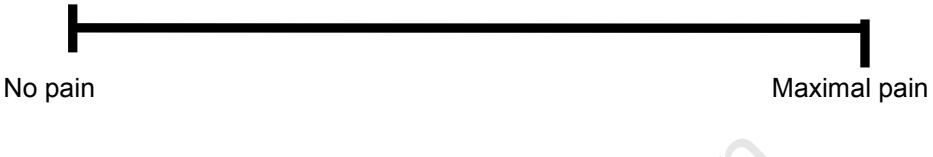
Pain during passive stretch

No pain Maximal pain

Pressure pain

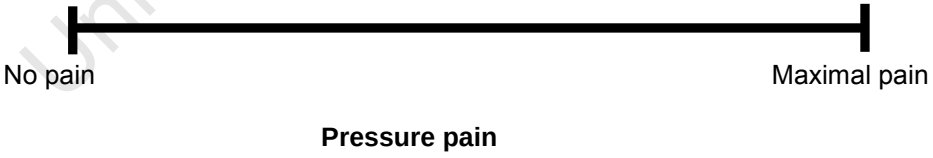
Hamstrings

Pain at rest



Pain during normal daily activities

Pain during passive stretch



Pressure pain

Gastrocnemius (Calf)

No pain Maximal pain

Pain at rest

No pain Maximal pain

Pain during normal daily activities

No pain Maximal pain

Pain during passive stretch

No pain Maximal pain

Pressure pain

(Seven days of these sheets will be provided in the logbook)

Daily Log of dietary details and training information:

Please fill out this table for the duration on the study:

Day	Breakfast	Lunch	Supper	Type of exercise if any	Medication taken	Use of any recovery methods/treatment (list)
21/05/2011						
Test session1 22/05/2011						
23/5/2011						
24/5/2011						
25/5/2011						
26/5/2011						
27/5/2011						
28/5/2011						
Comrades Day						
30/5/2011						
31/5/2011						
1/6/2011						
2/6/2011						
3/6/2011						
4/6/2011 Test session 2						
5/6/2011						
6/6/2011						
7/6/2011						
8/6/2011						

9/6/2011						
10/6/2011						
11/6/2011 Test session 3						
12/6/2011						
13/6/2011						
14/6/2011						
15/6/2011						
16/6/2011						
17/6/2011						
18/6/2011 Test session4						

Appendix VIII:

Compliance Questionnaire:

Name:_____

Please answer the questions as truthfully as possible.

	Yes	No
1) Have you participated in any type of exercise since the completion of the Comrades Marathon? If yes what have you done_____		
2) Have you used and medication to alleviate muscle pain?		
3) Have you massaged or rubbed your stiff and sore muscles?		
4) Have you put ice or heat-packs on your muscles?		
5) Have you stretched your stiff muscles?		
6) Have you made use of compression garments?		
7) Have you done anything else to alleviate stiff and sore muscles? If yes what have you done?_____		

Thank you for taking the time to complete this

Appendix IX:

Modified Borg Scale: Rare of Perceived Exertion

Rating scores for relative perception of effort (RPE) (BORG, 1982)

Score	Description
6	
7	Very Very light
8	
9	Very Light
10	
11	Fairly Light
12	
13	Somewhat Hard
14	
15	Hard
16	
17	Very Hard
18	
19	Very. Very Hard
20	Maximal exertion

Appendix X:

MRC/UCT RESEARCH UNIT FOR EXERCISE SCIENCE AND SPORTS MEDICINE

Participant feedback

Differences in running performance during the recovery period after an ultramarathon race

SPORTS SCIENCE INSTITUTE OF SOUTH AFRICA

essm EXERCISE SCIENCE AND SPORTS MEDICINE

MRC/UCT

RESEARCH UNIT FOR EXERCISE SCIENCE AND SPORTS MEDICINE

14th November 2012

Dear

Thank-you very much for participating in this study. We realise that the testing procedure was both time-consuming and strenuous, and cannot adequately express our gratitude to you for completing the study. We really appreciated your continued good humour and patience throughout the testing procedure.

We also hope that you enjoyed the experience, and thank you for excellent results that you gave us. We trust that you will find the information contained in this folder both interesting and exciting, and that you will be able apply some of this information to your training and racing.

Should you require any further details regarding the results of the study, please do not hesitate to contact Tamlyn at 083 703 2143 *(c)*, tamlynjane@mweb.co.za *(email)*.

Thank-you once again for completing the study!

Wishing you good running,

Best regards

Tamlyn Guest

Personal Information

Body mass:	78.5	kg
Stature:	182.0	cm
Sum of seven skinfolds:	57mm	
Body fat percentage:	12.6 %	
Lean body mass:	68.6	kg
Lean thigh volume:	5195	cc
Maximum heart rate (HR_{max})	191	

Time trial results:

Kilometers	Time trial 1 (before Comrades)	Time trial 2 (1 week after Comrades)	Time trial 3 (2 weeks after Comrades)	Time trial 4 (3 weeks after Comrades)
1	4.36	4.19	4	4.38
2	9.13	9.35	8.56	10.3
3	13.4	13.41	12.53	15.08
4	17.57	17.44	16.49	19.44
5	22.54	21.43	20.42	24.24

Perceived exertion:

Kilometres	Time trial 1 (before Comrades)	Time trial 2 (1 week after Comrades)	Time trial 3 (2 weeks after Comrades)	Time trial 4 (3 weeks after Comrades)
1	11	12	12	11
2	12	13	13	11
3	12	14	14	12
4	13	16	16	12
5	15	17	18	15

SUMMARY OF STUDY RESULTS:

The results of this study showed no significant difference between the groups for the 5 km time trial performance and that both groups' performance improved during the testing period from post-race TT1 to post-race TT3. The results of this study do not allow for investigation of the differences in performance for the groups during the time trials. Further studies using larger sample sizes may allow for the differences between the groups to be determined. No alterations in heart rate were noticed despite this being found to be delayed and exaggerated in a previous study for up to 25 days following an ultramarathon (179). There was a change of rate of perceived exertion throughout the testing procedure which may have initially been low in the experimental group as a result of the tapering process.

The Comrades Marathon induced muscle pain in the experimental group consistent with delayed onset muscle soreness. The muscle pain in the gastrocnemius, quadriceps and hamstring muscles of the experimental group occurred within the first 24 hours after the race, and returned to a pain free state after seven days. These findings are supported by other studies investigating the delayed muscle soreness as a result of exercise induced muscle damage (64)(65)(60). An interesting finding of this study is that the changes in early performance following the Comrades showed a positive relationship to previous pacing strategies used in 10 km and 42 km races which may support the role of central regulation and prior experience affecting performance in the presence of fatigue and muscle damage. This is supported by Burgess's (12) findings that there may be an interaction between protective adaptations and central mechanisms during the recovery period after an ultramarathon race.

CONCLUSIONS

Based on the findings of this study it can be seen that the muscle pain induced by the ultramarathon race was resolved in seven days. Heart rate was unchanged. The change of the rate of perceived exertion increased over time this may have initially been low as a result of the tapering process; or as a protective mechanism to avoid injury prior to the race. The 5 km performance of both groups improved throughout the recovery period, this may have been affected by the effects of delayed onset muscle soreness initially and the recovery of the muscles structurally, teloanticipation as well as the feedback participants received during the time trials. This study may provide insight into the amount of time needed to safely recover from ultramarathon events, thereby potentially reducing the risk of overtraining or injury. The findings of this study may also be of practical relevance to health professional and coaches, and may contribute to the development of evidence-based guidelines regarding return to training and competition after an ultramarathon event.

Feedback explanation form:

Anthropometry

Anthropometry is the process of measuring physical dimensions of the human body. These measurements are then used to either describe size and proportions, or to indirectly estimate body composition.

In this study, body mass, stature, skinfold thicknesses, and girth measurements were recorded. From these measurements, the sum of seven skinfolds, estimated body fat (%), lean body mass, and lean thigh volume were recorded.

i. Body fat percentage

The Durnin and Wormersley technique is used to estimate body fat percentage. The calculation involves measuring four skinfold sites, being triceps, biceps, subscapular and suprailiac, and substituting the log of their sum into an equation.

ii. Sum of seven skinfolds

Body fat may be described as the sum of seven skinfolds. The seven sites used are triceps, biceps, subscapular, suprailiac, calf, thigh and abdomen.

There is a tendency in laboratories around the world to move away from expressing an athlete's body fat as a percentage, but rather to express body fat as a sum of seven skinfolds (mm). This is because the use of skinfold thicknesses to predict body fat percentage is a "doubly-indirect" procedure and therefore has inherent inaccuracies. It is also assumed that the densities of the fat and fat-free mass are constant. However, these assumptions are not always met. It is therefore recommended that the sum of seven skinfolds be used to assess body composition.

iii. Lean body mass

Lean body mass (or fat-free mass) is calculated as:

lean body mass = (body mass) - (fat mass)

where fat mass = body mass x % body fat

iv. Lean thigh volume

The measurement of lean thigh volume is adapted from a technique previously described by Katch and Katch (1974). The assumptions associated with this technique are that the thigh is a truncated cone, and that fat deposition on the thigh is evenly distributed. The calculation involes the measurement of the triceps skinfold, sub-gluteal, mid-thigh and above-knee girth measurements, and the sub-gluteal to above-knee length measurement. This measurement is important for the EMG recordings, as a high lean thigh volume measurement may interfere with the EMG signal.

Typical anthropometry measurements for elite distance runners are presented in the table below.

Measurement	Male	Female
Body fat percentage (%)	8 ± 1	13 ± 1
Sum of seven skinfolds (mm)	< 40	< 45
Lean thigh volume (cc)	2500 – 6000	2000 - 5000

III. Maximum heart rate (HR_{max})

One method of determining effort during running is to monitor heart rate during exercise. It is known that maximum heart rate falls with age. A simple equation to remember is that maximum heart rate (in beats/minute) can be calculated as 220 minus age in years. It is thought that the maximum benefit from training is achieved by training at between 60 % to 90 % of HR_{max} . Ideally heart rates should fall between these values for most of the training time. Values higher than these should be achieved only during short duration speed training;

lower values only when one is jogging during the days of recovery from hard training or racing.

To control exercise intensity using this method, the pulse rate must be measured accurately. This can be done either with the use of a heart rate monitor, or by counting the pulse rate at any convenient spot where you feel a pulse. For example, the artery on the thumb side of your wrist. Start counting immediately after exercise and count for 10 seconds only, since the pulse rate returns to slower resting levels very rapidly. Then multiply the 10-second count by six to get a heart rate in beats per minute.

The table below shows maximum heart rates and target range (60-90% of HR_{max}) for different ages.

Age	**HR_{max} (beats/min)**	**Target HR range (beats/min)**	**Target HR range (beats/10sec)**
20 – 29	200	120 - 180	20 - 30
30 – 39	190	114 - 168	19 - 28
40 – 49	180	108 - 162	18 - 27
50 – 59	170	102 - 150	17 - 25
60 – 69	160	96 - 144	16 - 24
70 +	150	90 - 132	15 - 22

It should be noted that there are no scientific data to support an ideal specific heart rate for different types of training, and much of what is written is based on anecdotal experiences. There is no doubt that future studies will refine this area, making the prescription of training heart rate a more exact science. Many studies have documented that heart rates measured during races are consistently higher than the heart rates measured at the same running speeds during training. This finding has important implications for runners using heart rate monitors during competition as a gauge of their running pace because the athlete will run slower than expected during a race, should a racing target heart rate be calculated on a heart rate determined during training. Indeed, this finding has already been discussed in the lay press after a competitive runner ran slower than expected in the Comrades marathon after setting his pace according to his heart rate (Green, 1994).

Therefore, monitoring heart rate during competition is not an accurate indication of running speed, and further research will need to identify the cause of the elevated heart rate during competition, before heart rate monitors can be used with accuracy in competition.

Appendix XI:

Correlational analysis for delta performance and maximum training distance

There were no significant relationships between early delta performance and the maximum training distance for the total group (r = -0.13; p = 0.48), the experimental group (r = 0.05; p = 0.96) or for the control group (r = -0.35; p = 0.20). There also were no significant relationships between mid delta performance and the maximum training distance for the total group (r = -0.26; p = 0.15), the experimental group (r = -0.16; p = 0.56) or for the control group (r = -0.26; p = 0.35). In addition, there were no significant relationships between late delta performance and the maximum training distance for the total group (r = - 0.20; p = 0.29), the experimental group (r = - 0.12; p = 0.67) or for the control group (r = - 0.03; p = 0.92). A summary of the relationships between delta performance and the maximum training distance is provided in Table 4a. A negative correlation indicates that as the maximum training distance increases, delta performance decreases. A positive correlation indicates that as the maximum training distance increases, delta performance increases.

Table 4a: Relationships between Δ performance early, Δ performance mid, and Δ performance late and maximum training distance for the total group, experimental group and control group for post-race.

Correlation	Total		Experimental		Control	
	r	p	r	p	r	p
Early Δ performance	-0.13	0.48	0.05	0.96	-0.35	0.20
Mid Δ performance	-0.26	0.15	-0.16	0.56	-0.26	0.35
Late Δ performance	-0.20	0.29	-0.23	0.67	0.03	0.92

3.6.4 Delta performance and maximum training frequency

There were no significant relationships between early delta performance and the maximum training frequency for the total group (r = - 0.11; p = 0.54), the experimental group (r = 0.18; p = 0.50) or for the control group (r = - 0.47; p = 0.08). There also were no significant relationships between mid delta performance and the maximum training frequency for the total group (r = - 0.07; p = 0.70), the experimental group (r = 0.16; p = 0.55) or for the control group (r = - 0.39; p = 0.15). In addition, there were no significant relationships between late

delta performance and the maximum training frequency for the total group (r = 0.002; p = 0.99), the experimental group (r = 0.19; p = 0.47) or for the control group (r = - 0.28; p = 0.32). A summary of the relationships between delta performance and the maximum training frequency is provided in Table 4b. A negative correlation indicates that as the maximum training frequency increases, delta performance decreases. A positive correlation indicates that as the maximum training frequency increases, delta performance increases.

Table 4b: Relationships between Δ performance early, Δ performance mid, and Δ performance late and maximum training frequency for the total group, experimental group and control group for post-race.

Correlation	Total		Experimental		Control	
	r	p	r	p	r	p
Early Δ performance	-0.11	0.54	0.18	0.50	-0.47	0.08
Mid Δ performance	-0.07	0.70	0.16	0.55	-0.39	0.15
Late Δ performance	+0.002	0.99	0.19	0.47	-0.28	0.32