

# THE EFFECTS OF ENDURANCE TRAINING ON NEUROMUSCULAR CHARACTERISTICS IN MASTERS RUNNERS



**Karen Sharwood**

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**THE EFFECTS OF ENDURANCE TRAINING ON NEUROMUSCULAR  
CHARACTERISTICS IN MASTERS RUNNERS**

By

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**SOUTH AFRICA**

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## **DECLARATION**

I, Karen Sharwood, do hereby declare that the experiments presented in this thesis were conceived and executed by myself except where otherwise indicated.

Neither the substance nor any part of this thesis has been submitted in the past, or is being, or is to be submitted for a degree in the University of Cape Town or any other university.

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**Karen Ann Sharwood**

**August 2003**

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**Sharwood K**, Lambert M, St Clair Gibson A and Noakes T. Changes in Oxygen Consumption During and After a Downhill Run in Masters Long Distance Runners. *Clinical Journal of Sports Medicine*. 12(5): 308-312, 2002 (**CHAPTER 5, appendix 12.12.**).

**Sharwood K**, Paavolainen L, Nummela A, Lambert M, St Clair Gibson A, Rusko H, Noakes T. Reduced EMG activity and fatigue during a 5 km time trial. *European Journal of Applied Physiology*. In review (**CHAPTER 7**).

### III. CONFERENCE PRESENTATIONS ASSOCIATED WITH THE THESIS

**The 8<sup>th</sup> Biennial South African Sports Medicine Association conference, Johannesburg, South Africa – free communication (September, 1999):**

Changes in Muscle Power and Neuromuscular Efficiency After a 40 minute Downhill Run in Veteran Long Distance Runners.

**The 27<sup>th</sup> Annual Physiology Society of South Africa conference,**

**Stellenbosch, South Africa, free communications (September, 1999):**

Changes in Muscle Power and Neuromuscular Efficiency After a 40 minute Downhill Run in Veteran Long Distance Runners.

**The 5<sup>th</sup> Annual Conference of the European College of Sport Science,**

**Jyväskylä, Finland, poster presentation (July, 2000):** Changes in Muscle Power and Neuromuscular Efficiency After a 40 minute Downhill Run in Veteran Long Distance Runners.

**The 10<sup>th</sup> Biennial South African Sports Medicine Association conference,**

**Johannesburg, South Africa, symposium (February, 2003):** Neuromuscular Characteristics During Running.

**The 50<sup>th</sup> Annual Meeting of the American College of Sports Medicine, San Francisco, USA, free communications (May, 2003):** Reduced EMG Activity and Fatigue During a 5 km Time Trial.

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## **VI. ABSTRACT**

### **Background**

Despite the well documented positive effects of regular participation in endurance exercise, there are a number of case reports and anecdotal observations of masters runners, who have accumulated high volumes of training and racing mileage, who experience a precipitous decline in running performance. Furthermore, there is substantial clinical data available to support theories of muscle damage and morphological alterations as a direct consequence of chronic endurance training. Therefore, the principle aim of this thesis was to determine whether there is a relationship between exposure to high mileage racing and training and alterations to neuromuscular characteristics, which together, contribute to an accelerated decline in running performance in masters athletes.

### **Study one**

The aim of this study was to establish whether there was a relationship between the total accumulated volume of training and racing in masters runners and the neuromuscular efficiency of the quadriceps muscles, before and after a downhill run. Twenty male masters long distance runners (45 - 50 years) with a range of training (1 300 km to 111 280 km) and racing (0 km to 9 737 km) experience were recruited to participate in this study. The subjects performed a 40 minute downhill run (- 10 % decline) on a treadmill, at a speed corresponding to 70 % of the subject's peak treadmill

running speed (PTRS). Two isometric maximal voluntary contraction (MVC) tests, lasting 5 seconds and 25 seconds, and a drop jump test were performed before and immediately after the downhill run.

Electromyographic (EMG) activity from the vastus medialis (VM) muscle was recorded continuously during both of the isometric tests and the drop jump.

The difference between the EMG/mean force relationship over the 5 second MVC, before and after the downhill run, was calculated as the delta ( $\Delta$ ) neuromuscular efficiency. This was related to the total kilometres trained, current training distance, total kilometres raced and number of races > 56 km. The difference in drop jump height before and after the downhill run was measured as well as changes in heart rate throughout the run. There was a significant curvilinear relationship between the  $\Delta$  neuromuscular efficiency and total kilometres raced ( $R^2 = 0.53$ ,  $P < 0.05$ ), and a significant inverse relationship between  $\Delta$  neuromuscular efficiency and the number of races > 56 km ( $r = -0.50$ ,  $P < 0.05$ ). Drop jump height decreased after the downhill run, and heart rate increased during the run. The conclusion from this study was that runners who have raced an accumulated distance of > 5 000 km show a significant dissociation in the  $\Delta$  neuromuscular efficiency after a downhill run, compared to less experienced runners.

## **Study two**

The aim of this study was to determine whether oxygen consumption during submaximal running increases in proportion to years of accumulated

training and racing in masters runners after a bout of downhill running. Seventeen male masters distance runners (45 - 55 years) with a range of training (3 536 km to 79 320 km) and racing (205 km to 12 218 km) experience were recruited to participate in this study. Subjects were asked to perform a 40 minute continuous treadmill run, at 70 % of PTRS, consisting of two horizontal runs of 10 minutes each, separated by a 20 minute downhill (- 10 %) run. Heart rate and oxygen consumption were measured continuously during the run. Data were analysed to identify correlations between the end of the first horizontal section (minute 10) and the first minute of the second horizontal run (minute 31). Delta values were related to current training mileage, total accumulated racing distance and total accumulated training distance. The results showed that there were significant changes in both heart rate ( $P < 0.001$ ) and oxygen consumption ( $P < 0.001$ ) over time during the 40 minute run but that there were no significant relationships between the change in oxygen consumption (delta) between minute 10 and minute 31 and total accumulated training mileage, total accumulated racing mileage and current training mileage. The results of this study suggest that either submaximal oxygen consumption is not a sensitive marker of changes in neuromuscular activity or that the downhill protocol did not impose a sufficient "eccentric" stress for the subjects.

### Study three

The aim of this study was firstly, to identify a measurement technique and protocol that would accurately measure preactivation during running, and secondly, to determine whether the measurement of preactivation could be used as a sensitive measure of neuromuscular changes, associated with fatigue and muscle stiffness regulation, during dynamic exercise. Eighteen subjects performed a 5 second MVC and a 20 m sprint before a 5 km time trial (5K). The sprint was repeated during the last lap of the 5K and the MVC immediately after the 5K. EMG activity from the VM, vastus lateralis (VL), rectus femoris (RF), biceps femoris (BF) and gastrocnemius (GA) was recorded continuously during all tests. The results of this study showed that EMG in the 20 m sprint decreased for preactivation ( $P < 0.0001$ ) and ground contact ( $P < 0.0001$ ) during the 5K. There was an increase in sprint time ( $2.63 \pm 0.14$  s to  $3.16 \pm 0.18$  s) and contact time ( $138.0 \pm 16.5$  ms to  $172.0 \pm 12.2$  ms) ( $P < 0.00001$ ) after the 5K. The change in 20 m time was correlated to changes in contact time ( $r = 0.77$ ,  $P < 0.0001$ ), preactivation ( $r = 0.59$ ,  $P < 0.05$ ) and stride length ( $r = 0.64$ ,  $P < 0.01$ ). Average force decreased immediately after the 5K ( $881.5 \pm 242.6$  N to  $745.1 \pm 215.5$  N) ( $P < 0.01$ ), as did EMG from VL ( $P < 0.05$ ), RF ( $P < 0.01$ ) and BF ( $P < 0.01$ ). There was a linear relationship between the change in average force and average EMG during the MVC ( $r = 0.90$ ,  $P < 0.0001$ ). Based on these results, it was concluded that the use of muscle preactivation to determine the effects of fatigue on the neuromuscular system during a 5 km time trial was a valid measure and that changes in muscle preactivation may illustrate sensitive

alterations in muscle recruitment patterns during functional activities such as running.

#### **Study four**

The aim of this study was to determine whether there was a relationship between the accumulated volume of training and racing and alterations to muscle preactivation and EMG recruitment patterns during a 5 km time trial in masters runners. Eighteen male masters (45 - 65 years) endurance runners, were recruited to participate in this study. Subjects were separated into two groups based on their exposure to high mileage training. Experimental subjects had been running for > 20 years, and control subjects had been running for < 15 years. Subjects performed a maximal 20 m sprint before, and during the last lap of a 5K. EMG activity was recorded continuously from the VL, VM and GA, muscles during the 5K. According to the study design, there were significant differences between groups in the volume of accumulated training and racing. There were significant increases in sprint time ( $3.32 \pm 0.30$  s to  $3.70 \pm 0.38$ ,  $P < 0.0001$ ) and contact time ( $160 \pm 2$  ms to  $180 \pm 2$  ms,  $P < 0.0001$ ) and significant ( $P < 0.0001$ ) decreases in stride length ( $0.07 \pm 0.11$  m) and stride frequency ( $0.20 \pm 0.15$  strides. $s^{-1}$ ) for the total group following the 5K, whilst there were no group or interaction effects. The change in sprint time was correlated to the decrease in stride frequency ( $r=0.77$ ,  $P < 0.001$ ) and stride length ( $r=0.68$ ,  $P < 0.05$ ). Preactivation for the total subject group decreased significantly ( $P < 0.001$ ) in all muscle groups. There was a significant group x time interaction

( $P < 0.01$ ) for VL preactivation, suggesting that the preactivation decreased more (30 %) in the experimental group compared to the control group (26 %). The change in combined lower limb muscle preactivation was correlated to 5K running velocity ( $r = -0.68$ ,  $P < 0.01$ ), the change in sprint time ( $r = -0.71$ ,  $P < 0.01$ ), current training ( $r = -0.74$ ,  $P < 0.001$ ) and total accumulated training mileage ( $r = -0.55$ ,  $P < 0.05$ ). These data suggest that athletes exposed to high volumes of racing and training appear to have an altered muscle activation strategy initiated by the central nervous system, which may act as a protective mechanism against muscle damage and injury.

## **Conclusions**

Based on the findings in this thesis, a model to characterise runners who are able to remain competitive for more than 20 years has been proposed. This model suggests that runners who experience an alteration in EMG recruitment patterns, either in anticipation of a chronic training stimulus or as an adaptation to it, may represent a group of "resistant" athletes. This alteration in muscle recruitment acts as a protective mechanism against chronic muscle injury, although may also contribute to a decline in performance over many years.

## CHAPTER 1

# INTRODUCTION

University of Cape Town

There is substantial evidence to show that aging is associated with significant reductions in skeletal muscle mass and strength (193-195;293; 352;358;426;478;537), alterations to metabolic (293;426), oxidative (293;426) and contractile function (426) as well as changes to the neuromuscular system (148;325;537). Together, these changes reduce exercise capacity and neuromuscular efficiency and result in a gradual decline in athletic performance.

However, these alterations in skeletal muscle morphology, innervation and function associated with increasing age can be attenuated by the addition of a regular endurance training stimulus (96;182;298;378;426;478;479;504; 521;582). Indeed, much of the decline in skeletal muscle function associated with age appears to be related more to a progressive reduction in the demands placed onto, rather than absolute changes within the muscle itself (293).

Endurance exercise improves endurance capacity and performance by altering the enzymatic, biochemical and morphological characteristics of skeletal muscle (125;212;251;317;374;571). Furthermore, regular endurance exercise is associated with increased longevity (450) and has been used as a preventative treatment modality for cardiac and other chronic diseases of lifestyle (104).

Notwithstanding the positive effects of regular endurance exercise, examination of differences in age-adjusted world-record results indicate that endurance capacity decreases gradually from the third through sixth decades, following which the drop-off in endurance exercise capacity rapidly accelerates (200).

However, despite this natural decline in running performance, there are a number of anecdotal reports (426) and case studies (328;513) of well-trained athletes with a number of years of running training and racing experience that undergo a precipitous decline in running performance, which occurs at a faster rate than expected for their age (426). In support of these observations are clinical data on long distance runners which show mitochondrial abnormalities (143;513), muscle fiber damage (513), irregularly shaped muscle fibers and peripheral nuclei (558) to suggest that there may be chronic alterations in the ultra structural muscle fibers, or muscle recruitment patterns, as a result of chronic endurance training that may contribute to the decline in running performance.

Taken collectively, these observations of changes in running performance in aging athletes have raised the following question: is there a relationship between exposure to high mileage racing and training and alterations to neuromuscular characteristics, which together, contribute to an accelerated decline in endurance running performance? The aim of this

this is, therefore, to characterise this relationship and to propose possible mechanisms that may contribute to a decline in running performance.

To introduce this thesis, case reports of endurance athletes who have experienced a sudden decline in performance that occurs at a faster rate than expected for their age are discussed, together with clinical data collected from endurance athletes before, during and after endurance running events. In addition, physiological alterations associated with aging, regular physical activity and muscle damage are also discussed in detail. Thereafter, the specific questions addressed in this thesis are identified, followed by the experimental section.

University of Cape Town

## CHAPTER 2

# THE EFFECTS OF AGE, TRAINING AND DAMAGE ON SKELETAL MUSCLE

University of Cape Town

## **2.1. REVIEW OF THE LITERATURE**

Although participation in ultra endurance running events has risen exponentially since the late 1970's (426), there are few examples of athletes who are able to maintain constant age-group record performances for many decades. This suggests that high volumes of training and racing sustained over several years might have negative consequences on physical performance in endurance runners. Indeed, athletes who compete in these events undergo hours of rigorous training and racing in preparation for their event and it has been suggested that this continual training stimulus may account for an acceleration in the aging process (330).

### **2.1.1. CHANGES IN PERFORMANCE IN ENDURANCE ATHLETES ASSOCIATED WITH AN INCREASE IN AGE**

Widrick et al (571) have shown that weekly training distance, running pace and training frequency declines over time (15 years) in elite competitive masters runners (see Table 2.1). Similar reductions in training volumes have been reported by Trappe et al (545) and Pollock et al (463) and more recently by Lambert et al (328) and Lambert and Keytel (329).

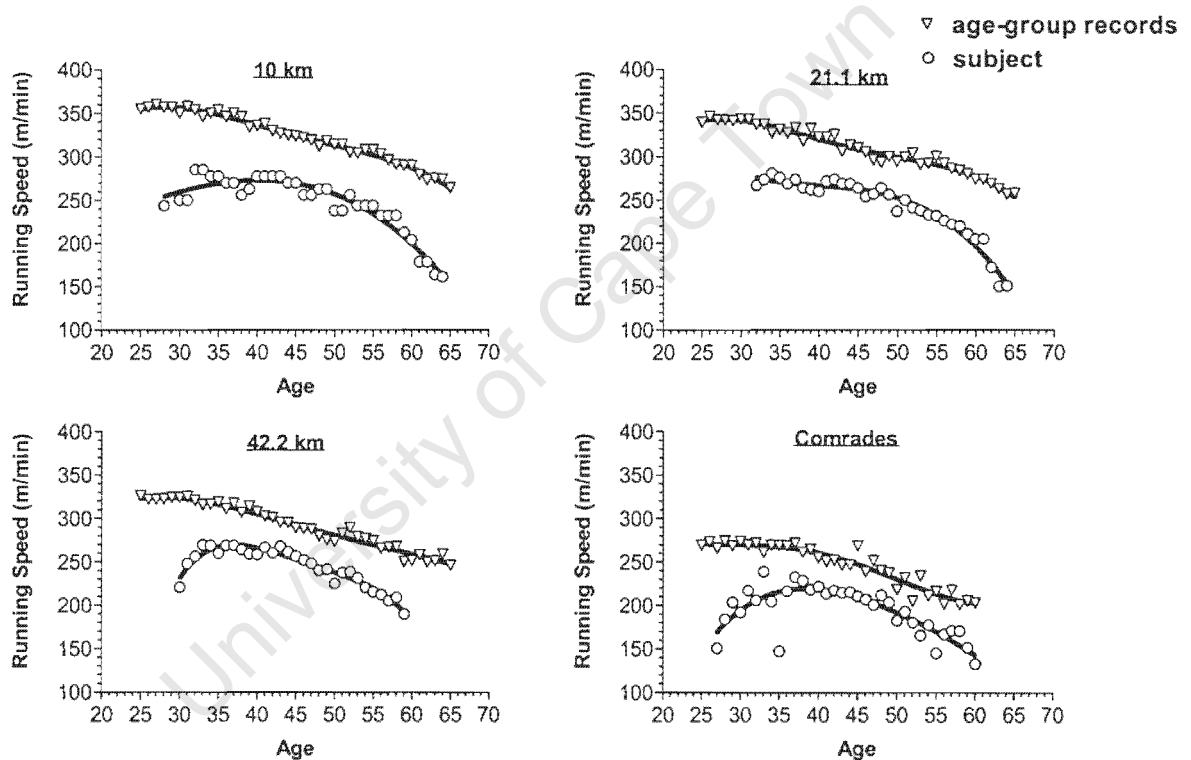
In addition, Lambert and Keytel (329) have shown that the age-group winners in a 56 km footrace had all been running for approximately 15 years, even those who set these records over the age of 60 years. This finding suggests that peak age-group racing performance can be sustained for approximately 15 years after which athletes begin to experience a decline in their performance.

**TABLE 2.1:** Changes in endurance training distance, intensity and frequency in elite competitive masters runners (571)

	<b>DISTANCE</b> (km.wk <sup>-1</sup> )	<b>INTENSITY</b> (km.hr <sup>-1</sup> )	<b>FREQUENCY</b> (sessions.wk <sup>-1</sup> )
<b>1966 – 1976</b> (~20-30 yrs)	164 (81 - 225)	14.9 (13.8 – 16.1)	7 (6 - 7)
<b>1977 – 1987</b> (~ 30-40 yrs)	108 (48 - 161)	14.2 (12.1 – 14.9)	6 (5 - 7)
<b>1988 – 1992</b> (~ 40 – 50 yrs)	77 (40 – 121)	13.6 (11.4 – 14.9)	6 (5 - 7)

Noakes (426) has discussed a number of reports of sudden changes in performance in well-trained masters athletes. Fifty year old Bill Rodgers, a successful competitive runner for nearly 30 years, retired from the Boston Marathon after 30 km. Noakes (426) inferred that cumulative damage from intensive training and frequent racing in his earlier years had become permanent and was directly affecting his ability to compete.

A case-report (328) describes a runner who had competed more than 122 standard marathons (42 km), 34 Comrades marathons (90 km) and 56 ultra marathons ranging in distance from 50 to 58 km, accumulating a training distance of 153 944 km and a racing distance of 16 604 km over 37 years of running. An analysis of his racing times for distances from 10 – 90 km (figure 2.1) showed that running performance started to decrease at a faster rate than was expected for his increasing age, after he had been competing for approximately 15 years (328).



**Figure 2.1:** The average running speed (m/min) for the runner for 10-, 21.1-, and 42.2-km races and the Comrades marathon ( $\pm 90$  km) from the age of 27 to 64 years. These are compared with the U.S. national age-group records for the 10-, 21.1-, and 42.2-km events and the Comrades marathon age-group records. Reprinted, by permission, from M.I. Lambert et al., 2002, "Accelerated decline in running performance in a master runner with a history of a large volume of training and racing," *Journal of Aging in Physical Activity*, 10 (3): 317.

This subject reported that after 15 years of racing and training, athletes who were of a similar age, but who had less training experience, were able to easily beat him during races. Interestingly, this athlete also noted that after the age of 50 years, his body was no longer able to absorb high mileage training and that his muscles were increasingly stiff. This anecdotal observation suggests an alteration in the shock absorbing qualities of his muscles and tendons.

In another case study, the running performance of an endurance athlete who completed 3 529 km over seven weeks began to continuously decline after this event (502). These data cannot be interpreted in more detail, as the duration of the period over which this decline in performance took place was not reported.

Other observations have suggested that those athletes who begin training and racing later on in their lives are able to perform at a higher standard compared to athletes of the same age who have been competitive for many years. Noakes (426) provides the examples of New Zealanders John Campbell and Jack Foster, and South African Titus Mamabola, who have achieved world class age group records for 40 and 50 year olds. These athletes did little running in their twenties and early thirties and only began to increase their training and racing mileage after the age of 40.

## **2.1.2. STUDIES INVESTIGATING MUSCLE DAMAGE AND ENDURANCE RUNNING**

In addition to anecdotal reports and case studies, there are also several studies that provide clinical evidence for chronic muscle damage associated with endurance exercise (105;191;248;317;502;513;558), which are discussed in detail below.

### **2.1.2.1. Warhol et al (558)**

Warhol et al (558) studied skeletal muscle injury and repair in 40 male distance runners after a standard marathon. These authors obtained gastrocnemius muscle biopsy samples from two or more runners at each time interval after the race as follows: same day, and 1, 2, 3, 5, 7, 10, 14, 21, 28, 42, 56, 70 and 84 days after the race.

The subjects in this study showed evidence of cell injury (interstitial collagen deposition and thickened capillary basal lamina) before the race, possibly as a result of the training involved in the preparation for the race.

Within two days of the race, the muscle samples from all subjects showed depletion of both glycogen and lipid stores. There was profound myofibrillar lysis, endothelial cell damage and disappearance of the sarcoplasmic reticulum. Mitochondrial damage was evident, with dissolution of cristae

and loss of the mitochondrial matrix. Biopsy samples obtained seven days after the race showed satellite cells and interstitial cells resembling fibroblasts present in the muscle tissue, evidence of acute injury resolution. Samples obtained four weeks after the marathon illustrated myofibrillar damage resolution with abundant mitochondria and central cell nuclei, indicative of regeneration. By weeks eight and 10 after the race, there was further indication of regeneration and repair, including prominent satellite cells, suggested to be the precursor of new skeletal muscle cells, although abnormalities of mitochondrial size and shape persisted. This study also showed evidence of prior cell injury and loss by variable amounts of interstitial collagen deposition and thickened capillary basal lamina.

Ten to 12 weeks after the race, the samples had central nuclei and an increased content of endoplasmic reticulum in the muscle, previously shown to be morphologic correlates of the regenerative process. The observed damage in this study was focal and confined to individual sarcomeric units, suggestive of repetitive cell injury, but not of necrosis.

#### **2.1.2.2. Hikida et al (248)**

Hikida et al (248) obtained muscle biopsy samples from 10 male runners on the morning of a 42 km marathon, within 15 minutes of completing the marathon and again at days 1, 3, 5 and 7 following the marathon.

Perhaps the most important finding from this study was that in 80 % of the pre-marathon biopsy samples, there were signs of repetitive trauma, which included erythrocytes and mitochondria in the extracellular space, and disruptions in the sarcolemma. Similarly, there was evidence for leukocytic and phagocytic activity, whilst a number of these samples also showed "contractile knots" and "disorientated myofibrils".

The samples collected after the marathon had similar characteristics to those of Warhol et al (558). In addition, erythrocytes and mitochondria were observed in the muscle extracellular spaces, as well as crystalline inclusions within the mitochondria, Z-line streaming and empty basal lamina tubes.

These structural abnormalities gradually increased during the week following the marathon, peaking between days one and three after the race. There was evidence of muscle necrosis with morphological changes, including sarcolemmal disruption, which created large gaps within the muscle fibers. This study also showed mitochondria being actively engulfed by phagocytes in the basal lamina in the samples collected after the marathon.

The runners in this study presented with degenerating peripheral myofibrils, reflected by a loss of Z-lines, abnormally aligned sarcomeres and the presence of bodies containing only microfilaments in the muscle samples. These runners also had atrophic fibers, distributed between the normal

fibers, as well as satellite cells, which became more prevalent in the later biopsy samples.

The authors concluded that because morphological abnormalities persisted for at least seven days after a marathon and because many of these same observations were seen in the pre-race sample, both the intensive training for the marathon and the marathon itself must induce inflammation and fiber necrosis. It was also suggested that the inflammatory reaction that accompanies these activities might be a major contributing factor to the sensation of delayed onset muscle soreness.

These findings have been confirmed more recently by Goodman et al (214) who showed evidence of pleomorphic mitochondria with increased density, subsarcolemmal accumulation of mitochondria and lipofuscin granules in muscle samples from runners before a 21 km race. These data provide further evidence of muscle fiber damage associated with endurance running training and the suggestion of permanent skeletal muscle and connective tissue damage associated with chronic endurance racing and training.

### **2.1.2.3. Kuipers et al (317)**

Kuipers et al (317) monitored the histological and ultra structural features of the vastus lateralis muscle of untrained subjects over an 18 - 20 month

training period with a 15 km, 25 km and 42 km race equally dispersed over this time. Muscle biopsies from the vastus lateralis were obtained five days before each race and again 0.5 - 6 hours and 8 - 9 days after the race.

Ultra structural examination revealed irregular Z-lines and "hypercontraction" immediately after the start of the training period. These alterations persisted throughout the training period, increasing in frequency with the increase in the length of the training runs. This study therefore concluded that the extent of changes in morphological characteristics of skeletal muscle was related to the total training distance, rather than to the intensity of exercise. This study also emphasised the combined effects of racing and training on muscle and the possible accumulated effects of muscle damage.

#### **2.1.2.4. Chambers et al (105)**

Chambers et al (105) studied athletes participating in the 90 km Comrades Marathon. They used a three jump testing protocol (squat jumps, a measure of muscle power which excludes the stretch shortening cycle, counter-movement jumps and drop jumps, measures of muscle power that both include the stretch shortening cycle) to assess the effect of an ultra marathon on muscle power and the stretch shortening cycle. These measurements were an indirect assessment of the ability to use stored elastic energy in the quadriceps muscle. Athletes were asked to perform

the three jump tests immediately after the race, daily for five days following the race and then weekly for a further four weeks following the race.

Immediately after the race, all three jump heights were significantly reduced, with the squat jump height remaining significantly lower for 18 days, the counter-movement jump height lower for 11 days and the drop jump height lower for three days. By the end of the testing period, four weeks after the race, jump height for all three tests had returned to pre-race values (105).

The results of this study also showed that pre-race counter-movement jump height was only one centimetre higher than squat jump height before the race, suggesting a low stretch shortening cycle activity and by implication a low ability to use the stored elastic energy in the quadriceps muscles. This finding suggests that the elastic component of the quadriceps muscles may have somehow been reduced before the race, possibly as a result of the high mileage training performed by these athletes during preparation for the race, a suggestion confirmed by Lepers et al (339). This also supports the findings of pre-race morphological changes in skeletal muscle associated with training in ultra endurance athletes (248).

It was speculated that the reductions in quadriceps muscle power as shown by Chambers et al (105) after an ultra endurance event, might also be associated with a decrement in endurance running performance.

### 2.1.2.5. Sjöström et al (502)

Sjöström et al (502) obtained muscle biopsy samples from the gastrocnemius muscle of a 46 yr old endurance athlete before and after an ultra endurance running event (3 529 km over seven weeks – an average of 70 km per day).

In contrast to the results in the study of Hikida et al (248), pre-race muscle samples did not show significant evidence of any pathological changes. However, immediately after the run, muscle fibers were varied in size and shape and were tightly packed in well-defined fascicles. The perifascicular connective tissue was increased and infiltrated by inflammatory cells, and there was an increased amount of connective tissue in the fascicular region. The authors also observed an increase in the amount of central nuclei, evidence of necrotic fibers and some fibers undergoing regeneration. Many fibers from this sample also appeared to be uneven and had a “moth eaten” appearance, suggesting possible architectural disturbances. These findings have subsequently been confirmed by Matin et al (373).

Sjöström et al (502) concluded that the focal and generally diffusely spread increase in connective tissue, together with the fiber size variation and degenerated and regenerating muscle fibers were indicative of a reactive

healing process, suggesting that fiber damage and disintegration had been in progress for a long period of time (502).

#### **2.1.2.6. Fridén et al (191)**

Fridén et al (191) have described morphological changes in skeletal muscle of subjects with severe delayed onset muscle soreness following a series of muscle lengthening exercises. A biopsy sample was obtained from the soleus muscle of each subject two weeks before the exercise which caused muscle damage, and again on days two and seven following the exercise. By the second day after the damaging exercise, there was marked broadening, streaming and at some places, total disruption at the myofibrillar Z-band. Based on these observations, the authors concluded that the contractile machinery of overloaded muscle fibers is distorted for several days following exercise that causes muscle damage.

#### **2.1.2.7. St Clair Gibson et al (513)**

St Clair Gibson et al (513) describe a case report of a 28 year old male who complained of a progressive decline in running performance, associated with an increasing inability to tolerate high-mileage training. The skeletal muscle symptoms that he described were that his legs became progressively weaker when his training distance increased to above 100 km/week. A muscle biopsy was performed on the patient's left vastus

lateralis muscle. Four months later, a second muscle biopsy was performed on the same vastus lateralis muscle and also on the left triceps muscle.

Histologic analysis of the first vastus lateralis biopsy sample revealed no inflammation, necrosis, or regeneration of muscle fibers. The muscle interstitium, capillary vessels, lipid and glycogen content all appeared normal. However, histochemical analysis of the same muscle biopsy sample showed that there was uneven mitochondrial distribution with subsarcolemmal mitochondrial aggregation and that there were several muscle fibers that had a ragged, red appearance. The NADH and SDH stains showed similar subsarcolemmal mitochondrial accentuation. These results were consistent in the second vastus lateralis muscle biopsy, yet no such abnormalities were present in the biopsy of the triceps muscle.

Electron microscopic analysis showed that the mitochondria, in both the first and second vastus lateralis muscle biopsy samples, displayed variation in size and contained a dense matrix with an increased number of coarse and broad cristae. The abnormal mitochondria were observed in large subsarcolemmal aggregates as well as along the sarcomere.

The authors suggest three explanations for these findings: (i) mitochondrial myopathy, (ii) unknown infective or toxic agents (iii) an excessive exercise routine that occurred for much of the subject's adolescent and early adult life. The authors concluded that exercise-induced damage is a likely

explanation as the mitochondrial abnormalities were limited to the lower limb muscles and were present in both muscle biopsy samples taken four months apart. This finding indicates consistent damage associated with a long-term training effect and that the mitochondrial accumulation in the subsarcolemmal space is an exaggerated example of the normal response to endurance training. Furthermore, these authors speculated that these mitochondrial abnormalities might explain the reported decrements in this athlete's running performance.

#### **2.1.2.8. Summary of the studies on muscle damage and endurance running**

Collectively, these studies show that regular endurance running results in ultra structural muscle fiber damage that may occur as a result of both training for the event and from the event itself. These studies do, however, acknowledge that there is large inter-individual variation in muscle damage between subjects. This raises the possibility that there may be a variable response in the vulnerability of a runner's muscle to incur this muscle damage. It follows that there may be runners who incur damage and ultra structural changes and other runners who are somehow able to resist the accumulative effects of high mileage racing and training and who are thus more likely to be able to maintain age-group record times.

Despite the individual differences in vulnerability to incur damage, there are a number of well-documented alterations in skeletal muscle that are associated with both age and training. Specifically, aging and training have been associated with significant reductions in muscle mass and strength (466) and altered metabolic, oxidative and contractile function (123;200), which together, influence running performance. These aspects will be discussed next as a background to the experimental phase of the thesis.

### **2.1.3. THE EFFECT OF AGE ON SKELETAL MUSCLE**

#### **2.1.3.1. Aging and muscle strength**

Muscle strength reaches peak values between the ages of 25 and 35, is maintained between the ages of 35 and 40 and begins to show a gradual decline thereafter (54;352;466). After the age of 60, the loss of force generating ability of muscle is accelerated (54;148;334;352;480) and declines at approximately 1 – 2 % per year (505). These reductions in strength have been reported to occur during shortening (concentric), isometric and lengthening (eccentric) phases of movement.

### 2.1.3.1.1. Shortening (concentric) muscle force

Frontera et al (193) identified significant reductions in strength ranging from 24 % to 30 %, at both slow and fast angular velocities, in knee extensors and flexors in male subjects (mean starting age of 65 years) over a 12-year period. Similar findings have been reported by Aniansson et al (9;10) who showed isokinetic strength losses of up to 35 % in subjects between 73 and 83 years. Frontera et al (193) concluded that the decline in isokinetic strength ranged from 1.4 % to 5.4 % per year, depending on the muscle group and the speed of contraction of the muscle. These reductions infer an annual loss in concentric muscle strength of approximately  $3.2 \text{ Nm}^{-1} \cdot \text{yr}^{-1}$  due to the aging process (9;10;193;505).

### 2.1.3.1.2. Isometric muscle force

Similarly, other studies have shown that isometric force generated during a maximal voluntary contraction (MVC) in older men (mean age  $80 \pm 5$  years) is approximately 50 % of that of younger (mean age  $26 \pm 4$  years) subjects (480;550). These findings are supported by Connelly et al (123) who showed mean ankle dorsiflexor MVC torque to be 26 % lower in older subjects (80 - 85 years) compared to younger subjects (20 - 22 years) who were matched for height, mass and physical activity profiles. Greig et al (221), however, could not show any significant differences in isometric strength between subjects (79 - 89 years) tested over an eight year period. This discrepancy

suggests that the significant declines in maximal force production may occur gradually over a longer period of time, for example 40 years (480;550) compared to eight years (221).

#### **2.1.3.1.3. Lengthening (eccentric) muscle force**

There are equivocal data available reporting the effects of increasing age on lengthening muscle force. Lindle et al (352) showed a 31 % difference between old (mean age 70 years) and young men (mean age 30 years) in lengthening force of the knee extensor muscles, whereas there are a number of studies suggesting that lengthening muscle force in humans may be less affected by age than concentric strength (165;258;465;467).

Highgenboten et al (247) have suggested that a possible factor explaining the preservation of lengthening muscle force with age may be age-associated changes in mechanical and elastic properties of connective tissue. Age-related increases in connective tissue (342) and collagen cross linking (139) may enhance the elastic potential of skeletal muscle and thus may allow an increase, or maintenance, of muscle lengthening force production, despite decreases in maximal force generating capacity of aging muscle.

### **2.1.3.2. Possible mechanisms of the reduction in muscle force associated with increasing age**

The exact mechanisms responsible for the decline in force output associated with an increase in age have yet to be conclusively identified. The main mechanisms proposed to account for the changes in muscle strength include: (i) changes in muscle mass and cross sectional area (54;89;195), (ii) changes specific force or whole muscle quality (peak force corrected for muscle cross sectional area) (89;195;297), (iii) changes in neuromuscular innervation (54;89;195), (iv) changes to skeletal muscle connective and viscoelastic properties (216;217;585) and/or (v) other factors such as changes to skeletal muscle contractile characteristics and muscle cell structure and function (194;292).

#### **2.1.3.2.1. Changes in muscle mass and cross sectional area**

The age related decline in force output has been associated with a decrease in the cross-sectional area (CSA) of the muscle similarly associated with increasing age (89;292;411;582). Young et al (583;584) have shown reductions ranging between 25 - 35 % in the cross sectional area of the knee extensors in older (70 - 79 years) compared to younger (21 - 28 years) subjects. Similar age-associated reductions in muscle cross sectional area have been shown in the quadriceps femoris (298), in the plantar flexors (474) and in the elbow flexor and extensor muscle groups (297).

Frontera et al (193) concluded that approximately 90 % of the variability observed in strength changes over a 12 year period could be explained by these changes in muscle cross sectional area. In this experiment, the total cross sectional muscle mass of the quadriceps femoris decreased by 16 % and knee flexors decreased by 15 %. These changes are significantly higher than those reported by Greig et al (221) who showed a 6 % decrease in the quadriceps cross sectional area after eight years in a group of relatively active subjects. The differences between these two studies could be explained by the activity profiles of the subjects that participated in each trial. Frontera et al (193) used healthy sedentary subjects in their study, whilst participants in the study of Greig et al (221) were physically active. This difference provides further support to the suggestion that regular physical activity slows the decline in skeletal muscle function that is associated with age.

Changes in muscle mass and cross sectional area may be directly linked to alterations in skeletal muscle morphology, specifically, alterations in muscle fiber type and distribution, fiber size and fiber number.

(a) *Fiber type and distribution*

Lieber (349) has provided a summary of the functional characteristics of the different skeletal muscle fiber types. Type I (slow twitch) muscle fibers have high oxidative enzyme activity, low glycolytic enzyme activity and are

associated with an extensive capillary density and high concentrations of myoglobin. This metabolic profile allows these fibers to be relatively resistant to fatigue. Type IIa (fast twitch) muscle fibers have intermediate levels of glycolytic and oxidative enzyme activity, whilst type IIb fibers fatigue rapidly and have high glycolytic enzyme activity. Type IIb fibers have fewer mitochondria and lower myoglobin concentrations, compared to slow twitch fibers (109;349).

The literature is equivocal on whether there are significant fiber-type distribution changes associated with an increase in age. Some studies have shown increases in the percentages of type I fibers (332;334;347;442;544), a preferential loss of type II fibers (334;335) or no change in fiber composition associated with increasing age (119;170;225;332;345;442).

Often the discrepancies between these studies can be explained by the use of different histochemical techniques and methodologies (needle biopsy technique vs. radiological techniques), study designs (cross-sectional vs. longitudinal) and subject groups (active vs. sedentary).

(b) *Fiber size*

There is also conflicting data regarding changes in fiber size associated with aging. Although acknowledging a large degree of inter-individual variation, Trappe et al (544) and Coggan et al (119) have shown reductions of  $9.081 \pm$

1 233 to  $6\,595 \pm 231 \mu\text{m}^2$  and  $6\,765 \pm 1\,083$  to  $6\,014 \pm 321 \mu\text{m}^2$  in type I and II fiber areas respectively in distance runners in a longitudinal study lasting 20 years (544) and in a cross sectional study comparing old (mean age 63 years) to young (mean age 26 years) runners. Conversely, Frontera et al (193) did not show any changes in mean fiber area in either type I or type II muscle fibers over a 12 year period.

Whilst reductions in the size of type II fibers associated with an increase in age have been confirmed by a number of studies (54;223;298;344;469), Booth et al (54) have proposed that there may be an equivalent loss of both type I and type II muscle fibers, but whereas there is no change in the size of the remaining type I fibers, type II fibers appear smaller compared with their peak adult size. Similarly, the collective findings of studies using needle biopsy techniques or whole muscle cross sectional investigations performed on human specimens post-mortem, have suggested that the cross sectional area of type II fibers are significantly reduced with age, whilst type I fibers seem to be less effected (10;224;335;347;469).

(c) *Fiber number*

A reduction in total muscle fiber number may also account for the reduction in force production (225). Indeed, Lexell et al (347) found a reduction of up to 50 % in the number of both type I and type II fibers in post-mortem samples from 80 year old subjects compared to 60 year old

subjects. More recently, these authors have concluded that, with specific reference to the vastus lateralis muscle, cross sectional area is largely determined by the total number of fibers and to a lesser extent by the size of type II fibers (340;343).

These studies show that there is large inter-individual variation in the changes in muscle fiber morphology that are associated with age and which may contribute to the reduction in muscle force output.

#### **2.1.3.2.2. Changes in specific force**

Specific force, or muscle quality, refers to strength per unit of muscle mass (358). There is some evidence to suggest that there may be a reduction in force per unit of cross-sectional area of muscle in older individuals compared to younger controls (77;279).

The possible mechanisms responsible for the reduced specific force associated with aging remain unclear. In rodent muscle, qualitative changes occurring within the muscle fiber may directly affect force generation during cross-bridge formation (74). This suggestion has been supported by Frontera et al (195) who found lower specific force in the vastus lateralis muscle of older men (mean age 74 years) compared to younger men (37 years), and confirmed that the intrinsic ability of muscle fibers to generate force is reduced with age.

However, another study compared muscle strength and specific force between untrained young (mean age 21 years) and middle aged (mean age 54 years) men (89). This study showed that although peak force was significantly higher in the younger subjects, when force was corrected for muscle cross-sectional area, the difference between the two groups was eliminated. Similar findings have been reported by other studies (123;195;291;480).

It would appear that the discrepancy between the data available in the literature, on changes in specific force, is indicative of differing age and physical activity profiles of the subjects recruited to participate in these studies. Accordingly, there is a distinct need for a longitudinal study to clarify these issues (89).

#### **2.1.3.2.3. Changes in neuromuscular activation patterns associated with aging**

Any alterations to the neural drive to skeletal muscle will directly influence its force-generating capacity (88;89;149;282). Aging in human skeletal muscle is associated with changes in the integrity of the neuromuscular system. These changes include a decrease in the number of motor units (537;550), an increase in the size of motor units (481;537), an increase in the amplitude of motor unit action potentials (325), reductions in motor neuron conduction velocities (123;168;325;480) and a decline in the number of

active alpha ( $\alpha$ ) motor neurons (148;542). Other reported age-related morphological changes to the neuromuscular system include alterations to the neuromuscular junction (440) and changes related to the excitability of the sarcolemma (137).

Accordingly, the age-associated motor unit remodelling theory has been proposed (75;148;347). This theory suggests that there are preferential losses of fast twitch muscle fibers and their parent motor neurons with increasing age. This change results in slowed contractile properties, higher innervation ratios of slow-twitch motor units and reductions in muscle fiber number and size (88;123;148;415;416) which all contribute to a reduction in maximal force output. Furthermore, there is also evidence to suggest that as a consequence of larger motor units being preferentially affected (282; 333;346), explosive muscle power is also significantly reduced (57;111; 236;237;242;268;291).

Connelly et al (123) used intramuscular measuring techniques to investigate the firing rates of over 900 motor units in the tibialis anterior muscle. This study showed that mean motor unit firing rates in healthy, active older men (80 - 85 years) were approximately 30 - 35 % lower at all torque levels compared with those from younger men (20 - 22 years). These authors proposed that increasing age does not necessarily alter the strategy employed by the central nervous system to produce muscle force, but

rather than the firing rates are readjusted, equally, to a lower level at all torque levels tested (123).

Therefore, the reduction in firing rate associated with an increase in age could be associated with weaker and slower contracting muscle fibers, providing indirect support for the concept of age-related neuromuscular remodelling. This model of neuromuscular adaptation also illustrates the marked adaptive capacity for the surviving motor units to compensate for motoneuronal loss (88;123;148;415;537;550).

Campbell et al (88) has supported to the concept of age-related neuromuscular remodelling by suggesting the possibility of a progressive loss of functioning motor units in older subjects, along with a slower twitch duration in the remaining units, which renders them less efficient (325). Similarly, Tomlinson and Irving (542) have shown that up until the age of 60, there does not seem to be any evidence of a reduction in motor neuron numbers. In contrast, after 60, there are significant reductions in the number of motor neurons. In addition, Edström and Larsson (160) showed a substantial decrease in the number of motor units in the soleus muscle of rats with an increase in age. In this experiment, young rats had  $49 \pm 10$  motor units, whereas older rats had  $29 \pm 10$  motor units. These changes resulted in a decrease in muscle force and an increased innervation ratio (motor unit: muscle fiber) in older rats (160).

There are, however, conflicting reports in the literature regarding alterations in voluntary activation patterns associated with increasing age. Bilodeau et al (50) showed that older subjects (mean age 71 years) are unable to activate their muscles to the same extent as younger (mean age 26 years) subjects at the end of a fatiguing task. This finding suggests that there is a decreased capacity to recruit additional motor units with the onset of fatigue in older subjects. In addition, this study showed no evidence of neuromuscular task failure in older subjects, contrary to studies showing impairments in neuromuscular propagation (198).

However, Cannon et al (89) did not show any significant differences in muscle activation patterns between young (mean age 21 years) and middle aged (mean age 54 years) subjects. These authors calculated that these age-related difference in peak force corresponded to a 34 % reduction in peak force between the ages of 20 and 50 years. They concluded that these alterations could be caused by changes in muscle mass, rather than changes in neural innervation or specific force (89). Similarly, it seems that at least during an isometric maximal voluntary contraction, the quadriceps muscle can be activated to the same degree in young and old men (480), supporting the suggestions that the age-associated muscle weakness may be more as a consequence of peripheral limitations such as reductions in quantity and quality of muscle (341;474), decreases in specific tension (72;77;460) and disuse atrophy, rather than changes in central activation and firing rate properties.

The findings of Jakobi and Rice (269), and others (89;123;325;480) support the suggestion that maximal activation patterns do not differ between old and young subjects. However, these authors (269) suggest that older subjects show more variation in maximal activation patterns, and are thus less consistent than younger subjects, which may often explain differences in results between subjects of varying ages.

Based on these studies, it is unclear as to whether there are significant alterations in neuromuscular activation patterns that are associated with an increase in age. The discrepancies between studies may be as a result of variability in study design, subjects recruited and muscle group tested, or due to absolute differences in muscle activation between subjects of differing age.

#### **2.1.3.2.4. Connective tissue and viscoelastic properties of skeletal muscle associated with aging**

Collagen provides a foundation for the maintenance of muscle-tendon integrity and is involved in the transmission of muscular forces (97;216;336). There are several factors related to collagen content that may contribute to the passive viscoelastic properties of skeletal muscle. These factors include the amount of collagen, its phenotypic distribution (377), the extent of collagen cross-linking, and the architectural organization of the collagen fibrils (312;377).

There is substantial evidence to suggest that the quality of connective tissue deteriorates with age (216;217;585). Maturation of collagen alters both the mechanical and the chemical properties of the protein, resulting in an increase in stability and tensile strength (175;585). These alterations in the biochemical properties of collagen have been positively correlated with muscle stiffness (335;585), such that there is an increase in stiffness with a concomitant increase in age (310). This finding has been supported by Akiya et al (4) who have shown that there is evidence of alterations in the properties of connective tissue with increasing age and after exercise training (216;551), such that there is a lower peak ground reaction force/body weight. This results in a decreased ability of skeletal muscle to absorb shock (4).

#### **2.1.3.2.5. Other factors that may contribute to a decline in force production associated with aging**

There are reports in the literature that have suggested that the aging process may directly affect the contractile characteristics of skeletal muscle and that the reductions in muscle strength are also associated with increases in non-contractile material such as fat and connective tissue (194). Kent-Braun et al (292) showed that the intramuscular non-contractile content of muscle increased up to threefold over an age span of approximately 40 years. This study also showed that the ratio of contractile to non-contractile tissue was associated with physical activity levels, such

that the more physical activity undertaken by the subjects, the less intramuscular fat accumulation, and thus proportionately lower change in non-contractile component (292).

Other factors that have been proposed to attribute to the decline in force generating capacity associated with an increase in age include alterations in sarcoplasmic reticulum function (141), inadequate muscular energy supplies (533), abnormalities in contractile and regulatory proteins (31), and alterations in the internal architecture of the cell, including alterations to excitation-contraction mechanisms (75:369).

### **2.1.3.3. Conclusions**

A survey of the literature has yielded conflicting results of the effects of age on skeletal muscle. This can be attributed to differences in study design and the subject age selection and activity profiles.

Many of the studies that have been conducted to assess the effects of age on skeletal muscle have largely been cross-sectional in nature, and thus have limitations in describing the changes occurring with increasing age over many years. Similarly, there are very few studies in the literature that have identified skeletal muscle changes in middle aged subjects (40-55 years).

Taken collectively, with the limitations described above, it can be concluded that an increase in age is associated with a reduction in muscle force and efficiency. Specifically, age-associated declines in muscle strength up to 50 years of age are primarily related to quantitative changes in muscle mass. As age increases beyond 50 years, there appears to be additional reductions to neuromuscular innervation patterns and deterioration in muscle quality (89).

There is evidence, however, to suggest that these age-associated alterations can be altered by the addition of a regular endurance training stimulus. Indeed, It is well documented that endurance exercise training alters the enzymatic, biochemical and morphological characteristics of skeletal muscle (125;212;317;374;571), which together contribute to increased muscle efficiency and enhance performance capabilities. These consequences of endurance training will be described in the next section.

#### **2.1.4. ADAPTATION OF SKELETAL MUSCLE TO ENDURANCE EXERCISE**

One of the most noticeable and significant effects of endurance training is the ability to perform submaximal exercise for prolonged periods. The adaptation of skeletal muscle to endurance exercise can be categorised into (i) metabolic and biochemical adaptations, (ii) muscle power, (iii) neuromuscular adaptations, (iv) adaptations of muscle fiber types, strength

and size, (v) cross sectional area of muscle and (vi) muscle force and velocity.

#### **2.1.4.1. Metabolic and biochemical adaptations**

Noakes (426) has summarized consequences of the biochemical adaptations associated with endurance training as (i) an increase in maximal oxygen consumption, (ii) an increase in the capacity to store muscle and liver glycogen (iii) an increase in the rate of fat usage with a concomitant reduction in glycogen utilization at all work rates and (iv) a shift in the lactate turn point coinciding with a higher running speed.

Many of these adaptations can be explained by changes in skeletal muscle oxidative capacity (260;461). There are significant increases in both the number and size of skeletal muscle mitochondria after endurance training (294). There is also a concurrent increase in mitochondrial enzyme content (461;547), particularly in those enzymes associated with fatty acid metabolism and the shuttle systems responsible for transporting hydrogen ions into the mitochondria for utilization in the respiratory chain (273;547).

This increase in endurance appears to be related to the changes in enzyme activities, per unit tissue, for the metabolic systems associated with terminal oxidation of both fats and carbohydrates (95;374).

Mitochondria from endurance trained muscle have an increased capacity to produce energy at higher free fatty acid concentrations. Therefore, at any rate of exercise intensity, trained muscles are less dependent on carbohydrate metabolism to produce the same amount of energy than untrained muscles (426). As a result, fat becomes a more readily available source of fuel for energy at higher exercise intensities and the associated hydrogen ion production, which is a consequence of carbohydrate metabolism, is reduced (273;290). Therefore, the disturbed muscle contractility, which occurs in the presence of increased hydrogen ion concentration, is minimised (271;273;374). Furthermore, a consequence of these enzymatic changes is a conservation of muscle glycogen stores (95;374).

#### **2.1.4.2. Muscle power**

Paavolainen et al (447) have shown that muscle power, or the ability of individual cross-bridges to generate force, is closely related to running performance. Furthermore, Noakes (426) has suggested that regular endurance training may contribute to these changes in muscle cross-bridge activity.

Trappe et al (546) showed that the contractile function of individual slow and fast twitch muscle fibers in older subjects (mean age 74 years) could be improved with specific exercise training. Specifically, the authors

showed increases in single cell diameter, peak tension and shortening velocity after 12 weeks of progressive resistance training, which together, contribute to an increase in muscle power in both type I and type II fibers. These changes in muscle cross-bridge activity will also have an effect on running economy (493), thereby enabling runners to become more efficient at the same relative running speed.

#### **2.1.4.3. Neuromuscular adaptations**

In addition, Noakes (426) has suggested that improvements in endurance performance as a consequence of endurance training must result from an increased capacity of the brain to recruit a larger muscle mass for longer (238;241). These training-induced neural adaptations have been reported to be the primary source of increases in force and power production within the first eight weeks of training (238;575).

Häkkinen et al (238) showed that after six months of explosive strength training in habitually active subjects, maximal isometric and dynamic leg extension strength increased by 36 % and 22 % respectively, accompanied by significant increases in vastus lateralis and vastus medialis muscle activation. The authors (238) concluded that the increase in muscle strength could be explained only in part by changes in muscle cross sectional area. Indeed, the increase in EMG activation from the quadriceps muscle would suggest that neural adaptation plays a fundamental role in

improving muscle power during regular exercise training. These findings have been confirmed by Paavolainen et al (444) who have shown that explosive strength training is successfully able to alter patterns of neuromuscular activity, resulting in an increase in muscle stiffness and thus improvements in running economy and efficiency.

#### **2.1.4.4. Adaptations of muscle fiber types, strength and size**

Although previous studies have shown that training alters the metabolic and oxidative capacity of skeletal muscle (212;426), no studies have shown consistent alterations in muscle histochemical characteristics (94;212;317; 477;525;535). The majority of studies seem to suggest that on average, elite distance runners possess significantly more slow twitch (type I) muscle fibers than either untrained subjects or middle distance runners (110;469;477).

Coggan et al (118) showed that after 10 months of endurance training, there were no changes in the percentage of type I fibers from the lateral gastrocnemius muscle in healthy (mean age 64 years) subjects. Type IIb fibers decreased by approximately 8 %, and type IIa fibers increased by 9 %. The conclusion from this study was that there had been a gradual conversion of type IIb fibers to type IIa as a consequence of endurance training, a finding supported by Andersen and Henriksson (8) and Rodriguez et al (477). Conversely, Howald et al (260) showed a 12 % increase in type I fibers and a 24 % decrease in type IIb fibers after six weeks of endurance

cycling, without any evidence of fiber type conversion, whilst Kuipers et al (317) and Gollnick et al (212) could find no shifts in the ratio between fiber types in athletes training for a marathon or after a five month endurance training programme.

Jansson and Kaijser (270) compared untrained arm (deltoid) and well-trained leg (gastrocnemius and vastus lateralis) muscles of elite orienteers to a group of control subjects (16 - 18 year old boys). This study showed that the elite athletes had an equal distribution of type I fibers in the gastrocnemius, the vastus lateralis and the deltoids, but that the percentage of type I fibers was significantly higher in the elite athletes compared to the control group, highlighting the role that genetic factors may play in influencing skeletal muscle morphology.

The physiological profiles of single muscle fibers in highly trained athletes and the cellular adaptations that occur in these fibers with various exercise training programmes, suggest that there may be a high degree of plasticity within the specific fiber types (543).

#### **2.1.4.5. Cross sectional area of skeletal muscle**

The cross sectional areas of skeletal muscle fibers varies considerably among individuals (125;574). Costill et al (125) showed that in elite distance runners, type I fibers were 29 % larger than type II fibers, whilst no differences

in fiber size were shown in middle distance runners and untrained men. These authors concluded that the differences in fiber size must be as a consequence of the training distance, with middle distance running requiring a larger component of strength-type training, compared to predominantly endurance type training performed by long distance runners.

Similarly, Coggan et al (118) showed increases in the lateral gastrocnemius muscle cross sectional area of type I, IIa and IIb muscle fibers of 12 %, 6 % and 12 % respectively, in 60 - 70 year old men after 10 months of endurance training.

Other studies, however, have been unable to identify any alterations in muscle fiber size associated with endurance performance (212;317).

#### **2.1.4.6. Muscle force and velocity**

There has only been one laboratory study (572) investigating skeletal muscle alterations associated with endurance training, using a control group matched specifically for age. Widrick et al (572) compared the force-velocity and power-velocity properties of single muscle fibers between endurance trained masters runners (mean age  $44 \pm 1$  year) and age-matched sedentary controls. These authors found that the running group's type I and IIa fibers were significantly smaller in diameter than those

measured in the sedentary group. Similarly, the peak power output was 13 % and 27 % lower in type I and type IIa fibers respectively, compared to the sedentary group. Accordingly, the absolute force associated with the lower peak power output in the active group was also significantly lower. This study also showed that the maximal shortening velocity produced by the runners was higher than the sedentary controls.

Widrick et al (572) concluded that even though the runners in their study were unable to produce the same amount of force as the sedentary controls, the elevated shortening velocity in the active group allowed these muscle fibers to maintain a greater level of force production during rapid muscle shortening. In this way, type I fibers make a greater contribution to the total power output of the whole muscle and thereby reduce the reliance on the more fatigable type IIa fibers.

Collectively, these data thus suggest that after the age of 50 years, the extent to which age alters skeletal muscle morphology, innervation and function, can be altered, slowed or partially reversed by endurance exercise training (182;298;478;479). Indeed, it has been shown that aging muscle responds to endurance training in a similar manner to that of younger subjects (293).

### **2.1.5. THE COMBINED EFFECT OF AGE AND ENDURANCE TRAINING ON SKELETAL MUSCLE**

Accordingly, the combined effects of endurance training with increasing age on muscle fiber composition, muscle fiber size and skeletal muscle contractile properties have been identified and are discussed below.

#### **2.1.5.1. Muscle fiber composition**

Trappe et al (544) studied distance runners over 20 years. The athletes were divided into three groups based on their current participation in competitive running events: (i) competitive runners, (ii) runners who trained for physical fitness, and (iii) runners who no longer participated in physical activity. The combined data from all three groups showed a significant increase in type I fibers from approximately 61 to 68 %. This response was, however, attenuated in the competitive runner group, where mean muscle fiber composition remained unchanged (544). The authors concluded that muscle fiber changes associated with aging might be influenced by the degree of physical activity throughout life and/or the muscle fiber composition in early adulthood (544), a suggestion supported by Larsson et al (332;334).

Other studies have, however, reported that muscle fiber composition remains unaltered with age and that there are no differences in fiber

composition between masters runners and younger, performance-matched runners (119;170;223;344). The differences between these studies can be explained by the use of longitudinal study designs, able to identify absolute changes within subject groups (544) compared to cross sectional study designs (119;170;223;344).

#### **2.1.5.2. Muscle fiber size**

Widrick et al (571) and Trappe et al (544) have shown that type I and IIa muscle fibers obtained from athletes competitive in endurance training for long periods of time are smaller than those same fibers observed in sedentary controls (571) or obtained from the same athletes 20 years earlier (544). As a result of these smaller fibers, Widrick et al (571) showed that athletes produce 15 % less force in their gastrocnemius muscle compared to the sedentary subjects. However, they concluded that a smaller muscle diameter was not a disadvantage for endurance activities. Indeed, a smaller diameter would reduce the diffusion distance between the capillary and the mitochondria, resulting in a more efficient delivery of oxygen and fuel to the working muscle. Furthermore, it has also been shown that regular endurance training (469) can prevent the age-associated reduction in type II fiber size and oxidative capacity. This finding has important implications for masters runners, specifically.

### 2.1.5.3. Skeletal muscle contractile properties

Trappe et al (544;545) and Widrick et al (571;572) studied elite master runners who had continued a high level of training for more than 20 years. They used longitudinal test re-test designs to study myosin heavy chain (MHC) fibers, as an indication of muscle contractile properties and rate of cross-bridge cycling. On average, the shortening velocity measured in the runners was approximately 19 % faster than the sedentary subjects. These authors concluded that despite the increase in contractile speed in the runners, sedentary individuals are consistently more powerful in both type I and IIa muscle fibers (544;545;571;572). Furthermore, endurance trained athletes become progressively weaker over an extended period of time.

As mentioned above in section 2.1.3.2.5. (page 33); Kent-Braun et al (292) have identified a novel relationship between percentage non-contractile area of the tibialis anterior muscle and physical activity in older subjects (65 - 85 years). This relationship suggests that habitual physical activity level plays an important role in the age-related accumulation of intramuscular fat. This finding has been supported by others (499;500) who have shown that older athletes (66 - 85 years) have lower intramuscular fat than age-matched sedentary controls.

In summary, although it has been shown that years of accumulative endurance training causes single muscle fibers to be smaller, weaker and

able to produce less power compared to sedentary control muscle fibers, these data also provide further support to the suggestion that participation in regular physical activity may positively attenuate the age-associated alterations to skeletal muscle, which contribute to a decline in running performance.

The quantification of these age- and training-associated alterations in neuromuscular function is essential in the characterization of the decline in running performance. Despite the many difficulties that are encountered using non-invasive measurement tools to identify neuromuscular function, electromyography provides a valid and reliable tool in the determination of neural activation patterns and how these are associated with mechanical output. Therefore, the following section will discuss the use of electromyography and factors that may contribute to alterations in electromyographic activity during exercise.

#### **2.1.6. ELECTROMYOGRAPHY (EMG) ACTIVITY AND THE EMG/FORCE RELATIONSHIP**

Forces generated by skeletal muscle to execute intended movements are regulated by the central nervous system, using proprioceptive afferent information to modulate the commands (151;417;456). Descending command from the central nervous system regulates motor unit recruitment

and firing rate so that the required movement can occur.

Electromyography (EMG) is a technique used to measure electrical activity of a muscle and from which muscle recruitment is inferred (3). This technique has many applications during exercise, particularly for studying changes in neuromuscular characteristics (3;42;220;286).

During isometric muscle actions (179), motor unit recruitment follows the size principle described by Henneman et al (35;244). This theory suggests that neurons with small and slow conducting axons which innervate type I muscle fibers are recruited initially, before neurons with large and rapidly conducting axons, which innervate the type II muscle fibers. Depending on the muscle, recruitment of additional motor units continues until approximately 50 – 80 % of the maximal voluntary contraction, after which the additional force is achieved only by an increase in the firing rates of the active motor units (138;396;406). An increase in surface EMG together with an increase in muscle force thus indicates an increase in motor unit activation and exercise intensity (202;354). Whilst there are a number of studies that have confirmed a linear relationship between force and EMG activity (40;42;202;353;393), some studies, have shown inconsistent relationships between surface EMG variables and force production (169; 177;309;364;372). In addition to fiber type differences and the effects of age and training on neuromuscular characteristics discussed above, inconsistencies in the EMG/force relationship can also be explained by changes in whole limb recruitment patterns (5;163;568), muscle wisdom

(167;197;281) and alterations in muscle temperature (169;174;196;262;439;494) and intramuscular pressure (2;80;169;485;508).

#### **2.1.6.1. Muscle wisdom and changes in whole limb recruitment patterns**

There is substantial evidence to show that isometric muscle force declines rapidly with the onset of fatigue during sustained maximal voluntary contractions. This decrease in force production is usually accompanied by a reduction in motor unit discharge rate, which, during a sustained maximal voluntary contraction, can be as much as 50 % over a 60 second period (42;44;197;226). Associated with this reduction in discharge rate are progressive decreases in the rate of relaxation in whole muscle (45;261), single motor units (155;215) and single muscle fibers (337), and a reduction in the frequency of activation necessary to elicit maximum force (42;44). This concurrent decline in force, relaxation rate and motor neuron discharge rate has been referred to as "muscle wisdom" (167;197;202).

Two hypotheses have been proposed to explain this decline in reflex output. Bigland-Ritchie et al (43) and Garland et al (204) have suggested that this response relies on an inhibitory signal possibly provided by metabolically induced activity in small myelinated and unmyelinated muscle efferents, such as those belonging to groups III and IV (24). These afferents are sensitive to several parameters associated with either metabolic fatigue or muscle damage (483) and there is some evidence to

suggest that these receptors have powerful input to inhibitory interneurons (116). The fatigue-induced metabolic stimulation of these muscle afferents may lead to presynaptic inhibition of the Ia terminals and/or inhibition of interneurons in the oligosynaptic pathways (156). The alternative hypothesis suggests that there may be a disfacilitation of the alpha-motoneuron pool due to a progressive withdrawal of spindle-mediated fusimotor support (53;229).

Although the mechanisms underlying muscle wisdom remain unclear, it is suggested that this decrease in motor unit activity may serve to forestall muscle fatigue by (i) optimising the force output of motor units as their contractile speed slows (44;45;197) and (ii) protecting against peripheral conduction failure associated with prolonged, high discharge rates (276).

In addition to the alterations in motor unit discharge rates contributing to changes in the EMG/force relationship, there is also the possibility that different muscles have a non-uniform activation pattern during maximal work (5). Westgaard and De Luca (568) have suggested the possibility of motor unit rotation and substitution. This concept describes a mechanism by which motor units alternate their activity in a cyclical fashion, such that substitution of one motor unit for another would be followed by back-substitution of the original unit. This substitution phenomenon functions to protect motor units from excessive fatigue when there is a demand for constant muscle activation (568). These authors (568) have also shown

evidence of differing firing behaviour among concurrently active motor units. Similar differences in whole limb recruitment patterns have been shown using mechanomyogram (MMG) techniques (309;496) that have provided evidence of non-uniform mechanical activity of the individual (vastus medialis and vastus lateralis) knee extensor muscles. Together, these inconsistencies in motor unit firing properties would influence the overall EMG/force relationship.

#### **2.1.6.2. Muscle temperature and intramuscular pressure**

Esposito et al (169) have suggested that alterations to muscle temperature may also have an effect on muscle recruitment and rate coding, thereby altering the EMG/force relationship. Other studies have confirmed these suggestions and have shown that impaired exercise performance in the heat is associated with a reduction in voluntary activation (42;183;372;439).

In contrast, however, there are other studies that have shown that an increase in body and environmental temperature has only minor (196) or no effects (262) on neuromuscular performance. Hunter et al (262) measured EMG activity from the rectus femoris muscle at regular intervals during a cycling protocol in hot (35 °C) and cold (15 °C) environments. This study showed that although the heated conditions resulted in increased skin temperature and heart rate, there were no differences in muscle recruitment or maximal performance between each temperature extreme.

The discrepancies between these studies are likely to be explained by the protocols used to measure EMG activity. Nybo and Nielsen (439) measured EMG activity during a 2 minute maximal voluntary contraction immediately after a cycling intervention, whilst Ftaiti et al (196) and Hunter et al (262) measured EMG activity during a functional exercise protocol, and thereby perhaps obtained a more reliable measurement of alterations in EMG activity associated with exercise in the heat.

Furthermore, Masuda et al (372) and Sadamoto et al (485) have suggested that other factors contributing to alterations in the EMG/force relationship during maximal voluntary contractions are changes in intramuscular pressure and blood flow. Changes in pressure and blood flow associated with static contractions may alter the intramuscular pH, or the concentration of potassium ions. These ionic fluctuations alter the excitability of the muscle fiber membrane (11;408;472) thereby influencing surface EMG activity (80;169).

Collectively, these results suggest that centrally driven muscle recruitment strategies contributing to maximal force production can be inferred from the information provided by surface EMG data. However, there are a number of factors that may alter this relationship. These factors include differences in muscle recruitment strategies as a result of age and training, differences in muscle fiber properties, muscle wisdom and changes in

whole limb recruitment patterns, and alterations in muscle temperature and intramuscular pressure.

It is possible that in addition to increases in intramuscular pressure with long duration static contractions, swelling and inflammation associated with skeletal muscle damage may also affect the EMG/force relationship (2;80; 169;372;506;508).

### **2.1.7. LENGTHENING MUSCLE ACTIONS (ECCENTRIC EXERCISE)**

Lengthening muscle actions (179) imply an active lengthening of skeletal muscle whilst resisting force (288;435). Based on the cross bridge theory of muscle contraction, the force exerted by muscle is generated by the interaction between actin and myosin (165;245;426). However, when muscle fibers are lengthened during an "eccentric" action, the actomyosin bonds are thought to undergo a mechanical detachment rather than an ATP-dependent process (184). In addition, because of the higher efficiency associated with these actions (304;384), higher muscle strain is distributed over fewer muscle fibers (165;354;384). Indeed, lengthening muscle actions place high stresses and strains on the involved structures and has thus been associated with exercise-induced structural abnormalities (13;52;145;171-173;187;191;414;508).

The ability of skeletal muscle to generate force is greater during lengthening actions than during a concentric, or shortening, action (179;578). This increased ability of skeletal muscle to absorb energy during a lengthening action contributes significantly to the protection and reduction in damage of the less compliant elements of the skeletal system. These include bone, ligaments and connective tissue. Furthermore, lengthening muscle actions require lower levels of voluntary activation by the nervous system (as indicated by a reduction in EMG activity) to achieve a given muscle force (13-15;52;145;165;171-173;187;191;384;414;452;508).

#### **2.1.7.1. Neural activation during lengthening muscle actions**

Enoka (165) has reviewed the possibility that lengthening muscle actions may require a unique neural activation strategy. High threshold motor units are used minimally during resting conditions but are essential for exercise requiring high levels of muscle power. The function of a unique activation system would therefore be to maximise muscle activity, yet preserve the "health" of these high threshold motor units (165;569). This phenomenon can be illustrated by reductions in muscle activation during lengthening maximal voluntary actions, alterations in the recruitment order of motor units during submaximal lengthening muscle exercise, decreases in the size of the motor-evoked potentials by transcranial and peripheral nerve stimulation during lengthening exercise and increased fatigue resistance during repeated lengthening muscle activity (165;287;569).

The differences in neural recruitment patterns between different muscle actions (shortening vs. lengthening) may also be due to a modulation of the relative excitability within the populations of motor neurones innervating a muscle, it's synergists and the contra lateral muscle (165;355).

Notwithstanding the differences in recruitment strategies between shortening and lengthening muscle actions, there is a characteristic pattern of change that occurs within the contractile apparatus of skeletal muscle following repeated lengthening muscle actions (187;191;351). Typically, there is a threefold increase in focal disturbance of the striated band pattern at the ultra structural level (191). This focal disturbance appears to originate at the Z-disc. Z-disc streaming refers to Z-discs which appear broadened, smeared, or totally disrupted (187), with Z-discs of adjacent myofibrils out of register and running a "zig zag" course. In addition, other structural abnormalities associated with excessive lengthening activity include sarcolemmal disruption, dilation of the transverse tubule system, distortion of myofibrillar components, fragmentation of the sarcoplasmic reticulum, lesions of the plasma membrane, cytoskeletal damage, changes in the extracellular myofiber matrix and swollen mitochondria (105;188;189;191;192;248;317;502;513;558).

These clinical data link lengthening muscle actions to muscle fiber and connective tissue damage. The clinical signs that have been associated with this ultra structural damage are muscle discomfort, pain, swelling and

stiffness, collectively referred to as delayed onset muscle soreness (DOMS) (115;124;316;363).

### **2.1.8. DELAYED ONSET MUSCLE SORENESS (DOMS)**

Delayed onset muscle soreness is the sensation of discomfort or pain in the skeletal muscle that occurs one to two days following a bout of muscular exertion (13;115;316;363;508), especially when the exercise is relatively intense, is of long duration, is an unfamiliar mode and/or includes lengthening muscle actions (15;115;316;508).

Lieber et al (351) have shown that in rabbit muscle, structural changes accompanying lengthening contraction-induced injury take place within the muscle 5 - 15 minutes after the initiation of exercise. However, symptoms associated with this muscle damage are first evident between 8 - 24 hours after the exercise, peak between 24 and 72 hours after the exercise, and disappear gradually thereafter (13;14;171;414). The most frequently associated symptoms with DOMS include an increase in limb volume and swelling, increases in muscle stiffness, reductions in range of motion, a decrease in force production and a leakage of myofibril proteins into the blood (84;108;112;113;124;171;185;316;363;414;431;434;436;436;506).

### **2.1.8.1. Mechanisms underlying DOMS and skeletal muscle damage**

Armstrong (15) has proposed an integrated model of 4 phases of muscle injury resulting from lengthening actions: (i) initial events, (ii) autogenic processes, (iii) phagocytic phase, and (iv) regenerative phase.

Active lengthening of skeletal muscle results in damage to the muscle fibers. These fibers contain intrinsic proteolytic and degradative pathways that may then respond to this damage (15). Prior to the inflammatory response, when phagocytic cells invade the damaged tissue (363), autogenic processes associated with inflammation begin the degradation of the lipid and protein structures in the injured cells (15). Once the phagocytic stage is initiated, rapid breakdown of the damaged muscle fibers begins from lysosomal proteases and other substances produced by macrophages (15). The final stage involves regeneration of the injured cells.

#### **2.1.8.1.1. Initial events**

There are two primary hypotheses proposed for the initial events associated with muscle damage, (i) mechanical mechanisms and (ii) metabolic and biochemical mechanisms.

(a) *Mechanical mechanisms*

Stauber et al (516) and others (259) have suggested that DOMS is due to a complex reaction of extracellular matrix, cell and inflammation mediators associated with the disruption of muscle fibers and connective tissue.

Faulkner et al (180) have suggested that the mechanism of injury to skeletal muscle from lengthening muscle actions is the increased tension per individual cross bridge. The force that is developed during these muscle actions is significantly higher than which is developed during isometric actions, yet the total number of strongly bound cross-bridges during lengthening activity is only about 10 % greater (180;578). This increase of force per active muscle unit causes mechanical disruption of the ultra structural elements within the muscle fibers, such as the Z-line and contractile filaments (180;192).

Lieber and Fridén (350) have suggested that high tension alone may not cause muscle damage during lengthening exercise, and that damage may also be associated with muscle fiber strain during active lengthening. They suggest that active strain that exceeds the limits of the extrasarcomeric cytoskeleton framework, which joins the cytoskeleton to the sarcolemma, may also result in sarcolemmal damage.

Morgan (398) has proposed that, because of a lack of homogeneity in sarcomere length, lengthening actions cause some (shorter) sarcomeres to become over extended. This action pulls the actin and myosin fibers in these sarcomeres apart to such an extreme that on relaxation, interdigititation is lost and the sarcomere is said to have "popped". In addition, such "popping" sarcomeres may disrupt the sarcolemma and the sarcoplasmic reticulum of adjacent fibers, resulting in the characteristic pathology of muscle damage. During prolonged endurance exercise, repetitive lengthening muscle activity will contribute to the alteration in sarcomere uniformity, thus also contributing to the sarcomere "popping" (82;398).

(b) *Metabolic and biochemical mechanisms*

i. *Muscle temperature*

Kumazawa and Mizumura (318) showed that type III and IV nerve endings are sensitive to temperatures of 38 to 48 °C. Lengthening muscle actions have been shown to generate higher local temperatures compared to shortening actions (134;410) which may account for damage associated with this lengthening muscle activity. Accordingly, it has been suggested that elevated muscle temperatures may be associated with damage of the structural elements of the muscle, resulting in necrosis of muscle fibers and breakdown of connective tissues (13;15;173).

## ii. Metabolic waste product accumulation

Peripheral waste product accumulation, specifically increases in lactic acid concentrations have also been proposed to account for muscle damage (13-15;173;248;568) during lengthening, unaccustomed and long duration exercise. There are, however, considerable data that argue against this hypothesis. Indeed, exercise involving lengthening muscle actions requires less energy expenditure, a lower oxygen consumption and produces less lactate than during equivalent shortening muscle actions (13;14) and thus increases in the accumulation of lactic acid is unlikely to play a significant role in the initiation of skeletal muscle damage.

## iii. Adenosine triphosphate (ATP) and calcium concentrations

It has also been suggested that during exercise, the demand for ATP in skeletal muscle may exceed ATP production (13-15). Associated with a decrease of free ATP within the cell, are alterations in hydrogen ion concentrations, which reduce the capacity for calcium release from the cell via ATP dependent pumps (14;541). Subsequent increases in intracellular calcium concentration following the initial injury are proposed to contribute to the progression of muscle damage by stimulating calcium activated neutral proteases such as calpain (16;37;39;154;158;559). These proteases may initiate proteolysis by cleaving "susceptible" Z-line associated proteins such as desmin (39;79;248) and may possibly also have a preferential damaging effect on mitochondrial function (213;513).

The mechanisms for the increase in intracellular calcium concentrations following muscle injury are unknown, although it is likely that lesions in the sarcoplasmic reticulum, and/or a defect in the ability of the sarcoplasmic reticulum to reuptake calcium are contributing factors (158). It has been hypothesised that when actin and myosin are pulled apart during lengthening actions, the surface membrane is damaged allowing an entry of extracellular calcium (13;413). In addition, the possibility of an opening of stretch-responsive channels and/or alterations in triad and t-tubule orientation resulting in calcium entry via voltage-sensitive channels has also been proposed (37;113).

This damage to the muscle fiber results in an inflammatory response that causes a transfer of fluid and inflammatory cells into the damaged tissue.

#### **2.1.8.1.2. Autogenic processes**

Inflammation is characterised by the movement of fluid, plasma proteins and leucocytes into the tissue in response to injury (363;506). Signalling occurs between the injured muscle cells and the mononucleated cells that subsequently appear at the injury site (541). Inflammatory cells and myogenic cells both respond to muscle injury (363). Inflammatory cells are primarily involved in the removal of cellular debris, whilst myogenic cells initiate the replacement of the damaged tissue (506). Infiltration of inflammatory and myogenic cells into the muscle is orchestrated by

specific cytokines (471;507). Cytokines are small polypeptides, proposed to have a fundamental link between immunological and neuroendocrinal systems involved in inflammation, chemotaxis and the acute phase response (363;471;507).

An increased presence of monocytes and macrophages at the site of injury follows the inflammation associated with exercise-induced muscle damage (363).

#### **2.1.8.1.3. Phagocytic phase**

Macrophages and neutrophils produce a variety of cytotoxic enzymes and oxygen free radicals. Macrophages are capable of degrading and remodelling cells and are primarily responsible for the resorption of neutrophils in necrotic tissue and the sequestration of persistent foreign material or antigens (363). Indeed, several studies have reported that macrophages are the predominant inflammatory cell present in exercise induced muscle injury (17;277;363;382;471). Increased concentrations of lymphocytes, myogenic cells and mast cells have also been associated with exercise-induced muscle injury, following a marathon run (248;502) or forced muscle lengthening (517).

Monocytes and macrophages perform 3 major functions within the muscle damage and repair cycle (113;363;471;541): (i) breakdown of debris, (ii)

removal of cellular debris via phagocytosis, and (iii) assistance in skeletal muscle regeneration.

#### **2.1.8.1.4. Skeletal muscle regeneration**

Following these initial and autogenic events, macrophages, neutrophils and other smaller cells (471) break down and remove all traces of the originally damaged fiber during the phagocytic processes. The regeneration of a new fiber begins within the basal lamina of the original muscle fiber. Once a population of myoblastic cells is established, myoblasts fuse into multinucleated myotubes that differentiate into mature muscle fibers with peripherally located nuclei. This process is then followed by the formation of functional neuromuscular junctions (91).

The establishment of neuromuscular junctions during regeneration is a tightly controlled process consisting of 2 main phases (90;91;93;441). First, regenerating nerve fibers grow into the new muscle either through the cut ends of nerves leading to the muscle, or by the sprouting of nerves in tissues adjacent to the regenerating muscle (51;91;441). Second, this process is followed by the formation of a functional neuromuscular junction (90).

In the presence of an intact basement membrane and undamaged muscle fibers, complete regeneration of skeletal muscle may occur within seven days (106;263).

The skeletal muscle of endurance athletes undergoes continual breakdown and regeneration in response to regular lengthening muscle actions associated with long distance running. Although the regeneration and repair of skeletal muscle is an essential part of inducing skeletal muscle adaptations to training, it has been suggested that older runners may not be able to adapt to these ultra structural alterations to the same extent as younger athletes (228;330;370).

(a) *The effect of age on skeletal muscle regeneration*

Skeletal muscle has the ability to regenerate after a wide variety of injuries (93). However, there is some evidence from animal models to suggest that skeletal muscle regeneration is negatively affected by the aging process (228;370) and that complete and successful muscle regeneration is significantly less in older compared to younger animals (73;181;486).

Conversely, Carlson et al (92) used an *in vivo* rat model to show that under appropriate conditions, such as sufficient motor nerve supply, aged damaged muscle is able to regenerate as well as younger damaged muscle. In some cases, the older rat muscle exceeded the regeneration capacity of the younger muscle. Indeed, these authors argue that provided the satellite cells are regenerating in similar environments, there remains sufficient cellular reserve in older muscle to allow regeneration to occur as well as in young muscle (*in vivo*) (51;92;140).

In addition to the autogenic and regenerative processes that occur at the injury site discussed above, Fridén et al (1988) have presented evidence that implicates cytoskeletal disturbances as a major contributing factor to the observed ultra structural changes.

#### **2.1.8.1.5. Muscle damage and the cytoskeleton**

The myofibrillar cytoskeleton is comprised of two sets of filaments, the exosarcomeric cytoskeleton and the endosarcomeric cytoskeleton. The exosarcomeric cytoskeleton consists of longitudinal and transverse intermediate filaments comprised of the proteins desmin, vimentin and synemin. The longitudinal fibers run between Z-discs and serve as an attachment site for mitochondria, nuclei and the sarcolemma, as well as limiting the extensibility of the sarcomere whilst the transverse filaments link adjacent myofibrils at the level of the Z-disc.

The endosarcomeric cytoskeleton acts as a third filament system, coexisting with actin and myosin within the sarcomeres, and is comprised of titin and nebulin. It has been proposed that titin is responsible for resting muscle elasticity and the central position of myosin within the sarcomeres, whereas nebulin is primarily involved in the maintenance of actin's lattice array (562).

Lieber et al (351) and others (192) have identified that the structural protein, desmin, is a sensitive marker of muscle damage and have suggested that cytoskeletal disturbances of desmin precede, or are at least detectable much earlier than direct injuries to the contractile properties.

Fridén et al (188) have shown abundant longitudinal desmin extensions in muscle biopsies obtained three days following intense lengthening muscle exercise. As a result, 2 theoretical causes of cytoskeletal disorganization have been proposed (188;191;515). First, disorganization may reflect mechanical disturbances of the desmin filaments caused by high tensions developed during lengthening muscle actions or distension of the cytoskeleton caused by oedema. An increase in the intracellular pressure as a consequence of a release of osmotic components from disruptions in the Z-disks will therefore result in uneven distribution of forces on the cytoskeletal attachments (188;191;515).

Second, changes in the myofibrils are a subsequent response to the extensive myofibrillar lesions and may reflect sarcomeogenesis (188). Indeed, an increased synthesis of desmin and the reorganization of the cytoskeletal system may be fundamental in the reorganization of the distorted myofibrillar material. The desmin filaments would thereby possibly act as mechanical forerunners for the ultimate repair of sarcomeres (188).

Although the role of the myofibril cytoskeleton of skeletal muscle remains controversial, the Z-disc streaming, splitting and degradation observed following predominantly lengthening muscle actions may be as a result of disruption of both the endo- and the exosarcomeric cytoskeleton (562). This disruption would result in altered skeletal muscle mechanical properties and force production (538).

### **2.1.8.2. The effect of DOMS on muscle function**

Skeletal muscle function, including force production, is significantly compromised following injury and exercise-induced muscle damage (114; 136;159;187;356;379;414). After exercise involving active lengthening of the muscles, there is an immediate decrease in maximal force production, followed by a slow recovery (13;15;30;38;52;81;114;165;171;192;339;414; 432;436;508). The pre-exercise strength values may remain below baseline levels for up to 11 days following the damaging bout of exercise (80-82;108).

Investigation of the time courses for the development of soreness and for loss of strength suggests that there is little or no relationship between the 2 parameters (105;277;414;430;476). Newham et al (414) showed that the maximal voluntary force of knee extensors returned to normal 24 hours following downward stepping, whilst at the same time, muscle soreness was the most intense. More recently, Rodenburg et al (476) and Nosaka et al (430) have shown a poor relationship between muscle soreness and muscle

strength, for up to five days, following a bout of active lengthening arm exercise, which caused muscle damage.

The mechanism by which lengthening muscle action results in a loss of strength has not been clearly identified. Either the strength deficits are a secondary response to soreness perceptions, or the inherent capacity of the muscle to produce force is lowered (158). There is increasing evidence to support this latter possibility (6;38;80;399;414). Morgan and Allen (399) have suggested that the reduction in force and tension producing capabilities after lengthening muscle actions could be associated with one or more of the following factors: (i) changes in the central nervous system, motor nerves or at the neuromuscular junction, (ii) disorganization of the contractile machinery and calcium regulation, and (iii) selective muscle fiber damage. These suggestions have been further supported by others (38;339;470) and are discussed in more detail below.

#### **2.1.8.2.1. Central changes**

It has been suggested that the structural changes associated with lengthening muscle actions are accompanied by alterations in neuromuscular performance (307). Specifically, there is evidence to show a reduction in neuromuscular efficiency (83;144), reflected as a decrease in the EMG/force ratio, during and after lengthening muscle actions. These findings suggest that firing patterns in damaged muscles may be altered in

such a way that additional motor units, together with an increase in firing frequency, may occur in an attempt to compensate for changes in contractile function (47;76;158).

Newham et al (414) have shown an increase in EMG activity during a muscle lengthening stepping exercise. A submaximal knee extension test performed at specific intervals following the damaging exercise also showed increased EMG activity at all joint angles between 0 and 90 degrees. In addition, the neural activation necessary to maintain full extension for a period of 2 seconds was increased. Comparable results have been shown by Abraham (2) and McGlynn et al (383). Furthermore, Komi and Viitasalo (307) observed a decrease in maximum strength together with an increase in neural activity at a given muscle tension both immediately and two days after damaging muscle lengthening exercise.

Contrary to these results, however, Behm et al (38) have shown that even with significant losses in force after a lengthening exercise of the elbow flexors, there are no significant changes in EMG activity and that muscle activation is restored to pre-exercise values after only one day. This finding has been supported by Brown et al (76) who showed a significant inhibition of muscle recruitment for three days following a damaging, long-duration exercise bout.

The discrepancies between these studies could be explained by differences in the damaging exercise interventions that were used. For example, Behm et al (38) used a resistance exercise protocol, which would be unlikely to result in the same magnitude of structural muscle damage that has been shown to occur after specific muscle lengthening techniques used in other studies (2;307;414). Accordingly, these protocols might not have been sufficiently debilitating to significantly curtail neuromuscular activation.

#### **2.1.8.2.2. Disorganization of the contractile machinery and calcium regulation**

The relationship of force and myofibrillar damage following active muscle lengthening may indicate that there is a failure of the contractile machinery at the level of the sarcomere (112;117;172;414). A reduced efficiency of the excitation-contraction coupling process associated with a loss of maximal force generating capacity has been demonstrated in animals (265;561). These studies have confirmed a reduced rate of calcium release from the sarcoplasmic reticulum in maximally activated tetanic force, indicating that a failure to fully activate the contractile machinery is the primary reason responsible for the loss of force after lengthening muscle action. Furthermore, these investigators (265;561) estimated that at least 75 % of the reduction in maximal force production could be explained by excitation-contraction coupling failure immediately after the damaging

exercise bout, and these alterations contributed to the reductions in maximal force production for up to 5 days thereafter.

These results suggest that in addition to the structural changes that occur to the excitation-contraction mechanisms associated with lengthening muscle actions, calcium regulation and sensitivity may also be affected and will thus also contribute to the reduction in force generating capacity following damaging exercise (6;369;399).

#### **2.1.8.2.3. Selective muscle fiber damage**

Several investigations have reported selective damage to type II muscle fibers (80;173;189;190;192;277;339;384). Lepers et al (339) showed a greater loss in lengthening muscle force than concentric strength following a two hour run. These authors concluded that the greater loss in lengthening strength was due to specific damage to type II muscle fibers (339). These suggestions have been supported more recently by Byrne and Eston (80) who showed reductions in isometric strength for up to seven days (35 % at one hour post-exercise and 5 % on day seven post-exercise) after an exercise protocol, which caused muscle damage. The authors suggested that muscle damaged through lengthening muscle actions is characterised by an inability to generate high force and power, yet an improved ability to maintain force and power (80). Based on these functional outcomes, it was concluded that type II fibers were selectively recruited and subsequently

damaged during exercise involving predominantly lengthening muscle action (80).

Fridén and Lieber (189;190) proposed a mechanism for the selective type II fiber damage, suggesting that during the initial stages of lengthening, fast twitch glycolytic muscle fibers are instantaneously fatigued. These fibers, then unable to regenerate ATP, undergo systematic mechanical disruption. In addition, structural differences between fast and slow twitch muscle fibers may also predispose type II fibers to selective damage. Type II fibers are characterised by narrower Z-lines, reflecting a lower actin-myosin attachment, and thus a weaker sarcomere connection (189;190).

There is substantial evidence therefore to suggest that lengthening muscle actions result in a significant reduction in maximal and dynamic force production. The inability to generate an initial high force and power after damaging exercise will depend on events that occur at, or distal, to the neuromuscular junction (peripheral) and/or on the ability to activate the muscles voluntarily (central). It is likely that there is a continual interaction between central and peripheral factors which together result in a reduction in performance.

Furthermore, unaccustomed lengthening muscle activity, has been associated with leakage of intramuscular protein from the injured muscle fibers (32;38;81;82;158;231;283;434;436;510). The measurement of

concentrations of certain muscle proteins or activities of enzymes in the blood may serve as an indirect marker of muscle damage.

### **2.1.8.3 Biochemical markers of skeletal muscle damage**

Elevated circulating levels of creatine kinase (CK) activity, lactate dehydrogenase (LDH) and myoglobin concentrations (Mb) have all been well documented after various forms of exercise (158), including running distances ranging from 15 km (231) to 80 km (283). For practical reasons, creatine kinase has been widely used as an indicator of muscle damage following strenuous exercise (84;114;158;231;256;308;367;492;509).

Creatine kinase is a dimeric enzyme which catalyses the reversible phosphorylation of ADP, by creatine phosphate, to form ATP and free creatine (256). Since creatine kinase is a large molecule, it is unlikely to enter into the bloodstream directly from the cells and therefore increased activities of this enzyme in the circulation are usually only observed after muscle fiber damage accompanied by membrane leakage, or necrosis of the muscle fiber (423;510).

Creatine kinase activity in the blood can easily be influenced by a variety of factors including the intensity and duration of the exercise, the type of exercise, training status, gender, ethnicity, age and environmental conditions (256;423;510). Furthermore, there is considerable variation and

inter-subject variability in serum creatine kinase activity following exercise that results in muscle damage (112;256;368;428). Therefore, the appearance of creatine kinase activity in the blood can be used as a marker of muscle damage, but cannot be used to accurately quantify this damage.

In an attempt to identify more accurate markers of muscle damage, recent emphasis has therefore been placed on the predominantly bound proteins of the contractile apparatus of skeletal muscle, such as myosin heavy chains (contractile proteins of the thick filaments) (365) or skeletal troponin I (regulatory proteins of the thin filaments) (510;511). These proteins are both unique to skeletal muscle (510) and an increase in the circulation of either one after exercise would indicate cell membrane leakage and degradation of the contractile apparatus (510;511).

There is substantial evidence to suggest, however, that regular endurance exercise training attenuates the negative effects of muscle lengthening activity on muscle fiber damage and thus skeletal muscle enzyme activity (520).

#### **2.1.8.4. Effects of endurance training on the response to lengthening muscle actions and associated muscle damage**

Gibala et al (205) investigated the differences between trained and untrained individuals in their ability to respond and recover from muscle lengthening induced injury. These authors showed that the proportion of disrupted fibers and the magnitude of this disruption were significantly less in trained subjects compared to sedentary controls. Other studies have shown significantly fewer neuromuscular alterations in trained subjects for approximately 14 days following damaging lengthening exercise (277). Gibala et al (205) have therefore concluded that regular exercise training may attenuate the severity of lengthening-induced injury, and have suggested the possibility of an accelerated tissue repair process in the skeletal muscle of endurance trained individuals.

In addition, Fridén (188) has suggested two possible mechanisms of structural myofibrillar adaptation that may result from a regular muscle lengthening training stimulus: First, there may be an increase in sarcomere length, which would allow muscle fibers to stretch further, before "popping" (82;398;399) or resulting in a mechanical disruption. Second, sarcomeogenesis (188;359;398) may result in an increase in the number of longitudinal sarcomeres available for each lengthening action, and thirdly, there may be an increase in the synthesis of Z-band proteins or intermediate filaments that would act to strengthen existing myofibrils.

Together, these morphological alterations, associated with regular training, would attenuate the effects of high muscle lengthening forces on skeletal muscle.

Indeed, research has consistently shown that if this same exercise is repeated within several weeks of the initial damaging bout, significantly less structural and functional impairments occur after the second exercise session. This observation has become known as the repeat bout effect (107;112;158;386;432;432;433;436;437;449).

#### **2.1.9. THE REPEAT BOUT EFFECT**

The repeat bout effect describes an adaptation of skeletal muscle to a bout of exercise that causes muscle damage. For example, activities of circulating enzymes are lower, there is less swelling and muscle soreness, less abnormalities visible in ultrasound and magnetic resonance images, and a faster recovery of muscle strength measured in the subsequent bout, compared to measurements obtained immediately following the initial damaging exercise (107;112;158;386;432;432;433;436;437;449).

### 2.1.9.1. Proposed mechanisms to explain the repeat bout effect

Although the mechanisms underlying the repeat bout effects remain unclear, there have been a number of theories proposed to explain this phenomenon.

#### 2.1.9.1.1. The neural theory

The “neural theory” (66;82;208;385;429;560) proposes that following the initial bout of exercise which causes muscle damage, there is a more efficient recruitment of motor units, increased synchrony of motor unit firing, a more even distribution of workload over the active fibers, improved usage of synergist muscles and perhaps even an increase in slow twitch fiber recruitment (208;385;560). This theory was prompted by the observation that the force loss immediately after a second bout of exercise consisting of lengthening muscle actions was less than after the initial bout, suggesting that neural adaptations may be involved in reducing the force or the number of active muscle fibers utilized during lengthening muscle actions (208;560).

The influence of neural factors on functional parameters after active muscle lengthening exercise has attracted recent attention. It has been suggested that the development of a subjective maximal lengthening torque, under pre-fatigued and pre-damaged conditions, may in fact not

be maximal and is relatively incomplete, limited by an inhibitory or tension limiting mechanism (257;534). This submaximal muscle activation may represent a greater reserve for improvements in lengthening muscle force and thus may contribute to the enhanced neural strength gains following the initial exposure and adaptation to the damaging exercise (257).

Based on this theory, it could be argued that a centrally mediated increase, or maintenance, of force production after muscle damage may place more stress on the contractile apparatus. If this were the case, the severity of the muscle damage would be increased. There is some evidence, however, to suggest that the ability to adopt a more efficient motor unit recruitment strategy may favourably alter motor unit recruitment in such a way that the stress placed on each myofibril is reduced during the subsequent bout of exercise (208).

An alternative suggestion, proposed by Nosaka and Newton (432), is that the smaller magnitude of neuromuscular changes observed in the second bout of exercise may be due to an intrinsic mechanism which inhibits the recovering muscles from generating the same extent of force as they did during the initial bout which caused the muscle damage. Should similar levels of force be produced during the second bout, muscle damage would again occur, thereby retarding the recovery and regenerative process.

### 2.1.9.1.2. The connective tissue theory

The "connective tissue theory" (114;158;360;363;516) proposes that adaptation occurs after successful muscle repair and/or reorganization of several contractile and structural components such as the sarcomeres (360), the extracellular matrix (516) and the cytoskeleton (363), making the muscle less vulnerable to exercise induced muscle damage. It is likely that the repair process is partially dependent on the inflammatory response triggered by the initial mechanical damage (108;158;188;288;363;506;541). As discussed above, the inflammation removes damaged muscle tissue by recruiting macrophages as well as playing a role in muscle repair and/or reorganization (363;541). Thus, subsequent remodelling of the intermediate filaments and/or an increase in intramuscular connective tissue provides protection against further muscle damage during the second bout of exercise.

### 2.1.9.1.3. The cellular theory

The "cellular theory" (84;158;192;359;366) proposes that muscle damage is the result of irreversible sarcomere strain during lengthening muscle activity which stretches the sarcomeres beyond actin-myosin overlap and reduces contractile integrity. Following the initial damaging exercise, the repeat bout effect may occur through an increase in the number of sarcomeres in series in muscle fibers. This suggests that the sarcomeres remain on the

ascending limb of their length-tension curves over the range of muscle lengths used in the exercise, thus reducing the total sarcomere strain during the subsequent bout of exercise, and thereby limiting further muscle damage (359;398).

#### **2.1.9.1.4. Additional factors associated with the repeat bout effect**

Additional peripheral adaptations that could be associated with the repeat bout effect include strengthening of plasma membranes, removal of stress-susceptible fibers, adaptations in excitation-contraction coupling mechanisms, improvements in the ability to repair initial micro injury and/or a blunted inflammatory response (386).

It is unlikely that one theory can explain all of the various observations of the repeat bout effect that are discussed in the literature. It is therefore possible that the repeat bout effect occurs through the interaction of various neural, connective tissue and cellular factors that are dependent on the changes associated with the initial damaging exercise bout and the specific muscle groups used. The combination of these factors and the duration of their effects are important determinants in the effectiveness of running performance and adaptability to exercise training.

### 2.1.9.2. The duration of the repeat bout effect

Nosaka et al (436) measured maximal isometric force, range of motion, upper arm circumference, muscle soreness and plasma creatine kinase activity before and for five days after exercise consisting of lengthening actions of the elbow flexors. These measurements were repeated every three months for 12 months following the initial exercise bout, which caused muscle damage. The results of this study showed faster recoveries of strength, reductions in muscle swelling and soreness and smaller increases in plasma creatine kinase activity when the interval between damaging exercise bouts was less than six months. The repeat bout effect seemed to subside after six months as there were no differences in muscle force recovery, muscle swelling, soreness and plasma creatine kinase activity between the initial tests and the tests conducted after nine months.

Byrnes et al (84) studied the duration of the repeat bout effect by using two 30 minute bouts of downhill running, separated by three, six and nine weeks, to induce muscle damage. This study found no changes in measures of muscle soreness, plasma creatine kinase activity and myoglobin concentration between bouts for the nine week group, a significantly shorter protective effect compared to that shown by Nosaka et al (436).

Nosaka et al (436) attribute the differences between the two studies to different methodologies (i.e. leg muscles during downhill running, compared to the smaller elbow flexor muscles), or differences in the extent

of the muscle damage induced by each exercise (peak creatine kinase values reported by Byrnes et al (84) were significantly lower than those reported by Nosaka et al (436)). Both authors do note, however, that there was a large variability in the extent of muscle damage and response to the same exercise protocol among subjects.

In summary, the literature suggests that regular endurance training and exercise involving repeated lengthening muscle actions results in ultra structural muscle damage. Symptoms of this skeletal muscle damage include delayed sensations of pain and stiffness, reductions in maximal force production, increases in serum muscle enzyme activity and muscle swelling and inflammation. The inflammatory response associated with muscle damage serves to remove injured muscle tissue and initiate an effective regenerative process. It appears that both central and peripheral factors play an important role in restoring muscle function to pre-injured performance levels, both during the regenerative phase as well as initiating an efficient repeat bout effect.

#### **2.1.10. SUMMARY OF THE LITERATURE AS BACKGROUND TO THE THESIS**

Aging has been associated with morphological alterations and changes to motor control that together result in a progressive decline in force production, power and overall physical capacity. Despite these age-associated changes, participation in regular endurance exercise results in

an overall improvement in running performance through the interaction of central and peripheral factors. Indeed, the addition of a regular training stimulus to skeletal muscle is able to attenuate the negative consequences associated with increasing age.

There are however reports of endurance athletes, competitive for many years, who experience a sudden decline in their running performance. In this sample of ultra marathon athletes, it is unlikely that the reported reduction in performance capacity is attributed to aging alone. The literature shows many examples of skeletal muscle pathology associated with ultra endurance exercise. Studies also show skeletal muscle pathology in trained athletes before an event, suggesting that this may be chronic muscle damage rather than a transient pre-regeneration phase.

It has been questioned as to whether these neural and morphological changes are a functional form of chronic adaptation to long distance running, or whether they represent pathological alterations to skeletal muscle and result in an acceleration of the aging process. Thus, the aim of the first study in this thesis, in accordance with these questions raised from the literature, was to establish whether there was any relationship between the total accumulated distances of racing and training in masters long distance runners and changes in strength and neuromuscular characteristics after a damaging exercise intervention.

## CHAPTER 3

### STUDY ONE

# **CHANGES IN MUSCLE POWER AND NEUROMUSCULAR EFFICIENCY AFTER A 40-MINUTE DOWNHILL RUN IN MASTERS LONG DISTANCE RUNNERS**

### 3.1. INTRODUCTION

The review of the literature has discussed in detail the effects of both age and training on skeletal muscle. Briefly, habitual exercise is associated with increased longevity (450) and serves as a preventative modality for cardiac and other chronic diseases of lifestyle (104). Despite the positive effects of regular exercise, there are anecdotal observations of masters long distance runners with several years of running training and racing experience that have a precipitous decline in running performance which occurs at a faster rate than expected for their age (143;330;426).

Clinical data on long distance runners are also available to support theories of muscle damage and morphological changes as a direct consequence of chronic endurance training. In addition to the studies on skeletal muscle and the effects of exercise, it is also known that the properties of connective tissue change with age and after exercise training (216;551). Thus, another possibility is that the age-related decline in running performance may result from the decreased ability of the locomotor muscles to store elastic energy and convert this energy to mechanical work during muscle contractions (63). Furthermore, the possibility that this loss of elasticity in the muscle is a function of the accumulated stresses of training and racing rather than of age has not been evaluated.

Thus, the aim of this study was to establish whether there was any relationship between the total accumulated distances of racing in masters long distance runners and changes in maximal isometric strength, electromyographic (EMG) activity and the ability of the knee extensor muscles to absorb forces before and after a downhill run.

## **3.2. METHODOLOGY**

### **3.2.1. Subject selection**

Twenty healthy male veteran runners (age 45 - 50 years) with a range of running experience were recruited to participate in this study. Subjects were included if they were currently training more than twice a week and if they were able to complete a 40 minute downhill run. No upper limit was placed on accumulated mileage. Each subject signed an informed consent form at the beginning of the study. The study was approved by the Ethics and Research Committee of the Faculty of Health Sciences, University of Cape Town Medical School.

Subjects were asked to maintain their same physical activity pattern over the course of the study and not to begin any new recreational or training programmes. A full training history, which included information regarding physical activity and running performances over their entire running career,

was obtained from each subject. This history also included the subject's current training distance per week and an estimate of the total distance raced in their running careers.

### **3.2.2. Anthropometry**

During the first visit to the laboratory, mass, stature and an anthropometric assessment was conducted on each subject. Body fat was calculated using the equation of Durnin and Womersley (157) from the sum of seven skinfolds (482). Lean thigh volume for each subject was calculated according to the method adapted from Katch and Katch (284), based on the assumption that the upper lower limb has the shape of a truncated cone.

### **3.2.3. Maximal oxygen consumption ( $VO_2$ max) and peak treadmill running speed (PTRS) test**

Maximal oxygen consumption ( $VO_2$  max) and peak treadmill running speed (PTRS) were determined using a continuous, incremental running protocol on a motor driven treadmill (Quinton Instruments, Seattle, WA, USA).

Subjects began running on a horizontal treadmill at 10 km.hr<sup>-1</sup>. The speed was increased by 0.5 km.hr<sup>-1</sup> every 30 seconds thereafter (493). Oxygen consumption was measured continuously (Oxycon Alpha, Jaeger/Mijnhardt, Groningen, Netherlands). The test continued until the

subject was unable to maintain the pace of the treadmill.  $\text{VO}_2$  max was defined as the highest oxygen consumption recorded during the test over a 30 second period. The PTRS was defined as the fastest running speed the subject could maintain for 30 seconds. Heart rate was recorded every 5 seconds using a Polar Vantage XL heart rate monitor (Polar Electro, Finland).

#### **3.2.4. Drop jump (DJ) assessment**

On their second visit to the laboratory (7 - 10 days later), subjects performed a drop jump (DJ) test to assess the elastic recoil capacity of the quadriceps muscle. Subject's standing height, facing the wall, was measured with their arms fully extended above their head. Subjects were required to jump as high as possible up against a wall, after having jumped off a 50 cm bench. The horizontal displacement of the bench was standardised at 150 cm. The difference between the subject's stretched height recorded while standing and height achieved after the DJ was recorded. Subjects were allowed two practise jumps; the higher of the two DJ heights was recorded.

#### **3.2.5. Maximal isometric knee extensor force testing**

Within five to 10 minutes of the DJ tests, the peak isometric force of the right knee extensor muscles was measured using a Kin-Com isokinetic dynamometer (Chattanooga Group, Inc., Chattanooga, USA). Hand and

arm positions were standardised for each subject. Each subject underwent a standardised warm-up and familiarisation procedure, which included 4 isometric contractions of the knee extensor muscles at 50 % of their subjective maximum, followed by 4 contractions at 70 % maximum. All isometric tests occurred at 60° of knee flexion. An angle of 0° was regarded as full extension. Subjects exerted a maximal voluntary isometric force for 5 seconds, followed by 5 seconds of rest. This work/rest protocol was repeated on 4 occasions. After a one minute rest, subjects performed two 25 second isometric contractions separated by a 10 to 15 second interval. During each isometric test, subjects were instructed to exert maximal force as fast as possible. Peak and average force (Nm) and time to peak force (s) were recorded for each subject.

### **3.2.6. Electromyographic activity**

Prior to the first drop jump, an EMG triode electrode (Thought Technology Triodes MIEP01-00, Montreal, Canada) was attached to each subject's right vastus medialis (VM) muscle. The position was standardised for each subject, with the electrode placed three centimetres proximal to the medial angle of the superior border of the patella. The skin over each site was prepared by shaving off the hair with a disposable razor. After this, the outer layer of epidermal cells was removed using industrial sandpaper. Oil and dirt was removed from the skin using an alcohol swab. Electrodes were secured with self-adherent wrap (Coban 3M, 1582, St. Paul, MN, USA).

Electromyographic (EMG) activity was recorded during the second maximal contraction of each 5 second isometric test. Subjects were told that the EMG would be recorded during this contraction and motivation was then given to each subject. EMG activity was also recorded during the first 25 second isometric contraction and during the drop jump test.

The electrode was linked via fiber-optic cable to the Flexcomp/DSP EMG apparatus (Thought Technology, Montreal, Canada) and host computer. The EMG activity of VM during the drop jumps and isometric contractions were sampled at 1984 Hz, thereby providing raw data. EMG signals from the electrode were band-pass filtered (20 - 500 Hz) and amplified using standard differential amplifiers (Thought Technology, Montreal, Canada); common mode rejection ratio > 130 dB at one kHz, input impedance = one million MegOhms; adjustable gain up to 1600. A 50 Hz line filter was applied to the raw EMG data to prevent any external electrical interference from electrical sources. A toggle switch was activated at the initiation of the drop jump to mark the start point of the test procedure.

The raw EMG signals were full wave rectified to remove movement artefact using a high pass second order Butterworth filter with a cut-off frequency of 15 Hz. The data were then smoothed with a low-pass second-order Butterworth filter with a cut off frequency of 5 Hz. Temporal normalisation of the data was based on the toggle-switch initiation data and by expressing the EMG data as a function of the time period for the entire muscle

contraction. The time period for the isometric data was 25 seconds for the fatigue test, and 5 seconds for the maximal test. EMG activity was sampled for 3 seconds after the initiation of the toggle switch for the drop jump.

### **3.2.7. EMG Data analysis**

Total area under the curve for EMG (mV) vs. time (s) (IEMG) was analysed using GraphPad Prism software (GraphPad Software, Inc., San Diego).

IEMG, maximal and mean EMG values (mV) were calculated for each drop jump, the 5 second isometric test and the 25 second endurance test. The EMG fatigue index was calculated by subtracting the last 5 seconds of the 25 second contraction from the second 5 second period. The second 5 second section was chosen as the initial data point. This was done because of the possibility that there may have been a variation, or a possible lag phase, in the time to peak force output in the first few seconds of the test.

IEMG activity for the 5 second isometric contraction was divided by the average isometric force produced during this contraction at 60°.

Accordingly, IEMG (mV.s)/ torque (Nm) ratios, or neuromuscular efficiency, were calculated for each isometric contraction prior to and immediately after the "eccentric" challenge.

### 3.2.8. Downhill treadmill running test

Within five to 10 minutes following the isometric tests, subjects performed the downhill treadmill running test. Subjects were given the opportunity to warm up and stretch before they began the run. The test started with the subjects running for 3 minutes on a horizontal treadmill at a speed corresponding to 70 % of their peak treadmill running speed (PTRS), determined during the  $\text{VO}_2$  max test. The front of the treadmill was then lowered (-10 %), and the subjects continued to run downhill for 40 minutes at the same speed. Heart rate was recorded every 5 seconds (Polar Vantage XL heart rate monitor), and stride frequency ( $\text{steps}\cdot\text{min}^{-1}$ ) was recorded at 5 minute intervals throughout the run.

### 3.2.9. Post downhill running tests

Within five minutes after the downhill run, peak isometric force and EMG activity was again measured, as described previously. The drop jump, as discussed previously, was performed directly after the isometric tests.

### 3.2.10. Statistical analysis

A paired t-test was used to evaluate the differences between values conducted before and after the 40 minute downhill run. A Pearson's product moment correlation was calculated to determine relationships

between variables. All group data are represented by the mean  $\pm$  standard deviation. Statistical significance was accepted when  $P < 0.05$ .

### 3.3. RESULTS

The general characteristics of the subjects are listed in Table 3.1 and their training and racing histories in Table 3.2. Total training distance was calculated as being  $37\,618 \pm 35\,939$  km, with a minimum accumulated training mileage of 1 300 km and a maximum of 111 280 km (86 fold difference). The total accumulated racing distance was  $3\,411 \pm 3\,484$  km, with a range from 0 km (one subject had never raced) to 9 737 km. Together, the overall running training and racing distance was  $37\,486 \pm 37\,453$  km, ranging from 1 300 to 114 691 km.

Heart rate tended to increase during the downhill run ( $123 \pm 16$  after 5 minutes to  $133 \pm 17$  beats.min<sup>-1</sup> after 35 minutes). There was no significant change in stride frequency throughout the duration of the downhill run ( $170 \pm 13$  strides.min<sup>-1</sup>).

The drop jump height achieved by most subjects generally decreased after the downhill run, but these differences were not significant ( $-0.7 \pm 6.3$  cm).

The mean and maximum EMG activity of the right vastus medialis muscle measured during the 5 second and 25 second isometric contraction and during the drop jump, immediately before and after the downhill run, are listed in Table 3.3.

**TABLE 3.1:** General characteristics of experimental subjects (n=20)

VARIABLE	MEAN $\pm$ SD	RANGE
Age (years)	48.4 $\pm$ 1.7	45.0 – 52.0
Weight (kg)	78.5 $\pm$ 10.9	63.0 – 101.0
Height (cm)	177.7 $\pm$ 6.4	167.0 – 185.5
Lean thigh volume (cc)	3 911 $\pm$ 877	2 600 – 4 917
Sum of skinfolds (mm)	94.0 $\pm$ 32.1	40.8 – 135.5
Body fat (%)	23.4 $\pm$ 4.6	15.0 – 28.6
Lean body mass (kg)	59.8 $\pm$ 6.3	50.8 – 67.5
Maximum heart rate (b.min <sup>-1</sup> )	177 $\pm$ 15	151 – 200
VO <sub>2</sub> max (ml.kg <sup>-1</sup> .min <sup>-1</sup> )	50.8 $\pm$ 7.0	36.7 – 59.7
Peak Treadmill Running Speed (PTRS) (km.h <sup>-1</sup> )	15.9 $\pm$ 1.5	13 – 17.5

**TABLE 3.2:** Training and racing history of the experimental subjects.

VARIABLE	MEAN $\pm$ SD	RANGE
Total years of running	14.4 $\pm$ 11.8 (20)	1.0 – 45.0
Total training distance	37 618 $\pm$ 35 939 (20)	1 300 – 111 280
Current distance per week (km.wk <sup>-1</sup> )	52.3 $\pm$ 29.1 (20)	15.0 – 80.0
Current days of running per week	4.0 $\pm$ 1.6 (20)	2.0 – 7.0
Total years since first marathon	14.4 $\pm$ 9.1 (16)	0.0 – 31.0
Total km raced	3 411 $\pm$ 3 483 (19)	0 – 9 737
Number of 10 km races	33 $\pm$ 53 (19)	0 – 200
Number of 21 km races	36 $\pm$ 52 (19)	0 – 198
Number of standard marathons (42 km)	33 $\pm$ 36 (16)	0 – 100
Number of Ultra marathons (>56 km)	12 $\pm$ 6 (11)	0 – 23
Personal best 10 km time (min)	44.2 $\pm$ 10.6 (15)	32.5 – 65.0
Personal best 42 km time (min)	185.0 $\pm$ 40.0 (16)	110.0 – 270.0
Personal best 90 km (min)	478 $\pm$ 60.3 (10)	417.0 – 606.0

*Number in brackets denotes sample size. Sample size varies depending on whether or not the subject competed in the event.*

**TABLE 3.3:** EMG activity of the vastus medialis (VM) measured before and after the downhill run.

VARIABLE	PRE DOWNHILL RUN	POST DOWNHILL RUN
5 Second isometric IEMG (mV.s)	141 144 ± 93 266	127 313 ± 65 984
Maximum 5 second isometric EMG activity (mV)	294 ± 182	257 ± 128
Drop jump IEMG (mV.s)	81 231 ± 3 604	86 969 ± 39 874
Mean 5 second isometric EMG activity (mV)	178 ± 118	162 ± 82
Maximum drop jump EMG activity (mV)	485 ± 239	451 ± 194
Mean drop jump EMG activity (mV)	142 ± 60	146 ± 67

All values are mean ± standard deviation.

These differences were not significant. IEMG before and after the downhill run varied between subjects (decreased in eight subjects, increased in five subjects, and remained the same in six subjects. Data from one subject were missing). Similar varied responses were seen in isometric force values (Table 3.4) although there was a tendency for mean and peak isometric force to decrease by approximately 15 % in both the 5 second (12 subjects decreased, two subjects increased, and five subjects remained constant. Data in one subject were missing) and 25 second isometric contractions.

**TABLE 3.4:** Isometric data measured before and after the downhill run.

ISOMETRIC TEST	VARIABLE	PRE DOWNHILL RUN	POST DOWNHILL RUN
<b>5 seconds</b>	Peak force (Nm)	<sup>a</sup> 496 ± 158 (19)	430 ± 138 (19)
	Mean force (Nm)	431 ± 139 (19)	366 ± 125 (19)
	Time to peak (s)	2.34 ± 1.14 (19)	2.52 ± 1.62 (19)
<b>25 seconds</b>	Peak force (Nm)	456 ± 182 (18)	<sup>b</sup> 402 ± 170 (16)
	Mean force (Nm)	365 ± 166 (18)	309 ± 151 (16)
	Time to peak (s)	9.36 ± 9.06 (18)	4.8 ± 5.52 (16)

All values are mean ± standard deviation. Number in brackets denotes subject size.

<sup>a</sup> Note that only 19 subject's data were recorded due to equipment failure.

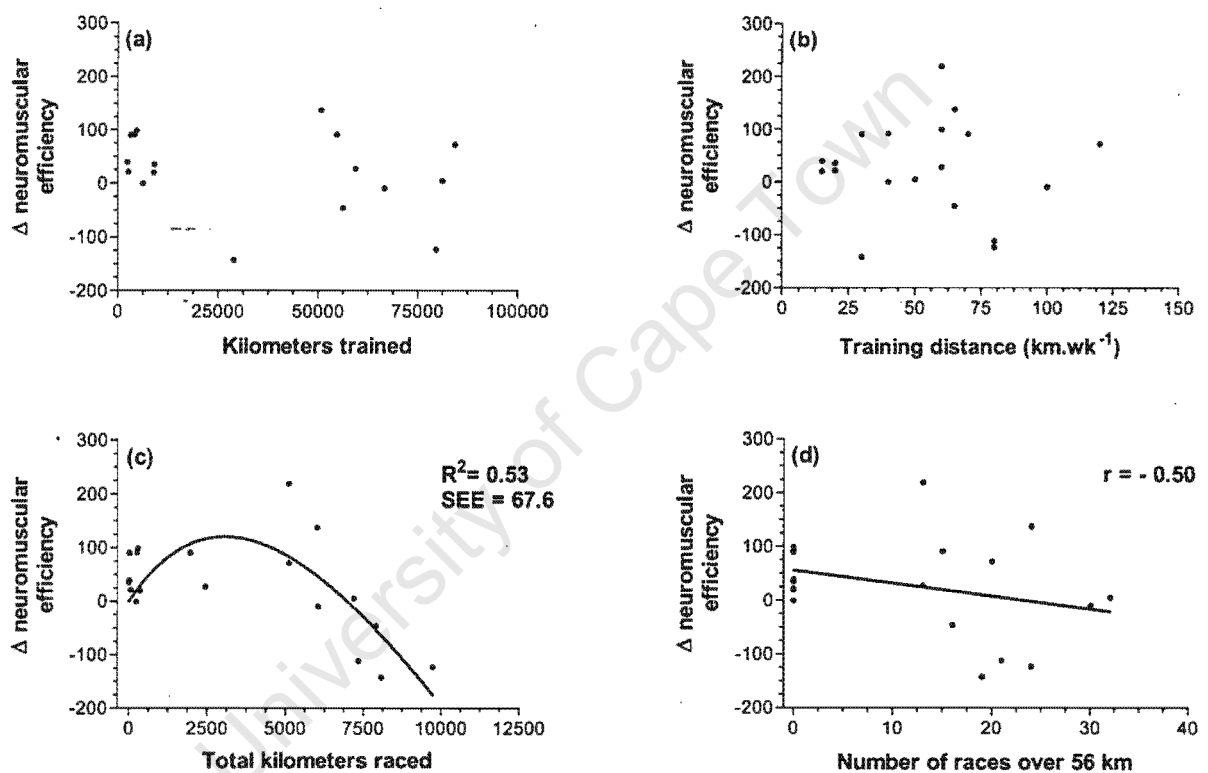
<sup>b</sup> Of the 18 subjects whose data were available after the downhill run, two subjects were unable to complete the second 25 second isometric contraction.

There were no significant relationships between total kilometres raced and the difference before and after the downhill run for heart rate ( $r = -0.06$ ) and drop jump height ( $r = 0.31$ ). In addition, there were no relationships between current weekly training distance and the difference before and after the downhill run for heart rate ( $r = -0.06$ ) and drop jump height ( $r = 0.05$ ).

The ratio between IEMG activity and the average isometric force (5 s) (neuromuscular efficiency) was calculated before and after the downhill run.

Figure 3.1 shows that there was no relationship between the change ( $\Delta$ ) in

neuromuscular efficiency and total kilometres trained (figure 3.1 a), or between  $\Delta$  neuromuscular efficiency and current weekly training distance (figure 3.1 b). However, there was a curvilinear relationship between  $\Delta$  neuromuscular efficiency and total kilometres raced (figure 3.1 c,  $R^2 = 0.53$ ) and an inverse linear relationship between  $\Delta$  neuromuscular efficiency and the number of races over 56 km in distance (figure 3.1 d) ( $r = -0.50$ ,  $p < 0.05$ ).



**Figure 3.1:** Relationship between the  $\Delta$  neuromuscular efficiency and (a) the number of kilometres trained (km), (b) current training distance per week ( $\text{km.wk}^{-1}$ ), (c) total number of kilometres raced (km) and (d) the total number of completed races > 56 km.

### 3.4. DISCUSSION

The important finding from this study was that the prolonged downhill running caused a dissociation in the  $\Delta$  neuromuscular efficiency, particularly in those runners who had accumulated more than 5 000 km in racing (figure 3.1 c). This suggests that these subjects responded differently to the downhill run than those subjects who had raced less than 5 000 km.

Although it is known that the neuromuscular system adapts differently depending on the different levels of fatigue (518), there are no data on the variations in the neuromuscular response in a group of subjects exposed to the same relative amount of lengthening muscle stress, but who differ in the total amount of running they have completed in their lives. Based on the results from this study, it is evident that the subjects responded differently to the downhill run. This was particularly apparent in the subjects who had accumulated more than 5 000 km in racing and was inversely related to the number of races over 50 km (figure 3.1 d). The varied response to the downhill run could not be explained by either total accumulated training volume (figure 3.1 a) or current training distance (figure 3.1 b).

A  $\Delta$  neuromuscular efficiency of zero represents a situation where EMG activity of the muscle and force produced by that muscle change in proportion to one another. Either an increase or decrease in EMG or force, or both can cause a deviation from zero for the  $\Delta$  neuromuscular efficiency. In this study, although there was a general tendency for EMG activity and

peak isometric force to decrease after the downhill run (Tables 3.3 and 3.4), these results were not significant. When the data for each individual subject were examined, EMG activity decreased in eight subjects, increased in five subjects, and remained the same in six subjects after the downhill run. Likewise with the isometric force data, isometric force decreased in 12 subjects, increased in two subjects and remained the same in five subjects before and after the downhill run. There was no consistent pattern in whether the EMG activity and force increased, decreased or remained the same when the race distance increased over 5 000 km. Therefore, the results shown in figure 3.1 c are difficult to explain based on the measurements conducted in this study. Given that the relationship between EMG and force for graded effort is unknown, a dissociation in the EMG/force ratio can be inferred to any deviation in the amount of fibers recruited to produce a given force, or vice versa.

This dissociation in the  $\Delta$  neuromuscular efficiency after the 40 minute downhill run in the subjects with the greater racing distances can possibly be caused by any of six different explanations. These are i) changes in whole limb recruitment patterns, ii) increased muscle stiffness, iii) unavailability of fast twitch muscle fibers for recruitment, iv) changes in motor unit firing rates v) changes in muscle temperature and vi) changes in intramuscular pressure.

Firstly, the quadriceps muscle group consists of 4 muscles - the rectus femoris and the 3 vasti, all of which contribute to force production during knee extension. As a result of continual training and racing, and the accompanying muscle damage (330;558), it is possible that the firing patterns of other associated muscles in the more experienced runners were different. Also, there may be a recruitment of additional motor units, together with an increase in firing frequency in order to maintain the required force (47). These changes would then contribute to a change in  $\Delta$  neuromuscular efficiency after the downhill run. Recruitment patterns of the vastus medialis were the only patterns recorded in this study, and thus, this theory of changes in limb recruitment patterns causing the  $\Delta$  neuromuscular efficiency could not be tested.

The next possible cause of the  $\Delta$  neuromuscular efficiency would be an increase in muscle stiffness. According to Bosco et al (63), the function of elastic tissue is to store elastic energy, and then to convert it into mechanical work. It does this by means of an afferent input to the central nervous system (CNS) (63). With muscle damage, however, there is an increase in muscle stiffness, and therefore an alteration to the visco-elastic properties of the muscle fibers (33). This then would impair the receptor mediated afferent input to the CNS, and thus contribute to the  $\Delta$  neuromuscular efficiency. The data from this study are consistent with the findings of these investigators: the runners in this study who had raced more than 5 000 km, and by implication therefore incurred repeated bouts of

muscle damage, showed a greater dissociation in  $\Delta$  neuromuscular efficiency.

Another explanation for the dissociation in  $\Delta$  neuromuscular efficiency is the unavailability of fast twitch muscle fibers for recruitment. Fast twitch muscle fibers are preferentially recruited to produce maximal force according to Henneman's size principle (35). These are also the first muscle fibers to fatigue and incur damage during prolonged endurance (169) and muscle lengthening activities such as downhill running. Accordingly, the more experienced runners may not be able to recruit their highly fatigable fast twitch fibers to the same extent, due to continual damage and fatigue, as those runners who have raced fewer kilometres. Costill et al (125) found that the muscles of elite distance runners were characterised by both a greater percentage and cross sectional area of slow twitch fibers resulting in a relatively smaller percentage of fast twitch fibers available to produce force. Experienced runners therefore may also have a smaller percentage of fast twitch fibers available for use prior to the downhill run, as a result of chronic adaptations to endurance training. However, one would expect runners who have this ultra structural muscle fiber type composition not to show significant changes following a downhill run.

The dissociation in  $\Delta$  neuromuscular efficiency can also be explained by changes in firing rates by means of "muscle wisdom" or central programming of neural input to the muscle fibers. Enoka and Stuart (167)

described that it was possible to optimise force by means of an economical activation of fatiguing muscle. During fatiguing exercise, there is a significant decrease in firing frequency that occurs via an effective proprioceptive feedback mechanism. This results in a selective recruitment of slow twitch fibers that have a higher resistance to fatigue (380;531). It has been suggested that "muscle wisdom" is directly related to either the available percentage of fast twitch fibers, and exists to protect these fibers from damage as a result of metabolite accumulation (St Clair Gibson et al, unpublished data), or to selectively recruit slow twitch fibers which are more fatigue resistant, thus maintaining force. As a result of fatigue, this property of "muscle wisdom" is applied, resulting in an increase in slow twitch fiber recruitment, and therefore a change in  $\Delta$  neuromuscular efficiency. In addition, if the runners who have accumulated greater distances in their training and racing have a different fiber composition, the muscle wisdom changes in firing frequency may have effected the  $\Delta$  neuromuscular efficiency due to alterations in recruitment patterns. This question could perhaps be answered by a study, which would relate the fiber composition in runners to the  $\Delta$  neuromuscular efficiency after downhill running.

It is unlikely that changes in muscle temperature, which have also been shown to effect muscle recruitment and rate coding (42), would have affected the  $\Delta$  neuromuscular efficiency in this study. Although body temperature may have varied between subjects, environmental conditions

were standardised, thus excluding increasing muscle temperature as a major factor causing the dissociation in  $\Delta$  neuromuscular efficiency.

The last explanation for the dissociation in the  $\Delta$  neuromuscular efficiency was an increase in intramuscular pressure. Muscle damage and delayed onset muscle soreness causes a swelling of the affected muscles (13;108). An increase in intramuscular pressure, similarly to an increase in temperature, has also been shown to alter motor unit recruitment patterns (169;183). In this study, however, measurements were recorded directly following the downhill run before any swelling would have appeared (174). Therefore, it is unlikely that pressure changes are the cause for the findings in this study.

Anecdotal observations show that functional skeletal muscle regeneration occurs after an ultra-endurance race, as runners with severe muscular pain for several days after a race make a seemingly full recovery and are able to race again after an adequate recovery period (426). These observations are supported by studies that show a progressive repair of the mitochondrial and myofibrillar damage three to four weeks after a marathon race (558). After eight to 12 weeks following a marathon there are still signs of muscle regeneration including central nuclei and increased satellite cells in the muscle biopsy samples (558). This raises the question of whether the regeneration process after muscle damage is complete, particularly when the muscle damage is severe as may occur after an ultra-

endurance race. It is tempting to speculate that the muscle regeneration is not complete and that after several bouts of muscle damage and repair, as would occur after racing several ultra marathons, changes occur in the muscle, which alter neuromuscular fatigue. Also, the quality of the regeneration process may vary between subjects. This might explain the findings of Sjöström et al (503) who studied national class runners and found that the overall morphology of the skeletal muscle of the marathon runners varied between subjects. Only one of the five runners in their study had normal muscle structure. The extent to which regeneration varies among different subjects needs to be addressed in further studies.

It needs to be emphasised that the subjects in this study, particularly those subjects who had accumulated many kilometres in training and racing, represent a unique group. Many runners are incapable of training and racing the volumes that are reported by the subjects in this study because of biomechanical factors that predisposes them to injury. Other subjects may also have a vulnerability to develop pathological changes in their muscles (143;330;513), and may, as a result, cease participation in marathon and ultra marathon races after relatively short careers. Therefore, the subjects in this study who had a history of high volume training and racing represent a group of runners who are, by definition, relatively resistant to the stresses of long distance running. Thus, the extent of any damage may, paradoxically be less in these runners than in others who are unable to race frequently without developing a progressive muscle

damage. It should also be noted that none of the subjects who were recruited for this study experienced any sudden decline in their running performance, which again may suggest a unique subject group.

The results in this study also need to be interpreted in the context that the downhill run, although stressful and in all cases severe enough to induce delayed onset muscle soreness in the subjects (158), was not as stressful as the downhill run reported in other studies. For example, in other studies the forces produced in knee extension after a marathon decreased by 26 % (421) and 36 % (495). In the present study there was no significant difference in peak torque after the 40 minute downhill run. It would be interesting to determine how the  $\Delta$  neuromuscular efficiency would change in an experiment in which peak force decreased significantly after the downhill run, and as occurs, for example, after an ultra marathon race. It should also be noted that the runners who participated in this study were also perhaps capable of completing the run with a relatively small motor unit recruitment of the tested muscles.

In summary, it can therefore be concluded that those runners who have raced an accumulated distance of > 5 000 km, show a significant dissociation in the  $\Delta$  neuromuscular efficiency after a 40 minute downhill run when compared to less experienced runners. Further research is needed in order to determine the exact extent of these differences, as well as other possible causes and their meanings.

### 3.5. QUESTION ARISING FROM THE STUDY

The question arising from this study is that if runners, who have been exposed to many years of racing and training, experience altered neural recruitment pattern after a stressful exercise challenge that is associated with muscle damage, would these changes also manifest themselves in alterations in oxygen consumption and therefore differences in running economy when exposed to a similar exercise challenge?

The basis of this argument assumes that additional muscle recruitment would be required in those athletes who have experienced chronic muscle damage associated with long term high mileage training and racing, to maintain a constant work load or running velocity. The additional muscle recruitment would be associated with increased oxygen consumption. This question will be discussed in more detail in Chapter 4.

**CHAPTER 4**

**FACTORS ASSOCIATED WITH  
RUNNING ECONOMY IN MASTERS  
RUNNERS**

University of Cape Town

## **4.1. REVIEW OF THE LITERATURE**

### **4.1.1. INTRODUCTION**

In the previous review, alterations to skeletal muscle associated with age and endurance training were discussed and the influence that these factors have on endurance performance. Running economy, defined as the steady-state oxygen consumption for a given submaximal running velocity (86;275;405), has also been accepted as an important determinant for performance in long distance running (41;67;121;130;131;313;401;405) in athletes with similar maximal oxygen consumption values. According to this relationship, the better runners are the more economical runners (427). This relationship is also influenced by both age and training status.

### **4.1.2. FACTORS ASSOCIATED WITH RUNNING ECONOMY**

Several factors have been associated with the variability in running economy between athletes. These factors will be discussed in the next section under the following headings: (i) endurance training, (ii) age, (iii) stride length and stride length variation, (iv) heart rate and ventilation, (v) muscle fiber type, (vi) body temperature, (vii) muscle power and strength, (viii) lengthening muscle actions and muscle damage, (ix) fatigue, (x)

motor unit recruitment patterns, (xi) muscle elasticity and (xii) muscle stiffness and joint range of motion.

#### **4.1.2.1. Endurance training**

The literature provides equivocal evidence for the effect of endurance training on running economy. A number of studies describe improvements in running economy after training (67;122;129;130;142;402;454;455;464;527-529), while other studies have shown no change (150;392;573) or even a decrease in running economy after regular endurance training (132;326). Indeed, it appears that there is a wide variation in the oxygen cost of running associated with a regular training stimulus (376).

Bransford and Howley (67) and others (400;402;455), concluded that the differences between trained and untrained subjects were mainly due to a repetitive training stimulus which induces an optimisation of motor unit recruitment patterns, improvements in running style and biomechanics, and the development of a more efficient oxidative energy supply.

Similarly, Morgan et al (400) suggested that the differences in running economy between trained and untrained subjects was a function of repeated exposure to a training load, since the best running economy values are often found in more experienced athletes, or those runners who complete the highest weekly training mileages (274;453).

Furthermore, Mayhew et al (376) found a significant inverse relationship between the efficiency of running and years of training.

Lake and Cavanagh (327) measured kinematic variables (stride length, vertical oscillation, trunk lean, knee angle, shank angle, max plantarflexion) and submaximal oxygen consumption, during running, in previously untrained subjects before and after a six week running training programme. They showed that these subjects were unable to demonstrate any improvements in running economy subsequent to endurance training. Although the subjects in this study showed no alterations in biomechanical variables or running mechanics, the change in training status in these athletes appeared to make running economy worse (327). These authors concluded that during the early stages of running training, running style is resistant to change and that improvements in running performance are predominantly as a result of metabolic and physiological adaptations, rather than alterations to running style. In addition, they also concluded that factors leading to improvements in running economy might only occur after a longer duration of exposure to a running training stimulus (327;400).

Indeed, Bailey and Pate (29) have suggested that alterations in training status may cause physiological changes that both positively and negatively affect running economy. For example, improved running biomechanics and intracellular oxidative capacity associated with

endurance training will favourably improve running economy (29), whilst training induced changes in mass distribution may have a negative impact on running economy (102;371). A redistribution of body mass to the extremities, associated with training induced alterations in muscle size, results in a significant increase in submaximal oxygen consumption (409).

The equivocal results reported in the literature may be explained by the varying length of training studies, typically lasting between six to 12 weeks. The duration of these studies may be too short to produce a measurable improvement in running economy, especially in individuals who already train consistently (275). There may also be a certain threshold of training, or a particular type of training necessary for inducing significant alterations in running economy. The level of fitness at the start of the study is an important variable that is often not well controlled and which has the potential to influence whether or not changes in running economy will occur (275).

Alternatively, it may also be possible that exercise training exerts only a minor influence on running economy and that more economical runners may have a biomechanical or anatomical predisposition that would allow for an economical running gait and would favour success in endurance events (67;130). In addition, genetic variables and age-associated changes may also have an effect on running economy (67;130;426).

#### 4.1.2.2. Age

Most of the studies that have associated changes in running economy with age have focussed on changes found in children and adolescents (12;313;314;548). These studies have suggested that differences in leg length, stride length, basal metabolic rate, body surface area to body mass ratio, reduced glycolytic capacity and training and growth related factors between children and adults may account for the observed variability in running economy (128;129;313;314;548).

A retrospective analysis performed by Morgan et al (400) has provided some evidence to suggest that within a limited age span (approximately 20 years), the aerobic demand of running remains relatively constant in active adult distance runners. Similarly, Åstrand et al (27) could not show any changes in oxygen consumption at a given submaximal intensity in physically active subjects after 20 years, confirming their original cross sectional studies (26).

Pate et al (453) measured oxygen consumption during level treadmill running in a heterogeneous group of habitual distance runners, ranging in age from 20 – 60 years. This study found that age was significantly and positively correlated with submaximal running oxygen consumption, suggesting that younger runners are more economical than older runners. These findings have been confirmed by others during cycling, walking and

running (12;497;563) and have been explained by reductions in muscle elasticity together with antagonistic relaxation during running that occurs with increasing age (331).

Differences in running economy between younger and older subjects (313;331;497;548;563) can also be explained by reductions in hip flexibility, shortened stride length, increases in body fat mass and increases in cardiac and respiratory demands, which have all been associated with increased age (331;497;548). These age-associated neuromuscular alterations may result in a reduction in the ability to store and re-use elastic energy, which will reduce running efficiency.

These studies are however limited in their application to runners under the age of 60 years. It would appear that regular exercise training might negate these alterations in flexibility, body composition and aerobic demands that are associated with reductions in running economy in athletes who are younger than 60 years (314). For these reasons, it could be suggested that in competitive masters runners (45 – 60 years), changes in running economy may be attenuated and an economical running gait will be more likely maintained, compared to an age-matched sedentary control group.

#### 4.1.2.3. Stride length and stride length variation

The simple process of shortening or lengthening the running stride has an important effect on active skeletal muscle function as any adjustment would place the muscles on a different region of its specific force-velocity curve (103;412;475). This change would alter the efficiency of the muscle during running and would result in an increase or decrease in the energy demand during running (103;130;405;475).

The process by which an athlete selects a particular running style and gait pattern appears to be subconscious (103), such that at every running speed, an optimal stride length exists for that athlete at which oxygen consumption is minimised (70;103;405;426).

Sjödín and Svendenhag (501) have suggested that long distance running training may result in alterations in running technique, such as small changes in stride length, which minimizes vertical movements and makes running more economical. Indeed, experienced runners are able to obtain a near optimal stride length for maximal efficiency by either altering their stride frequency in an effort to minimize perceived exertion, or by selecting a stride length and stride frequency combination that becomes the most optimal, physiologically, through conditioning or repetition (103). This finding has been supported by Bailey and Messier (28) who trained novice runners for seven weeks and compared changes in

running economy associated with either freely chosen or regulated stride lengths. This study showed that the variability in stride length inherent in novice runners has no relationship with running economy during the initial phases of exercise training. Therefore, any significant changes in stride length that alters running economy might be as a result of several months, if not years, of running training (28).

Morgan et al (405) have hypothesized that at lower stride frequencies, muscles need to develop relatively higher external power to achieve longer stride lengths, while at higher stride frequencies, the mechanical power associated with moving the limbs increases. At these extreme high or low stride frequency-stride length conditions, the reliance on the less economical type II fibers is increased, thereby increasing oxygen consumption and worsening running economy.

These results suggest that running economy may not be altered by small deviations in stride length and frequency, but can be substantially increased if these deviations are considerably different from the optimal combinations specific to each athlete and running velocity (371).

#### **4.1.2.4. Heart rate and ventilation**

Heart rate and ventilation both reflect the efficiency of oxygen supply to the active muscles and have been shown to positively correlate with

oxygen consumption (453;581). Indeed, myocardial and ventilatory work has been shown to account for 1 - 2 % and 7 - 8 % of the overall energy cost of exercise, respectively (295;394) and therefore has a direct affect on running economy (186;567).

Bailey and Pate (29) and others (152;488) have suggested that regular endurance training reduces both heart rate and ventilatory rate during submaximal exercise. Reductions in heart rate are associated with an increase in stroke volume (65;491), decreased intrinsic heart rate, decreased sympathetic tone or decreased circulating catecholamines (323), whilst reductions in ventilation are a result of a decrease in breathing frequency and an increase in tidal volume (29). Together, these alterations may contribute to an overall reduction in total body oxygen consumption.

#### **4.1.2.5. Muscle fiber type**

Contracting type II muscle fibers require more oxygen compared to type I fibers, as a result of a higher rate of cross bridge cycling and ATP consumption (109;315;403;473). In addition, it has been suggested that different muscle fibers have specific visco-elastic properties such that type I fibers are able to retain stored elastic energy for longer without cross bridge detachment (61). This decreases the reliance on energy

generated by oxidative phosphorylation and thereby reduces submaximal oxygen consumption (126).

In addition, Barstow et al (34), Bosco et al (61) and Coyle et al (126) have shown that fiber type distribution significantly affects the characteristics of the oxygen consumption response during exercise. Contrary to these findings, however, are those of Williams and Cavanagh (576) who have been unable to show a relationship between muscle fiber composition and running economy in a group of trained male runners.

#### **4.1.2.6. Body temperature**

A positive association between body temperature and oxygen consumption during prolonged, constant load exercise and submaximal exercise performed under hyperthermic conditions has been described (234;361;426;512). This association is presumably caused by the increased peripheral blood flow, increased ventilatory rate and the reduction in the efficiency of oxidative phosphorylation that occurs during exercise in the heat (361;512).

There are some studies, however that have shown no change, or a reduction in oxygen consumption associated with exercise in the heat (78;199;484), illustrating an enhanced muscular efficiency. Furthermore, training-induced alterations to exercise in the heat, such as increases in

plasma volume may attenuate the thermoregulatory response on running economy (29;489).

#### **4.1.2.7. Muscle power and strength**

Improvements in maximal leg strength, induced through regular resistance training, may improve running economy and endurance performance by reducing the proportion of the maximal force required for each contraction, and thus the recruitment of type II fibers (246). Conversely, strength training induced muscle fiber hypertrophy may negatively affect running economy through the redistribution of body mass (409), as discussed above (section 4.1.2.1., page 109).

Therefore, explosive strength training (444), using sprinting and jumping exercises combined with weight training using low loads and high to maximal velocities, may be a more effective means to improve running economy. Paavolainen et al (444) concluded that this specific type of training might induce alterations in motor control that would enable a muscle to resist the imposed stretch more efficiently, and as a result increase the accumulation of elastic energy by the entire muscle-tendon complex.

#### 4.1.2.8. Lengthening muscle actions and muscle damage

The observation of increases in oxygen consumption during submaximal exercise, using predominantly lengthening muscle actions, has received little attention. The energy cost of lengthening muscle activation is substantially less than that of shortening contractions (165;299;300;458;462;565) as fewer muscle fibers are recruited to perform the same work (49;165). Klausen and Knuttgen (296) were amongst the first authors to illustrate an "oxygen drift", or a gradual rise in oxygen consumption during active lengthening exercise, by showing a 25 % increase in mean oxygen consumption during 25 – 50 minutes of "eccentric" cycling. More recently, gradual increases in oxygen consumption of up to 40 % have been shown during downhill walking (133) and running (84;492;567).

Since oxygen consumption during running reflects the activity of the working muscle, it is likely that the cause of an increased oxygen consumption during muscle lengthening and prolonged endurance activities lies at the level of the exercising muscle (145). Dick and Cavanagh (145) measured oxygen consumption, EMG activity from the vastus lateralis and vastus medialis muscle groups, and stride length from experienced runners during a 40-minute downhill run. Their study showed significant increases in oxygen consumption and EMG activity after 20 minutes of downhill running without any significant changes in stride length (145). These results have been confirmed more recently by Perrey

et al (458) who concluded that the progressive increases in EMG activity for the rectus femoris and vastus lateralis muscles contributed to the increase in oxygen consumption during high intensity lengthening muscle exercise.

Based on the results of these studies it is proposed that an increase in oxygen consumption during downhill running, which is comprised almost exclusively of lengthening muscle activity, may be related to muscle fiber and connective tissue damage and/or local muscle fatigue (69;87;145; 324;458;458). As a consequence of this muscle damage and fatigue, the active skeletal muscle fibers are unable to generate sufficient force and as a result, additional motor units must be recruited to maintain a given level of work (145). This suggestion is supported by the 10 % increase in oxygen consumption that occurs simultaneously with an increase in EMG activity from the vastus lateralis and vastus medialis in the study of Dick and Cavanagh (145). The damaged fibers, however, would continue to use oxygen, in addition to the newly recruited fibers, which would contribute to an increase in submaximal oxygen consumption, and thereby would result in a reduction in running economy (145).

Westerlind et al (567) tested this hypothesis by including a second downhill running bout, separated by one week, into their protocol. They proposed that, based on the repeat bout effect, muscle damage induced during the second run would be significantly less and thus they would observe

fewer alterations in oxygen consumption. However, the results of this study showed a similar increase in submaximal oxygen consumption during the second downhill run. These authors (567) suggested that other factors, in addition to muscle damage, may be contributing to the changes in oxygen consumption during downhill running, such as increases in heart rate, expired ventilation, increases in rectal temperature and fatigue.

This suggestion has been confirmed by Gleeson et al (206) who showed higher heart rates, minute volumes, breathing frequencies and ratings of perceived exertion during submaximal cycling exercise, two days following a damaging exercise bout. These authors (206) concluded that submaximal exercise performed by subjects experiencing the associated symptoms of muscle damage, produces physiological responses that are indicative of a higher relative exercise stress. Accordingly, it is likely that such effects will significantly limit the level and duration of exercise that can be achieved in subsequent exercise training bouts and thus may contribute substantially to the sensation of fatigue (206).

#### **4.1.2.9. Fatigue**

The literature suggests that running economy deteriorates during prolonged duration running, and that the magnitude of this deterioration increases with both increasing exercise intensity and duration (101;232; 233; 512;579;581). Both central and peripheral factors have been

proposed to explain the gradual increase in submaximal oxygen consumption observed during prolonged treadmill running (135). These include an increased energy expenditure associated with dissipation of the heat generated during exercise (230;512), increases in blood catecholamine and growth hormone concentrations (280), increases in pulmonary ventilation (29), increases in fat metabolism and associated metabolic consequences (581), increases in heart rate (512;567;581), or as a consequence of skeletal muscle weakness and damage reflecting an increase in muscle fiber recruitment (69;87;135;145;324;458;581).

Morgan et al (404) investigated the effects of a fatiguing (30 minutes) maximal run (89 % of  $\text{VO}_2$  max) on running economy and running mechanics in 16 male runners. With the exception of the plantar flexion angle at the toe phase during the gait cycle, biomechanical analyses revealed little variation in 21 other temporal, kinematic and kinetic gait descriptors, previously linked to variation in running economy (576). Similarly, on days 1, 2 and 4 after the maximal run, there were no alterations in running economy, suggesting that there were no lasting effects of fatigue from the high intensity protocol on submaximal oxygen consumption (404). The conclusion from this study was that 30 minutes of high intensity running, equivalent to a 10 km road race, which resulted in some evidence of fatigue, does not alter running mechanics sufficiently to result in an increase in submaximal running economy. The finding that

fatigue does not affect running economy has been confirmed in other studies (71;153).

In contrast, Xu and Montgomery (581) have shown that a 90 minute treadmill run, performed at either 65 or 80 % of  $\text{VO}_2$  max, alters running economy and results in a significant increase in oxygen cost. Similarly, Calbet et al (87) measured running economy two and seven days following a competitive duathlon event. In their study (87), running economy was significantly impaired for two days following the event, but had returned to pre-race values one week later. Davies and Thompson (135) have suggested that the gradual increase in submaximal oxygen consumption might be related to a decline in force associated with fatigue. A decrease in muscle strength will lead to an increase in the recruitment of fibers to maintain a constant running velocity and thereby increase oxygen consumption (581).

Additional studies investigating the relationship between running kinematics and fatigue during middle- (162) and long-distance running (233;420) have shown that there is a reduction in stride length with a corresponding increase in stride frequency which occurs with the onset of fatigue in order to maintain a constant running velocity. Hauswirth et al (233) have shown that significant alterations in running kinematics at the end of a marathon run contributes to a decrease in running efficiency in well-trained triathletes. Specifically, these authors showed increases in the

extension of the knee at foot strike and in the maximal knee angle in the swing phase together with significant reductions in stride length. These authors, however, were unable to identify one isolated kinematic variable associated with changes in running economy following a marathon. Accordingly, it was suggested that the decrease in running efficiency measured during and after long duration running may be linked to alterations of many different biomechanical and physiological parameters, rather than to just one (233).

Kyröläinen et al (323) have shown significant reductions in maximal isometric force and in the maximal rate of force production of the plantar flexors and knee extensors following a marathon, which were associated with significant increases in submaximal oxygen consumption, ventilation and heart rate. Similarly, Davies and Thompson (135) have shown gradual increases in oxygen consumption in ultra marathon athletes during a high intensity, four hour run on a treadmill. In this study, the increase in oxygen consumption became more significant after 110 minutes of running. These authors concluded that the rise in oxygen consumption could be explained by a reduction in the contractility of the active muscle fibers. Indeed, Petrofsky (459) has shown that during cycling at workloads between 20 to 100 % of  $\text{VO}_2$  max, the number of active muscle fibres is directly proportional to oxygen consumption during submaximal exercise.

Accordingly, these studies (87;135;323;420) suggest that in overcoming the reduction in muscle function caused by either muscle damage or fatigue, more fibres are recruited to produce the required force or to maintain a constant running velocity. Thus, due to the relationship between EMG activity and oxygen uptake (48), it is likely that the altered function of the neuromuscular system associated with fatigue will also influence running economy. Indeed, it has been shown that at the end of a marathon, greater EMG activity to the muscle is required to produce the same resultant force during the push off phase (323;420;422).

#### **4.1.2.10. Motor unit recruitment patterns**

There have been few studies characterizing the relationship between motor recruitment patterns and running economy. Heise et al (243) and Kyröläinen et al (319) have identified that alterations to muscle activation are a major contributing factor to superior running economy and that there are significant differences in muscle recruitment patterns between economical and less economical runners (243). Specifically, economical runners have an earlier onset of rectus femoris activation during the swing phase, which remains active for a longer duration during each phase of the running cycle, compared to less economical runners (243;319). Heise et al (243) speculate that the more economical runners may rely more extensively on this efficient bi-articular muscle to contribute to the dual

function of hip flexion and knee extension, rather than the mono-articular hip flexors.

In addition, this study also showed that more economical runners have a shorter hamstring-gastrocnemius coactivation period during the swing phase, compared to less economical runners (243). Muscle coactivation has been directly related to joint stiffness (255) and as such, economical runners may use this to their advantage. Accordingly, economical runners may co-ordinate the activity of bi-articular muscles so that their combined coactivation during running will simultaneously increase joint stiffness in more than one joint, thereby requiring less metabolic energy to produce the same work (243). It has also been suggested that the optimisation of motor unit recruitment patterns may be as a consequence of long-term endurance training (403).

An increased muscle and joint stiffness during the stance phase may translate to a greater elastic energy return from both muscle and tendon, which contributes to effective propulsion during running.

#### **4.1.2.11. Muscle elasticity**

Bosco et al (61) have suggested that the differences in running economy between individuals may be caused by differences in the ability to store and re-use elastic energy. The improvement in muscular performance

following an initial lengthening muscle prestretch has been attributed to this storage and re-use of elastic energy (61), which is delivered, free of energetic cost from the recoil of previously stretched elastic elements (61). Thus, the recoil of elastic energy can be assumed to contribute markedly to running economy (61;102).

The elastic characteristics of the muscle-tendon units are of particular importance in determining running economy (532). Taylor (532) has suggested that the speeds and stride frequencies selected by both humans and animals are those where the storage and subsequent recovery of elastic energy are maximised. Williams and Cavanagh (576) have shown that the least economical athletes also show significantly less mechanical energy transfer between the legs and the trunk during running, compared to more economical athletes.

#### **4.1.2.12. Muscle stiffness and joint range of motion**

Muscle range of movement and flexibility may also play a role in affecting muscle elasticity and running economy. It has been suggested that elastic energy storage and return may be improved with a stiffer, or a less flexible musculotendinous system (18;207). Specifically, Gleim et al (207) found that higher "nonpathological musculoskeletal tightness", based on 11 measures of trunk and lower limb flexibility, was related to lower aerobic demands during submaximal walking and jogging. These authors

concluded that in subjects who are less flexible, elastic energy contributions to running economy may be enhanced and the need for neutralization of unproductive movements by active muscle reduced. A combination of these factors lowers the metabolic cost of producing movement.

#### **4.1.2.13. Other factors associated with running economy**

Intra-individual variation in running economy reported in the literature ranges from 3 % to 11 % (397;402;405). However, Morgan et al (577) has suggested that when treadmill running experience, footwear, time of day and current training status are controlled during testing protocols, stable running economy values can be obtained in trained runners.

Knowledge of the daily stability of running economy is essential if the efficacy of a testing protocol is to be reliably determined. Recently, Pereira and Freedson (457) showed that in highly trained distance runners, with adequate control of extrinsic factors, within subject variability did not differ across fitness levels. These authors therefore concluded that biological variation could account for approximately 94 % of the intra-individual variation observed in running economy.

Other factors that may have an effect on running economy include training specificity (272), gender (67;405), shoe weight (102;426), clothing

(131;426), wind and air resistance (131;426), foot terrain (102;131), circadian fluctuation (249;403) and psychological state (86;127) and therefore need to be controlled for comparative studies.

#### **4.1.3. SUMMARY OF THE LITERATURE**

Differences in running economy have been associated with anthropometric, physiological, metabolic and biomechanical factors but, as yet, no single isolated biomechanical or kinematic variable has been identified to explain differences in running economy between subjects and between research trials.

Alterations in running economy associated with endurance training may result from changes in muscle oxidative capacity and associated changes in motor unit recruitment patterns, exercise ventilation and heart rate and alterations in performance technique, which together may attenuate reductions in running economy that have been associated with age.

Whilst muscle damage, associated with exercise with predominantly lengthening muscle actions and fatigue, results in a gradual increase in oxygen consumption during submaximal exercise as additional motor units are recruited, there is evidence to suggest that running economy is

related to muscle elasticity and stiffness, and that "stiffer" muscle and tendons are better able to store elastic energy during the lengthening phase of stretch shortening activities than more compliant musculotendinous structures.

The question remains, however, as to whether there may be more significant changes in running mechanics and running economy after more prolonged running. Little is known regarding the accumulative effect of prolonged periods of rigorous training and frequent competitive distance racing on running economy. The previous study (Chapter 3) showed that runners who have accumulated high training and racing mileage use a different neural recruitment strategy to maintain maximal muscle force. It could be suggested that because of this alteration in muscle recruitment, there may also be alterations in running economy in these athletes.

Accordingly, the aim of the second study was to determine whether there was a relationship between exposure to high volume running training and racing and alterations in running economy.

**CHAPTER 5**

**STUDY TWO**

**CHANGES IN OXYGEN  
CONSUMPTION DURING AND AFTER A  
DOWNHILL RUN IN MASTERS LONG  
DISTANCE RUNNERS**

## 5.1. INTRODUCTION

As discussed in Chapter 3, experienced masters athletes with an accumulated racing distance of > 5 000 km showed a dissociation in the relationship between muscle recruitment and resultant force output after running downhill. The mechanism underlying this response to "eccentric" loading of the lower limb muscles, and its physiological significance, is unknown. It was suggested that these changes may be related to lower limb muscle pathology caused by excessive training and racing or changes in neuromuscular activity. One manifestation of these findings may be a change in oxygen consumption after downhill running. This is based on the premise that running economy, measured as oxygen consumption at a standardised submaximal running speed, is a surrogate predictor of successful distance running performance (131) in athletes displaying similar maximum oxygen consumption ( $\text{VO}_2 \text{ max}$ ) values (121; 130;405) such that the better athletes are usually the most economical runners (427). Despite large intra- (103) and inter- (121;130;405;576) individual variation, running economy may serve as a reliable performance criterion in endurance trained athletes (186).

Should the basis of this argument be correct, there might be an association between the cumulative training and racing load and an increase in submaximal oxygen consumption, particularly after a bout of downhill running which imposes an "eccentric" stress on the muscle.

Accordingly, the aim of this study was to determine whether oxygen consumption increases in proportion to the years of accumulated training and racing in masters runners after exposure to a short bout of downhill running. It was postulated that the muscles of masters athletes with greater training and racing distances would be more susceptible to alterations in muscle function during downhill running. As a result, these athletes would show a greater impairment in running economy when tested after a bout of lengthening muscle loading, induced by downhill running, compared to less experienced runners.

## **5.2. METHODOLOGY**

A similar group of athletes to those recruited for the first study (section 3.2.1., page 85) was selected to participate in this study ( $n = 17$ ). Subjects visited the laboratory on two occasions separated by no more than seven days. On both occasions, the time of day was standardised, and the subjects, who were all familiar with running on a treadmill, wore the same shoes for both testing sessions. During the first visit to the laboratory, subjects underwent an anthropometrical assessment, interview and maximal testing as described in Chapter 3 (section 3.2., page 85) and on their second visit they performed a submaximal oxygen consumption test. The study was approved by the Ethics and Research Committee of the Faculty of Health Sciences, University of Cape Town.

### 5.2.1. Test of submaximal oxygen consumption

Subjects performed a brief warm up, consisting of a 5 minute submaximal run on a horizontal treadmill at a self-chosen running pace. Following this warm up, subjects were able to stretch for a further 5 minutes before they began the test, which was designed to impose a varying lengthening and shortening load to the knee extensor muscles. Subjects were required to run continuously at a speed corresponding to 70 % of their peak treadmill running speed for 40 minutes. Subjects ran for 10 minutes at a 0 % gradient (horizontal). Thereafter, the treadmill gradient was decreased to -10 % while the subject continued to run at the same speed. This gradient was maintained for 20 minutes, after which time, the gradient was again raised to 0 % and the subject continued running for a further 10 minutes. This protocol was based on that used by Dick and Cavanagh (145) who showed that an upward drift of oxygen consumption occurred after running downhill (-10 %) for 15 minutes. Oxygen consumption was measured continuously during the run (Oxycon Alpha, Jaeger/Mijnhardt, Groningen, Netherlands), and heart rate was recorded every 5 seconds (Polar Electro, Finland). Stride frequency (steps.min<sup>-1</sup>) was also recorded at 5 minute intervals throughout the test, and stride length (SL) was calculated as  $SL (m) = \text{speed (m.min}^{-1}) / \text{stride rate (steps.min}^{-1})$ .

### 5.2.2. Data analysis

The heart rate and oxygen consumption data were analysed similarly. Absolute values were obtained at 5 minute intervals by averaging the values for 30 seconds before and after the actual time point. This was done at minutes 5, 10, 15, 20, 25, 30 and 35. For minute 0, values from the start of the test to minute one were used, and for minute 40, the final minute values of the test were used. Mean data for each 5 minute interval were obtained by averaging all values collected for 5 minute segments during the run. This was done for minutes 0 - 5, 5 - 10, 10 - 15, 15 - 20, 20 - 25, 25 - 30, 30 - 35 and 35 - 40.

Absolute values for each time point were compared between subjects and then compared as relative values as a percentage of either maximal oxygen consumption ( $\text{VO}_2 \text{ max}$ ) or maximal heart rate (HR max). This was repeated for each 5 minute mean value.

Data were then analysed to determine whether there were any correlations between the data collected at minute 10 (end of the first horizontal section) and the subsequent data collection points (minutes 11, 30, 31 and 40). These points were selected as they coincided with (i) the first minute of the downhill run (minute 11), (ii) the end of the downhill run (minute 30), (iii) the first minute of the second horizontal run (minute 31) and (iv) the end of the second horizontal run (minute 40). These

differences were referred to as the delta ( $\Delta$ ) heart rate or the  $\Delta$  oxygen consumption. These delta values were then related to current training mileage ( $\text{km}\cdot\text{wk}^{-1}$ ), total accumulated racing distance (km) and total accumulated training distance (km). The relationships between these variables were calculated to determine if these factors were confounding variables and were thereby influencing the results.

### **5.2.3. Statistical analysis**

All values are expressed as the mean  $\pm$  standard deviation ( $X \pm \text{SD}$ ). Data were analysed with a one-way analysis of variance with repeated measures test, with time as the main effect. A Scheffe's test was used for the post-hoc analysis. The relationship between variables was determined using a Pearson's Product moment correlation. A multiple regression analysis was then performed to determine which factors in combination best predicted the changes in oxygen consumption. Statistical significance was accepted when  $P < 0.05$ .

## **5.3. RESULTS**

The general characteristics of the subjects are listed in Table 5.1. These results suggest that this was a relatively homogenous group of runners with respect to age, maximal heart rate, peak treadmill running speed and

maximal oxygen consumption. This group of subjects was similar to those in the previous study (Chapter 3) based on their general characteristics and previous training and racing history.

Training and racing histories are listed in Table 5.2. The range of total training distance varied from 3 536 km to 79 320 km. The total accumulated racing distance was  $5\,462 \pm 4\,221$  km, with a range from 205 km to 12 218 km. Nine subjects had raced accumulated distances of greater than 5 000 km. Together, the overall training and racing distance was  $41\,056 \pm 28\,883$  km, ranging from 4 055 to 91 285 km. The number of standard and ultra marathons completed by the subjects and their personal best times (minutes) for 10 km, 42 km and 90 km are also shown in Table 5.2.

Figure 5.1 shows absolute heart rate (figure 5.1 a) and oxygen consumption (figure 5.1 b) values measured at 5 minute intervals throughout the 40 minute run. Minutes 0, 5 and 10 were recorded during the first horizontal run, while minutes 11, 20, 25 and 30 were measured during the downhill run. Similarly, heart rate and oxygen consumption were also measured at minutes 31, 35 and 40 during the second horizontal run. Two subjects were unable to complete the last 5 minutes of the run, and thus data from these subjects are not available for minute 40.

**TABLE 5.1:** General characteristics of experimental subjects (n=17).

<b>VARIABLE</b>	<b>MEAN ± SD</b>	<b>RANGE</b>
Age (years)	50.6 ± 4.5	41 – 60
Weight (kg)	78.7 ± 13.4	65.0 – 120.0
Height (cm)	178.5 ± 5.9	168.0 – 189.2
Lean thigh volume (cc)	4 000 ± 588	3 147 – 5 506
Sum of skinfolds (mm)	79.3 ± 25.2	46.6 – 142.0
Body fat (%)	21.0 ± 3.8	14.3 – 29.0
Lean body mass (kg)	61.9 ± 8.4	52.1 – 87.3
Maximum heart rate (b.min <sup>-1</sup> )	173 ± 9	156 – 194
VO <sub>2</sub> max (ml.kg <sup>-1</sup> .min <sup>-1</sup> )	51.2 ± 6.9	35.0 – 59.0
Peak Treadmill Running Speed (PTRS) (km.h <sup>-1</sup> )	16.3 ± 1.8	12.0 – 19.0

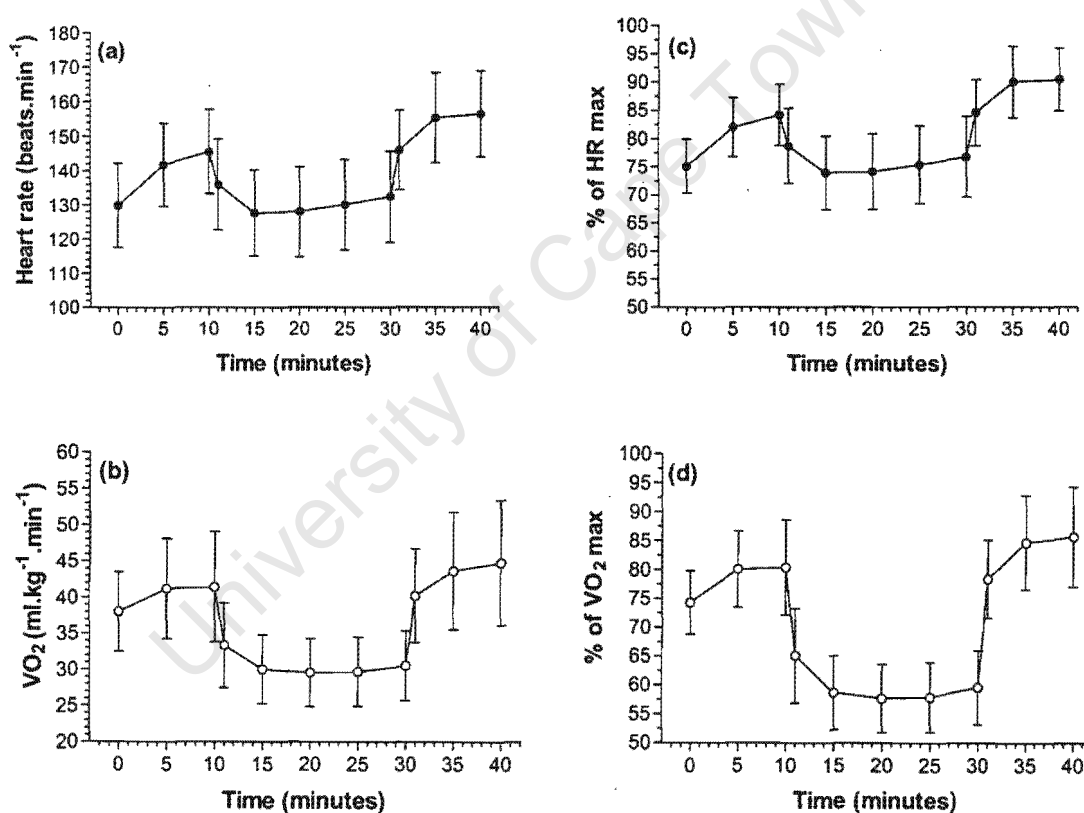
**TABLE 5.2:** Training and racing history of the experimental subjects.

<b>VARIABLE</b>	<b>MEAN ± SD</b>	<b>RANGE</b>
Total years of running	15.4 ± 9.9 (17)	1.0 – 30.0
Total training distance	35 593 ± 25 565 (17)	3 536 – 79 320
Current distance per week (km.wk <sup>-1</sup> )	56.5 ± 19.1 (17)	25.0 – 100.0
Total years since first marathon	14.3 ± 8.6 (15)	0.0 – 29.0
Total km raced	5 463 ± 4 221 (17)	205 – 12 218
Number of standard marathons (42 km)	41 ± 32 (15)	0 – 100
Number of Ultra marathons (>56 km)	9 ± 6 (10)	0 – 12
Personal best 10 km time (min)	42.02 ± 7.40 (15)	37.00 – 59.00
Personal best 42 km time (min)	195.4 ± 32.4 (15)	152.0 – 246.0
Personal best 90 km (min)	497.4 ± 91.6 (10)	364.0 – 642.0

*Number in brackets denotes sample size. Sample size varies depending on whether or not the subject competed in the event.*

There was a significant difference ( $P < 0.001$ ) in heart rate over time during the 40 minute run, with a decrease of  $18 \pm 5$  beats in the first 5 minutes of running downhill (minute 10 to minute 15) and an increase of  $23 \pm 5$  beats

when returning to horizontal running (minute 30 to minute 35). Similarly, oxygen consumption also changed significantly ( $P < 0.001$ ) over time with a mean decrease of  $11.4 \pm 3.8 \text{ ml.kg}^{-1}.\text{min}^{-1}$  during the first 5 minutes of running downhill (minute 10 to minute 15), and an increase of  $13.1 \pm 4.6 \text{ ml.kg}^{-1}.\text{min}^{-1}$  after 5 minutes of returning to the horizontal (minute 30 to minute 35). There were similar changes when the data were expressed as relative percentages of HR max (figure 5.1 c) and  $\text{VO}_2$  max (figure 5.1 d) ( $P < 0.0001$ ).

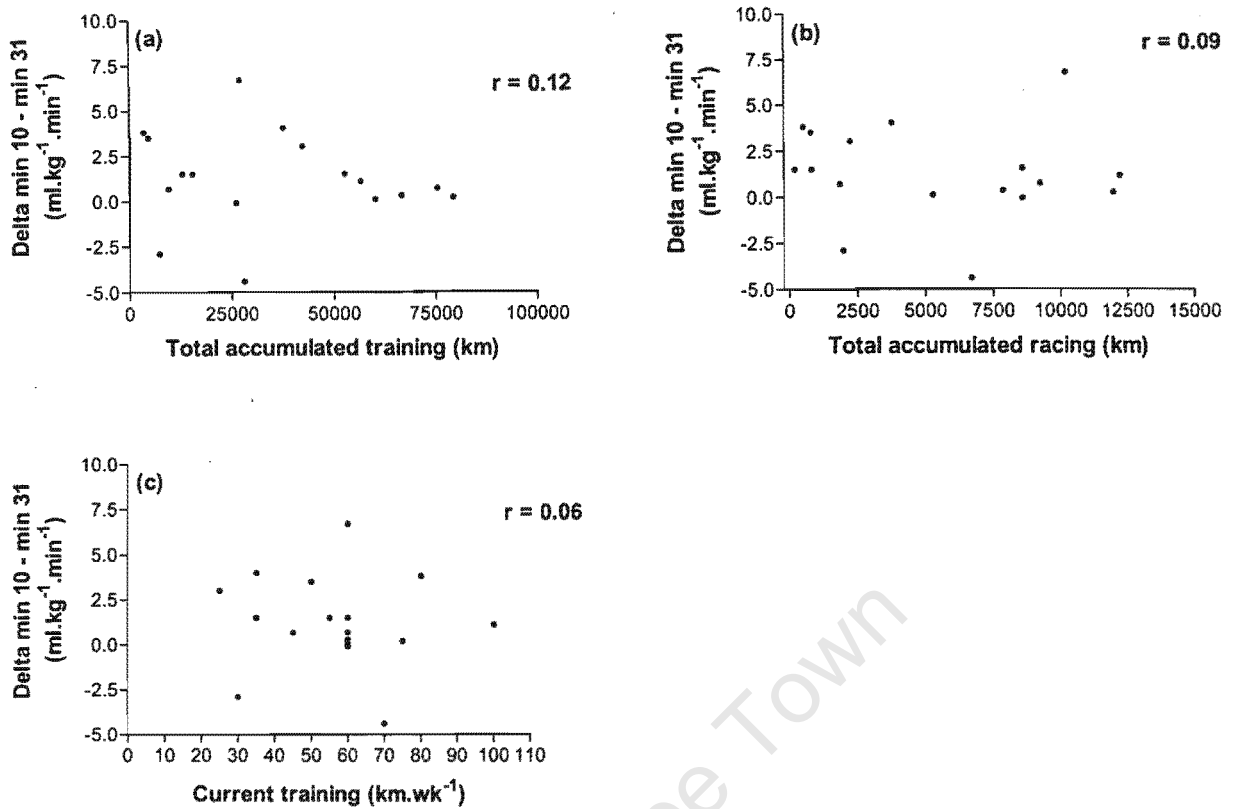


**Figure 5.1:** Changes in absolute (a) heart rate and (b) oxygen consumption during the 40 minute run and changes in (c) heart rate and (d) oxygen consumption as a percentage of HR max and  $\text{VO}_2$  max respectively.

Note: Two subjects were unable to complete the last 5 minutes of the run, thus data from these subjects were not available for minute 40.

Average stride length during the first horizontal run (minutes 0 - 10) was  $1.06 \pm 0.10$  m. During the first 10 minutes of the downhill run (minutes 10 - 20), mean stride length increased significantly ( $P < 0.0001$ ) to  $1.13 \pm 0.11$  m. Stride length then decreased during the second 10 minutes of the downhill (minutes 20 - 30) to  $1.12 \pm 0.11$  m ( $P < 0.05$ ). During the second horizontal run (minutes 30 - 40), average stride length was  $1.07 \pm 0.10$  m, significantly longer than during the first horizontal run ( $P < 0.01$ ).

Correlation analysis revealed that there were no significant relationships between the change in oxygen consumption between minute 10 and minute 31 (delta minute 10 – minute 31) and total accumulated training mileage (figure 5.2 a), total accumulated racing mileage (figure 5.2 b) and current training (figure 5.2 c). Furthermore, there were no significant correlations between changes in stride length and changes in oxygen consumption. Results of the multiple regression analysis suggested that current training was not a confounding variable and that this variable had no effect on the results of the correlation analyses between changes in oxygen consumption and accumulated training and racing mileage.



**Figure 5.2:** Relationship between the delta of minute 10 and minute 31 (the end of the first horizontal section and the beginning of the second horizontal section) and (a) total accumulated training kilometres, (b) total accumulated racing kilometres and (c) current training (km.week<sup>-1</sup>).

#### 5.4. DISCUSSION

The main finding of this study was that oxygen consumption was similar before and after 20 minutes of downhill running in a group of masters runners with a range of training and racing experiences (figure 5.1). This finding suggests that although oxygen consumption decreased as expected during the downhill phase of the protocol, there was no lasting

effect of this "eccentric" challenge on oxygen consumption upon resumption of level running. This finding is difficult to explain based on the previous study (Chapter 3) in which a dissociation in the EMG/force ratio of the knee extensors after a downhill run, in a group of masters runners who had raced more than 5 000 km was found. It was suggested that this altered neuromuscular efficiency of the knee extensor muscles would manifest as a change in oxygen consumption after the downhill run, based on assumptions made from previous studies discussed in the literature review.

Although there was a significant increase in stride length after the downhill run, there was no significant relationship between this change and any changes in oxygen consumption. It has previously been shown that any deviation from an optimal stride length results in an increase in energy demand during running (103). In this study, all subjects ran at a constant speed corresponding to 70 % of their peak treadmill running speed. Therefore, the possibility of changes in stride length being a major factor that would influence oxygen consumption in this study was excluded, as it was assumed that each subject would adopt their optimal stride length for their specific speed (103).

During downhill running, oxygen consumption decreases initially and then gradually increases (84;145;566;567). This may be because the lengthening role of the muscles acting against gravity (i.e. knee extensor

muscles: muscles of the anterior and posterior tibial compartments and hip extensors) is emphasized (172). The reasons why oxygen consumption did not change before and after the downhill run can be explained by the fact that the downhill protocol did not impose a sufficiently severe lengthening stress to disturb muscle function. This contrasts with the finding of Dick and Cavanagh (145) who showed that a similar protocol induced an upward drift in oxygen consumption after 15 minutes of downhill running in experienced male runners.

A different interpretation of the data might be that the subjects altered their muscle recruitment patterns after the downhill running, which compensated for any changes in neuromuscular efficiency of the knee extensor muscles that may have occurred. Changes in whole limb muscle recruitment patterns after downhill running have previously been reported (145). It is possible that alternative muscle groups were selectively recruited and rotated (390;568) during both the downhill phase of the protocol as well as during the second horizontal run in an attempt to maintain the required force needed to continue running at the same speed. If the total muscle mass recruited during the run had remained the same, there would be no differences in oxygen consumption before and after the downhill challenge.

Muscle stiffness is another factor that may have affected the changes in oxygen consumption before and after the downhill phase of the protocol

by regulating the storage of elastic energy before releasing it as mechanical work. It follows that muscles with increased stiffness have a reduced capacity to convert elastic energy into mechanical work and would therefore cause an increase in oxygen consumption. Barry and Cole (33) have shown an association between accumulated muscle damage and increased muscle stiffness. Accordingly, it could be suggested from this study that differences in muscle stiffness in athletes with varying degrees of muscle damage had no lasting effect on oxygen consumption after the downhill phase of the protocol. However, this conclusion would be based only on the assumption that muscle stiffness varied in these subjects, as this component was not measured in this study.

In conclusion, these data show that a short bout of downhill running does not cause a change in oxygen consumption which can discriminate between runners with varying amounts of training and racing. This suggests either that the downhill protocol did not impose a sufficient "eccentric" stress for the subjects, or that submaximal oxygen consumption is not a sensitive marker of changes in neuromuscular activity.

## 5.5. QUESTION ARISING FROM THE STUDY

It is interesting that in this sample of runners, there was no relationship between oxygen consumption during submaximal running and exposure to high mileage training and racing, yet in a similar group of athletes, there were significant neuromuscular alterations that occurred during maximal force production after an exercise that caused muscle damage. These findings suggest that despite possible neuromuscular alterations associated with ultra endurance racing and training, there may be a compensatory mechanism present which enables experienced athletes to continue to perform at a similar aerobic cost to runners who have not been exposed to a high volume of training and racing. Indeed, this suggestion has been confirmed by Paavolainen et al (444;446), who demonstrated significant neuromuscular alterations associated with explosive strength training, but did not show any alterations in running economy.

Should this be the case, a more sensitive indicator of neuromuscular activity during running would be required to identify possible differences and inconsistencies in muscle recruitment patterns between athletes exposed to differing amounts of racing and training. This would answer the question of whether experienced runners have a compensatory neural mechanism enabling them to run at the same running economy as less experienced runners.

Recently, Paavolainen et al (447;448) identified the use of measuring muscle preactivation as a reliable and sensitive technique to accurately assess muscle elasticity and stiffness. Preactivation of a muscle is calculated from the EMG activity 100 milliseconds before heel strike and has been shown to play a fundamental role in providing adequate shock absorption capabilities during running and contributing to an efficient running gait by increasing muscle stiffness and improving the return of stored elastic energy (302). Alterations to the level of muscle preactivation would thus have implications not only to running performance, but also to running economy by increasing the amount of elastic energy return.

These areas of neuromuscular characteristics, preactivation and muscle fatigue during running will be discussed in detail in Chapter 6. In particular, this discussion will provide background for the development of a research model designed to measure neuromuscular compensatory mechanisms during running, so that the questions arising from the second study (Chapter 5) can be studied in more detail.

## CHAPTER 6

# **NEUROMUSCULAR CHARACTERISTICS, MUSCLE PREAMBULATION AND FATIGUE DURING RUNNING**

## 6.1. REVIEW OF THE LITERATURE

### 6.1.1. INTRODUCTION

Running economy and the factors associated with running economy were discussed in detail in Chapter 4. Traditionally, it has been these measurements of cardio respiratory fitness (running economy, maximal oxygen uptake ( $\text{VO}_2 \text{max}$ ) and rates of blood lactate accumulation) that have been considered to be the most appropriate measures for the laboratory prediction of athletic performance since it is assumed that exercise performance can be explained exclusively by cardio respiratory and skeletal muscle factors (36;125;178;580). However, this conclusion ignores the potential role of the central nervous system in modulating endurance performance by either recruiting or de-recruiting a larger or smaller muscle mass during exercise under different conditions of exercise and differing environments (201;425).

Indeed, the results of our previous studies (Chapter 3 and Chapter 5) have shown that athletes who have been exposed to varying levels of high mileage racing and training display different neuromuscular characteristics yet are able to perform a submaximal run with a similar oxygen consumption compared to athletes exposed to significantly less training. These findings suggest that specific and sensitive alterations in neuromuscular function may contribute to the changes in performance

associated with high mileage training. To support this suggestion, are the findings of Komi (302) and Paavolainen et al (443;445;447;448), who have performed a series of studies to show that in addition to aerobic power, fatigue resistance and running economy, neuromuscular characteristics and muscle power are also related to running performance.

Furthermore, an important component of running ability is the nature of connective tissue, and it's ability to act as a spring to store and return elastic energy on landing (426). Repetitive stretch shortening cycle exercise (see section 6.1.2., page 151), e.g. marathon running, causes an alteration in muscle elasticity and muscle stiffness (421;422) and thus the ability of skeletal muscle and tendon structures to absorb shock is reduced. Similarly, repetitive maximal loading that occurs during marathon running may damage these shock-absorbing structures, causing a reduction in the energy return with each stride, decreasing running efficiency and having a negative influence on performance. It has been suggested that there may be a limit to the number of times these shock absorbing mechanisms may be damaged, after which the central nervous system makes alterations to neuromuscular recruitment patterns in an attempt to protect the body from further damage (426).

Accordingly, this review will discuss the contribution of neural, mechanical and muscular factors to running performance and how these factors interact to produce an efficient running gait cycle. Specifically, this

chapter will highlight the stretch shortening cycle and the functional role of preactivation during running, as well as mechanisms associated with elastic energy storage. This chapter will also discuss the effects of fatigue, muscle damage, age and specific training techniques on neuromuscular characteristics and how these variables may influence successful distance running performance.

### **6.1.2. THE STRETCH SHORTENING CYCLE (SSC)**

The stretch shortening cycle (SSC) refers to a shortening (concentric) muscle action preceded by a lengthening (eccentric) muscle action (219). Muscle elasticity plays an important role in human locomotion by improving the power output in maximal effort (18;99;303;540), and attenuating high impact forces. The effective storage and release of this elastic energy during stretch shortening cycle exercise contributes to the production of muscular force and mechanical effectiveness resulting in a movement more efficient than purely shortening or lengthening work performed alone (18;55;63;100;164;267;301;539).

#### **6.1.2.1. Factors determining the efficiency of the stretch shortening cycle**

The contribution of the stretch shortening cycle to improved running efficiency can be attributed to both mechanical and neural properties of

skeletal muscle. While the active muscle is stretched during the prestretch phase (eccentric), elastic characteristics are temporarily altered (98;99), thereby allowing potential energy to be stored that is partly recovered during the subsequent concentric contraction (303). Thus part of the positive work produced during the concentric muscle action is delivered, at no energy cost, from the recoil of previously stretched elastic elements (61). In addition to these mechanical contributions, segmental reflex activity, specifically the stretch reflex system will further reduce the energy cost during running (55;63). The combined use of this elastic energy and reflex activation of the neuromuscular system have been shown to be fundamental factors in the production of power during the shortening phase of the stride (63).

The maximal efficiency of the stretch shortening cycle depends on three predominant factors: (i) prestretch intensity, (ii) transition time between initial muscle lengthening and subsequent muscle shortening and (iii) muscle preactivation.

#### **6.1.2.1.1. Prestretch intensity**

Skeletal muscle length and velocity during the initial lengthening muscle action has considerable influence on the mechanical efficiency of the subsequent concentric work (19;58;63;99;278). With an increase in velocity of movement, the prestretch intensity also increases, thereby resulting in a

concomitant increase in concentric work production (19;278). In addition to the pure elastic consequences of the prestretch, this lengthening action also plays a role in facilitating a more efficient mobilization of the chemical energy by the contractile component (98). As elastic energy is partly stored in the contractile elements in the series of elastic compartments, any increase in the numbers of cross-bridges being activated would result in a larger capacity of elastic energy storage available for use during the following concentric movement (526).

Flitney and Hirst (184) and Huxley and Simmons (264) have suggested that during the lengthening prestretch, part of the developed tension is taken up by the elastic elements arranged in series with the sarcomeres. This occurs by means of a backwards mechanical rotation of the myosin heads, against their natural tendency, during the stretch to a new position of high potential energy. The lengthened cross-bridges will be detached if the stretch position is maintained for too long (98) or may cause sarcomere "popping" (398) and damage if the range of stretch is too large (184) (see section 2.1.7., page 52). This suggestion therefore infers storage of mechanical energy within the sarcomere cross-bridges and provides further evidence to show that a short transition time between lengthening and shortening phases favours an efficient stretch shortening cycle.

These suggestions are based on the assumption that the enhancement of muscle efficiency through the utilization of the stretch shortening cycle is primarily of mechanical origin associated with the elastic behaviour and attachment - detachment cycle of actin-myosin cross-bridges (58).

Accordingly, it has been estimated that the elastic contribution to the enhancement of the total power generated during stretch shortening cycle activities ranges between 20 % and 50 % (60), depending on intensity of the prestretch and the amplitude of the movement (63).

#### **6.1.2.1.2. Transition time**

The time interval between breaking and push off phases needs to be short (55;58;64;99;302;421), since a delay in this transition time results in a loss of the stored elastic energy (99). This has been illustrated during stretch shortening cycle exercise, which causes fatigue, where a delay in the transition between lengthening and shortening phases resulted in a subsequent decrease in elastic energy storage and a reduction in movement efficiency (58;100).

#### **6.1.2.1.3. Muscle preactivation**

Muscle preactivation represents a centrally driven feed forward, anticipatory mechanism (19;147;209-211;211;218;222;255;407), during which widespread areas of the cortex are involved in the planning and

execution of self-initiated movements, illustrated by pre-movement brain potentials (348), and onto which a reflex activity is imposed (25;147;387;407). Accordingly, muscle preactivation regulates muscle stiffness and the transition time between the prestretch and shortening components of the stretch shortening cycle (211;218;222), as a high pre-landing stiffness is directly responsible for a high post-landing stiffness (254;557).

During running, preactivation is calculated from the EMG activity recorded from lower limb muscles 100 milliseconds before heel strike (prestretch) (448). An increase in EMG activity immediately prior to the prestretch (preactivation) is negatively correlated to the total contact time of the foot on the ground as a result of improvements in muscle stiffness (250;418;443), and muscle recoil capacity (19;21;526).

In addition to its role in ensuring the effective utilization of elastic energy (19;21;250;418;443;526), preactivation also acts to tolerate and buffer high initial force peaks that occur on landing (209;211). Indeed, with the onset of fatigue following repeated stretch shortening cycle actions, contraction times for both lengthening and shortening phases are increased, the initial force peaks upon impact higher and the subsequent drop in force is more pronounced (211).

These findings highlight the importance of preactivation in contributing to the shock absorbing capabilities of skeletal muscle as well as providing

some evidence to suggest that alterations to muscle stiffness and preactivation may be an attempt of the central nervous system to protect skeletal muscle and tendons from muscle damage induced via high impact forces during repetitive stretch shortening cycle exercise. Furthermore, these results confirm the suggestion that preactivation plays a predominant role in determining efficient and superior running performance.

There is substantial evidence to suggest that the central nervous system regulates muscle preactivation by means of both central and reflex induced activation (147;209;407). Although mechanical systems cannot be ignored, Prochazka et al (468) have shown that a fast stretch of an active muscle causes substantial enhancements in stretch reflexes via Ia afferents from the muscle spindle. This reflex potentiation, together with an increase in motor neuron activity to the contracting muscles, would result in an increase in force at the end of the lengthening phase, resulting in an increase in muscle stiffness (24;58;418;552).

(a) *Stretch reflexes*

Bosco et al (55;63) have demonstrated that alterations in myoelectric or EMG properties, originating from centrally pre-programmed activation and segmental reflex activity, could play an important role in the

enhancement of stretch shortening cycle performance, in addition to the utilization of elastic energy (218).

The initial lengthening of the muscle-tendon complex occurs in the tendon (302). As soon as a "critical" tension is achieved, determined by the preactivation of the muscle prior to contact, the forceful "yielding" of the cross-links of the actin-myosin complex takes place with a concomitant loss of potential energy stored in the lengthened cross-bridges (184). In vitro studies have shown that the possible "over-yielding" of active cross-links, and thus loss of this potential energy, is prevented by intense muscular activation (302). This preventative activation is monitored most effectively by the stretch reflex system that is highly sensitive to the length and tension changes within the muscle-tendon complex (24).

Sinkjaer et al (498) have suggested that stretching a contracting muscle generates a large force increment, of which approximately half can be attributed to alterations to the stretch reflex. This suggestion has been confirmed by others (211;218;302;418) who have also shown an increase of segmental reflex activity immediately prior to ground contact. Thus, the appearance of higher stiffness at faster speeds can partly be attributed to segmental reflex activity (146;211;222;302), which therefore plays an important role in the subsequent stretch shortening cycle and significantly contributes to increased force generation during the concentric phase (302). Aura and Komi (19), have, however, suggested that there may be

an upper limit to the improvements in muscle stiffness characteristics and have concluded that this limit may be linked to the activation and chemo-mechanical behaviour of the skeletal muscle cross-bridges.

The effective contribution of the stretch reflex components becomes increasingly important in situations where stretching loads are high or efficient stretch shortening cycle behaviour is necessary (209;211). Under these conditions, muscle stiffness needs to be well regulated to meet external loading conditions (211;301) and to prevent biomechanical and skeletal muscle injury. The nervous system therefore attempts to compensate for mechanical deficiencies during stretch shortening cycle fatigue by increasing stretch reflex activity (209).

However, as the lengthening loads are increased, this compensatory mechanism is not effective enough to resist the fatigue effect on the mechanical performance of muscle. As a result, there is a subsequent reduction in reflex activation, which implies a mechanism in which fatigued muscles are protected from overly excessive stretch loads (209). Indeed, Gollhofer et al (209) and Nicol et al (419;421) have suggested that alterations in reflex regulation contribute to the observed reduction in elastic energy potential with associated alterations in muscle spindle and Golgi tendon organ sensitivity (421). These findings illustrate a clear role of neural and reflex activation strategies in influencing efficient stretch shortening cycle activity.

It is likely that there is a continual interaction and contribution of both mechanical and neural factors to the overall efficiency of the stretch shortening cycle. In addition to these integrative mechanisms, Bosco et al (63) have suggested that type I and type II skeletal muscle fibers may have different viscoelastic properties. Furthermore, there is some evidence to illustrate that muscle fiber content contributes to alterations in EMG activity during fatiguing exercise (554). These observations suggest that skeletal muscle structural components contribute to the interactive processes between neural control and mechanical responses during running.

### **6.1.3. STORE AND RECOIL OF ELASTIC ENERGY IN DIFFERENT FIBER TYPES**

Bosco et al (63) have shown that subjects with a high percentage of type II muscle fibers are able to store a greater amount of mechanical energy during the landing (lengthening) phase of vertical jumps with small angular displacement but not in jumps with larger angular displacements. Similar results have been shown by others (20;56;59;553) who have suggested that prestretch intensity and the rate of force development may be greater in subjects with more type II muscle fibers.

Bosco et al (63) concluded that the reasons for these discrepancies between subjects with predominantly fast twitch fibers and subjects with

predominantly slow twitch fibers could be explained by the attachment - detachment cycle of the sarcomere cross-bridges. For example, small amplitude jumps are characterised by a short transient period between stretch and shortening phases and thus during these jumps, subjects with a fast twitch fiber predominance would be at an advantage.

Alternatively, during large amplitude jumps there is a significantly longer transition time between the lengthening and shortening phases, and thus subjects having a greater percentage of slow type fibers would have a longer time to allow for further motor unit recruitment. This study concluded that longer coupling times made it easier for type I muscle fibers to retain their elastic energy for a longer time period without experiencing cross bridge detachment (63).

Similarly, the limiting effect of the coupling time on the recoil of elastic energy is more relevant in fast twitch muscle fibers than slow twitch fibers during running (62;63), again based on the differences in cross-bridge life time between the different fibers (61;63;530). Because type II fibers have a significantly shorter cross-bridge life time cycle, and the coupling time is longer than a few milliseconds, some of the cross-bridges in these fibers will be detached and their elastic potential lost. This finding would explain why the economy of running is higher in subjects with a type I muscle fiber predominance compared to subjects with a muscle fiber type II predominance, when running at slow and moderate speeds (63;530).

Moritani et al (407), using a hopping task to fatigue, showed that muscle groups with predominantly type II muscle fibers (gastrocnemius muscle) were affected by fatigue to a greater extent compared to predominantly type I muscle fiber groups (soleus muscle). These differences were illustrated by differing alterations in preactivation and the lengthening phases of the stretch shortening cycle in the two groups. This study concluded that due to the muscle fiber dependent electrophysiological responses to fatigue, the metabolic profile of different fiber types might play an important role in regulating muscle membrane excitability (407).

Collectively, these findings illustrate the contribution of neural, mechanical and muscular factors to an improved stretch shortening cycle capacity, which results in efficient endurance running performance. In addition, there is also substantial evidence to suggest that a greater ability to resist fatigue is a major contributing factor to superior running performance.

#### **6.1.4. NEUROMUSCULAR FATIGUE**

Fatigue in this context is defined as a decrease in muscle performance or a failure to maintain the required or expected force or power output (289;518). Fatigue is classified as being either central or peripheral in origin. Central fatigue is described as a reduction in neural drive or motor command to the muscle resulting in a decline in force or tension

development (286;426). Peripheral fatigue is defined as a decrease in the force generating capacity of the skeletal muscle due to action potential failure, excitation-contraction coupling failure or impairment of cross bridge cycling in the presence of unchanged or increased neural drive (286;426).

There are multiple physiological factors from the central nervous system through to the intramuscular contractile structures that may cause or be associated with fatigue (167;289). Depending on the site of fatigue, these mechanisms result in changes in amplitude of EMG signals (176), a shift in the EMG power spectra towards lower frequencies (519), a decrease in muscle force generating capacity, a decrease in muscle relaxation time (395) and a delayed transition time between the lengthening and shortening phases of motion during the stretch shortening cycle (421).

The relative contribution of central and peripheral fatigue in endurance exercise has yet to be confirmed. There appears to be a close relationship between changes in intracellular metabolites and force production during sustained maximal isometric contractions (85;161;391;564). This is compatible with the peripheral, skeletal muscle model of fatigue, which holds that intracellular metabolites regulate skeletal muscle contractile function (381). However, it is less certain that such peripheral factors can adequately explain the fatigue that develops during more prolonged exercise. For example, an exclusively peripheral model of exercise

regulation cannot explain how athletes can increase their pace near the end of a competitive event exactly when, according to the peripheral model of fatigue, metabolite accumulation or depletion should cause them to slow down (425). Nor can this model explain how skeletal muscle fatigue can develop during prolonged exercise when less than 25 % of the available motor units are recruited in the actively exercising limbs, or why such recruitment decreases progressively during prolonged exercise (514).

Kent-Braun (289) and Bigland-Ritchie et al (46) have shown that during a sustained isometric MVC, central activation failure contributes to approximately 20 % of the total fatigue, whereas the remainder of the performance decrement was attributed to intramuscular sources, primarily an increase in hydrogen ions. Conversely, Baker et al (30) compared short and long duration exercise protocols in an attempt to determine the relative contributions of central and peripheral fatigue. These authors found that during short duration exercise, most of the fatigue was due to a metabolic inhibition of contraction, whereas after longer duration exercise, there may be a non-metabolic component to fatigue acting beyond the cell membrane, at the level of the excitation-contraction coupling mechanisms.

It has also been proposed that both central and peripheral factors contribute to the development of fatigue (46), and reinforce the suggestion of Kent-Braun (289) that there is a constant feedback loop

between central and peripheral factors during fatiguing exercise and thus a direct link between intramuscular metabolism and central motor drive.

#### **6.1.4.1. Experimental techniques to induce and measure stretch shortening cycle fatigue**

The stretch shortening cycle provides a unique model to study neuromuscular fatigue. Studies investigating the effects of fatigue on the stretch shortening cycle have used models with force plate techniques (526) and sledge jump apparatus (19;23;209;306;518) for monitoring both upper (209) and lower body fatigue (22;23;253;419), or functional endurance type activities such as endurance cross-country skiing (305;555), marathon running (421;422;501), ultra marathon running (393) and explosive sprinting (438;447).

#### **6.1.5. THE EFFECTS OF FATIGUING STRETCH SHORTENING EXERCISE ON NEUROMUSCULAR CHARACTERISTICS AND PERFORMANCE**

Fatigue is a consequence of a combination of mechanisms. Therefore, fatigue observed during stretch shortening cycle activities may be complex in nature, since this neural control depends largely on a reflex-induced activation (518). Evidence for reductions in stretch shortening cycle performance following fatiguing activities is that contraction times increase for both the lengthening and shortening phases of the stretch

shortening cycle (210) which results in longer contact times. These changes may indicate an unsuccessful attempt by the neuromuscular system to compensate for the lost elastic potential (421).

Repeated stretch shortening cycle exercise is also likely to affect force production in both isometric (23) and dynamic performance (518). Such reductions in force have previously been reported during long duration skiing (305;555) and long (421;422) and short (438;447;448) duration running. Similarly, Millet et al (393) reported a decrease in maximal voluntary activation resulting in a reduction in maximal force production of the knee extensor and plantar flexor muscles in subjects who had completed a 65 km footrace.

#### **6.1.5.1. Proposed mechanisms to explain the reduction in force production associated with fatigue**

There have been two mechanisms proposed to explain the reduction in muscle function following fatiguing stretch shortening cycle exercise. Firstly, the failure of maximal muscle functioning might be due to some impairment of peripheral mechanisms (183). Secondly, as indicated by the reduction in EMG activity, there may be a lowered neural input to the muscle implying a centrally mediated component (23;24;202;425) encompassing supraspinal fatigue (68;202), peripheral inhibition (203;204) and/or disfacilitation of the alpha-motoneuron pool (53).

#### 6.1.5.1.1. Impairment of peripheral factors

Maximal contractile performance is partially determined by neuromuscular characteristics controlling the rate and force of myofibrillar cross bridge cycle activity (220;424). Viitasalo and Komi (554) and Gollhofer et al (210) suggested the reduced contractile characteristics of muscle are associated with a depression in calcium transport following exhaustive exercise. An inability to sustain calcium release from the sarcoplasmic reticulum would result in lower activation levels, whilst a reduction in the time taken to remove calcium from the cytosol would prolong the dissociation of actin and myosin and reduce the relaxation of muscle during the recovery phase (7;530).

A reduction in calcium transport is likely to be associated with an accumulation of hydrogen ions, as there is evidence to suggest that the sarcoplasmic reticulum binds more calcium with an increase in muscle acidity (14;210;388;530;541). As the pH is lowered, there is an increase in the requirement of calcium to produce tension. Furthermore, the increased hydrogen concentration may also reduce the effect of calcium on troponin (451), and therefore have a direct impact on the contractile process itself (530).

To examine possible sites of fatigue along the excitation-contraction pathway during stretch shortening cycle activity, Strojnik and Komi (518)

used maximal intensity drop jumps on an incline sledge apparatus and clearly showed a reduction in force production following the fatiguing intervention. The authors proposed that the reduction in force was as a result of impairments in contractile mechanisms following maximal stretch shortening cycle exercise and suggested that a possible mechanism contributing to these impairments was a decrease in calcium release from the sarcoplasmic reticulum and/or reduced capability of cross-bridges to form strong binding (518). Changes to calcium regulation and calcium sensitivity have been associated with alterations to the contractile function of the actin and myosin components (388;451;518;530).

#### **6.1.5.1.2. Centrally mediated factors**

Alterations in motor unit activation, stretch reflexes and stiffness regulation, as a result of successive stretching loads, has also been shown to contribute to the decline in force production associated with fatigue following repetitive stretch shortening cycle exercise.

Nicol et al (422) reported a 26 % reduction in maximal torque during a three second maximal voluntary contraction (MVC) following a 42 km footrace. The EMG recorded simultaneously decreased by approximately 36 % (422). The progressive reduction in motor unit activation during contractions at high force levels has been postulated to be a mechanism to minimise fatigue by avoiding neuromuscular transmission failure (167;

197;202). This provides evidence to suggest that a reduction in neural input contributes to the loss of maximal force production following intense stretch shortening cycle exercise (209). This finding was indirectly confirmed by Viitasalo and Komi (554) who showed similar reductions in maximal force, reflex and voluntary reaction times and EMG mean power frequency, following 100 maximal isometric leg extensions.

Centrally mediated preactivation as well as the activation during the lengthening phase are also affected by fatigue-induced changes (23;209;407;438). A reduction in muscle preactivation and neural input to the muscle decreases the efficiency of the contractile mechanisms (decreased preactivation) (23;209;210;407). This then results in a longer contact time and lower ground reaction forces. Ultimately, there is a longer time between lengthening and shortening phases, implying a decreased ability to store and use elastic energy, thereby decreasing running efficiency (443).

Furthermore, Avela and Komi (23) suggested that alterations in stiffness regulation might also play an important role in the reduction of muscle force and power. They used a protocol in which the subjects jumped on a sledge ergometer, before and after a 42 km footrace, to study this hypothesis. The results from this study showed that the reductions in peak force production corresponded to the reduction in EMG recruitment and decreases in muscle stiffness, indicating deterioration in the sensitivity of

the stretch reflex system after fatigue (23). Avela and Komi (23) concluded that the modulation of neural input was at least partly of reflex origin from the contracting muscle and that the reductions in centrally mediated muscle stiffness accompanying the decrease in stretch sensitivity may be responsible for the weakened muscle performance, due to an impairment of the use of elastic energy (23). Similarly, Komi (302) has suggested that repetitive impact loads may decrease the ability of the leg extensor muscles to sustain impact loads. This results in the muscle losing its recoil characteristics.

Since the enhancement of the stretch shortening cycle depends on the ability to tolerate and use stretch loads, a deterioration of the capacity to tolerate impact forces will influence endurance performance. Nicol et al (421;422) have shown reductions in sprinting and jumping performance, decreased isometric force production and an increase in time of the braking and push off phases, resulting in a net increase of total ground contact time and shorter flight times following a 42 km footrace (421). In addition to neuromuscular changes associated with fatigue, the possibility also exists that these reductions in performance capacity could also be associated with skeletal muscle damage.

### 6.1.6. THE EFFECTS OF SKELETAL MUSCLE DAMAGE ON STIFFNESS REGULATION

Fatigue induced through repetitive stretch shortening cycle exercise usually results in a reversible muscle damage process and has considerable influence on muscle mechanics, joint and muscle stiffness and reflex innervation (302). A reduction in neural input to the muscles accompanies the reduced maximal force production after either prolonged (cross country skiing) (305;555) or short duration (253;407;438) stretch shortening cycle activity. In addition, structural damage has been observed following marathon running with a large lengthening muscle component (Chapter 2) (105;191;248;317;502;513;558), suggesting that there may also be impairments in muscle fiber function (422) and a reduction in muscle contractile capabilities associated with damaging exercise (13;15;30;38;52;81;114;165;171;192;339;414;422;432;436;508).

Horita et al (253) have shown that stiffness regulation in the knee joint during drop jumping is significantly impaired after a repetitive stretch shortening cycle exercise. These authors suggest that myofibrillar disruption and/or connective tissue injury after stretch shortening exercise may affect the stiffness regulation of the entire muscle tendon complex (254). In a subsequent study, Horita et al (254) showed significant reductions in preactivation of the vastus lateralis muscle during a drop jump immediately following fatiguing exercise. Further reductions in

preactivation were observed two and four days thereafter, suggesting a neural compensatory mechanism employed to protect the muscle from additional damage. These authors concluded that mechanical behaviour during a drop jump (or perhaps any exercise with a stretch shortening cycle component) could be affected by modified motor control, pre-landing, in response to muscle damage (254).

Notwithstanding the reduction in muscle strength associated with muscle damage (section 2.1.8.2., page 66), some studies have shown no changes in EMG activity on the days following lengthening action muscle damage (38;384), suggesting that normal motor unit activation can be maintained despite symptoms of muscle damage. These data confirm the findings of Saxton and Donnelly (490), who demonstrated that strength loss after lengthening muscle exercise was unaffected by superimposing supramaximal stimulation during maximum voluntary isometric contractions.

Komi (302) has proposed the following two possibilities to explain the reduction in stretch shortening cycle performance associated with muscle damage. First, as a consequence of muscle damage, there is a decrease in the stretch reflex sensitivity, which causes a disturbance to the muscle and joint regulatory mechanisms, and in turn, to the efficiency of the stretch shortening cycle. Second, as a result of the deterioration in muscle function associated with muscle damage (section 2.1.8.2., page 66), there

is a reduction in the tolerance of the muscular system for high impact loads (reduction in preactivation) and a reduction in the elastic energy potential. These alterations combine to result in an increase in the work required during the concentric phase and thus a reduction in running efficiency (302).

#### **6.1.6.1. Recovery from stretch shortening cycle fatigue**

Recovery from fatigue induced by stretch shortening cycle exercise is a delayed process, follows a bimodal pattern and occurs in parallel with the recovery of maximal EMG activation and maximal force (22;180;254;302; 419).

The initial decline in performance immediately after the exercise is primarily contributed to mechanical injury, specifically myofibrillar disruptions (180;362), discussed in section 2.1.8.1. (page 56). This decline is followed by a short lasting recovery and a subsequent second performance decrement (254;302;362). The second decline in performance peaks 2 – 3 days after the damaging exercise (22;254;419; 421) and has been associated with inflammatory responses related to muscle damage (180;302). Indeed, Nicol et al (421) have shown that following a 42 km marathon, maximal force production was approximately 64 % of pre-race values, and that this reduction persisted for up to seven days following the race (421).

These findings infer that stiffness regulatory mechanisms may require an extended recovery time following muscle damage and fatiguing stretch shortening cycle exercise before they are able to return to optimal function (23;24).

Furthermore, these findings provide support to the suggestion that neuromuscular characteristics are closely associated with successful endurance performance, yet muscle preactivation and stretch reflex mechanisms are affected by muscular fatigue and skeletal muscle damage. Previous studies have shown that although there is a relative decrease in the efficiency of neuromuscular performance associated with fatigue in both high and low calibre runners, high calibre runners have consistently higher levels of preactivation and lower EMG contributions during the shortening phase (448). This then raises the question of whether this component of the stretch shortening cycle may be specifically isolated and trained in order to improve overall endurance performance.

#### **6.1.7. THE EFFECTS OF SPECIFIC EXERCISE TRAINING TECHNIQUES ON PREAMPACTIVATION AND MUSCLE STIFFNESS CHARACTERISTICS**

Although the maximal force that a muscle can produce is positively correlated to its cross sectional area (375;582), some studies have shown that there is a weak relationship between training-induced increases in

strength and muscle size (164;166). This dissociation between strength and size associated with training occurs because force is produced not only from the quantity of muscle recruited, but also by the ability of the nervous system to activate the appropriate muscle fibers (164). In addition to voluntary neural control, training-induced changes in reflex potentiation may also take place (487), specific to the type of action and movement velocity, which could effect motor unit recruitment within the muscle (239;487). Training induced neural adaptations are also related to an increase in the activation of the large motor neurones, an improvement in the co-contraction of synergists and/or a decreased co-activation of the antagonist muscles (166;235).

In addition, athletes with different performance capabilities often present with similar aerobic profiles ( $\text{VO}_2$  max). This has been well documented during maximal performance tests (424;426;447) and suggests that neuromuscular characteristics are also important determinants of endurance performance.

Regular endurance training selectively enhances the function of the cardio respiratory system, oxidative capacity and glycogen stores of the muscles (252;424;426). However, in addition to superior aerobic fitness, endurance athletes also need to produce increased muscle power to maintain high running velocities over the course of a race. These findings

further highlight the importance of specific exercise training on neuronal control mechanisms and on muscular spring characteristics (557).

The sensitivity of EMG electrode placement makes longitudinal trials and training studies investigating neuromuscular characteristics both technically and logistically challenging. There is only one well conducted training study investigating the longitudinal effects of endurance training on neuromuscular characteristics (357). This study measured EMG from the vastus lateralis muscle in professional cyclists, during a ramp protocol to exhaustion, over three defined periods of the season (rest, pre-competition and competition). The results showed increases in surface EMG during the season, with significant differences mainly between the rest phase and competition phase, illustrating an enhanced recruitment of motor units in active muscles, at all exercise intensities, with an increase in endurance training (357). These results provide some evidence to suggest that endurance training enhances the ability to recruit "reserve" motor units. These units are presumably not readily available for recruitment, but part of the training response may be a central adaptation that allows them to be activated (138;166).

Paavolainen et al (444;446) introduced explosive strength training as supplementary training to the endurance based exercise programme of cross-country skiers (446) and orienteers (444). The experimental subjects in these studies underwent explosive and heavy resistance training,

comprising 34 % of the total training programme for six (446) and nine (444) weeks, with the remaining 66 % of the programme being endurance training. Explosive strength training included jumping exercises, sprints and resistance exercise with low loads and high velocities. Control subjects maintained regular endurance training with no additional explosive strength training exercises.

In both studies (444;446), the explosive strength training resulted in specific positive changes in neuromuscular performance, illustrated by improved running velocities, higher jump heights and shorter contact times during a constant velocity run. These neuromuscular alterations occurred without any significant changes to aerobic characteristics.

More recently, Turner et al (549) trained distance runners for six weeks using explosive strength training, similar to that used in the studies by Paavolainen et al (444;446), concomitantly with regular endurance exercise training (549). This study showed that regular explosive strength training improves running economy at different speeds, confirming the results of Paavolainen et al (444;446), even though the training did not effect jump height (549). These authors attribute this finding to the intensity of the training programme, suggesting that the jumping exercises used in this study were of a lower intensity with less risk of injury to the subjects (549) compared to the protocol used by Paavolainen et al (444;446).

A shortcoming of these studies was that EMG was not measured during the jumping and running tests. Although no EMG measurements were obtained from these studies, it was suggested that the increases in neuromuscular performance characteristics associated with explosive strength training might be due to neural adaptations (444;446). Specifically, explosive strength training results in an increase in the amount of neural input to the muscle, illustrated during rapid dynamic and isometric contractions (240;444;446). This suggests that the increase in overall excitation of motoneurons could result from increased excitatory input, reduced inhibitory input, or both (1;166). Accordingly, it is likely that training induced alterations in neural control during stretch shortening cycle exercise may take place in both voluntary activation and inhibitory and/or facilitatory reflexes (1;235;320).

Viitasalo et al (557) compared neuromuscular function between jumpers and non-jumpers and showed that athletes who undergo specific jump training have increased EMG activity (preactivation) earlier before landing compared to the non-jumper controls. In addition, the neuromuscular system of the jumping athletes was more able to resist high muscle lengthening and ground reaction forces, illustrated by a larger force production in the propulsion phase and in jump height (557).

These results have been confirmed by Kyröläinen et al (322) and Kyröläinen and Komi (321) who have shown that a specific stretch

shortening cycle training programme improves mechanical efficiency and that the rate of EMG development is faster during the preactivation phase for power trained athletes.

#### **6.1.7.1. Possible alterations to connective tissue associated with endurance training**

Large forces and short braking phases transmit great forces to the connective tissue (e.g. tendons) (557) and thus specific explosive strength training may also have an effect on connective tissue characteristics. Tendons are able to store and release high amounts of elastic energy if exposed to high stretching loads in a sequence of stretch shortening cycle exercise (266;285). It has also been shown that specific training is able to modify the metabolism of connective tissue (311;523;524), causing mild hypertrophy and thus improving its mechanical properties (522;523).

Thus, in addition to possible differences in the structure and functioning of the neuromuscular contractile apparatus, differences in connective tissue properties may also account for superior performance in athletes trained with specific stretch shortening cycle exercises. In addition, differences in connective tissue properties have also been associated with increasing age (see section 2.1.3.2.4., page 32) and accordingly, an increase in age may also affect skeletal muscle preactivation and stiffness regulation (57;255).

### 6.1.8. THE EFFECT OF AGE ON MUSCLE STIFFNESS

Chapter 2 discussed aging of human skeletal muscle and its association with changes in the integrity of the neuromuscular system including alterations to motor unit numbers and size (123;137;148;168;325;440;480;537;542;550). However, there are few studies in the literature illustrating changes in muscle stiffness associated with increasing age (57;255). Hortobágyi and De Vita (255) compared preactivation in young (mean age 21 yrs) and old (mean age 69 yrs) women during downward stepping. The results of this study showed that older subjects had 64 % greater leg stiffness compared to younger subjects, and on average, preactivation (in this study, measured 200 ms before heel strike) was 136 % greater in the older subjects. In young and old subjects, the magnitude of preactivation of the knee extensors was 18 and 63 % respectively, of the total EMG measured during a maximum lengthening muscle action (557). Accordingly, this study concluded that older subjects might scale muscle preactivation in anticipation for the demands of impact. In addition, there is also some evidence to suggest that age may increase muscle preactivation (236;255) as the slowed contractile properties of aged muscles may make it more effective for the nervous system to regulate stiffness characteristics.

These results therefore suggest that the functional goal of an increased leg stiffness associated with an increase in age is to compensate for

neuromuscular impairments (255) such as reductions in muscle strength and slower rates of tension development (54;57;89;195;536). Additional research, however, is needed to further assess this hypothesis.

#### **6.1.9. SUMMARY OF THE LITERATURE**

The stretch-shortening cycle has a well-defined purpose: to enhance performance during the final phase (concentric contraction) of the gait cycle. During the prestretch phase of the stretch-shortening cycle, the active muscle is lengthened. During this phase, the imposed energy is stored as potential energy and then transferred to kinetic energy during a subsequent shortening of the muscle.

In addition, muscle preactivation, regulated by a central drive, increases muscle stiffness and stretch reflexes in both muscles and tendons thereby increasing the ability of the musculotendinous structures to tolerate high impact loads. However, these reflexes are reduced when the musculotendinous system is repeatedly loaded. Consequently, high impact loads become more difficult to tolerate as this neural form of fatigue develops. As a result, neuromuscular characteristics are well regulated by the central nervous system to protect skeletal muscle from chronic overuse damage and injury. These characteristics can be altered through specific training techniques that may enhance muscle stiffness and

improve running efficiency through a more effective use of elastic energy stored during the lengthening phase of the stretch shortening cycle.

This review has highlighted the importance of muscle preactivation firstly, to enhance the efficiency of the stretch shortening cycle, and secondly, as a primary shock absorption mechanism to eliminate high impact forces on landing during running.

Previous studies measuring muscle preactivation during running have provided valuable information regarding central patterning of muscle recruitment during running. Given the sensitivity of muscle preactivation, the techniques used to measure this variable need to be sensitive, yet practical, to allow their inclusion into dynamic running protocols.

Thus, the aim of the next study was firstly, to identify a measurement technique and protocol that would accurately measure preactivation during running, and secondly, to determine whether the measurement of preactivation could be used as a sensitive measure of neuromuscular changes associated with fatigue and muscle stiffness regulation during dynamic exercise.

**CHAPTER 7**

STUDY THREE

**A RESEARCH MODEL FOR  
NEUROMUSCULAR FATIGUE DURING  
RUNNING**

## 7.1. INTRODUCTION

The review of the literature in Chapter 6 focused on the potential role of the regulation of muscle recruitment, specifically the ability of the skeletal muscle to store and re-use elastic energy during the stretch shortening cycle, in determining athletic performance and in the development of fatigue. Paavolainen et al (448) showed that better performing runners in a 10 km time trial maintained optimal levels of neural preactivation, and as a consequence, shorter ground contact times, longer stride lengths and faster running speeds. These findings illustrate the potential importance of centrally regulated neuromuscular factors in determining endurance running performance.

Most previous studies have measured preactivation after prolonged exercise lasting between 35 (448) and 510 (393;422) minutes or high intensity, maximal sprinting (438). No studies have examined these EMG changes associated with short duration, high intensity endurance exercise (15 – 20 minutes) to determine whether these changes might have any practical application in investigating neuromuscular fatigue. Therefore, the aim of this study was to use a dynamic technique to determine whether changes in muscle preactivation occur during short duration (15 - 20 minutes), high intensity endurance exercise, and whether these alterations can explain changes in running performance, including the development of fatigue. The overall goal of this study was to develop a

research model that could be used to study neuromuscular changes in runners with a history of a high volume of running training and racing.

## **7.2. METHODOLOGY**

### **7.2.1. Subject selection**

Eighteen healthy, male distance runners were recruited to participate in this study. Subjects were included if they were able to complete 10 km in under 38 minutes. Each subject signed an informed consent form at the beginning of the study. The study was approved by both the Ethics and Research Committee of the Faculty of Health Sciences, University of Cape Town.

### **7.2.2. Experimental design**

Subjects were required to visit the laboratory on three separate occasions over a 10 day period. Subjects were asked to maintain their regular physical activity pattern for the duration of the study and were requested not to exercise on the morning prior to their testing.

On their first visit to the laboratory, subjects were given the opportunity to become familiar with equipment and testing protocols that would be

used during the trial. This familiarisation was performed in an attempt to reduce error associated with subjects performing unaccustomed exercise. A personal racing history was also obtained from each subject.

On their second visit, subjects ran a 5 km time trial during which the EMG activity of 5 muscles and the stride parameters of the subjects were measured. The 20 m sprint time and maximal voluntary contraction of the vastus lateralis (VL), vastus medialis (VM), biceps femoris (BF) and rectus femoris (RF) muscles were measured before and after the 5 km time trial.

On their third visit to the laboratory, not more than 7 days later, peak treadmill running speed (PTRS) was measured using a continuous, incremental running protocol on a horizontal (0 % gradient), motor driven treadmill for the determination of ( $\text{VO}_2$  max) and maximum heart rate (HR max) used for descriptive purposes. These methods have been described in Chapter 3 (section 3.2.3., page 86). The starting speed for the maximal test for this study was  $12 \text{ km}\cdot\text{hr}^{-1}$ .

#### **7.2.2.1. Anthropometry**

These methods have been described in Chapter 3 (section 3.2.2., page 86).

### 7.2.2.2. Maximal 20 m Sprint Test

Subjects performed three to five maximal 20 m sprints on an indoor running track (447;448). Subjects were given a running start of 15 m to ensure a normal and maximal running gait throughout the 20 meters and to exclude gait changes associated with acceleration during the sprint. Each 20 m sprint was separated by a brief recovery period during which subjects returned to the start of the sprint course. Twenty meter running time was recorded using two photocell gates connected to an electronic timer (Newtest, Ltd, Oulu, Finland). The 20 m sprint producing the fastest time was taken as the non-fatigued, pre 5 km time trial value, and was thus used for all subsequent data analysis. During the final lap of the 5 km time trial, subjects repeated the maximal 20 m sprint test down the straight section of the track.

### 7.2.2.3. Maximal voluntary contractions (MVC)

Ten minutes after completing the final 20 m sprint, subjects performed three 5-second maximal voluntary contractions (MVC) separated by 5 seconds rest on a custom-built seated leg press machine (Hur Ltd., Kokkola, Finland) with a knee flexion angle of 70°. Subjects were asked to exert maximal force against the footplate for 5 seconds during which standardized verbal encouragement was provided by the investigators. The single 5 second contraction that produced the highest force was

taken as the non-fatigued, pre 5 km time trial value. The single highest force value as well as the average of all force values recorded over 5 seconds during this contraction were used for all subsequent data analysis. Immediately after finishing the 5K, subjects went directly to the leg press machine where they performed one 5-second MVC. This test occurred within 10 seconds after finishing the 5 km time trial.

Verbal encouragement was standardized during all MVC testing. Subjects rested for 20 minutes before the start of the 5 km time trial.

#### **7.2.2.4. 5 km time trial (5K)**

Subjects performed a 5 km time trial (5K) on a 140-m indoor track. Subjects were instructed to run "as fast as possible" and were provided standardized verbal encouragement during the run. Split times were given to the athletes at each kilometre.

#### **7.2.2.5. Electromyographic activity and stride parameter measurements**

Before the start of testing, each subject had bipolar EMG electrodes (Beckman miniature skin electrodes, Illinois, USA) placed onto the vastus lateralis (VL), vastus medialis (VM), rectus femoris (RF), biceps femoris (BF) and gastrocnemius (GA) muscles of the right leg. The skin was shaved, rubbed with sandpaper and cleaned with alcohol. The electrodes were

positioned longitudinally on the belly of each muscle and carefully taped. All EMG data were amplified and recorded telemetrically (Biomes 2000, Glonner, Germany) and simultaneously, with the signals obtained from the leg press and the contact mat data, on a microcomputer using Labview 5.1 (National Instruments, Texas, USA). During the MVC, EMG from VL, VM, RF and BF was recorded simultaneously with the force that was recorded during the leg press.

A photocell contact mat to measure stride parameters (contact time and flight time) (556) was placed on the side straight of the track closest to the leg press machine. During the 20 m sprints, contact times and flight times were measured simultaneously with EMG from VL, VM, RF, BF and GA. Stride frequency (SF) ( $\text{strides}\cdot\text{s}^{-1}$ ) was calculated by using contact times (CT) and flight times (FT) as  $[1/(\text{CT}+\text{FT})]$ . Stride length (SL) (meters) was calculated by using velocity (V) and stride frequency (SF) as  $(V/\text{SF})$ . Both EMG and stride parameter data collected during each stride were averaged for the number of strides taken along the 20 m straight.

The non smoothed EMG signals were rectified, integrated and time normalised for the two phases of running: preactivation (100 ms before ground contact) and total ground contact time (448).

The percentage change in preactivation for the total lower limb was calculated by combining the percentage change in preactivation from each individual muscle group.

### **7.2.3. Statistical analysis**

Data were analysed using Statistica 5.5 (Statsoft, Inc., Oklahoma, USA). A dependent t-test was used to evaluate differences before and after the 5K, while a Pearson's product moment correlation coefficient determined relationships between variables (change in sprint time, change in contact time, change in stride length, change in preactivation, change in stride frequency and change in average MVC force production). Values are expressed as mean  $\pm$  standard deviation. Statistical significance was accepted as  $P < 0.05$ .

## **7.3. RESULTS**

The descriptive and performance characteristics of the research subjects are shown in Table 7.1. These data suggest that this group of athletes displayed a relatively wide range of both anthropometrical and performance variables.

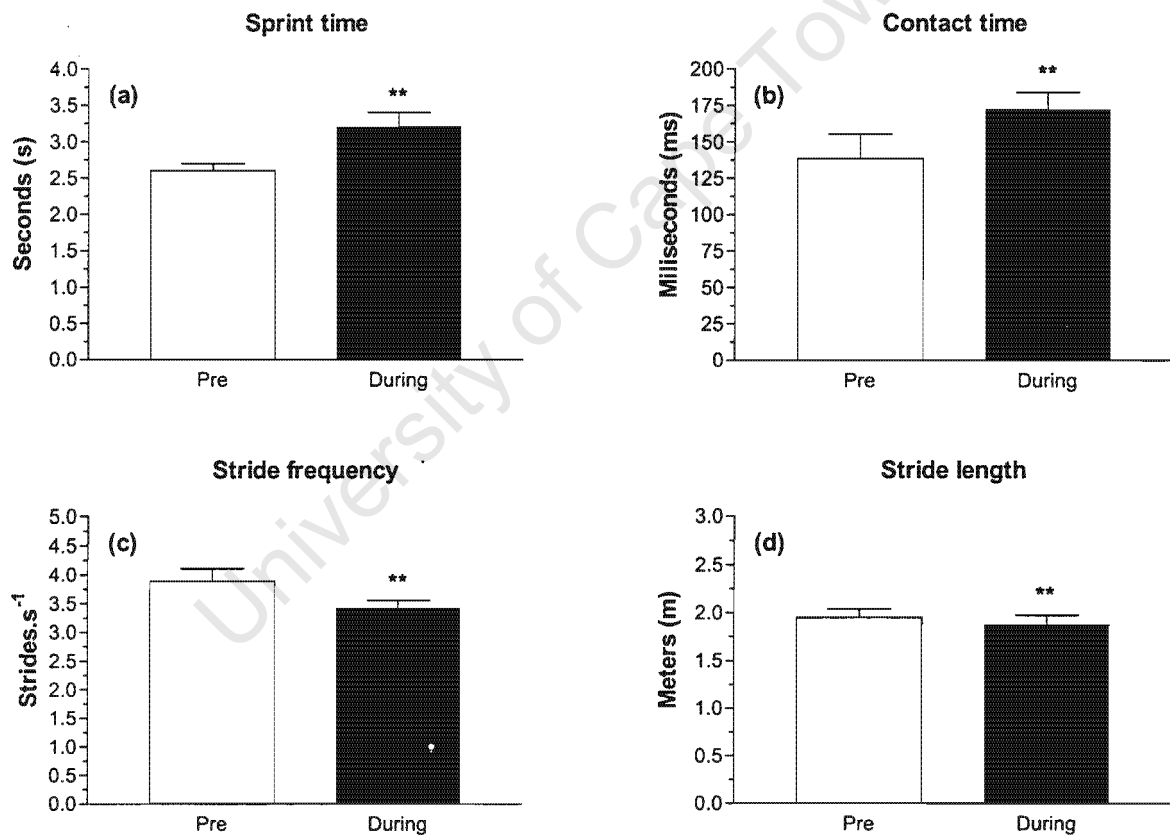
**TABLE 7.1:** Descriptive and performance characteristics of the research subjects (n = 18).

<b>VARIABLE</b>	<b>MEAN <math>\pm</math> SD</b>	<b>RANGE</b>
Age (years)	23.9 $\pm$ 6.5	16.0 – 34.0
Weight (kg)	59.6 $\pm$ 4.7	50.3 – 67.1
Height (cm)	168.8 $\pm$ 4.7	161.0 – 180.0
Sum of skinfolds (mm)	44.5 $\pm$ 11.7	31.0 – 70.7
Body fat (%)	10.7 $\pm$ 3.0	6.6 – 17.7
Maximum heart rate (b.min <sup>-1</sup> )	186.0 $\pm$ 10.0	168.0 – 205.0
VO <sub>2</sub> max (ml.kg <sup>-1</sup> .min <sup>-1</sup> )	64.0 $\pm$ 4.0	56.3 – 70.7
Peak treadmill running speed (PTRS) (km.h <sup>-1</sup> )	20.8 $\pm$ 1.8	19.0 - 23.5
10 km personal best running velocity (m.s <sup>-1</sup> )	5.0 $\pm$ 0.40	4.5 – 5.5
5 km time trial running velocity (m.s <sup>-1</sup> )	4.9 $\pm$ 0.3	4.3 – 5.3

### 7.3.1. Twenty meter sprints

Athletes were significantly slower during the second 20 m sprint performed during the last lap of the 5K. Sprint time increased significantly from 2.64  $\pm$  0.14 seconds in the pre 5K sprint to 3.16  $\pm$  0.20 seconds ( $P < 0.0001$ ) in the

20 m sprint during the 5K (figure 7.1 a). Similarly, there was also a significant increase in ground contact time from  $139 \pm 16.5$  milliseconds to  $172 \pm 12.2$  milliseconds ( $P < 0.0001$ ) (figure 7.1 b) in these 20 m sprints. Flight times increased from  $119 \pm 8.6$  milliseconds to  $122 \pm 12.0$  milliseconds, but this was not significant. Conversely, both stride frequency and stride length decreased significantly by  $0.49 \pm 0.20$  strides. $s^{-1}$  ( $P < 0.0001$ ) (figure 7.1 c) and  $0.07 \pm 0.06$  m ( $P < 0.0001$ ) (figure 7.1 d) respectively.

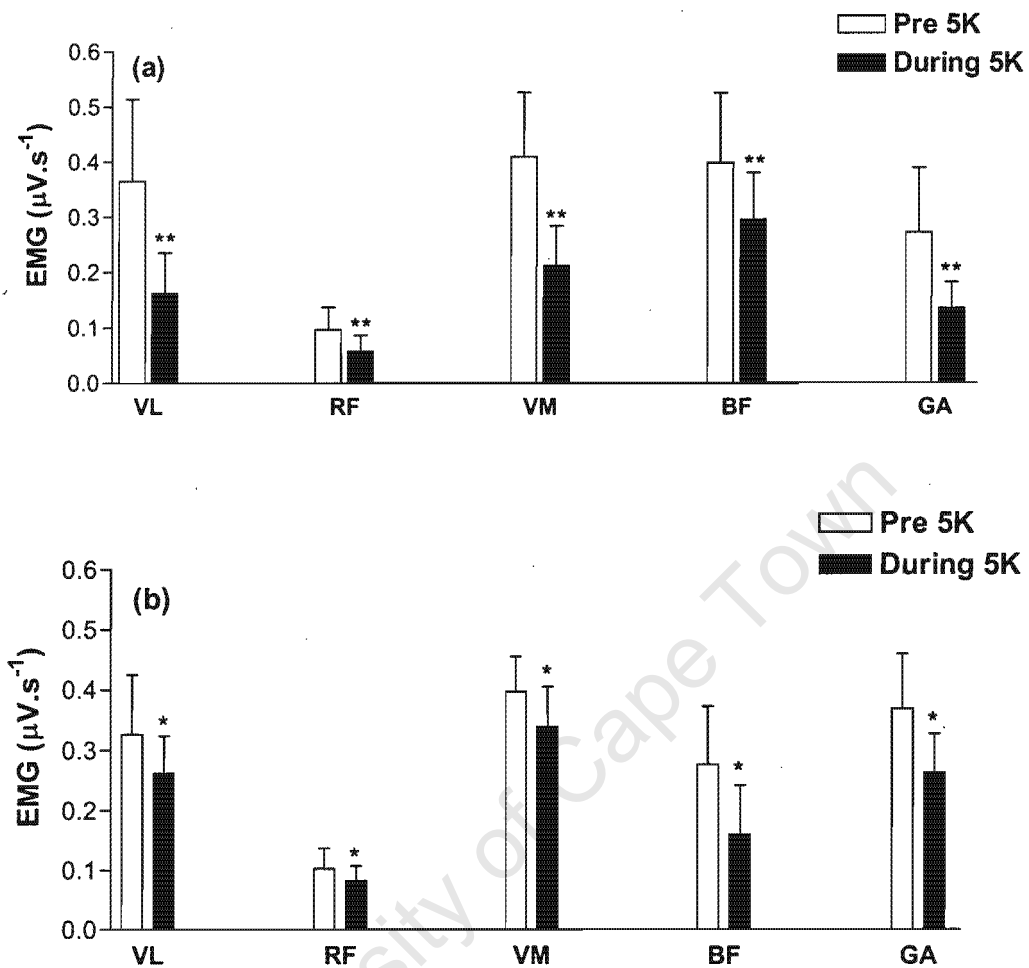


**Figure 7.1:** Changes in (a) 20 m sprint time, (b) ground contact time, (c) stride frequency and (d) stride length measured during the 20 m sprint test before the start of, and during the final lap of the 5 km time trial (\*\* $P < 0.0001$ , pre vs during).

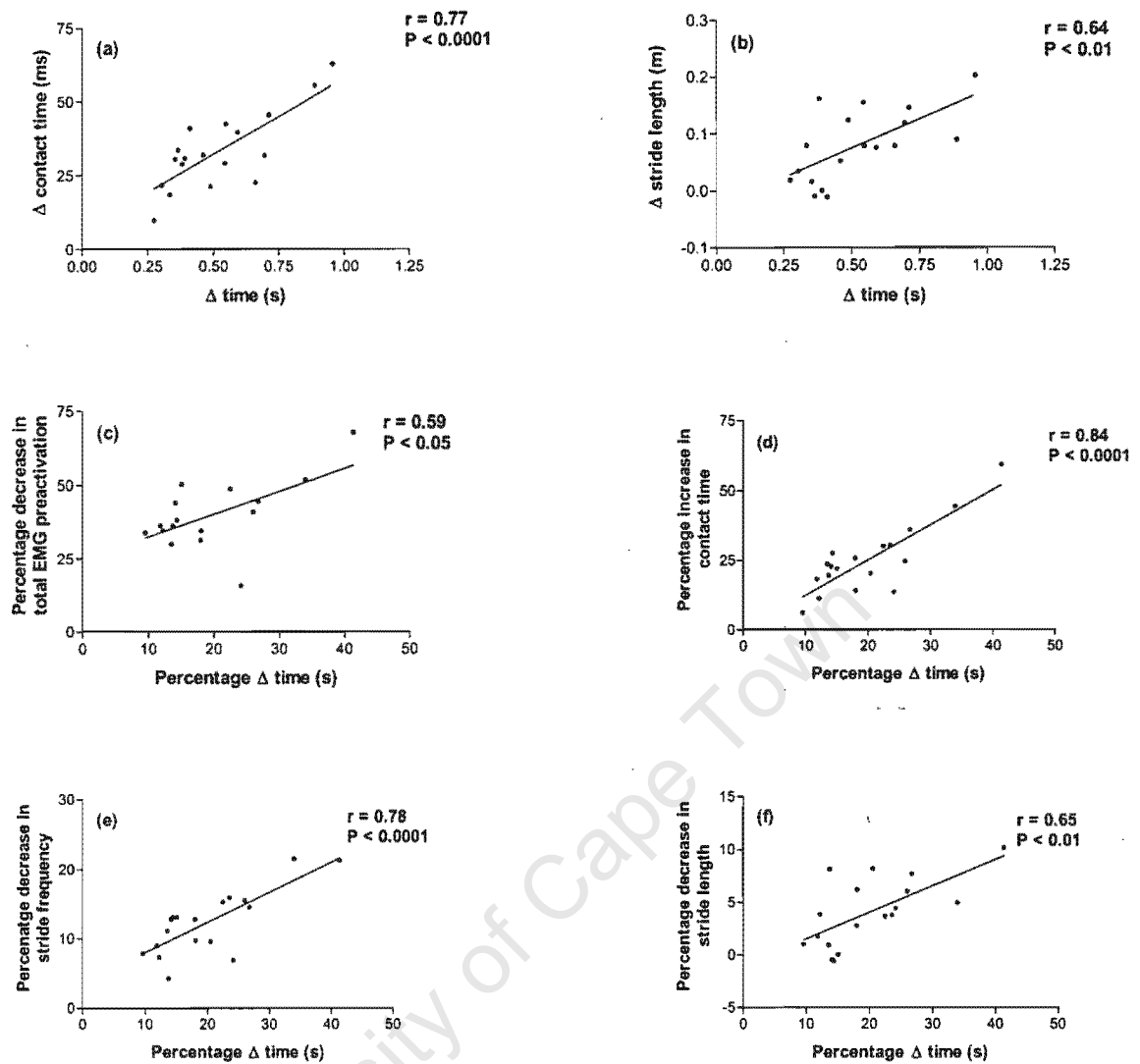
Mean EMG recorded from the vastus lateralis (VL), vastus medialis (VM), rectus femoris (RF), biceps femoris (BF) and gastrocnemius (GA) muscles decreased significantly ( $P < 0.0001$ ) (figure 7.2 a) during the preactivation phase. Similarly, EMG activity during the contact phase also decreased significantly in the second 20 m sprint ( $P < 0.01$ ) (figure 7.2 b).

Correlation analysis revealed that the change in 20 m sprint time was significantly related to the change in contact time ( $r = 0.77$ ,  $P < 0.0001$ ) (figure 7.3 a) and the change in stride length ( $r = 0.64$ ,  $P < 0.01$ ) (figure 7.3 b). In these figures, a greater change or percentage change in sprint time indicates a decrease in performance. The percentage change in the combined preactivation of all muscle groups (VL, VM, RF, BF, GA) was also significantly related to the percentage change in 20 m sprint time ( $r = 0.59$ ,  $P < 0.05$ ) (figure 7.3 c).

Related correlations were seen between the percentage change in sprint time and the percentage changes in contact time ( $r = 0.84$ ,  $P < 0.0001$ ) (figure 7.3 d); stride frequency ( $r = 0.78$ ,  $P < 0.0001$ ) (figure 7.3 e) and stride length ( $r = 0.65$ ,  $P < 0.01$ ) (figure 7.3 f).



**Figure 7.2:** Average EMG changes in the vastus lateralis (VL), rectus femoris (RF), vastus medialis (VM), biceps femoris (BF) and gastrocnemius (GA) muscles for all subjects during (a) the preactivation phase and (b) contact phase of the 20 m sprint before the start of and during the final lap of the 5 km time trial (\*  $P < 0.01$ , \*\*  $P < 0.0001$ , pre vs during).



**Figure 7.3:** Relationships between (a) the change in 20 m sprint time, between the initial and second 20 m sprint, and change in contact time, (b) the change in 20 m sprint time and change in stride length, (c) the percentage change in 20 m sprint time and the total combined percentage change in preactivation of vastus lateralis (VL), rectus femoris (RF), vastus medialis (VM), biceps femoris (BF) and gastrocnemius (GA) muscles, (d) the percentage change in 20 m sprint time and percentage change in contact time, (e) the percentage change in 20 m sprint time and the percentage change in stride frequency, and (f) the percentage change in 20 m sprint time and the percentage decrease in stride length.

Note that a positive change in 20 m sprint time indicates a decrease in performance.

### 7.3.2. Maximal voluntary contractions

Table 7.2 shows the changes in maximal and average force during the MVC. Average force during the MVC decreased significantly from  $881 \pm 242$  N to  $745 \pm 215$  N ( $P < 0.01$ ) after the 5K. Maximal force produced during the MVC decreased insignificantly from  $1107 \pm 247$  N to  $1051 \pm 308$  N (570), after the 5K.

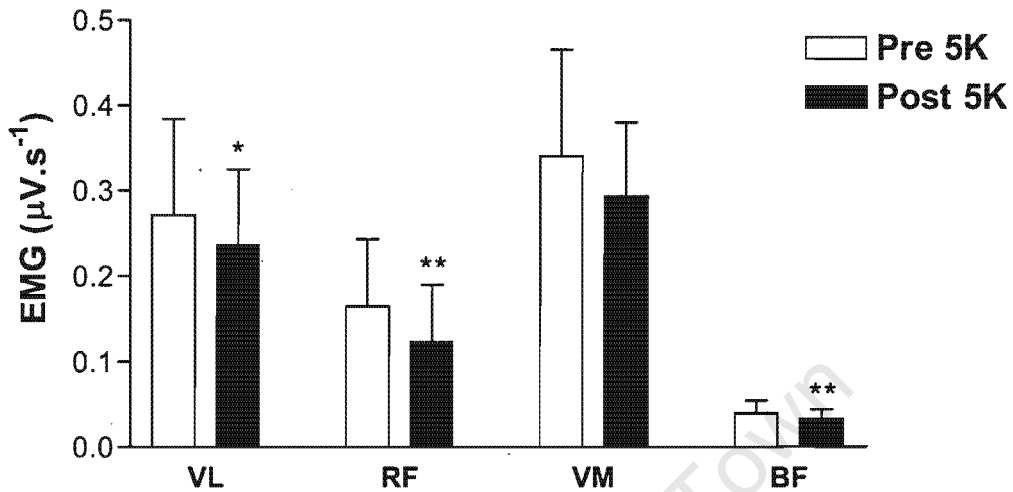
**TABLE 7.2:** Force changes in the maximal voluntary contraction (MVC) before and after the 5 km time trial.

VARIABLE	PRE 5K	POST 5K
	MEAN $\pm$ SD (range)	MEAN $\pm$ SD (range)
Maximal force (N)	$1107 \pm 248$ (697 – 1478)	$1051 \pm 308$ (521 – 1712)
Average force (N)	$882 \pm 243$ (464 – 1273)	$745 \pm 215$ ** (451 – 1205)

\*\*  $P < 0.01$ , pre vs post

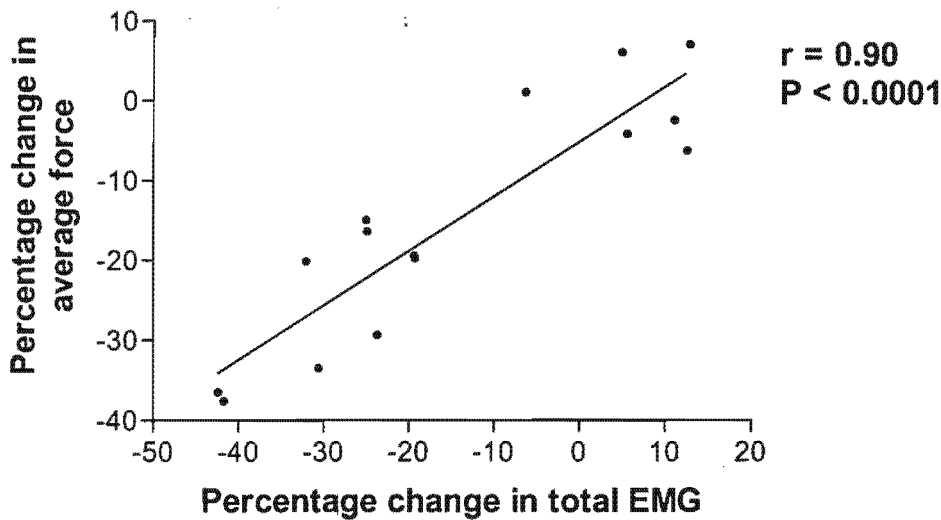
There was also a significant decrease in mean EMG, averaged over 5 seconds during the MVC, after the 5K (338). Integrated EMG was significantly lower after the 5K in VL ( $P < 0.05$ ), RF ( $P < 0.01$ ) and BF ( $P <$

0.01) and tended to decrease in VM although this latter change was not significant.



**Figure 7.4:** Average EMG changes in the vastus lateralis (VL), rectus femoris (RF), vastus medialis (VM) and biceps femoris (BF) muscles for all subjects in the maximal voluntary contraction (MVC) test before and after the 5 km time trial (\*  $P < 0.05$ , pre vs post; \*\*  $P < 0.01$ , pre vs post).

There was a positive linear correlation between the percentage change in average force production and the percentage change in average EMG recruitment during the MVC ( $r = 0.90$ ,  $P < 0.0001$ ) (389). This shows that approximately 81 % of the reduction in force during the MVC could be explained by a reduction in the EMG activity and, thus possibly, the extent of muscle recruitment.



**Figure 7.5:** Relationship between the percentage change in average force production and the total combined percentage change in EMG recruitment measured during the MVC performed before and immediately after the 5 km time trial.

#### 7.4. DISCUSSION

The first important finding of this study was that both the preactivation of the lower limb muscles (figure 7.2 a) and EMG activity during the ground contact phase of the maximal running stride (figure 7.2 b) decreased during a 5 km time trial. These findings are similar to those after a 400 m sprint (438), a 10 km time trial (448) and 42 km marathon (422).

These studies have all used exercise protocols of different intensities and durations to induce fatigue. The metabolic profiles associated with fatigue with these different protocols would also be different (425). Interestingly, despite the differences in metabolic profiles in each of these studies, the

changes in EMG activity with the onset of fatigue have been remarkably similar (23;422;438;448).

Associated with a decreased preactivation of the lower limb muscles is a decreased muscle stiffness and duration of the transition between the lengthening and shortening phases of the stretch shortening cycle (58; 301). Alterations to preactivation levels may then directly be associated with an increased ground contact time and an increase in the energy requirement during the propulsion phase of the running stride (58).

Accordingly, the second major finding of this study was that the decrease in stride length during the 20 m sprint test correlated with the reduction in sprint time (figure 7.3 b) and could be explained by the increase in ground contact times (figure 7.1 b, figure 7.3 a, figure 7.3 d). Similar findings were measured during a 400 m sprint (438) and can be explained by a prolongation of the transition time between the lengthening and shortening phases of the stretch shortening cycle associated with a reduction in the stored energy available for the next stride. Accordingly, stride length shortens (figure 7.1 d) as there is a decrease in both muscle stiffness and stored elastic energy, both of which assist in propelling the athlete a greater distance in subsequent strides.

Indeed figure 7.3 c shows that those athletes who developed the greatest reductions in preactivation also showed the greatest decline in sprint

performance during the 5 km time trial. These neuromuscular changes may therefore be associated with the alterations in running performance and fatigue that developed in this study.

The third finding of this study is that both EMG and force decreased significantly during the MVC performed immediately after the 5 km time trial. Whilst the analysis of EMG changes during an MVC following a fatiguing exercise does not measure the dynamic, temporal recruitment of different muscle groups, as was done in this study, similar force and EMG changes have been shown after fatiguing maximal leg extensions (353), high intensity cycling (40), and after a 65 km footrace (393). These studies have concluded that the reduction in EMG and the corresponding decrease in isometric force during an MVC represent central adjustments to fatigue (202). Similarly the changes in EMG activity shown during the MVC that followed the 5 km time trial in this study could also be interpreted as central neural fatigue.

Finally, there are a number of similarities between the findings of the present study and those performed previously (447;448) despite the differences in research subjects (orienteers compared to distance runners). This suggests that these findings represent a normal physiological response to running and occur in all athletes regardless of their speciality.

The results of this study show that EMG activity in the muscles of the lower limb is decreased during high intensity exercise of short duration lasting approximately 16 minutes. These changes in EMG are similar to those observed during both long duration endurance exercise and short duration, explosive exercise suggesting that there may be a common mechanism responsible for these alterations during running of any intensity or duration. Alterations in EMG activity are also present immediately after the completion of exercise, suggesting that there may be an alteration of the maximal recruitment potential that is associated with fatigue. These patterns of fatigue are closely related to changes in running performance since a decrease in neural preactivation has been associated with a reduction in muscle force production and mechanical efficiency. Collectively, these results suggest that running requires a unique and complex sequencing of muscle activation that can be altered after a relatively short intervention such as a 5 km time trial.

Finally, this study showed that the research protocol was able to detect relatively small neuromuscular changes after a short bout of high intensity running exercise. This protocol therefore has the potential to study neuromuscular changes in runners with a high volume of running training and racing.

**CHAPTER 8**

STUDY FOUR

**EMG ACTIVITY DURING A 5 KM TIME  
TRIAL IN MASTERS LONG DISTANCE  
RUNNERS**

## 8.1. INTRODUCTION

The results of the first study of this thesis (Chapter 3) showed that runners who have accumulated more than 5 000 km of racing have a different neural recruitment strategy to maintain maximal muscle function after a fatiguing intervention compared to runners who have raced significantly less. These same runners, however, are able to complete a submaximal run with no alterations in running economy and with similar oxygen consumption compared to the less experienced runners (Chapter 5).

These studies highlight two important issues. First, runners who are able to sustain a competitive training and racing career for over 20 years represent a unique subset of the running population and may be referred to as "resistant" runners. There are many runners who are incapable of training and racing the volumes that are reported by these highly motivated, resistant subjects. Although these "non-resistant" runners have not been studied systematically, their inability to tolerate a high volume of training and racing may be attributed to biomechanical factors, which predispose them to injury. Some runners may also have a susceptibility to develop pathological changes in their muscles (120;143;227;330;513), which effects muscle function, reduces their ability to train and results in them stopping marathon and ultra marathon races after relatively short careers.

Second, these findings raise the possibility that muscle recruitment patterns in runners may be altered, in some way, based on the level of exposure to a chronic running training stimulus. Indeed, it has been shown in the third study (Chapter 7), and by Nicol et al (421;422), that repeated stretch shortening cycle muscle action, as occurs during endurance running may modify neuromuscular behaviour so that the regulation either increases muscle stiffness or favours a damping mechanism. There are no studies, however, that have investigated whether this modification in neuromuscular function becomes a permanent alteration in runners who are exposed to ultra endurance running for many years.

Accordingly, the hypothesis for this study is that runners who have a history of high training and racing volume represent a group of "resistant" runners. In accordance with this hypothesis, these runners experience a centrally driven alteration, or down-regulation, in EMG recruitment patterns during weight bearing exercise, which acts as a protective mechanism against repetitive loading and damage to the muscle and tendon stretch reflex system. This down-regulation of preactivation results in a decrease in running speed, whilst protecting the muscle, enabling runners to continue training and racing in ultra distance events over an extended period of time.

Therefore, the aim of this study was to test this hypothesis and to identify whether there was a relationship between the volume of training and

racing and EMG recruitment patterns during a 5 km time trial in masters runners.

## **8.2. METHODOLOGY**

The experimental design for this study was similar to the methodology used in the previous study (section 7.2.2., page 184). Briefly, subjects ran a 5 km time trial during which the electromyographic activity of the vastus lateralis (VL), vastus medialis (VM), and gastrocnemius (GA) muscles were measured, and stride parameters (contact time, flight time, stride length, stride frequency) calculated. A maximal 20 m sprint was performed before and after the 5 km time trial.

This study did not include a maximal voluntary contraction (MVC) as described in the previous study (Chapter 7). Other differences in methodology between the previous study and this study are highlighted below.

### **8.2.1. Subject selection**

Eighteen male masters (45 - 65 years) endurance runners, who were currently training more than twice a week, were recruited to participate in this study. Subjects were divided into two groups based on their training

and racing histories. The experimental group consisted of 10 subjects who had been running for > 20 years, and the control group consisted of eight subjects who had been running for < 15 years.

### **8.2.2. Electromyographic activity and stride parameter measurements**

Before the start of testing, each subject had two EMG electrodes (Blue Sensor, Medicotest, Denmark) per muscle attached to the vastus lateralis (VL), vastus medialis (VM), and gastrocnemius (GA) muscles of the right leg. A one centimetre foot sensor (Norswitch bilateral telemetric footswitch system, Noraxon, Arizona, USA) was placed and securely taped onto the heel, the toe and the first and fifth metatarsals of the right foot. All EMG data were amplified and recorded telemetrically and simultaneously with the signals obtained from the foot sensors (Telemetry Research System 900, Noraxon, Arizona, USA).

The raw EMG signals were filtered using a 10 - 200 Hz bandwidth filter and integrated using the root mean square of 50 ms. Filtered, integrated EMG (iEMG) data were then time normalised for the two phases of running: preactivation (100 ms before ground contact) and total ground contact time (448).

### 8.2.3. Stride parameter calculations

Contact time, flight time, stride frequency and stride length were calculated using the data collected from the foot sensors placed onto the right foot during the 20 m sprints as follows:

$$\text{Contact time (CT) (ms)} = (\text{toe off} - \text{heel strike})$$

$$\text{Flight time (FT) (ms)} = [(\text{Right foot heel strike}_2 - \text{right foot heel strike}_1) - \text{CT}]/2$$

$$\text{Stride frequency (SF) (strides.s}^{-1}\text{)} = [1/(\text{CT} + \text{FT})]$$

$$\text{Stride length (SL) (m)} = (\text{velocity (m.s}^{-1}\text{)})/\text{SF}$$

### 8.2.4. Statistical analysis

Data from the total group, the experimental group and the control group were analysed using Statistica 5.5 (Statsoft, Inc., Oklahoma, USA). An ANOVA for repeated measures was used to determine the differences between the two groups over time and any possible interaction effects (group x time). A Pearson's product moment correlation coefficient determined relationships between variables (change in sprint time, change in contact time, change in stride length, change in preactivation, change in stride frequency, total accumulated racing mileage, total accumulated training mileage and current training distance). Values are expressed as mean  $\pm$  standard deviation. Statistical significance was accepted as  $P < 0.05$ .

To identify changes in total lower leg preactivation levels, the percentage change in preactivation data recorded from the VL, VM and GA was averaged and expressed as a single value, as described in Chapter 7.

### **8.3. RESULTS**

The descriptive and performance characteristics of the control, experimental and total groups of research subjects are shown in Table 8.1. There were no differences in descriptive or performance variables between groups. Total accumulated training and racing mileage and current training distances are shown in Table 8.2.

The subjects in the experimental group had accumulated significantly more mileage in both racing ( $P < 0.001$ ) and training ( $P < 0.0001$ ) and had been running for a significantly ( $P < 0.0001$ ) longer time compared to the control subjects. There were no differences between groups for current training distance. The total grouped data represent a range of racing and training distance from 590 – 26 183 km and 2 792 – 127 160 km respectively.

**TABLE 8.1:** Descriptive characteristics of control (n = 8), experimental (n = 10) subjects and total group average (n = 18). Values are expressed as mean  $\pm$  SD (range).

VARIABLE	CONTROL	EXPERIMENTAL	TOTAL
Age (years)	52.3 $\pm$ 5.8 (46.0 – 64.0)	54.4 $\pm$ 4.3 (46.0 – 62.0)	53.4 $\pm$ 5.0 (46.0 – 64.0)
Weight (kg)	68.3 $\pm$ 16.2 (48.9 – 99.1)	70.6 $\pm$ 6.1 (60.9 – 84.0)	69.6 $\pm$ 11.4 (48.9 – 99.1)
Height (cm)	170.8 $\pm$ 7.4 (161.5 – 185.0)	171.5 $\pm$ 4.3 (166.0 – 180.5)	171.2 $\pm$ 5.7 (161.5 – 185.0)
Lean thigh volume (cc)	3 399 $\pm$ 607 (2 242 – 3 945)	4 019 $\pm$ 927 (2 869 – 5 710)	3 744 $\pm$ 841 (2 242 – 5 710)
Sum of skinfolds	70.4 $\pm$ 39.2 (40.3 – 156.5)	76.1 $\pm$ 21.8 (49 – 115.8)	73.5 $\pm$ 29.9 (40.3 – 156.5)
Body fat (%)	18.0 $\pm$ 5.5 (13.2 – 27.8)	21.5 $\pm$ 3.9 (16.0 – 28.7)	20.0 $\pm$ 4.9 (13.2 – 28.7)
Lean body mass (kg)	55.4 $\pm$ 10.1 (41.5 – 71.6)	55.3 $\pm$ 3.5 (49.2 – 59.9)	55.3 $\pm$ 6.9 (41.5 – 71.6)
Maximum heart rate (b.min <sup>-1</sup> )	175 $\pm$ 10 (151 – 184)	169 $\pm$ 9 (154 – 186)	172 $\pm$ 10 (151 – 186)
VO <sub>2</sub> max (ml.kg <sup>-1</sup> .min <sup>-1</sup> )	48.0 $\pm$ 11.5 (31.9 – 61.5)	47.5 $\pm$ 5.5 (36.6 – 55.0)	47.7 $\pm$ 8.4 (31.9 – 61.5)
Peak treadmill running speed (PTRS) (km.hr <sup>-1</sup> )	16.6 $\pm$ 0.9 (15.0 – 18.0)	17.1 $\pm$ 1.9 (14.5 – 20.0)	16.9 $\pm$ 1.5 (14.5 – 20.0)
5 km velocity (m.s <sup>-1</sup> )	3.7 $\pm$ 0.5 (3.1 – 4.3)	3.9 $\pm$ 0.4 (3.3 – 4.3)	3.8 $\pm$ 0.4 (3.1 – 4.3)

**TABLE 8.2:** Total accumulated training and racing mileages, number of years running and current training distance for the control (n = 8) and experimental (n = 10) subjects and for the total subject group (n = 18).

VARIABLE	CONTROL	EXPERIMENTAL	TOTAL
Total kilometres raced	2 322 ± 2 388 (590 – 8 040)	11 232 ± 5 749* (6 194 – 26 183)	7 272 ± 6 371 (590 – 26 183)
Total kilometres trained	11 879 ± 5 438 (2 792 – 18 680)	78 584 ± 25 614** (43 756 – 127 160)	48 937 ± 39 023 (2 792 – 127 160)
Current training mileage (km.week <sup>-1</sup> )	45.6 ± 19.5 (20.0 – 75.0)	52.0 ± 8.5 (30.0 – 80.0)	49.2 ± 19.0 (20.0 – 80.0)
Total years running	6.4 ± 4.3 (2.0 – 14.0)	27.1 ± 5.6** (20.0 – 37.0)	17.8 ± 11.6 (2.0 – 37.0)

\*  $P < 0.001$  (experimental vs control)

\*\*  $P < 0.0001$  (experimental vs control)

### 8.3.1. Sprint time and stride parameters

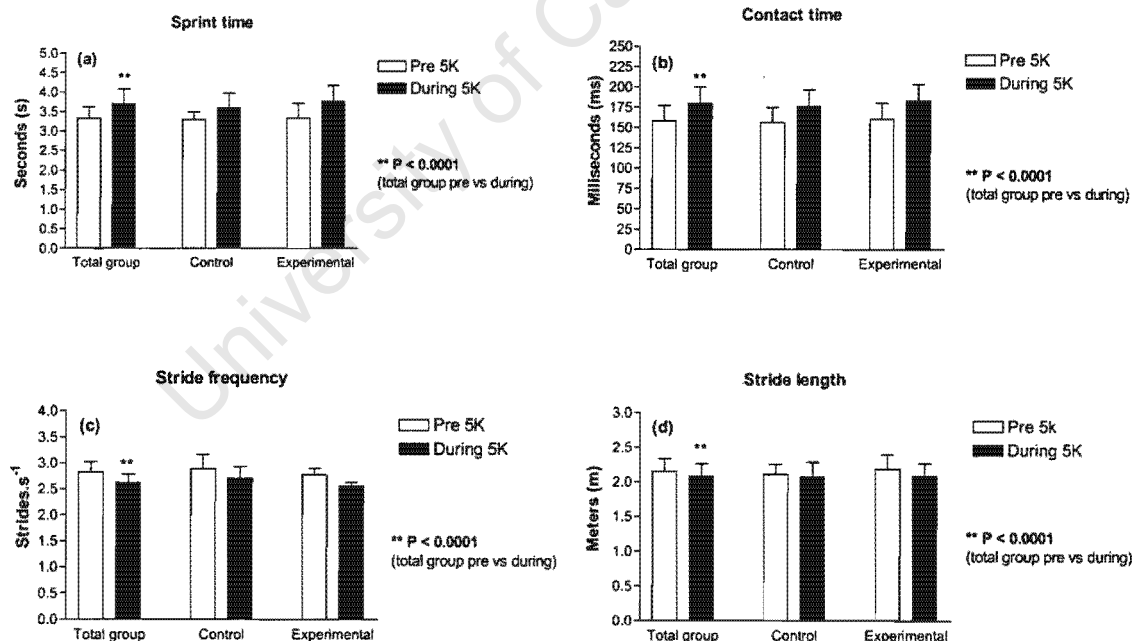
The 20 m sprint time for the total group increased significantly from  $3.32 \pm 0.38$  s in the pre 5K sprint to  $3.70 \pm 0.38$  s ( $P < 0.0001$ ) in the 20 m sprint during the 5K (figure 8.1 a). Sprint time increased by  $0.29 \pm 0.33$  s in the control group and by  $0.43 \pm 0.19$  s in the experimental group. There was a significant ( $P < 0.0001$ ) increase in contact time (figure 8.1 b) for the total group from  $158 \pm 19$  ms before the 5K to  $180 \pm 20$  ms during the 5K.

Contact time increased in the control group by  $20 \pm 19$  ms and increased

in the experimental group by  $23 \pm 19$  ms. There were no differences between groups for sprint time or total contact time.

Stride frequency and stride length decreased significantly ( $P < 0.01$ ) for the total group by  $0.2 \pm 1.5$  strides. $s^{-1}$  (figure 8.1 c) and  $0.07 \pm 0.11$  m (figure 8.1 d), respectively. There were no differences between the control and experimental groups for stride length and stride frequency. There were no changes in flight time for any of the groups (data not shown).

There were no differences between groups and no interactions of group  $\times$  time for any of these variables.



**Figure 8.1:** Changes in (a) 20 m sprint time, (b) contact time, (c) stride frequency and (d) stride length measured during the 20 m sprint test before the start of, and during the final lap of the 5 km time trial.

### 8.3.2. EMG activity – preactivation

Preactivation for the total subject group decreased significantly ( $P < 0.001$ ) by 27 % in the VL muscle (figure 8.2 a). There was a significant group x time interaction ( $P < 0.01$ ) for VL preactivation, suggesting that the preactivation decreased more (30 %) in the experimental group compared to the control group (26 %) (figure 8.2 a).

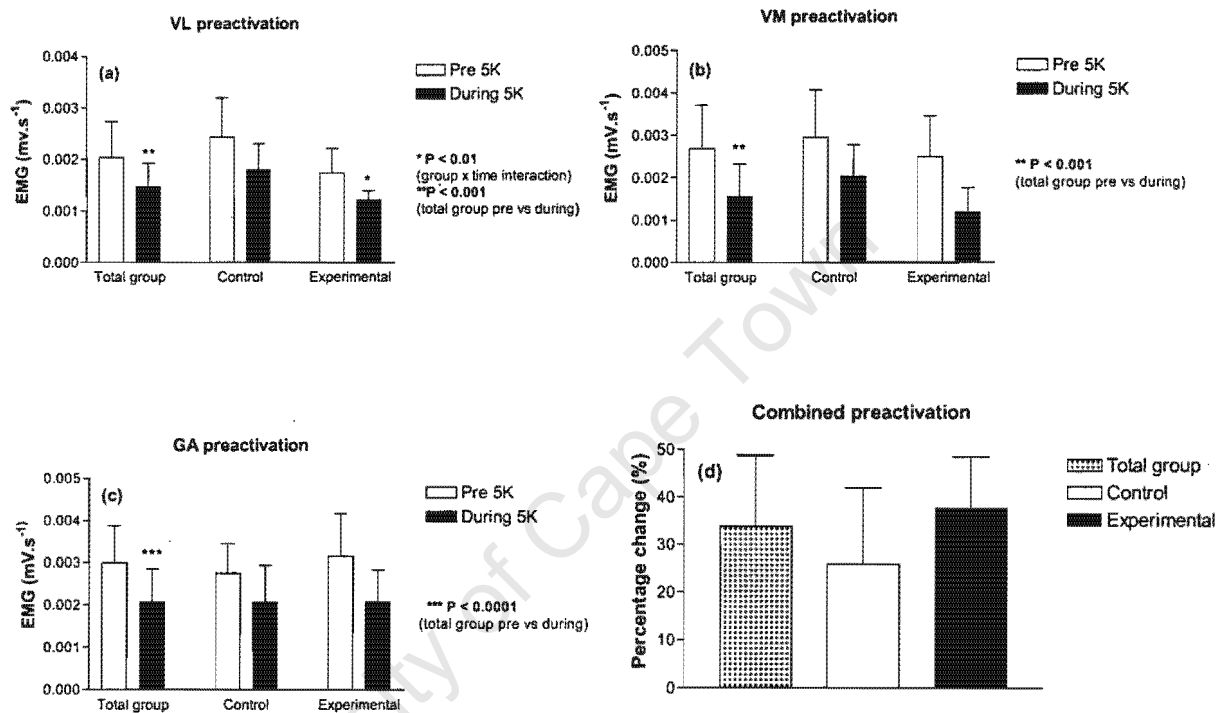
Preactivation for the total subject group decreased significantly by 42 % in the VM ( $P < 0.001$ ) (figure 8.2 b), and 31 % in the GA ( $P < 0.0001$ ) (figure 8.2 c) muscles. There were no significant interactions (group x time) for the VM and GA muscles.

Accordingly, these changes represent an overall decrease in total combined preactivation of 32 % for the total group, 27 % for the control group and 35 % for the experimental group (figure 8.2 d). These differences were not significant.

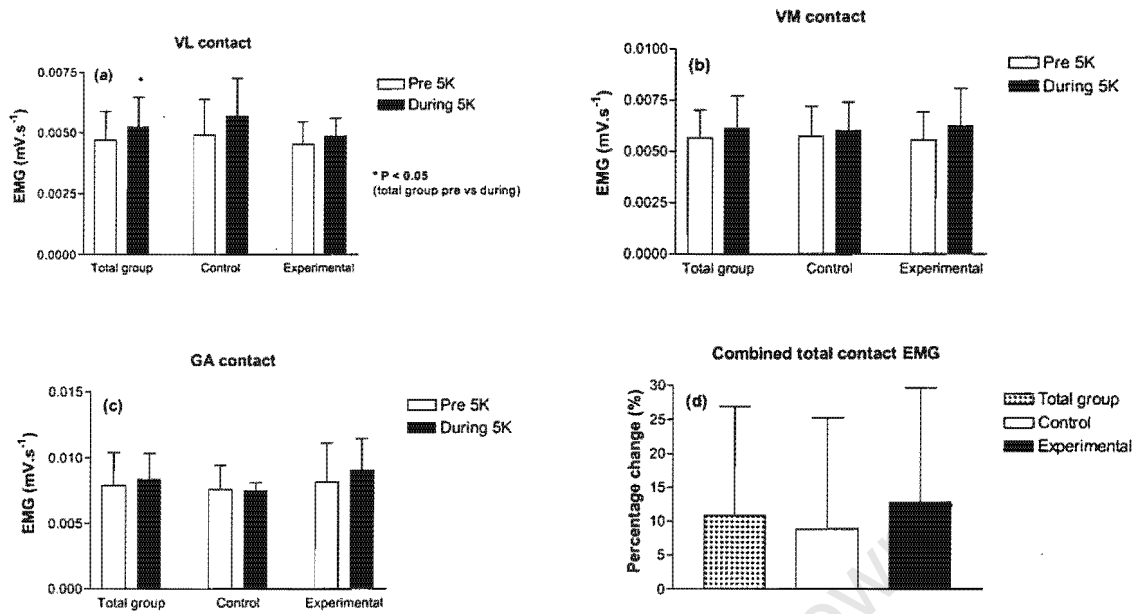
### 8.3.3. EMG activity – total contact EMG

EMG activity measured during the contact phase of running, increased significantly ( $P < 0.05$ ) for the total group in the VL muscle (figure 8.3 a) but there were no significant interaction (group x time) effects for the VL

muscle group. There were no significant time or group differences for the VM contact EMG activity and the GA contact EMG activity.



**Figure 8.2:** Changes in preactivation measured from the (a) vastus lateralis (VL), (b) vastus medialis (VM), (c) gastrocnemius (GA) muscles and (d) percentage change in combined VL, VM and GA preactivation during the 20 m sprint test before the start of, and during the final lap of the 5 km time trial.



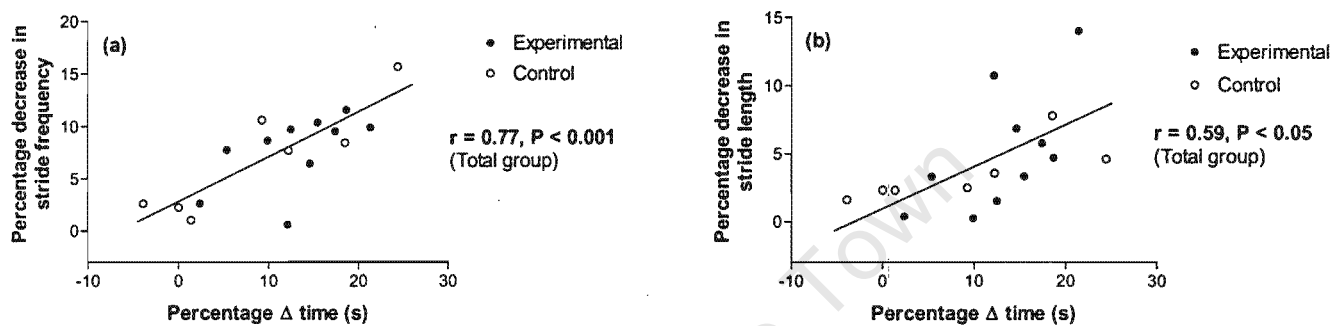
**Figure 8.3:** Changes in EMG activity during total contact time measured from the (a) vastus lateralis (VL), (b) vastus medialis (VM), (c) gastrocnemius (GA) muscles and (d) percentage change in combined VL, VM and GA contact time EMG during the 20 m sprint test before the start of, and during the final lap of the 5 km time trial.

The total contact EMG for the 3 muscle groups combined increased by 11 % for the total group, 9 % for the control group and 13 % for the experimental group (figure 8.3 d).

#### 8.3.4. Percentage changes

There were significant correlations for the total group between the percentage change in 20 m sprint time from before the 5 km time trial

and during the time trial and percentage decrease in stride frequency ( $r = 0.77$ ,  $P < 0.001$ ) (figure 8.4 a) for the total group. There were similar relationships between the change in 20 m sprint time and the percentage decrease in stride frequency for the experimental subjects ( $r = 0.68$ ,  $P < 0.05$ ) and the control subjects ( $r = 0.78$ ,  $P < 0.05$ ).



**Figure 8.4:** (a) Relationship between the percentage change in 20 m sprint time and the percentage decrease in stride frequency for the total group ( $r = 0.77$ ,  $P < 0.001$ ). This relationship was also significant for experimental subjects ( $r = 0.68$ ,  $P < 0.05$ ) and control subjects ( $r = 0.78$ ,  $P < 0.05$ ) when they were analysed separately. (b) Relationship between the percentage change in 20 m sprint time and the percentage decrease in stride length for the total group ( $r = 0.59$ ,  $P < 0.05$ ). There was a similar relationship between these variables for the control group ( $r = 0.91$ ,  $P < 0.01$ ), but not for the experimental subjects ( $r = 0.52$ , NS) when analysed separately.

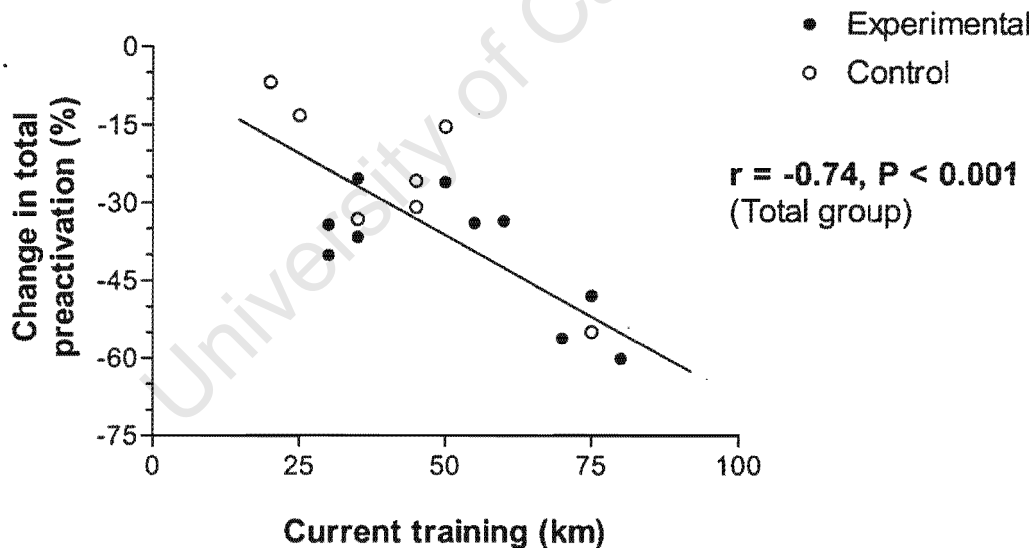
Note that a positive change in 20 m sprint time indicates a decrease in performance.

There was also a significant relationship between the percentage change in 20 m sprint time and percentage decrease in stride length (figure 8.4 b) for the total group ( $r = 0.59$ ,  $P < 0.05$ ) and the control group ( $r = 0.91$ ,  $P < 0.01$ ), but not for the experimental group.

A positive change in 20 m sprint time indicates a decrease in performance, such that subjects experiencing the greatest increases in sprinting speed (decreased performance) also showed the largest changes in stride frequency.

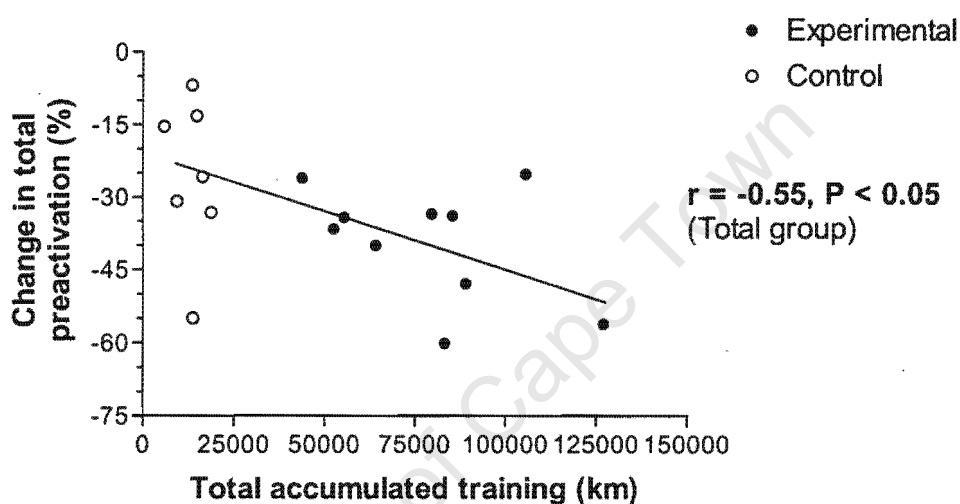
### 8.3.5. Accumulated training and racing distance correlations

There were significant inverse correlations between the percentage change in combined preactivation and current training distance for the total group ( $r = -0.74$ ,  $P < 0.0001$ ), the experimental group ( $r = -0.78$ ,  $P < 0.05$ ) and the control group ( $r = -0.84$ ,  $P < 0.05$ ) (figure 8.5).



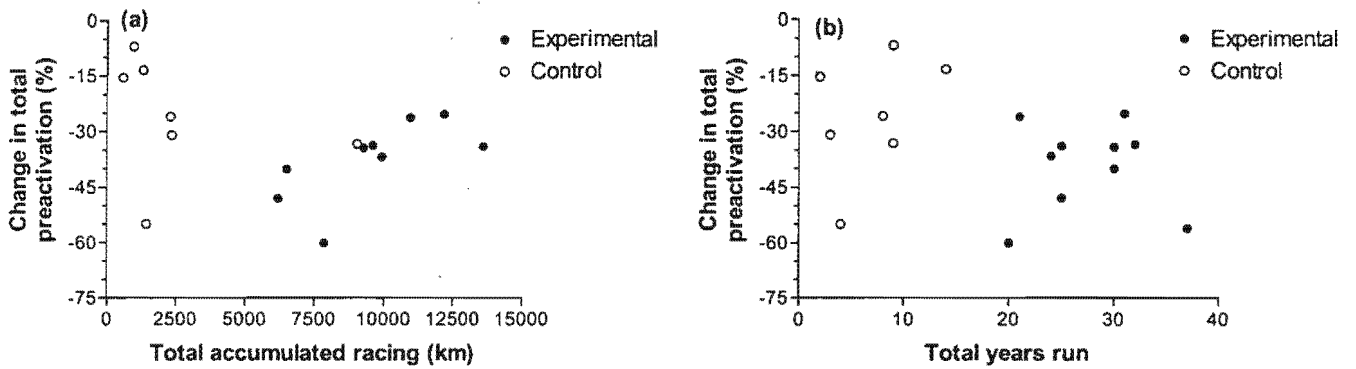
**Figure 8.5:** Relationships between the percentage change in total combined lower limb preactivation and current training distance for the total group of subjects ( $r = -0.74$ ,  $P < 0.001$ ). This relationship was also significant for the experimental ( $r = -0.78$ ,  $P < 0.05$ ) and the control ( $r = -0.84$ ,  $P < 0.05$ ) groups when they were analysed separately.

There was also a significant inverse correlation between the percentage change in total preactivation and total accumulated training mileage for the total group ( $r = -0.55$ ,  $P < 0.05$ ) (figure 8.6). There were no significant correlations between the percentage change in preactivation and total accumulated training distance for the experimental group or the control group when data from these groups were analysed separately.



**Figure 8.6:** Relationship between the percentage change in total preactivation and total accumulated training mileage in the total group of subjects ( $r = -0.55$ ,  $P < 0.05$ ). This relationship was not significant in the experimental and control groups when the groups were analysed separately.

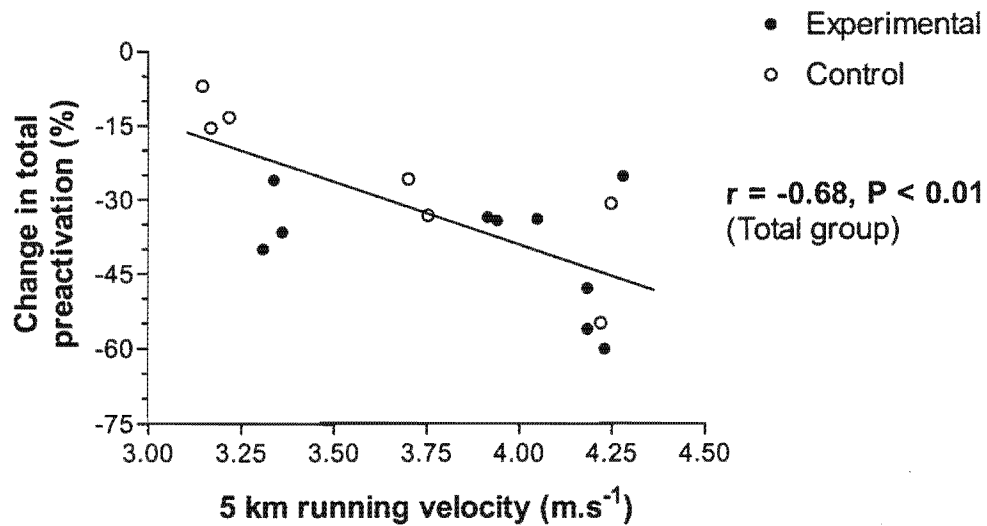
There were no significant relationships between the change in total preactivation and total accumulated racing distance (figure 8.7 a) and the total number of years running (figure 8.7 b) for any of the subject groups.



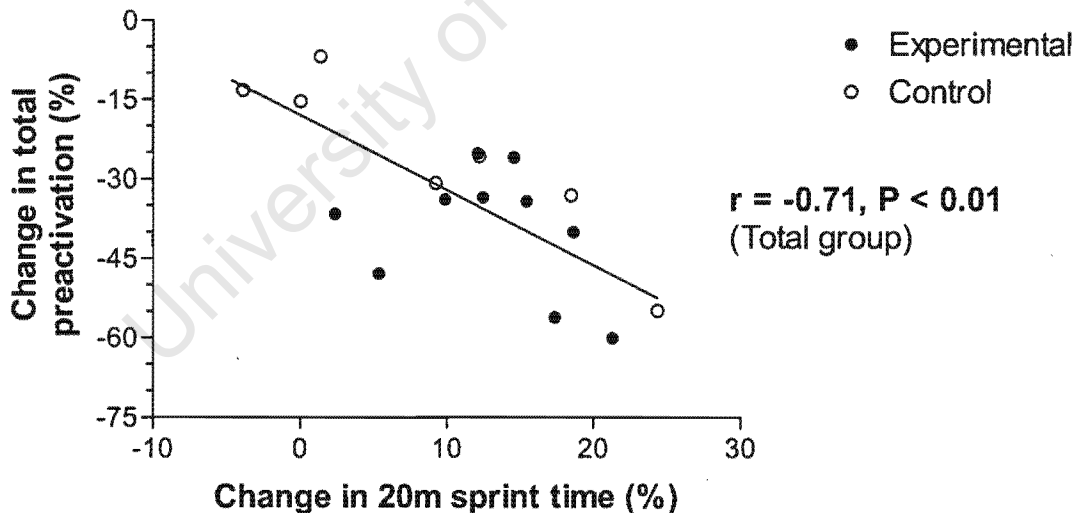
**Figure 8.7:** The relationship between the percentage change in total preactivation and (a) total accumulated racing distance and (b) total years of running in the total group of subjects. These relationships were not significant for any of the subject groups.

There was a significant inverse correlation between the change in preactivation and 5 km running velocity for the total group ( $r = -0.68$ ,  $P < 0.01$ ) (figure 8.8). When the groups were analysed separately, there was an inverse relationship between the change in preactivation and 5 km running velocity for the control group ( $r = -0.88$ ,  $P < 0.01$ ), but not for the experimental group.

The percentage change in preactivation also had a significant inverse relationship with the change in sprint time for the total group ( $r = -0.71$ ,  $P < 0.01$ ) and the control group ( $r = -0.92$ ,  $P < 0.01$ ) but not for the experimental group (figure 8.9).

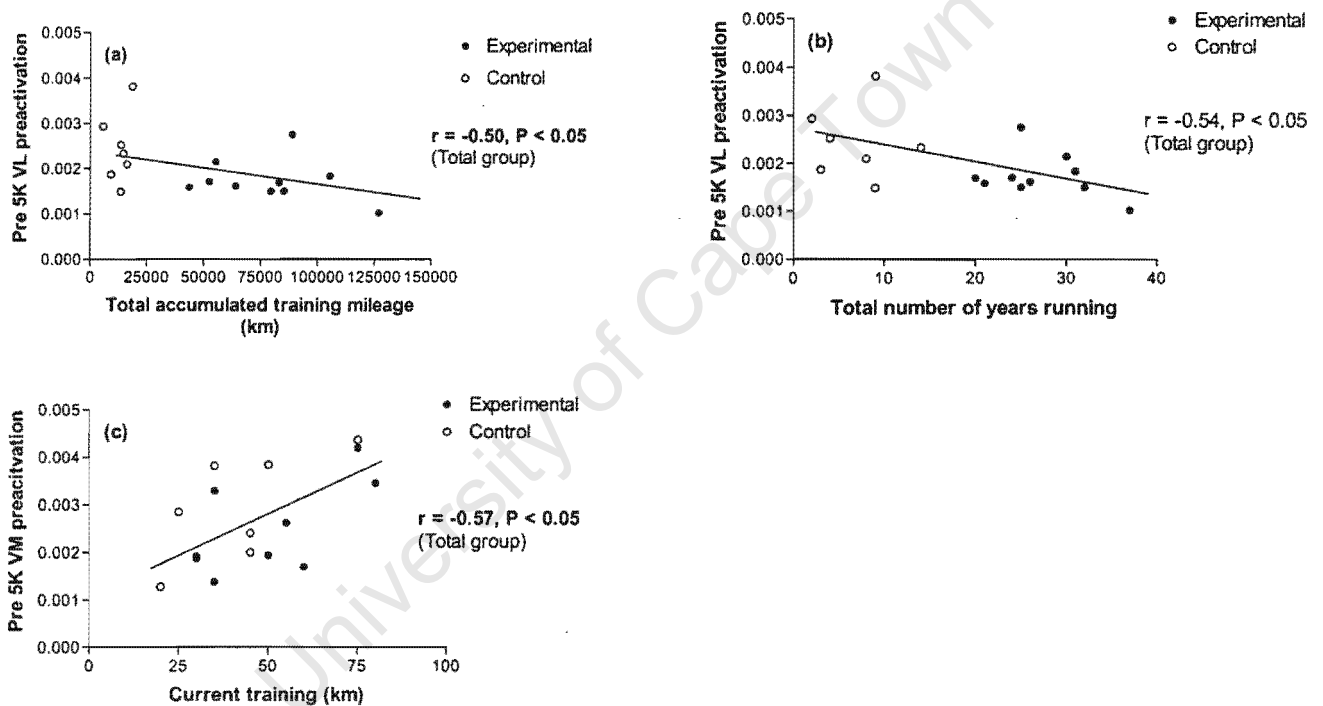


**Figure 8.8:** Relationship between the percentage change in total preactivation and 5 km running velocity in the total group of subjects ( $r = -0.68, P < 0.01$ ). This relationship was significant for the control subjects ( $r = -0.88, P < 0.01$ ), but not for the experimental subjects when the groups were analysed separately.



**Figure 8.9:** Relationship between the percentage change in total preactivation and percentage change in 20 m sprint time for the total group of subjects ( $r = -0.71, P < 0.01$ ). This relationship was significant for the control subjects ( $r = -0.92, P < 0.01$ ) but not for the experimental subjects when these groups were analysed separately.

There was a significant negative correlation between VL preactivation measured during the pre 5K sprint test and total accumulated training mileage ( $r = -0.50$ ,  $P < 0.05$ ) (figure 8.10 a) and total number of years running ( $r = -0.54$ ,  $P < 0.05$ ) (figure 8.10 b) for the combined group. The total group of subjects also showed a significant relationship between VM preactivation measured before the 5K and current training distance ( $r = -0.57$ ,  $P < 0.05$ ) (figure 8.10 c).



**Figure 8.10:** Relationships between pre 5K preactivation measured from the VL muscle and **(a)** total accumulated training mileage ( $r = -0.50$ ,  $P < 0.05$ ) and **(b)** total number of years running ( $r = -0.54$ ,  $P < 0.05$ ) and **(c)** the relationship between pre 5K preactivation measured from the VM and muscle current training mileage ( $r = -0.57$ ,  $P < 0.05$ ) for the total subject group. There were no significant relationships between any variables when the experimental and control groups were analysed separately.

#### 8.4. DISCUSSION

The most important finding from this study was that the 5 km time trial was successful in causing significant fatigue in all athletes. This was illustrated by increases in running speed (figure 8.1 a), increases in total contact times (figure 8.1 b) and decreases in stride frequency (figure 8.1 c) and stride length (figure 8.1 d). In addition, there were significant reductions in preactivation in all muscles that were measured (figure 8.2) and increases in EMG measured from the VL muscle group during the ground contact phase.

The increase in 20 m sprint time for the total group could be explained by increases in ground contact times (figure 8.1 b) and decreases in stride frequency (figure 8.1 c) and stride length (figure 8.1 d), and confirm the findings of Paavolainen et al (448), Nummela et al (438) and the previous chapter (figure 7.1, page 191), suggesting that neuromuscular characteristics and EMG activity of the lower limbs are altered during high intensity exercise of short duration. These findings further support the suggestion that there may be a common neuromuscular alteration associated with running of differing intensities and durations (Chapter 7) as similar neuromuscular changes have also been observed during both long duration endurance exercise (23;393;422) and explosive sprinting (438).

In addition, similar to the findings of the previous study (Chapter 7), the decreases in stride frequency (figure 8.4 a) and stride length (figure 8.4 b) during the 20 m sprint test correlated with the reduction in sprint time, and could be explained by the increase in ground contact times (figure 8.1 b). Similar findings have been measured during a 400 m sprint (438) and can be explained by a prolongation of the transition time between the lengthening and shortening phases of the stretch shortening cycle associated with a reduction in the stored energy available for the next stride. Accordingly, stride length shortens (figure 8.1 d, figure 8.4, figure 8.5) as there is a decrease in both muscle stiffness and stored elastic energy, both of which assist in propelling the athlete a greater distance in subsequent strides.

An alternative suggestion is that this process is reversed and that the reduction in stride frequency occurs in order to decrease the high impact forces on landing, thereby reducing the need for high levels of preactivation. It is likely that these two possibilities are reciprocally linked and act together to reduce the forces that occur on landing. What is unclear, however, is which mechanism occurs first – the reduction in preactivation or the reduction in stride frequency. In both cases, the function of the centrally regulated reductions in stride frequency and preactivation remains constant, a protective mechanism against excessive muscle damage and injury.

Alterations to EMG activity during running are closely related to changes in performance, since a reduction in preactivation has been associated with a reduction in muscle force production (421;448), mechanical efficiency (421;448) and running velocity (figure 7.3 c, page 194). Indeed, those runners who developed the greatest reductions in preactivation also showed the greatest decline in sprint (figure 8.8) and overall performance (figure 8.9) during the 5 km time trial.

Interestingly, although there were no differences between groups, when analysed along a continuum (figures 8.5, 8.6, 8.7 and 8.10), athletes who had been exposed to low volumes of training responded differently to athletes who had accumulated high volumes in training and racing. The results from the first study (Chapter 3) showed that athletes who had accumulated more than 5 000 km in endurance racing, demonstrated no consistent changes in EMG activity during a maximal voluntary contraction following a 40 minute downhill run, whilst the less experienced runners showed close relationships between variables following the run. Similarly, in this study, those athletes who had not accumulated high mileage in training and racing were more consistent in their responses to a 5 km time trial and showed stronger relationships and correlations between the different variables, compared to athletes who had been exposed to many years of competitive running. This provides further evidence to suggest that this group of “resistant” runners have significant alterations in muscle activation strategies during a fatiguing intervention.

Both age (255) and training (444;446;549;557) have been shown to affect muscle preactivation. It has been well documented that aging in human skeletal muscle is closely associated with changes in the integrity of the neuromuscular system including alterations to motor unit numbers and size, (123;168;325;325;480;481;537;550). There is also some evidence to suggest that age may have a positive affect on preactivation (236;255) as the slowed contractile properties of aged muscles may make it more effective for the nervous system to regulate stiffness characteristics. However, subjects recruited for this study were of similar age, and thus changes associated with an increase in age cannot fully explain the differences in neuromuscular characteristics between these athletes.

A more likely explanation for the range in neuromuscular response seen in this study is that training-induced changes in reflex potentiation (487), affecting the subsequent motor unit recruitment, may have taken place (239;487). An effective prestretch during the stretch shortening cycle causes substantial stretch reflex potentiation via Ia afferents from the muscle spindles (468). This reflex potentiation, together with an increased motor neuron activity to the contracting muscles results in an increase in muscle force production at the end of the lengthening phase and will thus contribute to the maintenance of optimal neuromuscular functioning.

Figure 8.5 shows the relationship between the change in preactivation during the 5 km time trial and current training mileage. Interestingly, there was a similar response in the experimental and control groups. This finding suggests that there may be consistent alterations to the regulation of preactivation that occurs either as an initial response to endurance exercise training, or in response to current training mileage, in all athletes, regardless of their exposure to high mileage training.

There are no studies on the effect of regular endurance training on muscle preactivation. However, studies on explosive strength training (444;446;549;557) have shown that athletes who undergo this specific type of training have higher preactivation levels compared to control subjects. These findings suggest a possible acute training induced increase in muscle stiffness regulation and stretch reflex sensitivity associated with low mileage, high intensity training, and support the findings from this study. However, as the training volume is increased, the regulation of muscle preactivation will be reduced.

Indeed, figure 8.6 illustrates that in the total group of athletes with varying exposure to endurance training, those athletes who had accumulated a high volume of training and racing, experienced more variation in muscle preactivation over a 5 km time trial compared to less experienced runners. This finding suggests that in addition to an acute alteration in preactivation regulation associated with weekly training, there may also

be a more long term and chronic response to endurance training, which results in an altered regulation of preactivation.

Although this study is cross sectional in design and thus cannot illustrate possible longitudinal changes associated with endurance training, grouped data showed that those athletes who had accumulated a high training volume (figure 8.10 a) and who had been running for many years (figure 8.10 b) showed lower levels of VL preactivation, whilst athletes who had higher weekly training distances had higher levels of VM preactivation in the 20 m sprint before the 5K (figure 8.10 c). These results provide some evidence to suggest that endurance training may increase the regulation of preactivation in particular muscle groups, but that this potentiation may have a "critical" point, after which high weekly training mileage accumulated over a number of years may contribute to a reduction in preactivation. Interestingly, it appears that preactivation in different muscle groups may be affected by a different quality of training stimulus (figure 8.10). These findings confirm that training and racing impose differing stressors to each muscle group (513) and highlight the possibility that each of these muscle groups have a specific role in facilitating an efficient running cycle. Specifically, it would appear that the VL muscle might have a predominant role in the shock absorption function of the knee extensor muscles.

An alternative explanation to explain the differences between athletes exposed to differing levels of training and racing could be related to inherent, or training induced, alterations to skeletal muscle morphology (554) and muscle fiber composition (63;407). Moritani et al (407) and Bosco et al (63) have shown that slow twitch and fast twitch muscle fibers are characterised by different visco-elastic properties such that there are specific muscle fiber dependent electrophysiological responses to exercise, and that predominantly fast twitch muscle groups are affected by fatigue to a greater degree than predominantly slow twitch fibers (63;407;554). Based on the methodology used in this study, however, this suggestion cannot be confirmed.

## **8.5. CONCLUSION**

Under conditions of high volume endurance training and racing, the role of muscle preactivation in altering muscle stiffness to buffer high impact forces is highlighted. These findings also suggest that runners who have not experienced years of competitive racing and training are able to maintain an explosive muscle power to a greater extent compared to more experienced runners, and thus appear to be more efficient in their ability to resist fatigue during a 5 km time trial. An alternative interpretation is that these findings could be a muscle activation strategy, initiated by the central nervous system, unique to those athletes who remain

competitive for many years and which acts as a protective mechanism against muscle damage and injury. However, based on the data collected in this study, we are unable to conclude whether these alterations are an adaptation to many years of endurance training, or a prerequisite to it.

The following chapter will discuss a proposed model, based on the findings in this thesis, to characterise this unique group of masters athletes.

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## CHAPTER 9

# PROPOSED MECHANISMS DEVELOPED FROM THE STUDIES IN THIS THESIS

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## 9.1. INTRODUCTION

There is a decline in athletic performance after the age of approximately 40 years (54;329;352;426;466) that has been associated with physiological alterations that occurs in skeletal muscle with an increase in age.

Participation in regular physical activity results in improvements in health profiles and may also be able to slow the rate of age-related decline in endurance performance. However, there is evidence to suggest that, in some runners, chronic exercise training may reduce performance at a faster rate than that predicted for their age. The principle aim of this thesis was to identify a relationship between exposure to a chronic, endurance training stimulus and alterations in neuromuscular characteristics that may be associated with this decline in performance.

This thesis has identified a unique group of athletes who had a history of high volume training and racing and therefore represented a group of runners who are, by definition, relatively resistant to the stresses of long distance running. These athletes were able to maintain a competitive running training and racing schedule for more than 20 years. These athletes were in contrast to other athletes who, after a few years, are forced to withdraw from high volume training and competitive racing because of injury or because of a vulnerability to develop pathological changes in their skeletal muscle (120;143;227;513).

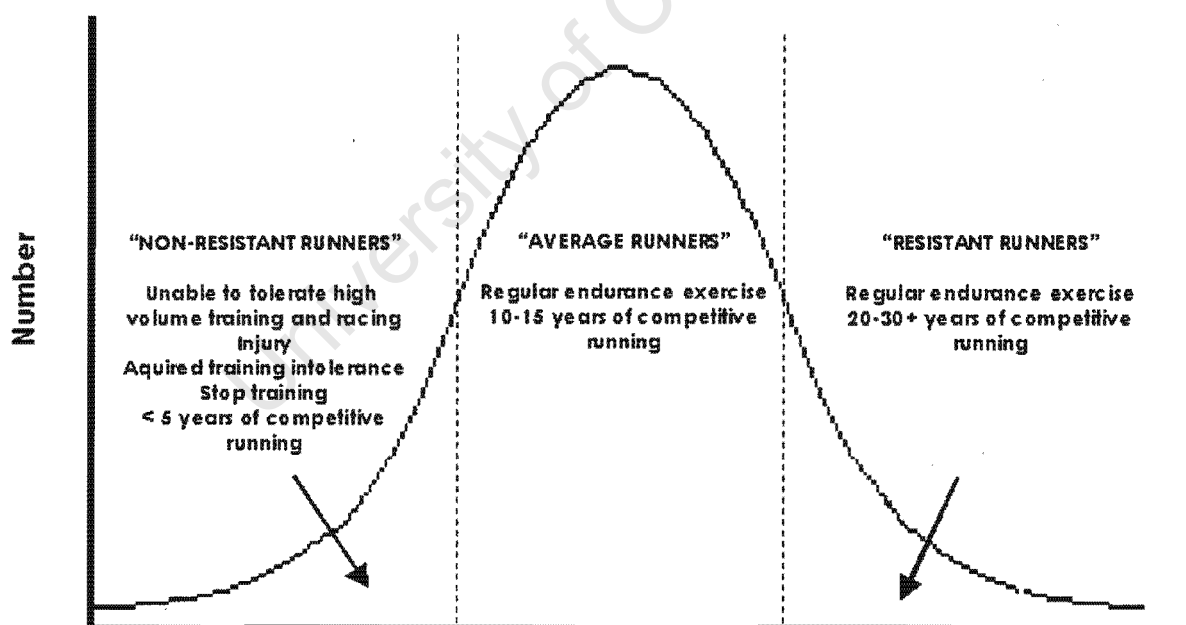
Compared to inexperienced runners of the same age, these "resistant" runners display similar aerobic profiles (Chapter 5), yet showed altered neuromuscular characteristics during and after fatiguing interventions (Chapters 3 and 8). These neuromuscular alterations have been interpreted as a compensatory mechanism that acts to protect the musculotendinous system against excessive damage associated with high muscle lengthening forces, which occur at heel strike during running.

The series of studies in this thesis has led to the theory, which still has to be subjected to rigorous testing, that athletes who participate in regular endurance training lie along a continuum, illustrated below in figure 9.1. In accordance with this theory, the centre of the curve is occupied by "average" runners who enjoy a productive running career lasting between 10 – 15 years (329;426). These runners usually withdraw from relatively high volumes of training and racing after this time period after sustaining and recovering from minor injuries.

To the left side of the curve are "non-resistant" athletes who are only able to tolerate high volume training and racing for approximately 5 years before sustaining repetitive musculoskeletal injuries. It is proposed that these athletes therefore either choose to stop training, or attempt to continue with high volume training in the presence of injuries and symptoms of over training. It is believed that these athletes develop "acquired training intolerance" with symptoms such as chronic fatigue, muscle pain and

exercise intolerance (143;227) that will ultimately force them to discontinue high mileage training.

The athletes on the right of the curve represent the group of "resistant" runners discussed in this thesis. These athletes are able to maintain a high volume of competitive training and racing and remain competitive in their age-groups for an extended period of time. Indeed, these athletes are unique in their ability to endure many years of accumulated endurance training without incurring serious and chronic injuries or biomechanical alterations that otherwise would force them to retire, as perhaps occurs in the "non-resistant" and "average" runners.



**Figure 9.1:** Speculation on the distribution of "non-resistant", "average" and "resistant" runners.

Although this is a theoretical model which still needs to be tested, it can serve as a basis for a discussion on the proposed mechanisms explaining the interaction between high volume training and racing and increasing age.

## **9.2. PROPOSED MODEL TO CHARACTERISE "RESISTANT" RUNNERS**

Based on findings of this thesis, the following model, illustrated by figure 9.2 is proposed. This model characterises the runners on the right and left extremes of the curve in figure 9.1, who represent "resistant" and "non-resistant" runners respectively.

### **9.2.1. The acute training response**

In response to an acute training stimulus, stiffness regulation of the muscle is altered. This alteration occurs in response to the high stresses associated with heel strike and landing during repetitive stretch shortening cycle exercise. Interestingly, this response seems to be consistent for all athletes, irrespective of their exposure to high volume training and can be illustrated by the relationship between the change in muscle preactivation levels during a 5 km time trial and current weekly training distance (figure 8.5, page 215).

The continual interaction between afferent and efferent signals to and from the central nervous system and skeletal muscle regulates recruitment patterns to alter muscle stiffness. These alterations result in changes to select stride parameters (figure 7.1, page 191 and figure 8.1, page 210). This can be interpreted as a buffering system against high impact forces, thereby providing some degree of protection against the possibility of chronic muscle damage.

### **9.2.2. Chronic endurance training and racing**

As the training and racing distance and intensity is increased, further adaptations in the neuromuscular system are required to protect against the risk of accumulative muscle damage. Aura and Komi (19) have raised the question that although an increase in EMG during the preactivation and lengthening phases of the stretch shortening cycle infers an improvement in muscle stiffness, there must be an upper limit to these stiffness characteristics. Indeed, the results of the studies in this thesis have shown that both stiffness regulation and selected stride parameters are affected by training distance over several years (figure 8.1, page 210 and figure 8.6, page 216).

It is proposed that these alterations are central in origin and are unique to "resistant" runners (figure 9.1). In addition, it may be proposed that there are two possibilities to explain the source of these alterations.

### 9.2.2.1. Adaptations to central regulatory mechanisms

This scenario suggests that as a direct consequence of many years of chronic endurance running training and racing, there is an adaptation within the central nervous system of some runners. This adaptation down-regulates EMG activity, and acts as a protective mechanism against repetitive loading and damage to the muscle and tendon system.

### 9.2.2.2. Intrinsic central regulatory mechanisms

The second scenario suggests that in some runners, there is an innate central mechanism that carefully regulates neuromuscular control and muscle recruitment. This mechanism is a prerequisite for any runner to progress to become a "resistant" runner.

It is also likely that there may be an interaction between these two scenarios, such that athletes who have the intrinsic capacity for down-regulating muscle recruitment may also be more likely to adapt to the chronic stresses of repetitive stretch shortening cycle exercise.

In both instances, it is suggested that the decline in motor unit activation is a reflex, which depends on signals from the contracting muscle. In this way, it may be possible, that the nervous system makes attempts to compensate for mechanical deficiencies by changing the stretch reflex activity, resulting

in alterations in stiffness regulation and changes to select stride parameters. This compensation attenuates the effect of fatigue on preactivation, thereby enhancing the shock absorption capacities and improving the ability of the muscle to buffer high impact forces associated with the foot landing on the ground.

Although this mechanism would then allow these runners to continue with high mileage training and racing, a reduction in muscle preactivation and neural input to the muscle decreases the efficiency of the contractile mechanisms (decreased preactivation). Consequently, contact times become longer (figure 8.1, page 210) and there is a reduction in ground reaction forces, ultimately resulting in a longer transition time between lengthening and shortening phases. These changes imply a reduced ability to store and use elastic energy, which may result in a decrease in running speed over an extended period of time.

### **9.3. RUNNERS WHO DEVELOP "ACQUIRED TRAINING INTOLERANCE" ("NON-RESISTANT RUNNERS")**

Although this group of athletes was not studied, the following assumptions can be made. Runners who do not have the regulatory mechanism of the neuromuscular system are unable to adapt to high volume training. These athletes experience high forces on landing, as preactivation and stretch reflex components in these runners may be insufficiently downregulated.

These high landing forces are associated with active lengthening muscle damage, resulting in injury and long term pathological changes to muscle morphology (120;227), often rendering these athletes unable to compete. These athletes represent the “non-resistant” running population illustrated on the left of the curve in figure 9.1. They subsequently either develop symptoms associated with “acquired training intolerance” (120;227), or they are forced to stop training and competing.

This proposed model of “resistant” and “non-resistant” runners obviously needs further investigation and still needs to be studied systematically

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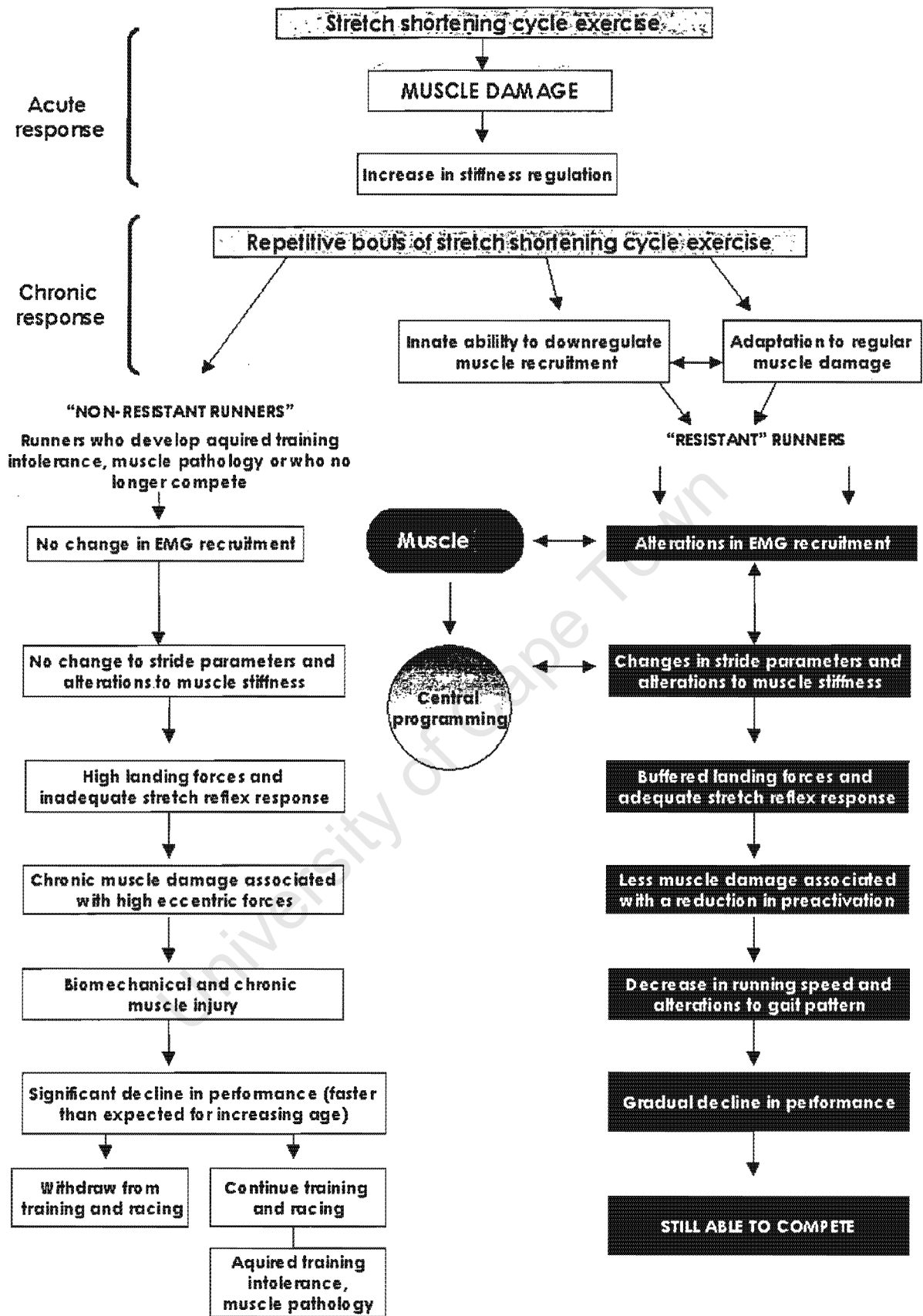


Figure 9.2: Proposed model to characterise "resistant" and "non-resistant" runners

## CHAPTER 10

# CONCLUSIONS

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The overall aim of this thesis was to determine whether there is a relationship between exposure to high mileage racing and training volumes and alterations to neuromuscular characteristics in masters runners. Based on the evidence provided in this thesis, the research questions can be answered as follows:

1. *Is there a relationship between the total accumulated distances of training and racing in masters long distance runners and changes in maximal isometric strength, electromyographic (EMG) activity and the ability of the knee extensor muscles to absorb forces before and after a downhill run?*

Runners who have accumulated more than 5 000 km of racing use a different neural recruitment strategy to maintain (subjective) maximal function after a 40 minute downhill run compared to runners who have accumulated significantly less racing distance. In addition, runners who are exposed to high volume training and racing show no consistencies in EMG activity following a lengthening muscle challenge compared to less experienced runners who show close associations between force production and muscle recruitment.

2. *Can changes in running economy, after a short bout of downhill running, be used as an indirect marker of chronic muscle damage after years of accumulated training and racing in masters runners?*

Running economy does not differ between athletes who vary in their exposure to high volume running training and racing and thus may not be a sensitive marker of possible accumulative muscle damage in these subjects. Alternatively, experienced long distance runners may be able to alter recruitment strategies in such a way that running gait is maintained and which is not reflected in changes in running economy.

3. *Can lower extremity muscle preactivation be used as a sensitive marker of neuromuscular activity and muscle stiffness?*

Centrally regulated lower limb muscle preactivation levels show consistent changes associated with fatigue, despite differences in the metabolic profiles of the fatiguing interventions. This suggests that preactivation can be used as a sensitive measure of central changes associated with fatigue and muscle stiffness regulation during dynamic exercise.

4. *Is there a relationship between the volume of training and racing and EMG recruitment patterns during running in masters runners?*

Lower limb muscle preactivation levels differ between subjects of the same age who have been exposed to differing levels of running training and racing. These differences appear to be associated with a down-regulation of EMG activity that serves to protect the muscle from damage that occurs as a result of high landing forces during running.

5. *Do years of accumulated endurance racing and training have an effect on neuromuscular characteristics?*

Based on the results of these studies, it can be concluded that runners who are able to continue with high volume training and racing for many years represent a unique group of "resistant" runners. It may be speculated that years of accumulated endurance running will alter EMG recruitment patterns, either in anticipation of a chronic training stimulus or as an adaptation to it. The origins and possible mechanisms of these alterations remain unclear and extend beyond the limits of this thesis. It is likely that there is an interaction between these two proposed possibilities, and that those athletes who have an innate capacity for altering neural control may also be more flexible in their adaptation to a chronic overload stimulus. Accordingly, further research will be required to address this issue and identify which of these possibilities enables this unique group of masters runners to continue to accumulate high volumes of training and racing.

## CHAPTER 11

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## CHAPTER 12

## APPENDICES

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### 12.1. INFORMED CONSENT (example: study one)

The MRC/UCT Research Unit for Exercise Science and Sports Medicine are conducting a study to determine the effects of a downhill run on electromyography (EMG) activity and muscle power in experienced and inexperienced masters runners.

What we hope to determine, is whether or not runners who are exposed to high mileage racing and training lose the elasticity in their skeletal muscle and whether these changes impair total muscle function.

You will be required to visit the laboratory on 2 separate occasions, separated by no longer than 7 days.

**The following tests will be performed on your first visit to the laboratory:**

1. You will be required to record a full running training and racing and injury history, which will be conducted by means of an interview and questionnaire.
2. An anthropometrical assessment will be conducted which will include the measurements of height, weight, girths and skin folds. This information will be used in the description of body size and proportions, as well as to estimate total body composition and skeletal muscle mass.
3. Prior to the start of the testing, you will have the opportunity to become familiar with the equipment that will be used during the testing.
4. A maximal oxygen consumption ( $VO_2$  max) test will be conducted on a motor driven treadmill. This test will require that you begin running at a relatively low speed, which will then be advanced in stages. A face mask will be placed over your nose and mouth and you will be asked to wear a belt around your chest to measure your heart rate. It is a maximal test, which means that it will terminate at exhaustion. However, this test may also terminate because of feelings of dizziness or any other discomfort.

**The following tests will be performed on your second visit to the laboratory:**

1. A small area of skin on your right leg will be shaved and cleaned with an alcohol swab. A small electrode will then be placed onto the area and secured with elastic strapping. The electrode will remain there for the duration of the trial.
2. You will be able to warm up and stretch before the start of the testing.
3. The drop jump test will require you to jump off a bench and immediately jump up again to touch a place on the wall.
4. You will then perform two tests on an isokinetic dynamometer. The first will require you to exert maximal force against a static lever arm for 5 seconds. This will be repeated 4 times. The second test will require you to exert maximal force against a static lever arm for 25 seconds. This will be repeated twice.
5. You will then be asked to run for 40 minutes on the treadmill. The speed of the treadmill will be adjusted to 70 % of the fastest speed you obtained during the maximal test. Your warm up will last for 5 minutes and will be at a gradient of 0 %. The gradient will then be decreased to - 10 % and you will be required to complete the 40 minute run at the same speed.
6. Immediately following the treadmill run, the drop jump and maximal strength tests will be repeated.

The downhill run included in this protocol may result in slight pain and discomfort in the thigh and calf muscles and is likely to persist for a short while following the run, but will subside gradually.

You will be asked to maintain your normal training regimen during the week prior to the test, and refrain from attempting any training which may be physically too strenuous, which may affect your results. You will also be required to report all training done during this period.

At the end of the testing period, you will be provided with a summary of your personal results, and how you compared with the other participants of this research trial. You will also be provided with comparison graphs and tables for the general population and an overall summary of the results and interpretations of the study.

Any questions about the procedures and equipment used during the testing, or the results of the testing are encouraged. Your permission to participate in this study is purely voluntary, and you are free to withdraw at any point if you so wish.

I \_\_\_\_\_ having read this form, understand that I may ask questions at any time during the testing procedure, realise that my permission to perform the above tests is voluntary, and am aware that I am under no obligation to give my consent. I further understand that I may withdraw from the tests at any time. I am also aware that neither the researchers at the UCT/MRC Research Unit for Exercise Science and Sports Medicine nor the University of Cape Town will be responsible for anything other than on site emergency care, should any medical problem occur during the course of testing.

Name of volunteer: \_\_\_\_\_

Signature: \_\_\_\_\_

Name of investigator: \_\_\_\_\_

Signature: \_\_\_\_\_

Name of witness: \_\_\_\_\_

Signature: \_\_\_\_\_

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**12.2. ANTHROPOMETRY**Age Height Weight **a) Skinfolds**Biceps Triceps Subscap Suprailiac Abdominal Thigh Calf **b) LTV**Sub gluteal Above knee Sub glut to knee Mid thigh **c) Hamstring flexibility**Right Left

**12.3. MAX TEST**

Warm up speed for 5 minutes

Treadmill gradient at 0

Increase speed by 0.5 km/hr every 30 seconds

**Peak treadmill running speed:**

**Peak VO<sub>2</sub>**

**RPE**

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## 12.5. RACING HISTORY

RACE	NUMBER COMPLETED	TOTAL
160 km		
Comrades		
Two Oceans		
Puffer		
100 miler		
80 km		
Standard marathon		
36 km		
30 km		
Half marathon		
18 km		
15 km		
10 km		
8 km		
5 km		
Other		
TOTAL		

## 12.6. INJURY HISTORY

INJURY	FREQUENCY, L/R, MUSCLE	DURATION
Stress fractures		
Iliotibial band syndrome (ITB)		
Muscle strain (acute/chronic)		
Muscle tear (acute/chronic)		
Plantar faciitis		
Achilles tendonitis		
Runners knee		
Compartment syndrome		
Shin splints		
Trauma		

Recurring injuries: \_\_\_\_\_

Amount of time taken off running: \_\_\_\_\_

Muscle pain after racing: \_\_\_\_\_

## 12.7. DATA COLLECTION SHEET: STUDY ONE

## a) Pre-downhill run ISOKINETIC

## i) 5 seconds

EMG file: Average force: 65°  60° Max force: 65°  60° Time to peak: 

## ii) 25 seconds

EMG file: Average force: 65°  60° Max force: 65°  60° Time to peak: 

## b) Pre-downhill run DROP JUMPS

EMG file: Standing height: Jump height: Change in jump height

**c) Downhill run**

Treadmill speed:

Time	5	10	15	20	25	30	35	40
HR								
RPE								

**d) Post-downhill run ISOKINETIC**

**i) 5 seconds**

EMG file:

Average force: 65°  60°

Max force: 65°  60°

Time to peak:

**ii) 25 seconds**

EMG file:

Average force: 65°  60°

Max force: 65°  60°

Time to peak:

**e) Pre-downhill run DROP JUMPS**

EMG file:	<input type="text"/>
Standing height:	<input type="text"/>
Jump height:	<input type="text"/>
Change in jump height	<input type="text"/>

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