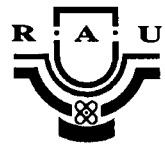

THE EFFECT OF REPEATED BOUTS OF DOWNHILL TRAINING ON RUNNING PERFORMANCE AND RECOVERY AFTER A 30-KM TIME TRIAL

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Submitted in fulfillment of the degree
Master of Science in Medicine

ABSTRACT

Purpose: The present study was designed to examine the effect of repeated bouts of either downhill or level running on running performance in, and recovery from, a 30-km time trial. **Methods:** Sixteen male subjects with a mean (± SD) age of 33.8 ± 5.8 years, body mass of 72.0 ± 7.3 kg and a stature of 176.6 ± 4.5 cm were randomly allocated to either a downhill (n=9) or a level group (n=7). The protocol consisted of a training phase, followed by a 30-km time trial and a recovery phase. During the training phase subjects ran either at a –10% grade (downhill group) or a 0% grade (level group) on a treadmill for nine 40 minute training runs [70% of peak treadmill running speed (PTRS)]. Thereafter all the subjects participated in a 30-km time trial (70% of PTRS), where heart rate (HR), rate of perceived exertion (RPE) and stride length (SL) were recorded, followed by five 15 minute submaximal recovery runs. The first recovery run was performed before the start of the training phase and again on four occasions after the 30-km time trial. HR, RPE, SL, minute ventilation (Vi), oxygen consumption (VO_2), carbon dioxide production (VCO_2) and respiratory exchange ratio (RER) were recorded during these 15 minute runs. Plasma creatine kinase (CK) activity and muscular soreness were assessed for the duration of the study. **Results:** HR decreased in the downhill group during the training phase, suggesting a HR training effect. Muscle pain and plasma CK activity in the downhill group increased after the first 40 minute downhill training run. These indicators of muscle damage did not show any further increases during the training phase, suggesting a "repeated bout effect". Towards the end of the 30-km time trial the level group, showed a greater heart rate drift (HRD) and an increased RPE, suggesting that they were not able to resist fatigue to the same extent as the downhill group. HR and RPE recorded during the recovery phase suggested that the downhill group showed a better recovery after the 30-km time trial. During the recovery phase the downhill group experienced no increase in muscle pain after performing the 30-km time trial, in contrast to the level group who experienced muscle pain for five days after the 30-km time trial. Plasma CK activity, was blunted after the 30-km time trial in the downhill group in contrast to the level group. **Conclusion:** The results of the investigation support the hypothesis that the inclusion of downhill training into a training program cause changes, which can be interpreted as enhancing performance during an endurance event and recovery after the event.

DECLARATION

I, Lynne Schutte, hereby declare that the work on which this thesis is based is my original work (except where acknowledgements indicate otherwise), and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university. I empower the University of Cape Town to reproduce for the purpose of research either the whole or any portion of the contents in any manner whatsoever.

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ACKNOWLEDGEMENTS

Special thanks to Professor Mike Lambert for his assistance, guidance, patience and encouragement as my supervisor.

Thanks are also due to Mr. Rian Lombard and Professor Geoff Rogers for the important part that they played in the investigation.

Nicky, thank you for your support and friendship.

Thanks are also due to Judy Belonje and Fikile Koetle who helped with data capturing and sample analysis.

This study was supported by grants from the South African Medical Research Council and by the Brunow Myer & Harry Research Funds of the University of Cape Town.

LIST OF ABBREVIATIONS

Adenosine triphosphate	ATP
Alanine aminotransferase	ALT
Analysis of variance	ANOVA
Aspartate aminotransferase	AST
Calcium	Ca^{2+}
Cardiac output	CO
Carbon dioxide production	VCO_2
Coefficient of variation	CV
Creatine kinase	CK
Creatine phosphate	CP
Delayed onset muscle soreness	DOMS
Difference in heart rate	$HR_{difference}$
Electromyographic	EMG
Heart rate	HR
Heart rate drift	HRD
Kilogram	kg
Least significant difference	LSD
Magnetic resonance	MRI
Maximal oxygen consumption	VO_{2max}
Maximum heart rate	HR_{max}
Myosine heavy chain	MHC
Newton	N
Non-steroidal anti-inflammatory drugs	NSAIDS
Oxygen	O_2
Oxygen consumption	VO_2
Peak treadmill running speed	PTRS
Personal best	PB
Profile of mood states	POMS
Rate of perceived exertion	RPE
Respiratory exchange ratio	RER

Serum lactate dehydrogenase	LDH
Skeletal troponin I	sTn I
Standard deviation	SD
Stretch-shortening cycle	SSC
Stride frequency	SF
Stride length	SL
Stroke volume	SV
Submaximal oxygen consumption	$VO_{2submax}$
Troponin I	Tn I
Velocity	v
Ventilation	Vi

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CHAPTER ONE

MOTIVATION AND AIMS OF THE STUDY

1.1 INTRODUCTION

Based on the cross-bridge theory of muscle contraction, the force exerted by muscle is generated by the interaction of actin and myosin which results in the myofibrillar proteins translating relative to one another. However, when the muscle fibers are lengthened in an eccentric muscle action, the actomyosin bonds are probably disrupted mechanically rather than undergoing an ATP-dependent detachment. This loading profile undoubtedly places high stresses and strains on the involved structures and contributes to the tissue damage that occurs with eccentric muscle actions (Schwane et al., 1983a; Enoka, 1996).

Numerous structural abnormalities are evident in muscle after exercise, especially exercise that involves eccentric muscle actions. These abnormalities include sarcolemmal distortion, dilation of the transverse tubular 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 (Byrnes et. al., 1985; Dick and Cavanagh, 1987; Westerlind et al., 1992; Balnave and Thompson, 1993; Enoka, 1996).

Eccentric exercise has been particularly associated with delayed onset muscle soreness (DOMS). Delayed onset muscle soreness is a sensation of discomfort or pain that occurs in response to unaccustomed exercise, or in response to large increases in the volume of exercise. DOMS is first felt between 8–24 hours after exercise and peaks in intensity between 24 and 72 hours. The soreness is typically accompanied by muscle stiffness, pain on active movement, reduced flexibility and tenderness when palpated and usually disappears by five days after exercise. Decrements in muscle strength occur immediately and last for several days (Balnave and Thompson, 1993; Smith et al., 1994a; Gleeson et al., 1995; Eston et al., 1996)

Several studies have shown that although strenuous eccentric exercise results in muscle damage, the performance of a repeated bout of the same exercise produces only modest changes in the indicators of damage. This phenomenon is known as the "repeated bout" effect (Nosaka and Clarkson, 1995; Smith et al., 1998).

Specifically, after the first bout of eccentric exercise there is a prolonged loss in muscle strength and range of motion, an increase in muscle proteins, such as creatine kinase (CK), in the blood and the development of muscle soreness. After a repeated bout performed between a few days to several weeks later, the recovery of strength is significantly faster than that found after the first bout, soreness development is less and muscle protein increases in the blood are blunted (Byrnes et al., 1985; Smith et al., 1994a; Smith et al., 1998). It appears that performance of one bout of damage-inducing exercise results in an adaptation in the muscle such that it is more resistant to the effects of a subsequent bout of intense eccentric exercise. A review by Enoka (1996), concluded that the short- and long-term consequences of including eccentric muscle actions in an exercise program can be to induce structural adaptations in muscle, to activate an inflammatory response and to modify the neural commands used to control movement. Although these adaptations can also be induced by other types of muscle contractions, they seem to be maximized by eccentric muscle actions.

Studies have attempted to quantify the effect of eccentric exercise training on the development of muscle damage. For example, one study was designed to investigate the effect of eccentric training on the appearance of muscle damage indicators, DOMS and impaired muscle function due to prolonged eccentric exercise (Balnave and Thompson, 1993). The study involved a 40 minute eccentric walk down a 25% gradient on a treadmill at 6.4 $km.h^{-1}$ once a week for eight weeks. Balnave and Thompson (1993) concluded that a single bout of downhill walking is sufficient to significantly abolish DOMS one week later. In agreement with previous reports (Byrnes et al., 1985; Nosaka et al., 1991) a long lasting repeated-bout adaptation to eccentric muscle action was displayed.

These data clearly have an applied aspect as they indicate that certain training strategies may induce adaptations, which make the muscle more resistant to damage. However, further research is needed before training recommendations can be made with some degree of confidence, derived from evidence based research.

1.2 AIMS OF THE THESIS

The main aim of this study was to examine the effect of including downhill training into a long distance training program.

More specifically, the aim of this study was to examine running performance in, and recovery after, a 30-km time trial, with either downhill or level training (nine x 40 minute runs) in the weeks prior to running the time trial. It is hypothesized that by including downhill training there will be a performance enhancing effect during endurance events and improved recovery after the event.

A secondary aim of the study was to investigate delayed onset muscle soreness and the markers of muscle damage as well as the repeated bout effect. To examine the time course and the extent of muscle damage during repeated bouts of downhill running and to provide possible insight into the mechanisms underlying delayed onset muscle soreness and the repeated bout phenomenon.

1.3 RESEARCH QUESTION

"Does downhill training (running at 70% of peak treadmill running speed for nine x 40 minute training sessions on a –10% slope) cause adaptations which make the muscles more resistant to fatigue and damage in a 30-km time trial designed to mimic a road race?"

CHAPTER TWO

LITERATURE REVIEW

2.1 INTRODUCTION

The review of the literature will be presented in the following order. First the available literature with regards to exercise-induced muscle damage will be presented and thereafter running economy will be discussed.

Exercise-induced muscle damage will be discussed under the following headings:

- mechanisms underlying muscle fiber damage;
- different methods used to induce muscle damage;
- factors associated with damage following repetitive eccentric muscle actions;
- skeletal muscle regeneration;
- repeated bout effect;
- fiber type involvement;
- effects of marathon and ultra-marathon race; and
- cardiovascular drift

Running economy will be presented under the following headings:

- factors affecting running economy; and
- relationship between running economy and performance

2.2 EXERCISE–INDUCED MUSCLE DAMAGE

Based on the cross-bridge theory of muscle contraction, the force exerted by muscle is generated by the interaction of actin and myosin, which results in the myofibrillar proteins translating relative to one another. However, when the muscle fibers are lengthened in an eccentric action, the actomyosin bonds are thought to disrupt mechanically rather than undergo an ATP-dependent detachment. This loading profile places high stresses and

strains on the involved structures and may contribute to the tissue damage that occurs with eccentric muscle actions (Enoka, 1996).

Studies of muscle damage following exercise have generally shown that eccentric muscle action is more likely to cause injury to active muscle fibers than either isometric or concentric muscle action (Dick and Cavanagh, 1987; Westerlind et al., 1994). Eccentric muscle actions involve actively resisting lengthening of the muscle and are characterized by high tension on the muscle fibers and connective tissue relative to concentric muscle actions (Schwane et al., 1983b; O'Reilly et al., 1987; Clarkson et al., 1992; Miles and Clarkson, 1994). Eccentric muscle actions are characterized by elongation of the muscle at the same time as contraction. With a given submaximal contraction power, an eccentrically contracted muscle uses less oxygen and ATP than a corresponding concentric action. Also fewer motor units are activated for any given load in eccentric work (Fridén et al., 1983; Fridén, 1984).

Numerous structural abnormalities are evident in muscle after exercise, especially exercise that involves eccentric muscle actions. These abnormalities include sarcolemmal disruption, dilation of the transverse tubular system, distortion of myofibrillar component, Z-line streaming, fragmentation of the sarcoplasmic reticulum, lesions of the plasma membrane, cytoskeletal damage, changes in the extracellular myofiber matrix and swollen mitochondria (Enoka, 1996; Lieber et al., 1996).

Other manifestations of damage include muscle soreness, prolonged losses in muscle strength and range of motion, changes in substrate levels, limb swelling, increases in muscle proteins in the blood, such as creatine kinase and decrements in motor control (Mair et al., 1995; Clarkson, 1997; Smith et al., 1998).

When the same exercise is repeated between a few days to several weeks later, there is significantly less delayed onset muscle soreness (DOMS), significantly lower circulating levels of creatine kinase and an enhanced recovery of strength and range of motion. This altered response is referred to as the "repeated bout" effect, and might reflect a rapid adaptation to the initial injury (Smith et al., 1998).

2.2.1 MECHANISMS UNDERLYING MUSCLE FIBER DAMAGE (CAUSE OF DAMAGE)

Many types of exercise have been shown to produce muscle damage. Damage has been observed after isometric exercise, marathon running and eccentric muscular activity (Ebbeling and Clarkson, 1989). Two basic mechanisms have been offered to explain how exercise initiates damage. One mechanism describes a disturbance in metabolic function and the other mechanism addresses mechanical disruption of the cell (Newham et al., 1983a; Ebbeling and Clarkson, 1989).

2.2.1.1 Metabolic hypothesis

One possible cause of muscle cell injury could be referred to as "metabolic overload", in which the demand for ATP in the fiber exceeds ATP production. Since muscle homeostasis is dependent upon ATP-dependent ion pumps, lowered ATP levels in the cell could result in altered ion concentrations in the cell. For example, abnormally elevated Ca^{2+} in the muscle fiber is known to trigger a cycle of events in which the mitochondria sequester some of the excess Ca^{2+}, which reduces mitochondrial respiration, which further reduces ATP availability. Thus, low ATP levels could initiate a vicious cycle starting with lowered ability to extrude Ca^{2+} from the cell via ATP-dependent Ca^{2+} pumps, and ending in cell death (Armstrong, 1986). According to this hypothesis, reduced ATP in the muscle during exercise could initiate this cycle. There is clear evidence of alterations in muscle mitochondria following exercise (Gollnick and King, 1969; Dohm et al., 1975). Gollnick and King (1969) reported changes in mitochondria in muscles of rats following prolonged exhaustive treadmill exercise. Also, skeletal muscle mitochondria in rats have been shown to accumulate significant amounts of Ca^{2+} during prolonged low intensity exercise (Tate et al., 1978).

A corollary to the metabolic hypothesis is that high metabolite levels in the muscles during exercise may lead to injury and pain. In particular, lactic acid has been suggested to have a toxic effect on muscles when present in high concentrations, such as during intense

muscular exercise (Newham et al., 1982; Armstrong, 1984). This idea has been particularly popular with respect to muscular soreness and is commonly offered as the explanation for delayed onset muscle pain (Armstrong, 1986).

There are several lines of support for the metabolic hypothesis. Perhaps the strongest evidence that exercise induced muscle injury might have a metabolic basis comes from studies of muscle ischaemia, in which oxygen delivery to the muscles is reduced by occluding their blood flows (Makitie and Teravainen, 1977). This prevents the cells from maintaining normal cellular respiration and hence, normal concentrations of ATP. In these experiments the muscle fibers are injured and the process and sequence of cell degeneration are very similar to those observed in muscles injured by exercise. Another general observation that could be used to support the idea of a metabolic basis for muscle injury is that the extent of injury, and consequent soreness, is proportional to the intensity of the exercise (Tiidus and Ianuzzo, 1983). Brooke et al. (1979) also found a direct relationship when peak blood lactate levels, during concentric cycling exercise, are plotted against changes in blood CK following exercise. De Vries (1966) reported that electromyographic (EMG) activity is elevated in muscles that are sore from exercise. They suggested that exercise might initiate a positive feedback cycle in which ischaemia leads to muscle spasm, which in turn causes, increased ischaemia. The progressive increase in ischaemia resulting from this cycle would reduce oxygen availability in the muscles.

The metabolic hypothesis is intuitively attractive as an underlying mechanism for exercise induced muscle injury, but there are several lines of evidence that argue against it. First, during submaximal exercise ATP levels in the active muscles appear to be maintained at near resting values (Saltin and Gollnick, 1983). Therefore, even though ATP use by the myosin cross-bridges increases dramatically, the metabolic pathways in the muscle cells are able to synthesize ATP rapidly enough to maintain stable ATP concentrations in the active fibers. Secondly, it has been demonstrated that muscle fiber damage is most apt to occur in the muscles that undergo eccentric actions (Armstrong et al., 1983). In eccentric muscle actions energy use is relatively low compared to that in concentric muscle actions in which the same muscle forces are produced (Bonde-Peterson et al., 1972) and lactic

acid concentrations are markedly lower during eccentric exercise (Schwane et al., 1983a). Therefore, the fact that eccentric muscle actions, that require low energy expenditure, cause greater muscle damage argues against a metabolic basis for the initial events in muscle fiber injury during exercise (Armstrong, 1986).

2.2.1.2 Mechanical strain hypothesis

From his experiments on muscle soreness at the turn of the century, Hough (1902) concluded that delayed soreness results from ruptures of muscle fibers or connective tissue that occur during the exercise, since the degree of soreness is related to the peak forces produced by the muscles, but not to the extent of acute fatigue of the muscles during the exercise bout. There are histological and biochemical evidence that structural damage does occur during exercise, particularly eccentric exercise (Fridén et al., 1981; Jones et al., 1986b). A study by Armstrong et al. (1983) showed that immediately after rats walked downhill on a treadmill for 90 minutes, lesions in fibers, in both the sarcolemma and the banding pattern, were apparent in the deep extensor muscles performing the eccentric muscle actions. Similar disruptions in fiber banding patterns have been observed in human muscles after eccentric exercise (Fridén et al., 1983).

Arguments against the metabolic hypotheses are based on the finding of greater damage following eccentric muscle actions compared with concentric actions (Armstrong et al., 1983; Newham et al., 1983c). Muscles that develop active tension eccentrically require less energy but experience greater injury than muscles that contract concentrically, as indicated by morphological changes (Armstrong et al., 1983), delayed onset soreness (Talag, 1973), performance changes (Talag, 1973) and increases in plasma proteins (Armstrong et al., 1983). EMG activity is also lower during negative work (maximal and submaximal) suggesting that relatively fewer fibers are recruited to produce large forces. Therefore, under comparable workloads, eccentric actions produce greater tension per cross-sectional area of active muscle than concentric muscle actions (Ebbeling and Clarkson, 1989).

Newham et al. (1983c) employed a step test to compare eccentric and concentric exercise. During the test, the quadriceps of one leg performed only concentric actions by stepping up, while the contralateral muscles developed active tension eccentrically by stepping down. No ultra-structural abnormalities were seen in the muscles that had contracted concentrically. The muscles that had developed active tension eccentrically, however, showed marked myofibrillar disorganization. Maximum voluntary force decrements and pain were also greater in these muscles.

Although there was a greater increase in electrical activation for a given muscle tension following eccentric compared with concentric stepping, there was no evidence of spontaneous electrical activity when the muscles were at rest (Newham et al., 1983c). This observation contradicts the spasm theory of De Vries (1966).

Eccentric muscle actions have been shown to produce more heat than concentric muscle actions at the same workload (Davies and Barnes, 1972). These elevated temperatures could damage structural and functional components within the cell. There is evidence from myopathy conditions that elevated temperatures can cause severe rhabdomyolysis (Bartsch et al., 1977).

2.2.2 DIFFERENT METHODS USED TO INDUCE MUSCLE DAMAGE

Research has established that unaccustomed physical activity results in delayed onset muscle soreness and increases in the concentration of muscle specific enzymes (Byrnes et al., 1985). Laboratory models to study these related phenomena have included such novel exercise regimens as prolonged isometric exercise (Clarkson et al., 1982; Clarkson et al., 1985), downhill running (Schwane et al., 1983b) and specific eccentric muscular exercise (Newham et al., 1983c).

Schwane et al. (1983b), tested the hypothesis that running down an incline, during which muscles primarily perform eccentric actions, causes greater muscle soreness and greater increases in plasma enzyme activities than does running on a level surface, during which muscles perform similar amounts of concentric and eccentric actions. It was concluded

that following downhill running delayed onset soreness was experienced in gluteal, quadricep, anterior- and posterior leg muscles and creatine kinase activity increased significantly. In contrast, following level running, no significant soreness occurred in any of the muscle groups and plasma creatine kinase activity was not elevated.

To compare eccentric and concentric exercise, Newham et al. (1983c) employed a step test. During the test, the quadriceps of one leg performed only concentric actions by stepping up, while the contralateral muscles developed active tension eccentrically by stepping down. Following the test, no ultra-structural abnormalities were seen in the muscles that had contracted concentrically. The muscles that had developed tension eccentrically, however, showed marked myofibrillar disorganization.

A study by Fridèn et al. (1983) showed that following eccentric cycle exercise, all the subjects suffered from severe soreness in their thigh muscles 18–72 hours after the exercise.

Kyröläinen et al. (1998) studied muscle damage induced by stretch-shortening cycle (SSC) exercise. The SSC exercises included 200-drop jumps and the same amount of SSC exercises on a sledge ergometer. The SSC exercise caused increases in the activity of serum creatine kinase and concentrations of serum carbonic anhydrase and also myoglobin. These proteins in the blood serum are used to estimate protein leakage from skeletal muscle.

Clarkson et al. (1992) used a series of maximal (high-force) eccentric actions of the forearm flexor muscles to induce muscle damage. Peak soreness was experienced two to three days post-exercise while peak swelling occurred five days post-exercise. Maximal strength and the ability to fully flex the arm showed the greatest decrements immediately after exercise with a linear restoration of those functions over the next ten days.

Thompson et al. (1999) examined the impact of prolonged intermittent high intensity shuttle running on soreness and markers of muscle damage. There was a marked

increase in both muscle soreness and markers of damage after the Loughborough Intermittent Shuttle Test, whereas these variables remained unchanged in a resting control group. The authors concluded that individuals who partake in a multiple-sprint sport on an irregular basis are at a high risk for muscle soreness and damage.

Lastly, post exercise soreness is also experienced following prolonged endurance events such as marathons, triathlons and other ultra-endurance events. Following events of these durations, particularly if running is involved, athletes experience stiffness and pain in the muscles for days or perhaps weeks (Miles and Clarkson, 1994).

2.2.3 FACTORS ASSOCIATED WITH DAMAGE FOLLOWING REPETITIVE ECCENTRIC MUSCLE ACTIONS

2.2.3.1 Morphological changes

Histological and ultrastructural analysis of muscle samples provide the most direct evidence of muscle damage (Fridén et al., 1984; Jones et al., 1986b; Clarkson and Tremblay, 1988). In normal skeletal muscle, the Z–disc appears as a well organized woven basket or square lattice (Hoppeler, 1986). Electron micrographs have shown marked broadening, streaming and in places total disruption of Z–discs following eccentric exercise (Fridén, 1984; Kirwan et al., 1992). Changes to the Z–discs are one of the most commonly found abnormalities and Fridén et al. (1981) have suggested that the Z–disc may be the weak link in the myofibrillar contractile chain.

In addition to Z–disc alterations, structural changes include myofibrillar and sarcolemmal disruptions (Armstrong et al., 1983), widening of the A– and I–bands, displacement of organelles (Fridén et al., 1983), increase in mitochondrial volume density (Fridén et al., 1983) and cytoskeletal changes (Fridén et al., 1984). Increased cellular infiltration has also been noted (Jones et al., 1986b).

The sequence of events associated with muscle damage and repair is not well defined, due to different methods used to damage muscle and the variable periods of assessment

following the insult (Ebbeling and Clarkson, 1989). For example, Fridén et al. (1981) obtained biopsies from the soleus muscle two and seven days after repeated stair descents. They found markedly less Z–disc disruption on day seven than on day two indicating that repair was taking place. The same researchers (Fridén et al., 1984) also found evidence of immediate damage to the vastus lateralis muscle following eccentric cycle ergometer exercise. However, three days after exercise the damage had progressed and separated myofibrils, swollen mitochondria and cytoskeletal alterations were noted. Lipofuscin granules indicative of lysosomal activity (Fridén et al., 1984) and polyribosome complexes representative of protein systhesis and muscle repair (Fridén et al., 1983) were seen in areas of disruption. Fibers appeared essentially normal by day six after exercise.

Using two different eccentric exercise protocols, Jones et al. (1986b) observed fiber necrosis and cellular infiltration in humans. Forearm flexors were forcibly extended using a winch with a mechanical advantage of about 10:1. Calf muscles were exercised as the subject walked backward, down an inclined treadmill. Although there was variability in the response among subjects, degenerating fibers and infiltrating mononuclear cells were not evident until seven days after exercise. By 20 days after exercise, many small regenerating fibers with basophilic cytoplasm and internal nuclei were present.

2.2.3.2 Delayed onset muscle soreness

Many types of muscle pain and soreness have been documented (Layzer, 1986). As mentioned previously Hough (1902) first hypothesized that delayed onset muscle soreness (DOMS) following unaccustomed exercise was a result of muscle damage due to mechanical stress. His theory, which has been supported by evidence from muscle biopsies (Fridén et al., 1983; Jones et al., 1986b), is now widely accepted (Ebbeling and Clarkson, 1989).

DOMS can be distinguished from temporary soreness. Temporary soreness, characterized by only moderate pain, is felt during the final stages of fatiguing exercise

(Talag, 1973; Fridén, 1983; Fridén, 1984). The primary cause of this temporary soreness is thought to be metabolic waste accumulation (Talag, 1973; Fridén, 1983).

In contrast, DOMS is the feeling of pain, tenderness, deep ache and stiffness in muscles that begins several hours after exercise (Newham et al., 1983c; Armstrong, 1984; Byrnes et al., 1985; Newham et al., 1987; Balnave and Thompson, 1993; Mac Intyre et al., 1996). Typically, one might be unpleasantly reminded of the previous day's exercise with any or all of the above symptoms when getting out of bed in the morning (Miles and Clarkson, 1994). The severity of this type of soreness varies, ranging from mild discomfort to extreme soreness limiting normal use of muscles (Jones et al., 1986b). The onset of DOMS occurs in the first 24 hours following exercise and the intensity will generally peak by 48 to 72 hours (Clarkson et al., 1992). In cases of extreme severity, peak soreness may be delayed as long as four to five days post–exercise (Jones et al., 1986b). DOMS tapers off thereafter and may disappear anywhere from 48 hours to two weeks later, depending on the severity.

(a) Methods used to evaluate muscle soreness

(i) Rating scale techniques

The method most commonly used to evaluate muscle soreness has been a questionnaire. Subjects are required to rate perceived soreness on a verbally anchored, fixed ordinal scale (Talag, 1973; Dick and Cavanagh, 1987; Clarkson and Tremblay, 1988; Costill et al., 1990; Clarkson et al., 1992; Gleeson et al., 1995; Pizza et al., 1995; Semark et al., 1999).

Gleeson et al. (1995) examined whether DOMS–inducing exercise affected physiological responses to subsequent submaximal dynamic exercise. Soreness was rated on a scale of one (normal) to ten (very, very sore) for overall muscle soreness. Soreness was assessed in the following areas: front lower leg, back lower leg, front thigh, back thigh and buttocks. The mean soreness rating for each area from both legs was calculated. The authors noted that although the method they used to rate muscle soreness was not the

most sensitive or reproducible method, it showed a clear difference in soreness ratings of the subject after eccentric compared with concentric exercise.

Pizza et al. (1995) assessed muscle soreness pre-exercise, post–exercise and at 1.5, 12, 24 and 48 hours of recovery from either level or downhill running. Muscle soreness was assessed by having the subjects perform concentric and eccentric actions in the upper and lower leg and also the upper torso. Subjects were asked to rate their soreness using a one (normal) to ten (very, very sore) scale. A composite score for the three areas was again used for analysis. It was concluded that the composite score for muscle soreness was significantly higher post–exercise and at 1.5, 12, 24 and 48 hours of recovery from downhill running compared with level running.

Muscle soreness has also been quantified using a probe connected to a strain gauge (Newham et al., 1983c; Jones et al., 1987; Semark et al., 1999). Newham and co–workers (1983c) measured the severity and distribution of muscle tenderness in the quadriceps after eccentric muscle actions by means of a pressure probe wrapped around the thigh. Their results showed that tenderness was localized primarily at the distal, medial and lateral parts of the quadriceps while the central and proximal regions were relatively spared. They also noted that at peak intensity the distribution of tenderness was more diffuse. It was concluded that eccentric muscle actions result in uneven tension over the myotendinous junction thereby, causing mechanical damage. When comparing the subjective sensations of muscular soreness following level and downhill running, Schwane et al. (1983b) found that every subject reported soreness in some muscle group at 24, 48 and 72 hours after the downhill run, exclusively.

Recently Thompson et al. (1999) used a novel method of evaluating muscle soreness. They examined the impact of prolonged intermittent high intensity shuttle running on soreness and markers of muscle damage. Before the test and for three days after, the subjects used a colored pen to label a diagram of the body's musculature to indicate exactly where they experienced soreness. It was concluded that the intensity of general soreness and in the specific muscles investigated, was greater than baseline for 72 hours after the shuttle test, peaking 24–48 hours post–exercise.

(ii) Morphological techniques

Previous studies have used isolated muscle biopsy samples to examine the relationship between muscle injury and DOMS after exercise (Fridén et al., 1983; Jones et al., 1986b; Manfredi et al., 1991). However, Mac Intyre et al. (1996) noted that muscle biopsies are restricted to a small and specific location of the muscle and are often limited in numbers from each subject.

After repeated maximum eccentric muscle actions, Komi and co–workers (1974) could not find any ultrastructural changes neither of the sarcoplasmic reticulum nor the organization of the contractile material. Van Linge (1962) was able to demonstrate both degenerative and regenerative changes in muscle from rats subjected to strenuous exercise.

According to Mac Intyre et al. (1996) previous studies (Fridén et al., 1983; Manfredi et al., 1991) have only shown isolated muscle injury because the technique of microscopic examination of muscle biopsies restricted the area of the quadriceps that could be sampled. Furthermore, examination of muscle biopsies may also be limiting in the locale as well as the size of the sample. It was also noted that biopsies are often taken from the vastus lateralis muscle, because large vessels could be more easily avoided in this region. However, a biopsy from vastus lateralis may not provide a comprehensive representation of the quadriceps, because in their study Mac Intyre et al. (1996), who assessed the presence of ^{99m}Tc–labeled white blood cells in exercised muscle, found the presence of ^{99m}Tc–labeled white blood cells throughout the muscle.

2.2.3.3 Inflammation and swelling

Conflicting results have been reported with regards to whether acute inflammation is the underlying mechanism for delayed onset muscle soreness (Pizza et al., 1995). Investigators have reported higher (Smith et al., 1989) or similar (Schwane et al., 1983b; Bobbert et al., 1986) leukocytes and neutrophils following eccentric exercise relative to concentric exercise. There are many inflammation like responses following damaging

exercise including pain, swelling, elevation in white blood cell count, particularly the neutrophils (Schwane et al., 1987; Smith et al., 1989), increased intramuscular and circulating levels of acute phase mediators and accumulation of monocytes and lymphocytes in damaged and regenerating fibers (Jones et al., 1986b; Stauber et al., 1988).

According to Smith (1991), monocytes infiltrate the damaged tissue and differentiate to become macrophages to phagocytise debris. The macrophages also release prostaglandin's which sensitize local pain receptors to such an extent that a hyperalgesic state occurs and painful chemical and mechanical stimuli are intensified. A lowering of the mechanical threshold of group III and IV muscle afferents has been demonstrated in inflamed muscle and may be the factor responsible for the sensation of tenderness (Berberich et al., 1988).

The initial damage is followed by secondary changes, including swelling. There is rather strong evidence that Ca^{2+} plays a pivotal role for inducing these changes (Duan et al., 1990; Armstrong et al., 1991). It is assumed that the mechanical overload induces an increase in intracellular Ca^{2+} concentration which may trigger a chain of events after cessation of the exercise (Armstrong, 1990).

Increased sarcoplasmic Ca^{2+} concentration will lead to Ca^{2+} accumulation in the mitochondria, which will impair the ATP generating capacity (Duan et al., 1990; Armstrong et al., 1991). A lower ATP generating capacity may affect membrane pumps. This may decrease sodium extrusion and lead to swelling of muscle fibers (Fridén et al., 1986).

Clarkson et al. (1992) noted that swelling in itself does not produce soreness as the time course of soreness and swelling are vastly different. Soreness does not appear until hours after exercise and peaks 24 to 48 hours post exercise. Peak swelling may not occur until five to ten days post exercise. Swelling begins within the muscle and then spreads into the subcutaneous space starting about five days post exercise (Nosaka and Clarkson, 1996). Rodenburg et al. (1994a) reported that changes in soreness precedes changes in magnetic resonance (MRI).

(a) Non-steroidal anti–inflammatory drugs

Anti–inflammatory drugs are not successful in significantly reducing muscle soreness (Janssen et al., 1983; Donnelly et al., 1988). If delayed onset muscle soreness occurs with sufficient severity that analgesia is desired, at present the evidence upon which to suggest an effective analgesic treatment is equivocal (Miles and Clarkson, 1994).

One of the primary arguments against inflammation as the mechanism of delayed onset muscle soreness is that non–steroidal anti–inflammatory drugs (NSAIDS) are ineffective in alleviating this condition (Donnelly et al., 1988). The mechanism of action of NSAIDS is to block the synthesis of prostaglandin's, thereby eliminating sensitization of pain receptors. Despite the suggested ineffectiveness of NSAIDS, a few investigations have reported modest positive effects for NSAID treatments relative to placebo for aspirin (Francis and Hoobler, 1987) and ibuprofen (Hasson et al., 1993).

It has been suggested that taking NSAIDS may be counter–productive because the inflammatory process, which includes stimulation of protein turnover for repair by prostaglandin's, is inhibited (Evans, 1987). Mishira et al. (1995) studied the effect of flurbiprofen, a NSAID, on muscles that had been subjected to exercise-induced injury. Their study demonstrated that the effect of flurbiprofen was time-dependent. The contractile properties and histological data suggested that NSAIDS caused a short-term gain but a subsequent functional loss. It was not possible to determine if this loss persisted or to what extent recovery occurred in the long term. They concluded that their results could prompt rethinking of the liberal prescription of NSAIDS as treatment for muscle injury.

(b) Corticosteroids

Hasson et al. (1992) studied the effect of dexamethasone iontophoresis on the symptoms of DOMS. No significant effects were noted on muscle function in this study. There was a slight improvement in muscle soreness perception in the treated patients. However,

according to Almekinders (1999) no other studies have been reported that confirm this finding. The use of oral corticosteriods in the treatment of DOMS has not been studied.

(c) Massage therapy and other modalities

Other treatment forms to ameliorate the symptoms of DOMS continue to be the subject of recent investigations. These treatment forms have generally resulted in conflicting findings. It has been theorized that physical therapy techniques could improve the healing response following eccentric exercise-induced muscle damage by diminishing stiffness and improving local circulation (Almekinders, 1999). Mild, positive effects from massage have been reported (Rodenburg et al., 1994b; Smith et al., 1994b). However, other investigators (Weber et al., 1994; Tiidus and Shoemaker, 1995) were unable to find improvement as a result of massage therapy. In addition, modalities like microcurrent electrical stimulation, therapeutic ultrasound, transcutaneous electrical stimulation and intermittent pneumatic compression have not been shown to produce significant clinical improvement in DOMS (Almekinders, 1999).

2.2.3.4 Changes in muscle function

Gleeson et al. (1995) investigated cardiorespiratory, hormonal and hematological responses to submaximal cycling performed two days after either an eccentric or a concentric exercise bout. They concluded that dynamic submaximal exercise performed two days after an exercise bout with a large eccentric component produces physiological responses that are indicative of a higher relative exercise stress. The authors also noted that such an effect is likely to result in an earlier onset of fatigue. Other studies have reported impairment of maximal isometric and dynamic force producing capability (Sargeant and Dolan, 1987; Jones et al., 1989) and impaired glycogen repletion (O'Reilly et al., 1987) accompanying delayed onset muscle soreness. These effects are likely to limit both the intensity and duration of exercise that can be achieved in subsequent training bouts over several days.

(a) Loss in muscle strength

There is a prolonged loss in muscle strength such that immediately after eccentric exercise, strength losses of 50–60% are found and strength is not restored until ten or more days post–exercise (Clarkson, 1997).

Studies have shown that muscle fibers are damaged by eccentric exercise. Particularly evident is the disruption of the myofibrillar banding pattern (Fridén et al., 1983; Newham et al., 1983b). It is possible that the damaged myofibrils would result in the strength decrements that can be observed. However, the ultrastructural damage becomes worse in the days following exercise, during the time that strength is recovering (Clarkson et al., 1992). Fridén et al. (1983) and Newham et al. (1983b) found more extensive damage in biopsies taken three days and 30 hours, respectively, after eccentric exercise.

Whether the initial decline in strength is due to fatigue, muscle damage, or a combination of both must still be determined. One also cannot discount an involvement of the nervous system in attenuating the strength loss and facilitating recovery. A change could occur in neural activation patterns that would "bypass" the more severely damaged fibers. There is evidence to show that EMG patterns are altered immediately and up to 48 hours after eccentric exercise (Komi and Viitasalo, 1977; Newham et al., 1983c).

Another explanation for the prolonged strength loss may be that sarcomeres are stretched out by performance of the lengthening actions. Sarcomere length is not uniform over the length of the fiber, shorter sarcomeres are found towards the ends (Jones and Round, 1990). If the lengthening actions pulled some of the central sarcomeres apart, the overlap between the actin and myosin filaments would be reduced, thereby reducing the maximal number of cross bridges that could form. Perhaps subtle changes in sarcomere length could influence the ability to generate force (Clarkson et al., 1992). Newham et al. (1988) found that larger strength losses were produced when subjects performed an exercise with the muscle at a long length (more than resting length) than at a short length (less than resting length). The over-stretched sarcomeres could explain the immediate strength loss after exercise. Following the exercise, sarcomere length would be gradually

restored, reflecting the slow recovery of strength. However, presently there are no data to substantiate these suggestions (Clarkson et al., 1992).

A common belief is that the soreness and pain in the muscle prevents the subject from voluntarily producing maximal force. However, soreness cannot be used as an explanation for the decrements in strength since strength is still reduced when soreness is no longer apparent. The time course of soreness development and strength loss/recovery is very different. A key point in refuting that soreness is the cause of reduced strength is that electrically stimulated contractions, thereby bypassing voluntary effort, also show a decrement in force generation (Newham et al., 1987).

Performance of serial concentric or isometric actions will lead to a loss in the ability to produce force much like the strength decrement observed immediately after eccentric exercise. However, unlike eccentric exercise, the strength loss after concentric and isometric exercise is restored in the next several hours (Clarkson et al., 1992).

(b) Decrease in range of motion

A decrease in range of motion occurs at both ends of the range (Clarkson et al., 1992). There is an inability to fully flex as well as an inability to fully extend the muscle. The inability to fully flex may be due to a change in proprioception and/or to over-stretched sarcomeres. The reason for the inability to extend the arm may be explained by swelling, a change in properties of supporting connective tissue, and/or non–neurally mediated contractures (Clarkson, 1997). Chleboun et al. (1995) noted that intermittent pneumatic compression applied to eccentrically exercised muscle, reduced both swelling and stiffness.

(c) Neural activation

EMG studies have suggested that strength decrements may be a result of damage to the contractile apparatus (Komi and Viitasalo, 1977; Newham et al., 1983c). Newham et al. (1983c) found an increase in integrated EMG activity both during eccentric stepping

exercise and when pre-and post–exercise strength tests were compared. A submaximal knee extension test performed at intervals after exercise showed an increase in integrated EMG at all knee joint angles between 0° and 90°. In addition, the neural activation necessary to maintain full extension for two seconds was increased. Recovery occurred over 24 hours.

Following eccentric exercise on an electromechanical dynamometer, Komi and Viitasalo (1977) measured isometric knee extension strength and EMG parameter (integrated EMG, averaged motor unit potential). They observed a decrease in maximum strength and an increase in neural activity at a given muscle tension both immediately and two days after exercise. Mechanical stress produced during eccentric work may cause structural changes accompanied by neuromuscular performance decrements. Injury to initial recruited motor units may necessitate recruitment of additional units to produce required forces (Ebbeling and Clarkson, 1989).

(d) Low frequency fatigue

An interesting phenomenon is the feeling of weakness and instability experienced for a few hours immediately after exercise only in the muscle which had contracted eccentrically (Newham et al., 1983c). This sensation is probably an indication of the pain to follow, and is presumably a reflection of profound low–frequency fatigue with inappropriate forces being generated by relatively low normal physiological firing frequency (Milner–Brown et al., 1973). According to Ebbeling and Clarkson (1989) a significant decrease in force generation can be observed at low (10 to 20 Hz), compared with high (50 to 100 Hz), frequencies.

Immediately after exercise, Newham et al. (1983c) observed muscular weakness and instability accompanying low frequency fatigue only in quadriceps muscles that had developed active tension eccentrically during a step test. Edwards et al. (1977) noted that low frequency fatigue was not due to depletion of high-energy phosphates nor to changes in electrical properties of the muscle. Rather, low frequency fatigue may be caused by mechanical damage to the sarcoplasmic reticulum resulting in less Ca^{2+} release for each

excitatory action potential (Newham et al., 1983c). Newham et al. (1983c) hypothesized that weakness was a reflection of low frequency fatigue with inappropriate forces being generated by the relatively low physiological firing frequency.

A study done by Newham et al. (1983c) examined both low frequency fatigue and soreness. They found no relationship between these two parameters. Low frequency fatigue resulting from eccentric stepping was noted at two minutes, ten minutes, one hour and five hours after exercise. On the other hand, soreness (assessed using a strain gauge) was not perceived until eight hours after exercise and peaked at 48 hours.

(e) Changes in muscle glycogen levels and insulin resistance

Clarkson (1997) noted that endurance performance might be compromised in muscles damaged from eccentric exercise because of changes in muscle glycogen levels. Muscle glycogen is not fully restored several days after eccentric exercise (O'Reilly et al., 1987; Asp et al., 1995), and research has also shown an insulin resistance in subjects who exercised with sore muscles (Kirwan et al., 1992). GLUT 4 protein concentration was decreased by about 35% at 48 hours post–eccentric exercise (Asp et al., 1995) and it was suggested that the loss of this protein may partially explain the inability to resynthesize glycogen.

Kirwan et al. (1992) performed euglycemic–hyperinsulinemic clamps on six untrained individuals to determine whether exercise that induces muscle damage also results in insulin resistance. It was concluded that exercise involving primarily eccentric work resulted in marked transient insulin resistance that was evident 48 hours after the exercise bout. It was also noted that failure to resynthesize glycogen stores for several days after eccentric exercise may be due to impaired insulin–mediated glucose uptake by skeletal muscle.

Glycogen availability may play an important role in the repair process of damaged muscles. Kuipers et al. (1985b) found that the glycogen content of muscle biopsy samples taken 24 hours after eccentric cycle ergometer work was lower than the glycogen

content of samples taken immediately after exercise. O'Reilly et al. (1987) examined muscle glycogen levels of the vastus lateralis after eccentric cycle exercise. They found a 39% reduction in muscle glycogen immediately after exercise. Ten days after exercise, glycogen levels were not restored and were even lower than immediately post–exercise values. Therefore, although glycogen depletion may not initiate damage, the reduced capacity to resynthesize glycogen may play an important role in the repair process.

2.2.3.5 Elevation of muscle proteins in the blood

Unaccustomed physical exercise results in protein leakage from injured skeletal muscle fibers (Mair et al., 1992), which is more pronounced after eccentrically than concentrically biased exercise (Newham et al., 1983a). Plasma activities or concentrations of certain myofibrillar proteins have been used in addition to direct muscle biopsy studies, to estimate skeletal muscle damage and its magnitude (Clarkson et al., 1986). After exercise the muscle injury markers increase in plasma with a varying delay after exertion (Clarkson et al., 1986; Mair et al., 1992) and the increase also seems to be related to the type and the intensity of exercise (Schwane et al., 1983a) as well as previous activity levels of the subjects (Evans et al., 1986).

(a) Creatine Kinase

Creatine kinase (CK) is found almost exclusively in muscle tissue and serum or plasma CK is considered an indicator of muscle damage (Armstrong, 1986; Ebbeling and Clarkson, 1989; Kuipers, 1994). Elevated CK activity in the circulation has been associated with myocardial infarction (Apple et al., 1984), degenerative muscle diseases (Rowland, 1980) and exercise induced skeletal muscle damage (Byrnes et al., 1985; Clarkson et al., 1986).

Ebbeling and Clarkson (1989) noted that when measuring CK in the blood, total CK concentration represented both the efflux and clearance of the enzyme. Thus, interpretation of peak changes in CK should be done with caution.

Although exercise induced CK elevation may indicate muscle damage, it does not provide an index of the magnitude of the damage. Kuipers et al. (1985a) examined serum CK and the amount of damage following exercise in rats. Using serial sections of muscle with light microscopy, a three-dimensional reconstruction was made and damage was quantified. The correlation between serum CK and percentage of muscle volume affected was r=0.04.

(i) Function and structure

CK is a dimeric protein found as three principle isoenzymes: muscle (MM), heart (MB) and brain (BB). CK occurs mostly in the MM isoform (approximately 90% of total CK) and only minutely in the MB and BB forms (Lang and Wurzburg, 1982; Balnave and Thompson, 1993).

CK–MM is primarily responsible for the post-exercise rise in serum or plasma CK activity (Rogers et al., 1985). With isoelectric focusing, CK–MM can be separated into three isoforms. Following physical exertion, CK is released from damaged muscle as CK–MM_1. This isoform is subsequently transformed in the blood to CK–MM_2 and then CK–MM_3. The presence of CK-MM_1 in the circulation indicates new release from damaged tissue. CK-MM_1 has been shown to be an earlier indicator of exercise induced muscle damage for both isometric (Clarkson et al., 1987) and eccentric exercise (Apple et al., 1988) regimens than total CK activity.

CK–MB has been elevated in the blood of marathon runners following a race. It is probable that increases resulted from the eccentric component of running and perhaps were exacerbated by ischaemia (Armstrong, 1986).

(ii) Time course and mechanism of efflux

The increase in serum or plasma CK activity after exercise is delayed and the extent depends upon the type of exercise. After downhill running (Byrnes et al., 1985) or isometric exercise (Clarkson et al., 1987) CK activity significantly increases three to six

hours after exercise and usually peaks 18 to 24 hours after exercise. However, after local muscular eccentric exercise, a significant increase in CK activity may not occur until 48 hours after exercise and may not reach peak values until seven days (Jones et al., 1986b; Newham et al., 1986; Clarkson and Tremblay, 1988).

Apple et al. (1984) reported that following a marathon, CK activity in the blood was usually elevated for 24 hours after the race. In well-trained marathoners, the peak CK activity occurred one day after competition, and then subsided towards the pre-race level.

After enzymes are released from muscle into extracellular spaces, they are transported to the blood by the lymphatic system (Lindena et al., 1979). The lymphatic system may also be important in the inactivation and removal of abnormal enzyme levels. Lymph flow is relatively slow therefore, the transport of CK through the lymph could explain part of the delayed appearance of CK in the blood. However, it is unlikely that this would explain delays of 24 to 72 hours observed following high force eccentric exercise (Clarkson and Tremblay, 1988).

Early CK release and ischaemia

Hypoxia and ischaemia have been popular explanations for changes in membrane permeability resulting in enzyme release. With exercise, hypoxia and/or ischaemia resulting in energy shortage may occur in skeletal muscle (Armstrong, 1986). Energy depletion may contribute to membrane permeability and/or muscle damage.

Flower et al. (1968) had subjects perform different types of exercise at various workloads (blood samples were obtained five and ten minutes after exercise). Results showed that most enzymes increased in proportion to work intensity and that trained subjects had smaller increased enzyme levels than untrained subjects at progressively higher workloads. The authors suggested that hypoxia resulted in an increased membrane permeability.

Increased CK activity in the blood following isometric exercise suggests that ischaemia may play a role in enzyme release. However, a comparison between high and low-tension isometric exercise also suggested that mechanical stress might contribute to the response (Clarkson et al., 1982).

Although changes in membrane permeability associated with metabolic disturbances may play a role in CK efflux immediately after or for several hours following exhaustive work or high tension isometric exercise, it is unlikely that these factors are a principal cause of the delayed CK efflux following high force eccentric exercise. Peak CK efflux, observed at five or more days after high force eccentric exercise (Newham et al., 1987; Clarkson and Tremblay, 1988), occurs well after the time that it takes for ATP and CP to be resynthesized. The longer delay in the appearance of CK in the blood after high force eccentric exercise has been suggested to co-inside with necrosis (Clarkson and Tremblay, 1988).

Delayed CK release and necrosis

Clarkson and Tremblay (1988) proposed a mechanism to explain the delayed CK release after local muscular eccentric exercise. These authors suggested that exercise induced damage may cause an accumulation of Ca^{2+} resulting in the production of noxious stimuli such as bradykinin and histamine causing muscle soreness, muscle contractures leading to decreased range of motion, impairment of sarcoplasmic reticulum and mitochondrial functioning as well as activation of sarcoplasmic proteases resulting in loss of sarcolemmal integrity and delayed release of CK.

Since myofibrillar damage, soreness and strength decrements have been observed prior to release of CK into the circulation, it seems that fiber destruction is progressive and that sarcolemmal disruption occurs in the final stages of necrosis (Clarkson and Tremblay, 1988). Fridén et al. (1984) found an increase in the intermediate filament protein, desmin, three days following eccentric exercise and suggested that this was evidence for sarcomereogenesis. This observation is consistent with the point at which serum CK just begins to increase, soreness is maximal and range of motion is most reduced following

local muscular eccentric exercise. However, strength is only beginning to recover at this time (Ebbeling and Clarkson, 1989).

Perhaps the repair process is initiated by the removal of noxious agents and Ca^{2+} from the fiber resulting in reduced soreness and greater range of motion. Although sarcomereogenesis may begin as soon as three days after exercise, Lazarides et al. (1981) noted that complete structural reorganization of damaged muscle fibers may take a week or longer.

(iii) Variability

Markedly high blood CK values (often over 2, 000 $U.l^{-1}$) have been found after local eccentric exercise (Clarkson and Tremblay, 1988), whereas values less than 500 $U.l^{-1}$ are commonly found after downhill running or isometric exercise (Byrnes et al., 1985; Clarkson et al., 1985).

A large intersubject variability in the CK response to exercise has been well documented (Noakes, 1987; Newham et al., 1987). In a study done by Newham et al. (1983a), where subjects performed an eccentric exercise, some subjects showed increases of CK activity up to 34, 500 $U.l^{-1}$, while other subjects showed increases of less than 500 $U.l^{-1}$. This variability is puzzling since in most cases it is unrelated to general fitness and physical characteristics of subjects, amount of soreness induced by the exercise and amount of work done during exercise (Ebbeling and Clarkson, 1989).

Clarkson and Ebbeling (1988) investigated whether the presence of CK inhibitors could explain the large blood CK variability among subjects following eccentric exercise of the forearm flexors. Subjects were classified as: 'no CK responders', 'low CK responders' (mean peak CK after exercise = 400 $U.l^{-1}$) and 'high CK responders' (mean peak CK after exercise = 2, 800 $U.l^{-1}$). Serum from high responders were mixed with serum from no or low responders. In all cases, the differences between the expected and observed CK activity for the mixes were within expected variability for the assay. The study was unable

to demonstrate the presence of CK inhibitors in the serum from subjects who showed evidence of severe exercise induced muscle damage.

There is no clear explanation for the CK variability. Many factors have been suggested to influence the inter-subject variability, including age, gender, body composition and ethnic group (Balnave and Thompson, 1993). It has been suggested that the low CK response for some subjects may be a consequence of a similar exercise performed prior to the laboratory exercise (Clarkson and Tremblay, 1988). A prior exercise may produce a rapid training response that could result in a protective effect lowering the CK response following the laboratory exercise test. This is plausible since laboratory exercises of only one-third of the number of contractions have reduced the CK response to a subsequent exercise of longer duration employing the same muscles (Clarkson and Tremblay, 1988).

(b) Myoglobin

Myoglobin is a haem-containing protein involved in oxygen transfer and storage within muscle fibers (Armstrong, 1986). Following distance running, myoglobin increases in the blood (myoglobinaemia) similar to the intra-muscular enzymes. Some of the myoglobin in the blood is also secreted in the urine (myoglobinuria) (Hansen et al., 1982). Appearance of myoglobin in the urine is one of the primary clinical signs of the general muscle syndrome referred to as 'rhabdomyolysis'. Exertional rhabdomyolysis occurs when subjects are exposed to severe, prolonged exercise and is commonly diagnosed in military recruits in the early stages of basic training (Geller, 1973; Knochel, 1982).

Byrnes et al. (1985) examined serum CK activity and serum myoglobin levels following repeated bouts of downhill running. Although, serum myoglobin levels have been routinely used in the diagnosis of myocardial infarction, few studies have assessed serum myoglobin levels following exercise. The results of the Byrnes et al. (1985) study showed that myoglobin peaked at six hours and CK peaked at 18 hours after exercise. These values compare closely with the results of a pervious study done by Cairns et al. (1983) who assessed myoglobin and CK levels every two hours following myocardial infarction and found peaks at 9.9 and 21 hours, respectively. Byrnes et al. (1985) suggested that

the earlier appearance of myoglobin could be due to a more rapid efflux of the myoglobin molecule, since myoglobin is a smaller protein than CK.

Regardless of the differences in time course between CK and myoglobin, significant correlation's were found between the absolute increases and also the relative increases in CK and myoglobin. These results suggest that damage that may have occurred during the exercise had similar effects on the two muscle proteins (Byrnes et al., 1985).

Sorichter et al. (1997) noted that although measurements of plasma CK activity and myoglobin concentrations have been used to determine muscle injury, these markers have limitations. Neither of them are skeletal muscle specific markers. Exercise-induced release of the predominantly cytoplasmic proteins, such as CK and myoglobin, can be caused by either temporary muscle fiber damage accompanied by membrane leakage with subsequent resealing of the membrane or by final death of the muscle fiber (Mc Neil and Khakee, 1992). Therefore, recent interests have been focused on contractile proteins, such as myosin heavy chain.

(c) Myosin heavy chain

Myosin heavy chain (MHC) is a structurally bound contractile protein of the thick filaments and an increase in plasma MHC concentrations after exercise indicates both membrane leakage and degradation of the contractile apparatus (Mair et al., 1992).

However, MHC shows a delayed increase after exercise induced muscle injury (Mair et al., 1995). Sorichter et al. (1997) compared troponin I as a marker of skeletal muscle damage after exercise, against CK, myoglobin and MHC fragments. Myoglobin peaked earliest (median two hours), followed by troponin I (median six hours), CK (median one day) and MHC (median two days). Therefore, Mair et al. (1995) concluded that MHC is not suitable for early diagnosis of muscle damage. Additionally the complexity of MHC isoforms and their expression patterns in striated muscles make it difficult to develop an assay that specifically identifies skeletal muscle damage.

(d) Troponin I

Troponin I (Tn I) is an inhibitory protein of the troponin-tropomyosin regulatory complex, which regulates the interaction of actin and myosin in striated muscles (Sorichter et al., 1997). There are three Tn I isoforms, one for slow twitch skeletal muscle, one for fast twitch muscle and one for myocardium.

A recent study by Sorichter et al. (1997) measured the release of skeletal Tn I (sTn I) after prolonged eccentric exercise. These authors followed the time course of sTn I and other markers of muscle damage, including CK, after downhill running and prolonged eccentric contractions of the quadriceps. They concluded that marked increases in sTn I can be detected within two to six hours from the onset of exercise induced muscle injury, with greater responses occurring for eccentric exercise.

Skeletal troponin I has a broad diagnostic window, is an initial marker, peaks within 24 hours and stays elevated for at least one to two days. In contrast to all other markers, sTn I is a protein unique to skeletal muscle. Its early increase in plasma and short time to peak indicate rapidly occurring alterations of the thin-filament troponin complex after exercise induced muscle injury. These authors also noted that although CK release was not as rapid as that of sTn I, they generally increased in parallel. Therefore, these findings suggest that CK is a good marker of muscle damage after different kinds of activity (Thompson, 1999).

(e) Lactate dehydrogenase / Alanine aminotransferase / Aspartate aminotransferase

Serum lactate dehydrogenase (LDH) activity peaks immediately, or within eight hours after exercise and returns rapidly to control values. In contrast, during acute myocardial infarction, serum LDH activity peaks 48 hours after the onset of infarction (Noakes, 1987).

The serum activities of alanine aminotransferase (ALT) increase only moderately, even with prolonged exercise. In studies of very prolonged exercise (running events lasting 5 to 24 hours), the greatest increases of 10- to 20-fold occurred in serum CK activity.

Serum activities of ALT and LDH increased two- to eight-fold after similar events (Noakes, 1987).

The activities of aspartate aminotransferase (AST) peak 24 to 48 hours after prolonged exercise, very similar to the activities of CK. The serum activities then decrease and return to control values at varying times depending on the degree to which its activity increased with exercise and the individual studied (Noakes, 1987). Downhill running has been shown to increase AST activity by approximately 110% (Maughan et al., 1989).

In part these differences can be explained by the different tissue origins of the enzymes and their intracellular location. The activities of those enzymes that exist in high concentration in skeletal muscle increase the most with exercise and those who predominate in the liver increase little or not at all with exercise. Similarly, cytoplasmic enzymes which must only cross the sarcolemma are more likely to reach the blood stream during exercise than mitochondrial enzymes which must diffuse across both the mitochondrial and sarcolemmal membranes (Noakes, 1987).

2.2.4 SKELETAL MUSCLE REGENERATION

One of the long unrecognized adaptive responses of skeletal muscle is its ability to regenerate after injury (Carlson and Faulkner, 1983). Although, according to Field (1960), skeletal muscle regeneration was first described over a century ago, it was nevertheless commonly believed, until quite recently, that skeletal muscle fibers were unable to repair themselves after damage caused by injury or disease. The ability of skeletal muscle to regenerate in all mammalian species is now well established (Mauro et al., 1970; Mauro, 1979), and many fundamental structural and functional characteristics of regenerating muscle have been delineated in laboratory animals (Carlson and Faulkner, 1983).

Although information on muscle fiber regeneration after exercise induced damage has occurred is incomplete, evidence from animal studies (Armstrong et al., 1983; Kuipers et al., 1983; Salminen, 1985) indicates that the restoration of the injured fibers follow a

similar sequence and time course to the regenerative pathways described for other types of muscle trauma (Carlson and Faulkner, 1983). Hence, one to four days after exercise the damaged fibers are phagocytised by macrophages and other phagocytic cells from the blood and interstitium. By three days after exercise the phagocytised fibers are replaced by mononuclear cells and new myofibrils are evident in developing myotubes. From three to 12 days the numbers of cells in the basal lamina tubes decrease and by two weeks after exercise the muscles appear completely normal (Armstrong et al., 1983; Salminen and Vihko, 1983).

According to Armstrong (1986) there is no evidence of a cumulative detrimental influence of this form of muscle fiber injury in muscles. They also noted that considering the number of times the muscles become sore over a lifetime, if there was not a complete restoration the muscles would deteriorate rather quickly.

Evidence from animal studies (Armstrong et al., 1983; Kuipers et al., 1983) indicates that injury occurring in rat muscle fibers during exercise is usually restricted to relatively short segments of the fibers. Therefore, the entire muscle fiber does not degenerate and the necrosis is restricted to 150–1250 μm segments (Kuipers et al., 1983). This is physiologically important, because the injured fiber is able to maintain its neural innervation. As mentioned previously from human biopsy studies, it is more difficult to determine the extent of fiber injury because of the small sample size (Armstrong, 1986).

2.2.5 REPEATED BOUT EFFECT

Although strenuous eccentric exercise results in muscle damage, performance of a repeated bout of the same exercise produces only modest changes in the indicators of damage (Newham et al., 1987; Clarkson et al., 1992; Westerlind et al., 1992; Smith et al., 1994a; Mair et al., 1995; Nosaka and Clarkson, 1995; Eston et al., 1996; Hyatt and Clarkson, 1998; Smith et al., 1998). This phenomenon is known as the "repeated bout effect" and has been shown in animals as well as in man (Kuipers, 1994). Specifically, after the first exercise there is prolonged loss in muscle strength and range of motion, a dramatic increase in muscle proteins in the blood and the development of muscle

soreness. After a repeated bout of the same exercise performed one to ten weeks later, the recovery of strength and range of motion is significantly faster than that found after the first bout, muscle protein increases in the blood are extremely blunted and soreness development is less (Nosaka and Clarkson, 1994). It appears that performance of one bout of damage–inducing exercise results in an adaptation in the muscle to the extent that it is more resistant to the effects of a subsequent bout of intense exercise.

2.2.5.1 Proposed mechanisms for the repeated bout effect

The mechanism responsible for the repeated bout effect is not known so far (Mair et al., 1995). Armstrong et al. (1983) has suggested that an initial bout of novel exercise results in a temporary reduction in the pool of stress–susceptible fibers. Muscle fibers that undergo lethal injury with exercise may be more fragile or more susceptible to damage than other fibers. Fragile fibers may develop through disuse of a given motor unit recruitment pattern or may represent a small percentage of fibers that are constantly degenerating (or aging) making them more susceptible to stress. Stress–susceptible or degenerating fibers may embody a population of cells that are destroyed by an initial bout of exercise. Strong, healthy fibers that could withstand the effects of repeated bouts would survive (Ebbeling and Clarkson, 1989).

It should be noted that it would not be necessary for an entire fiber to be fragile. Since focal necrosis can occur (Carpenter and Karpati, 1984), only portions or small areas of a fiber may be fragile and susceptible to exercise damage. There is evidence from biopsy samples of healthy human subjects that a small percentage of fibers appear disrupted (Meltzer et al., 1976). It may be these fibers that are lethally damaged during exercise (Ebbeling and Clarkson, 1989).

Although the stress–susceptible fiber hypothesis is plausible, there are data to suggest that fibers are not destroyed but strengthened (Clarkson and Tremblay, 1988). If the repeated bout effect were the result of elimination of fragile fibers, injury to this population should be evident after a single initial bout of exercise. Schwane and Armstrong (1983) found that after 30 minutes of training in rats, the training protected the vastus intermedius

muscle from damage following a 90 minute bout of downhill running performed three days later. However, there was no indication of damage after the 30 minute run suggesting that necrosis did not occur and lethal damage was not a prerequisite for muscle adaptation.

Newham et al. (1987) proposed that the mechanism responsible for the repeated bout effect could be associated with connective tissue and not muscle fibers per se. Subjects performed three repeated eccentric exercise bouts, of the forearm flexors, separated by two weeks respectively. There was an increase in serum CK activity for all subjects following bout one, but no significant changes in CK values were found following bouts two and three. Each bout produced progressively less soreness. Force decreased immediately following each of the three bouts but recovered more rapidly following bouts two and three compared with bout one. Stimulated forces also showed a decrement after each bout with a faster return to baseline following bouts two and three. There was no evidence of an increase in strength. In, fact maximum voluntary force was still reduced by approximately 20% two weeks after the third bout. Because muscle became neither stronger nor less fatigued, changes may have occurred in the connective tissue. Any damage caused by the first bout of exercise might act as a stimulus for new collagen synthesis. In this way, the collagen structure would be strengthened and protected from further damage.

It is also possible that adaptation is partly caused by an alteration in the motor unit recruitment pattern. Golden and Dudley (1992) suggested that adaptation that takes place following eccentric exercise, such that the muscle is more resistant to damage from a subsequent exercise bout, is due to a more efficient recruitment of motor units. These neural adaptations would serve a "protective" function to set a limit for excessive force generation or better distribute the workload among the fibers. Further studies are needed to investigate changes in motor unit activation and recruitment during eccentric exercise when muscle is damaged by previous exercise (Nosaka and Clarkson, 1995).

The results of experimental studies (Maier et al., 1986; Wernig et al., 1990) suggest that the observed muscle fiber injury after exercise may be part of an adaptational

degenerating–regenerating repair mechanism that involves changes in fiber type composition and contractile and other protein gene expressions in the muscle. It is likely that a combination of cellular and neurological factors is involved in the adaptation response which up to now cannot be satisfactorily explained (Mair et al., 1995; Eston et al., 1996).

2.2.5.2 Adaptations following an exposure to eccentric exercise

(a) Muscle protein changes

(i) Creatine Kinase

Creatine kinase (CK) is often used as a biochemical marker for myocardial infarction or skeletal muscle damage after exercise (Clarkson and Tremblay, 1988; Ebbeling and Clarkson, 1990). Following high force eccentric exercise, CK is released into the circulation and remains elevated for several days after the exercise protocol (Clarkson et al., 1992). Plasma CK levels generally peak on the third or fourth day following high force eccentric exercise and enzyme clearance by the reticulo–endothelial system (Sobel et al., 1976) is responsible for the return of CK to pre-exercise levels. Muscle damage after exercise has also been documented directly by myofibrillar disruption and indirectly by the perception of soreness and a prolonged loss in strength and range of motion (Newham et al., 1987; Clarkson et al., 1992). Repeating the same bout of exercise (using the same muscle group) 2 weeks to several months later, results in a reduced response to the exercise to such an extent that the loss in strength and range in motion are not as prolonged and less soreness is experienced. However, the CK response is almost completely negated following a second bout (Clarkson and Tremblay, 1988; Clarkson et al., 1992).

Although it is not known what causes the dramatic "repeated bout effect" in CK, the reduced CK response has been attributed to an adaptation effect in the exercised muscle. Clarkson et al. (1985) examined the serum CK response to a repeated bout of forearm flexion isometric exercise. Compared with the second bout, a substantial reduction in the

serum CK response was found following the second bout that was performed one week later. In another study using knee extension isometric exercise, subjects did four bouts of exercise each separated by one week (Triffletti et al., 1988). Although there was a significant difference in CK and soreness responses between bouts one and two, there were no additional reductions with subsequent bouts.

However, Nosaka and Clarkson (1994) noticed a blunted response even when the second bout was performed with the opposite arm. They analyzed serum CK activity following a second bout of exercise performed on day five after the first bout, when CK levels were still elevated. Although it was expected that CK levels after the second bout would surpass peak CK activity observed from the first bout, the CK response never exceeded the first peak. These authors suggested that accelerated clearance might have influenced CK levels.

A recent study by Hyatt and Clarkson (1998) investigated CK release and clearance, using MM variants, following repeated bouts of eccentric exercise. The authors noted that analysis of CK–MM isoforms following two bouts of eccentric exercise suggest that clearance of CK is enhanced after the first exercise. This acceleration would contribute to the dramatic blunting of total CK if a damaging exercise is performed within days of the first bout. Although there were clear differences in total CK patterns following the second bout between the groups analyzed in the study, newly released CK was detected following the second exercise for both groups using MM isoform data. It was concluded that MM variants should therefore be employed in future repeated bout studies to detect additional CK release that may not be apparent when analyzing total CK alone.

(ii) Myoglobin

Balnave and Thompson (1993) conducted a study that involved a 40 minute eccentric walk down a 25% gradient on a treadmill at 6.4 $km.h^{-1}$ once a week for eight weeks. The results showed that the effect of training on serum myoglobin was similar to that of serum CK. The elevation in serum myoglobin after the first downhill walk was greater than after

walks three, six and eight. It was also noted that the increase in serum myoglobin during week three was greater than the increases of week six and week eight.

(b) Delayed onset muscle soreness

Balnave and Thompson (1993) noted that once delayed onset muscle soreness (DOMS) appears, there is no known method of hastening its recovery and the only recognized prevention for DOMS is prior training in the particular exercise.

Eston et al. (1996) examined the effects of a prior bout of maximal isokinetic eccentric exercise on DOMS following a downhill run. The subjects in the treatment group performed 100 maximal eccentric activations of the knee extensors in the dominant leg. Two weeks later a downhill run was performed, consisting of five bouts of eight minutes at a –10% gradient at 80% of predicted maximal heart rate. The other group also performed the downhill run but without the prior isokinetic session. Although there was considerable variation in the severity of muscle tenderness among subjects, which has been observed previously (Clarkson and Ebbeling, 1988), the findings suggested that a prior bout of isokinetic eccentric exercise provides some degree of protection against further muscle tenderness.

Nosaka and Clarkson (1995) researched muscle damage following repeated bouts of high force eccentric exercise. Twelve non-weight trained males performed three sets of ten eccentric actions of the elbow flexors using a dumbbell that was set at 80% of the pre–exercise maximal isometric force level. The same exercise was repeated three and six days after the first exercise. The results showed that repeated bouts of the same moderate intensity eccentric exercise performed three and six days after the first exercise did not affect recovery from the first bout. It was also noted that although muscle soreness developed after the first exercise, there was no indication of newly produced soreness by day three and six.

Balnave and Thompson (1993) studied the effect of training on eccentric exercise–induced muscle damage. The study involved a training regimen of 40 minute eccentric

walks down a 25% gradient on a treadmill at 6.4 $km.h^{-1}$ once a week for eight weeks. Results showed that all 16 subjects experienced DOMS after the initial downhill walk. Many of the subjects reported that the soreness was the greatest along the lateral margins of the quadriceps. Subjects displayed a decrease in soreness in the quadriceps, shin and gluteal muscle groups between the first and the seven subsequent downhill walks. No significant soreness was experienced in these muscle groups after downhill walks two to eight.

(c) Changes in muscle function

(i) Muscle strength

Ebbeling and Clarkson (1989) noted that although prior performance of a local eccentric exercise can prevent release of CK upon a repeated bout other indicators of muscle damage are only attenuated. Following a repeated bout of eccentric arm exercise, soreness develops and there is a loss of strength (Clarkson and Tremblay, 1988). However, when compared with the first bout, soreness disappeared and strength recovered more rapidly following the second bout. The adaptation must protect against damage in such a way that a final stage in the damage process, necrosis or loss of sarcolemmal integrity (as indicated by CK efflux), is prevented.

Hyatt and Clarkson (1998) examined non–weight trained males performing both bouts of 50 forced lengthening contractions of the forearm flexor muscles separated by six days either with the same arm or with one arm followed by the contralateral arm. Range of motion, arm circumference, maximal isometric strength and perceived muscle soreness was assessed. Results showed that performing a second damage–inducing exercise with the same muscle group six days following the first exercise had a substantial effect on changes of range of motion, isometric strength, arm circumference and perceived muscle soreness. The difference between bout one and bout two for these parameters indicated that changes were more dramatic following the first bout than the second. These results are similar to those observed in previous studies that have documented a repeated bout effect (Ebbeling and Clarkson, 1990; Evans and Cannon, 1991).

Smith et al. (1994a) examined the impact of a repeated bout of eccentric exercise on muscular strength. Twenty-six men were randomly assigned to either a control or an experimental group. Both groups performed three sets (12 repetitions per set) of the eccentric phase of a chest press, at 80% of one repetition maximum. The experimental group repeated this exercise 48 hours later. The results concluded that after a bout of unaccustomed eccentric exercise there was a reduction in strength, most likely due to a decline in the inherent capacity of the muscle to produce force. The authors also noted that repeating a bout of exercise during the time of DOMS would not influence the time course of DOMS, serum CK or strength decrements.

2.2.6 FIBER TYPE INVOLVEMENT

There is discrepancy concerning the location of damage, especially when comparing animal models with human models (Armstrong et al., 1983; Fridén et al., 1983; Jones et al., 1986b). Both fast and slow twitch fibers may be injured (Ebbeling and Clarkson, 1989). Damage occurs predominantly in the type I fibers of animals and type II fibers of humans.

In the study of Fridén et al. (1983), evidence was presented indicating that the fast–contracting fibers were selectively damaged during eccentric training. In the study the type II/type I ratio of micrographs containing Z–band disturbances was 2.8:1 and 3.0:1 immediately and three days after exercise, respectively. The type IIB/type IIA ratio of injuries at these two occasions was 2.5:1. The authors noted that it should be kept in mind that the total average volume density of disorganized material was only 1.6 and 2.4%, although one–third and one–half of the fiber population was affected at the two points of biopsy.

This fast–twitch fiber vulnerability was in contrast with the results of Armstrong et al. (1983) in their study of downhill running rats. They showed that the deeply located predominantly slow–twitch fibers were predominantly affected, although no detailed ultrastructural fiber typing was done in that study.

Although no clear explanation exists for this difference, it may be attributed to the type and severity of exercise. Individual work periods are much longer in animal studies than in human studies (Fridén et al., 1984). Recent evidence has shown predominant fast glycolytic (type II) fiber damage in rabbit muscle that had been electrically stimulated during force lengthening (Lieber and Fridén, 1988).

According to Byrnes and Clarkson (1986) and Fridén et al. (1988) selective damage may be related to motor unit recruitment patterns and/or structural differences between type I and type II fibers. There is, however, no evidence for selective fiber recruitment on the basis of PAS–staining (Fridén et al., 1988).

2.2.7 EFFECTS OF MARATHON AND ULTRA–MARATHON RACE

Hikida and co–workers (1983) published a description of the structural damage existing in the muscles of competitive marathoners before and after a race. Their electron microscopic study demonstrated that these athletes showed significant muscle fiber damage even before a race, presumably from training and that the marathon increased the extent of the injury. Both pre- and post–race muscle samples showed evidence of muscle fiber necrosis and the presence of phagocytic cells and erythrocytes in the interstitial spaces. The effects on the muscle fibers included disruptions of the sarcolemma, disintegration and streaming of the Z–discs, accumulation of erythrocytes and phagocytes within the muscle fibers. The degenerative process in the muscles of the marathoners peaked at one to three days following the event, but were still present seven days after the race.

Some literature reports that little or no damage is observed in biopsy samples from muscles after endurance exercise. For example, Oberholzer et al. (1976) did not note any significant damage to the muscles of participants in a 100-km race, even though marked changes in substrate enzyme activity levels occurred. Some of these apparent discrepancies may be due to the particular muscles sampled in the various studies (Armstrong, 1986). Hikida et al. (1983) studied gastrocnemius muscles in the

marathoners, whereas Oberholzer et al. (1976) sampled vastus lateralis muscles in the participants in the 100-km race. It is probable that the gastrocnemius muscle is more directly involved in running than the vastus lateralis (Armstrong, 1986).

2.2.8 CARDIOVASCULAR DRIFT (HEART RATE DRIFT)

Following the initial responses after the first few minutes of exercise at a constant work rate [for example, 70-80% of maximal O_2 uptake], there is a gradual decrease in stroke volume (SV) and increase in heart rate. These two responses, as well as a progressive reduction of arterial, pulmonary arterial and right ventricular end-diastolic pressures, are the salient components of the general phenomenon of cardiovascular instability during prolonged exercise, termed "cardiovascular drift" (Nielsen et al., 1984; Hamilton et al., 1991; Montain and Coyle, 1992; Grant et al., 1997).

A study done by Montain and Coyle (1992) about fluid ingestion during exercise and increases in skin blood flow, showed that for every 1% loss in body weight due to dehydration, heart rate increased by seven beats.min^{-1}. The mechanism for cardiac drift is not entirely clear and appears to be caused partially by hypovolaemia and by other factors which are not fully explained at present (Heaps et al., 1994).

Cardiac output (CO) has also been observed to decline during prolonged exercise when the decline in SV is relatively greater than the concomitant increase in heart rate. Two factors that contribute to the progressive cardiovascular drift during prolonged exercise are the concomitant body water loss that occurs throughout prolonged exercise and a peripheral vasodilation and shift in the distribution of blood volume from the central circulation to the periphery (Hamilton et al., 1991).

2.3 RUNNING ECONOMY

2.3.1 INTRODUCTION

Running economy is defined as the rate of oxygen consumption (VO_2) at a given submaximal running velocity (v) (Bailey and Pate, 1991; Morgan and Craib, 1992; Noakes, 1992; Brandon, 1995), or as the aerobic cost of a given rate or distance of locomotion (Daniels and Daniels, 1992; Xu and Montgomery, 1995; Craib et al., 1996; Franch et al., 1998).

2.3.2 FACTORS AFFECTING RUNNING ECONOMY

Bailey and Pate (1991) classified the factors that influence running economy into external, internal and other categories. External energy (energy expended in overcoming an external resistance; factors that a runners has limited control over) includes age, segmental mass distribution, stride length and biomechanical variables. Internal energy (energy associated with oxygen delivery to the working muscles, thermoregulation and substrate metabolism) includes heart rate, ventilation and temperature and "other categories" includes VO_{2max}, training status, fatigue and mood state.

Pate et al. (1992) investigated the importance of internal, external and other variables to running economy in a large sample of male and female runners. They found that the variables that best estimated running economy were, in order of importance, ventilation, heart rate, VO_{2max} and body mass. It is important to note that the participants in the Pate et al. (1992) study were average to good runners, therefore, it is unclear whether or not the same set of variables will best predict running economy in elite runners.

2.3.2.1 Intra–individual variability

Knowledge of within-subject stability in running economy is important in determining the number of tests required to obtain a stable criterion measure prior to experimental manipulation (Morgan and Craib, 1992). Studies have shown that the mean coefficient of variation (CV) in running economy over a wide range of speeds (2.83-4.47 $m.s^{-1}$) ranged from 1 to 4% in moderately and well-trained male and female runners measured over 2, 4, 20 and 28 days under conditions in which time of day, training activity, footwear and treadmill accommodation were controlled to some extent. Technological variation, or variability associated with measurement error, accounted for less than 10% of the total coefficient of variation (Armstrong and Costill, 1985; Morgan et al., 1990a; Morgan et al., 1991; Williams et al., 1991).

Williams et al. (1991) determined the variations in running economy, at three different running velocities (2.68, 3.13 and 3.58 $m.s^{-1}$), in moderately trained runners. The coefficient of variation between the three running velocities was not significant therefore one speed did not result in any more or less variation in running economy than another. The study concluded that running economy is a relatively stable physiological measurement.

These results suggest that reliable and representative group economy measures can be obtained in trained runners with a minimum of testing if subjects are evaluated at the same time of day and in the same footwear, are non-fatigued at the time of testing and have received some period of treadmill accommodation (Morgan and Craib, 1992).

2.3.2.2 Maximal oxygen consumption (VO_{2max})

Pate et al. (1989) described the relationship between running economy and VO_{2max} to be negative in nature. More specifically, the relationship between VO_{2max} and running economy at 160 $m.min^{-1}$ was found to be significantly negative. Similarly, Pollock (1977) found $VO_{2submax}$ (at 4.5 and 5.4 $m.sec^{-1}$) and VO_{2max} to be significantly lower in elite marathoners than in elite middle-distance runners.

The possible mechanisms underlying this negative relationship between running economy and VO_{2max} are numerous (Bailey and Pate, 1991). It is possible that a negative relationship between the two variables was observed because those runners who possessed high VO_{2max} values were more accustomed to running at greater velocities than those used to assess running economy in the above mentioned investigations. Consequently, runners with lower VO_{2max} values may have trained more frequently at running velocities similar to those used in the above investigations and were more mechanically efficient at these velocities. It is also possible that individuals possessing greater VO_{2max} values were more able to utilize fat for the production of similar amounts of energy. Since the amount of oxygen needed to produce a given amount of ATP is greater when fat is the fuel source as compared to carbohydrate or protein, oxygen cost for individuals better able to utilize fat for the production of energy would be greater. In addition, segmental mass distribution may have confounding effects on the relationship between running economy and VO_{2max}. Greater concentration of body mass in the trunk area appears to be advantageous in terms of running economy. Conversely, those individuals who possess greater percentages of their body mass in the arms and legs may be able to obtain higher VO_{2max} values because a greater proportion of their lean muscle mass is active during running. Therefore, segmental mass distribution may have opposite effects on running economy and VO_{2max} (Bailey and Pate, 1991).

In summary, among a heterogeneous group of runners, VO_{2max} does correlate with distance running performance. However, when a homogeneous group of runners is studied, VO_{2max} becomes poorly correlated and running economy, highly correlated with performance (Daniels and Daniels, 1992).

2.3.2.3 Age

Children are less economical runners than are adults, but become more economical as they age, partly owing to training but also because of their weight gain. Improvements in running performance in adolescents appear to be due to changes in running economy, not VO_{2max} (Bailey and Pate, 1991; Noakes, 1992).

A study by Åstrand (1952), involving males and females of various ages showed that youngsters are less economical than adults in running. Åstrand (1952) concluded that this lower "efficiency" prevents younger boys from running as fast as more mature boys over longer distances, even though there was no difference in their VO_{2max}. These differences have been suggested to be due to the greater internal energy demand in children due to higher basal metabolic rates and a greater reliance on lipolysis for energy production when compared to their older counterparts. In comparison external energy demand for a given resistance may be lower in adolescents due to greater leg and stride lengths as well as lower body surface area to body mass ratios (Bailey and Pate, 1991).

At the other end of the age scale, Sidney and Shephard (1977) have stated that older adults, as a result of loss of flexibility, are less economical than young adults. A study done by Pate et al. (1992) examined several potential physiological, anthropometric and training determinants of running economy in a heterogeneous group of habitual distance runners. Multiple regression analysis indicated that age was significantly and positively associated with oxygen consumption while running at 161 $m.min^{-1}$. These results lead to the conclusion that younger runners are more economical than older runners.

It has been hypothesized that locomotive economy decreases with age due to reduced muscle elasticity and antagonistic muscle relaxation during activity (Larish et al., 1987). These changes may result in a reduced ability to store and use elastic energy (Pate et al., 1992).

2.3.2.4 Gender

Gender comparisons of running economy among trained and untrained subjects have produced conflicting data, with some studies indicating no gender differences in economy (Bunc and Heller, 1989), and others demonstrating that males are more economical than females (Bhambani and Singh, 1985). From a practical standpoint, methodological difficulties encountered in matching male and female subjects on VO_{2max}, training

background and running performance have probably contributed to a lack of consensus on this issue (Morgan and Craib, 1992).

A study designed by Daniels and Daniels (1992), to evaluate running economy over a range of submaximal velocities, among elite female and elite male middle- and long-distance runners made the following conclusions: when compared in running economy, male runners used less oxygen than female runners at common absolute running velocities. At equal relative intensities (%VO_{2max} or %PTRS), there were no differences in VO_2 between male and female runners. When male and female runners of equal VO_{2max} values were compared, using any method of comparison, the male runners were significantly more economical than their female counterparts. When comparisons were made between male and female runners with equal running economies, it was still found that the male runners had a better aerobic profile.

Therefore, it may be concluded that, while VO_{2max} values do differ between the sexes, gender has no effect on running economy: trained men and women are equally economical (Noakes, 1992; Daniels and Daniels, 1992)

2.3.2.5 Ethnic group

According to Geissler and Aldouri (1985), ethnic group may influence running economy. Asians and Africans have been found to utilize 17% less energy than Europeans when lying, sitting or standing, but no studies were performed during exercise. In a study done by Noakes et al. (1990) on elite runners of different racial groups, no race-related differences in running economy was found.

2.3.2.6 Flexibility

Craib et al. (1996) performed a study to examine the association between measures of limb and trunk flexibility and running economy. Correlational analysis revealed that dorsiflextion and standing hip rotation were significantly associated with the mean aerobic demand of running, such that runners who were less flexible on these measures were

more economical. Although speculative, these results suggest that poor flexibility in certain areas of the musculoskeletal system may enhance running economy in sub-elite male runners by increasing storage and return of elastic energy and minimizing the need for muscle-stabilizing activity.

2.3.2.7 Training status

The most natural way to improve running economy would be through the alteration of training status. Several investigators believe that running economy is affected by training status, however, the relationship between the two is unclear (Bailey and Pate, 1991). Middle and long distance running programs have been shown to have positive (Frank et al., 1998) and neutral (Daniels, 1985) effects on running economy. Lake and Cavanagh (1996), found that change in training status had a worsening effect on economy, while running mechanics remained the same after six weeks of training, in previously untrained subjects. The use of interval or high intensity training has had some success in improving running economy (Conley et al., 1981; Sjodin et al., 1982). Improvements in running economy from this type of training have been attributed to alterations in running style and intracellular oxidative capacity.

It is possible that there may be a certain threshold of training or a particular type of training necessary for inducing a significant change in running economy (Daniels, 1985). The level of fitness of the subjects at the start of the study is quite probably an important factor in whether changes in economy will be found (Daniels et al., 1978).

Improvement in running economy through manipulation of training status may have potential. However, more research implementing several different training protocols must be completed before a conclusion can be made as to what is the best type of training for the improvement of running economy (Bailey and Pate, 1991).

The relationship between training and the VO_2 requirement of running has also been examined by comparing economy differences between trained and untrained subjects and between elite and sub-elite runners (Morgan and Craib, 1992). Results from these

investigators are equivocal, with some studies showing worse economy in untrained or sub-elite runners and others demonstrating no difference in economy between trained and untrained runners. In a similar fashion, long distance and elite marathon runners have been shown to exhibit better, equal or worse economy when compared with middle-distance runners and sprinters (Morgan and Craib, 1992).

It is possible that training exerts a minor influence on economy and that economical runners are endowed with an anatomical or genetic makeup that produces an economical running style and favors success in longer running events (Morgan and Craib, 1992).

2.3.2.8 Cardiopulmonary and peripheral factors

(a) Heart rate and ventilation

Two variables that play an integral role in the delivery of oxygen to the working muscles and that have been shown to be significantly related to running economy are heart rate and ventilation (Pate et al., 1989). Myocardial and ventilatory work have been shown to account for 1–2% (Kitamura et al., 1972) and 7–8% (Millic-Emili et al., 1962) of the overall energy cost of exercise, respectively.

In a study by Pate et al. (1989) involving 167 habitual distance runners, both variables were significantly and positively correlated with oxygen consumption, indicating that better running economy was associated with a lower heart rate and ventilation. As noted by Bailey and Pate (1991) training-induced reductions in heart rate and ventilation might produce an overall drop in total body VO_2 leading to lower aerobic demands during exercise. However, research into the potential interrelationships between heart rate, ventilation and running economy during the course of an endurance training program is lacking (Bailey and Pate, 1991; Morgan and Craib, 1992).

(b) Muscle fiber type

Oxygen is ultimately processed in the muscle to produce energy therefore interindividual variation in running economy may be linked to differences in muscle fiber type (Morgan and Craib, 1992). Data obtained from isolated muscle preparations have indicated that the energetic demands required to generate force in fast twitch fibers are high because of a high rate of cross-bridge cycling and adenosine triphosphate consumption (Rall, 1985; Kram and Taylor, 1990)

Williams and Cavanagh (1987) observed no differences in muscle fiber type amongst 31 trained male runners who exhibited good, medium and poor economy. In contrast, a significant relationship between percent fast twitch fibers and net oxygen uptake per unit distance traveled during submaximal running was observed by Bosco et al. (1987), in 17 athletes. To explain their results it was suggested that slow twitch fibers may retain stored elastic energy longer without cross-bridge detachment, thus reducing reliance on energy generated from oxidative phosphorylation. However, methods to evaluate the potential link between elastic energy and reduced aerobic demands during running is lacking. Nonetheless, these researchers reported a significant association between the net aerobic demand of running and the ratio of efficiency of muscular work performed during pre-stretch jumps compared with a no pre-stretch condition, implying that data on the elastic behavior of muscle during jumping might have applicability to running (Bosco et al., 1987).

2.3.2.9 Fatigue

Changes in economy following intense training workouts and distance racing have been examined in elite and recreational distance runners. While some researchers have demonstrated worsened economy consequent to competitive distance racing (Cavanagh et al., 1985) or prolonged downhill running (-10% grade) at a low relative intensity (48% VO_{2max}) (Wilcox et al., 1989), a investigation by Morgan et al. (1990b) revealed no short-term change in running economy measured at 3.33 $m.s^{-1}$ (%VO_{2max} range = 56–81%) following a 30 minute level run at a mean exercise intensity of 89% of

VO_{2max}. In this study VO_2 stability was accompanied by a near absence of change in temporal, kinematic and kinetic descriptors of the gait pattern selected primarily because of their previous association with running economy. Based on these findings, the authors speculated that a hard 30 minute training run or 10-km race would not elevate $VO_{2submax}$ or perturb the running mechanics of nonfatigued runners who engaged in subsequent short term, moderate intensity submaximal runs. Little is known, however, regarding the cumulative effect of prolonged periods of rigorous training and frequent competitive distance racing on running economy (Morgan and Craib, 1992).

2.3.2.10 Detraining and overtraining

Few studies have examined the effects of overtraining and reduced training on running economy (Morgan and Craib, 1992). Kuipers and Keizer (1988) suggested that insufficient recovery from short term overtraining would increase the aerobic demand of running due to added recruitment or stimulation of motor units.

Research examining the influence of short term reductions in training load on economy has produced mixed findings, with one study showing no change in VO_2 following a ten day period of reduced training (Houmard et al., 1989) and another showing improved running economy (associated with elevated carbohydrate utilization) after three weeks of reduced training (Houmard et al., 1990). These data suggest that racing performance and many endurance training adaptations are maintained with reduced training. This information is valuable to runners and coaches in dealing with preventing injury, staleness and/or overtraining (Houmard et al., 1990).

2.3.2.11 Biomechanical factors

Bailey and Pate (1991), noted that manipulation of the biomechanical variables may be the most realistic avenue through which running economy can be altered.

(a) Body mass and mass distribution

Several investigators have described significant inverse relationships between body mass and running economy (Pate et al., 1989; Williams and Cavanagh, 1987). These relationships have been proposed to result from individual differences in distribution of mass among limb segments (Cavanagh and Kram, 1985).

It has been hypothesized that a smaller individual possesses a relatively greater amount of body mass in the extremities and would thereby have to perform a relatively greater amount of work moving body segments during running than a larger individual.

In a study done by Pate et al. (1992), results of a multiple regression analysis indicated that body weight was significantly and negatively associated with oxygen consumption while running on a treadmill at 161 $m.min^{-1}$. These results indicated that heavy runners are more economical than lighter runners, supporting the previously mentioned hypothesis.

The hypothesis has also been supported by several investigators who indicate that the increased oxygen cost of carrying an added load is greater when it is carried on an extremity than when it is carried on the trunk (Bailey and Pate, 1991).

Although the effects of segmental mass distribution on running economy have been described, avenues for improvement in running economy through changes in segmental mass distribution are not obvious. For example, it would appear to be illogical to advise an athlete to gain weight in his/her trunk in an effort to improve running economy. Such a manipulation could have a positive effect on weight distribution, but would negatively affect weight-relative VO_{2max} and distance running performance. Weight reduction from

the extremities could have a positive effect on running economy however, this is not practical (Bailey and Pate, 1991).

(b) Stride length and stride frequency

During steady-speed running there is an infinite number of combinations of stride length and stride frequency which a runner may adopt. The process by which a runner selects a particular combination would appear to be both self-determined and subconscious (Cavanagh and Williams, 1982).

The variation of stride length (SL) and stride frequency (SF) with running speed has been well documented. Hogberg (1952) was the first to document that stride length variation from that which was freely chosen by a runner resulted in an increase in submaximal oxygen consumption. Consistent with Hogberg's results, Cavanagh and Williams (1982) reported that variations from an optimum stride length results in an increasingly greater energy demand of running. Shields (1982) found that a stride length equal to 80% of total leg length was more "efficient" than was a stride length equal to 70 or 60% of leg length.

Stride length and running economy have been shown to differ between experienced and novice runners, with experienced runners possessing longer stride lengths and greater running economy. Therefore, it can be hypothesized that with training a novice will develop a longer stride length and greater running economy than that observed at the onset of training (Bailey and Pate, 1991). Nelson and Gregor (1976) found that a group of distance runners, during four years of training, increased the length of their strides and reduced stride frequency. Bailey and Messier (1991) however, found that neither stride length nor running economy changed significantly over a seven week training period in novice runners. It may be that changes in stride length and running economy take several months, if not years to develop (Bailey and Pate, 1991).

Alterations in stride length also appear to have a significant effect on the perceived effort of experienced runners. Messier et al. (1986) noted that both local (legs) and general ratings of perceived exertion (RPE) were significantly greater during runs in which the

stride length of experienced runners was 14% longer that their freely chosen stride length. Significant increases in local RPE's were also observed during runs where the same subjects were asked to over-stride by 7% or under-stride by 14%.

2.3.2.12 Environmental conditions

Environmental conditions, including the running surface, wind speed and direction, up- and downhill running, have the largest effects on a runner's economy (Noakes, 1992).

(a) Running surface

Passmore and Durnin (1955) first noted the influence of running surface on the oxygen cost of running. They showed that the oxygen cost of walking across a ploughed field was 35% greater than the cost of walking at the same speed on a smooth, firm surface. Wyngard et al. (1985) reported that running on sand had a similar effect. Mc Mahon and Greene (1979) suggested that optimizing the spring constant of a running track might improve running performance and running economy.

(b) Air and wind resistance

At low to moderately fast running speeds (2.27–4.77 $m.s^{-1}$), no differences in economy have been reported between level track and treadmill running (Mc Miken and Daniels, 1976) or between treadmill and overground running when level and graded (5.7%) conditions were compared (Bassett et al., 1985). Conversely, Daniels et al. (abstract) indicated that at equivalent or faster speeds (4.47 and 5.37 $m.s^{-1}$), level overground running in calm air increased the aerobic demand of running by 7.1% compared with the energy requirement of level treadmill running. These authors also noted that with increasing wind velocities, the detrimental effects of running into a headwind increasingly outweighed the benefits of running with a tail wind.

A practical issue for the distance runner is the potential benefit of drafting. While energy savings associated with drafting one and two meters behind another runner have been

estimated at 6% and 3%, respectively little experimental data are available (Morgan and Craib, 1992).

(c) Altitude

Limited research indicates that VO_2 demands are lower at altitude compared with sea level. Exercise at altitude requires a lower aerobic demand, despite an increase in ventilatory effort and the absence of wind resistance. A lower overall effort to breathe is observed, which is thought to be due to the reduced air density at altitude (Morgan and Craib, 1992).

Roi et al. (1999), examined the effect of altitude up to 5 200 meter on marathon performances. These researchers reported that a lower VO_{2max} mainly affected marathon performance at altitude. At altitude, VO_{2max} decreases by about 1.5–3.5% for every 300 meters of additional increase above 1 500 meters (Cerretelli, 1980). Better performance of elite marathoners at altitude, compared with good runners were related to higher percentage (%) of VO_{2max} maintained during every marathon. Differences between expected and observed performances at high altitude depended on uneven running paths and poorer economy of running that was related to the higher mechanical work of breathing (Roi et al., 1999).

(d) Uphill and downhill running

Davies (1980) calculated the additional oxygen cost of running uphill or, conversely, the energy saving of running downhill. Results showed that energy saving when running downhill was only half of the energy that would be lost when running on an equivalent uphill gradient. Therefore, uphill running increased the energy cost by about 2.6 ml $O_2.kg^{-1}.min^{-1}$ for each 1% increase in gradient. Downhill running was associated with a reduction in the oxygen cost of running by about 1.5 $O_2.kg^{-1}.min^{-1}$ for each 1% gradient, equivalent to an increase in speed of about 0.35 $km.hr^{-1}$.

(e) Temperature

The effects of temperature on running economy are complex. Different investigators have reported $VO_{2submax}$ to be increased (Saltin and Steenberg, 1964), decreased and unchanged by elevations in core temperature (Bailey and Pate, 1991). Those investigators who found an increased $VO_{2submax}$ with an increased core temperature cite increased energy requirements for peripheral circulation, increased sweat gland activity, hyperventilation and reduced efficiency of muscle metabolism as possible mechanisms. Conversely, investigators reporting $VO_{2submax}$ to be reduced or unchanged with an increase in core temperature believe that it is possible that the efficiency of muscle metabolism is improved (i.e. the amount of O_2 needed to produce a given amount of ATP is reduced) as muscle temperature is increased. It is possible that $VO_{2submax}$ is reduced slightly as muscle temperature is moderately increased but eventually increases as the mechanisms involved in heat dissipation are activated to a greater extent (Bailey and Pate, 1991).

The economy of running in a heat stress environment should be improved by heat acclimatization. Acclimatization, accompanied by exercise training, can increase plasma volume up to 12%. Increased plasma volume assists in the maintenance of stroke volume and consequently minimizes myocardial work in a heat stress environment. Further, an increased plasma volume improves sweating capacity and enables the body to tolerate greater internal heat production (Bailey and Pate, 1991).

2.3.2.13 Differences in capacity of the body to store energy

With each running stride, the muscles of the landing leg store "impact energy" as they contract eccentrically to absorb the shock of landing (Williams, 1985). Most of the stored energy is then used during the concentric muscle action that propels the body forward during the next stride. It has been suggested that we use the impetus of landing to assist the muscular effort of takeoff. It is possible that the muscles of uneconomical runners have less ability either to store or utilize this form of energy (Noakes, 1992).

2.3.2.14 Substrate utilization

It may be possible to alter running economy by altering the type of fuel (i.e. carbohydrate or fat) used to produce energy. When carbohydrate is used for energy production 5.05 kcal of energy are produced for every liter of oxygen consumed, whereas when fat is used for energy metabolism 4.70 kcal of energy are produced for every liter of oxygen consumed (Brooks and Fahey, 1985). Therefore, greater carbohydrate use during running at a given speed should be associated with lower oxygen cost. This type of manipulation can potentially be accomplished by either increasing the supply of carbohydrate to the working muscles or by minimizing the availability of fatty acids to the muscles. Ingestion of carbohydrate during prolonged exercise has been shown to improve endurance performance and increase respiratory exchange ratio, indication that more carbohydrate is being used for energy production. However, oxygen cost has been minimally reduced under such manipulations, indicating that ingestion of carbohydrate during prolonged exercise probably has little effect on running economy (Bailey and Pate, 1991).

2.3.2.15 Differences in technique

Running economy may change for the same athlete during different types of exercise, for example uphill or downhill running (Gregor, 1970), or during different activities such as cycling or step climbing (Daniels, 1985). It is possible that runners, who are economical on the flat, may be uneconomical while running either up- or downhill. Also, some economical runners may be less economical at cycling than are other runners, who are less economical runners (Noakes, 1992).

2.3.2.16 Clothing and running shoes

Stevens (1983) has calculated that changing from nylon clothing (weighing about 150 grams) to cotton clothing (weighing about 240 grams) would increase the time taken by a world class runner to complete the marathon by about 13 seconds and that of the average 03:40 marathoner by about 23 seconds. However, laboratory experiments do not

necessarily substantiate these calculations. Cureton et al. (1978) found that the addition of up to 4 kg to the torso increased the oxygen cost of running by only about 2.5%, or about 0.5% for 1 kg of extra weight.

Extra weight added to the legs or feet appears to have a far greater effect on running economy. The addition of 0.5 kg to each thigh or to each foot increased the oxygen cost of running by 3.5 and 7.2% respectively (Martin, 1985). Jones et al. (1986a) noted that the increase is the same in men and women.

2.3.2.17 Psychological factors

Psychological state refers to a particular cognitive or emotional condition of the mind. Running economy is defined as the amount of oxygen consumed to run at a given submaximal speed. Therefore, the influence of psychological state on running economy will examine whether cognition's or emotions influence oxygen cost at a given workload (Crews, 1992). A review by Morgan (1985) presented three categories of factors that could possibly influence exercise metabolism: affect, perception and cognition.

(a) Affect (Mood state)

Affect refers to an emotion or feeling related to an idea or object (Crews, 1992). Williams et al. (1990) investigated the effects of mood state on within subject variability in running economy. These authors found that a more positive mood state, as measured by the Profile of Mood States (POMS), was significantly correlated with greater running economy. Furthermore, correlations determined between the six POMS subscales and running economy indicated that tension held the strongest association (Bailey and Pate, 1991). The correlation between running economy and mood state may be the result of a common underlying mechanism however, these results do not indicate that a cause and effect relationship exists between the two (Bailey and Pate, 1991).

(b) Perception

Perception involves the use of senses, awareness and comprehension to understand objects and qualities in the environment (Crews, 1992). Research attempting to alter perception and to influence the physiological response to exercise has not shown positive results. Several methodological considerations my help to explain this relationship. None of the studies in this area used running or walking as mode of exercise. Second, hypnosis was used in most studies to alter perception. During hypnosis the individual is passive rather than active in controlling cognitions and this may affect physiological responses during exercise. Third, Type A personality is difficult to define using psychological inventories (Crews, 1992).

Crews (1992) also noted that the personality characteristic, "hardiness" could relate to running economy. Hardy personalities tend to be curious, expect change, view obstacles as challenges and perceive to have control over their environment. These characteristics may relate to running economy, for they present a more positive, controlled approach to life.

(c) Cognition

Memory, judgements and perception are integrated categories of cognition, or the process of knowing. Three categories of cognition that possibly influence running economy are mental strategies, coping strategies and biofeedback (Crews, 1992).

Conscious thoughts devised specifically to modify behavior would be considered mental strategies. Morgan and Pollock (1977) conducted a descriptive study examining the mental strategies of elite distance and middle distance runners vs. non-elite runners. Elite runners showed reduced oxygen cost at 16 $km.h^{-1}$ compared with the other two groups (Crews, 1992).

Techniques used to counteract the effects of exercise stress are coping strategies. Benson's relaxation technique was used by Benson et al. (1978) to reduce anxiety while

subjects exercised at a fixed work intensity on a cycle ergometer. Using a multiple baseline approach, subjects were able to lower their oxygen cost by 4% during the relaxation phase. The opposite results occurred when Cadarette et al. (1982) used Benson's relaxation protocol. Subjects exercised for 40 minutes at 50 Watt. Compared with a control group, oxygen cost was not altered however, respiration rate, tidal volume and ventilatory minute volume were reduced.

Information provided to individuals representing their own physiological responses is commonly referred to as biofeedback. Hatfield et al. (1986) trained 10 athletes using respiratory biofeedback and compared this condition with a control and a distraction condition. Subjects ran for 36 minutes on a treadmill at a speed that was just below their anaerobic threshold. Results indicated improved ventilatory economy following training however, oxygen cost did not differ.

2.3.3 RELATIONSHIP BETWEEN RUNNING ECONOMY AND PERFORMANCE

The aerobic demands of submaximal running have been investigated for many years however, VO_{2max} has generally been the factor that has received the most attention when attempting to explain running performances and also when identifying talented endurance athletes. Among a heterogeneous group of runners, VO_{2max} does correlate highly with distance-running performance. However, when a homogeneous group of runners is studied, VO_{2max} becomes poorly correlated and running economy highly correlated, with distance running performance (Noakes, 1992; Daniels and Daniels, 1992).

Costill and Winrow (1970) studied two top veteran ultra-marathon runners, with similar VO_{2max} values but with different running performances. These authors noted that the amount of oxygen each runner utilized when running at each of four submaximal running speeds, of between 10.8 and 16 $km.hr^{-1}$, differed quite substantially. Daniels (1985) and Sjodin and Svedenhag (1985) have reported that running economy can differ by as much as 30% even in trained runners.

Conley and Krahenbuhl (1980) studied a group of 12 elite distance runners, with best 10-km times ranging between 30.31 and 33.33 $min.s^{-1}$. They found that these runners' VO_{2max} values (ranging from 67 to 78 $O_2.kg^{-1}.min^{-1}$) could not be used to predict their 10-km times. However, there was a good correlation between the runners' VO_2 values at each of three submaximal running speeds and their best time for the 10-km race. It was concluded that a high VO_{2max} (above 67 $O_2.kg^{-1}.min^{-1}$) assisted each athlete to gain membership of an elite performance group, but within this select group, running economy and not VO_{2max} was the factor determining success in the 10-km race (Conley and Krahenbuhl, 1980).

Noakes (1992) interpreted the data differently. To gain entrance to the above mentioned elite group, the athletes need muscles with superior contractility. These muscles then allow the athlete to achieve a high work rate during the maximum test to exhaustion. This high work rate demands a high rate of oxygen consumption, which is interpreted as a high VO_{2max}. However, the athletes' running economy will determine the exact VO_{2max} value; uneconomical runners will have high VO_{2max} values and economical runners will have lower values.

The contribution of running economy to performance in middle distance events differs from long distance events (Brandon, 1995). Middle distance runners have the ability to run at low and high velocities with similar economies, in some cases the runners are reported to be able to run at higher velocities with a better economy than at lower velocities (Daniels, 1985; Daniels and Daniels, 1992). This is different from the traditionally accepted concept that a linear relationship exists between running speed and energy cost (Brandon, 1995). However, Daniels (1985) noted that the theory of a linear relationship between velocity and energy cost appears to hold up during submaximal runs where the energy cost is based almost solely on aerobic energy sources. Running performance at high velocities is also dependent on anaerobic energy sources.

In summary, when comparing the running efficiencies of elite athletes, it becomes apparent that runners with high VO_{2max} values, but relatively poor running performances, are probably less economical runners, whereas runners with comparatively low VO_{2max}

values, with outstanding performances, are more economical runners. Lastly, differences in running economy also occur in athletes who are matched according to VO_{2max} values and levels of training. This suggests that, although training may improve running economy, there are inherent differences in running economy between different athletes and that these differences cannot be completely removed by training (Daniels, 1985; Noakes, 1992).

2.3.4 CONCLUSION

Intensive training for and competition in, endurance events like the marathon are often accompanied by injury to fibers in the active skeletal muscles. Evidence for these injuries comes from the increase in intramuscular enzymes and myoglobin found in the blood following such exercise, from the subjective sensation of soreness in the muscles in the post-exercise period and from direct histological examination of samples of the damaged muscles. Histological studies demonstrate that some muscle fibers undergo degenerative changes following exercise although this is then followed by regeneration so there is no net loss of fibers. Precisely what initiates the cellular damage is not known, but hypotheses suggested include "metabolic overload" and "mechanical strain" (Armstrong, 1986). Eccentric actions are known to cause the greater amount of damage in muscles, which suggests that high local tensions in fibers may be more important than metabolic considerations in the etiology of the injury. Training reduces the magnitude of the damage that occurs in response to a given exercise task (Armstrong, 1986). This phenomenon is known as the "repeated bout" effect (Nosaka and Clarkson, 1995; Smith et al., 1998).

Recently the main focus of research has been to investigate delayed onset muscle soreness, elevated serum activities and impaired muscle function following repeated bouts of eccentric muscle action. There appears to be limited research, however, that has investigated the effect of repeated bouts of eccentric exercise as part of a training program on performance in, and recovery after, endurance events. To determine if such an eccentric training program prior to running an endurance event could be successful,

the different variables that play a role during the training phase, the endurance event and the recovery period thereafter must be examined.

One such a variable is running economy, which is defined as the rate of oxygen consumption at a given submaximal running velocity. Several investigators have demonstrated that running economy is correlated with endurance performance (Bailey and Pate, 1991), particularly within groups that are homogeneous in terms of maximal aerobic power (VO_{2max}). Factors that have been shown to be associated with running economy are numerous and include heart rate, ventilation, VO_{2max}, gender, age, temperature, fatigue, training status, body and segmental mass distribution stride length and various other biomechanical parameters.

Alterations in a training program, such as including eccentric training (downhill running), may precipitate changes in some of the above mentioned variables that could then lead to positive affects on running economy. An enhanced training regime may favorably change running economy by improving running style and intracellular oxidative capacity.

In conclusion, the effect of repeated bouts of eccentric exercise as part of a training program on running performance in, and recovery after, endurance events has not been a common focus of research in the field of delayed onset muscle soreness. This dictates that the review of literature needs to include not only those studies which deal with exercise induced muscle damage but also those which examine the various factors associated with running economy specifically.

CHAPTER THREE

THE EFFECT OF REPEATED BOUTS OF DOWNHILL TRAINING ON RUNNING PERFORMANCE AND RECOVERY AFTER A 30-KM TIME TRIAL

3.1 MATERIALS AND METHODS

The methodology of the study will be presented in the following manner. First the characteristics of the subjects are shown. Next the study design will provide a brief summary of the testing schedule. Thereafter each individual test administered for the purpose of the project will be explained in detail.

3.1.1 SUBJECTS

Sixteen healthy male subjects (26 to 48 years) and licensed members of local running clubs were recruited to participate in the study. Subjects were recruited through advertisements in running magazines, on the Internet and at running clubs in the Central Gauteng region. The subjects were randomly allocated to either a downhill group (n=9) or a level group (n=7). Subjects in the downhill group performed nine 40 minute downhill runs (-10% gradient, 70% PTRS), whereas the subjects in the level group performed nine 40 minute level runs (0% gradient, 70% PTRS) during the training phase of the study. All the subjects must have been running competitively for at least two years and the subjects must have completed at least one marathon. The study was approved by both the Ethics and Research Committee of the Faculty of Health Sciences, University of Cape Town and the Committee for research on human subjects of the University of the Witwatersrand. All subjects were informed both verbally and in writing about the nature and the demands of the study and asked to sign an informed consent (Appendix A) before commencing the trial.

Subjects were asked to maintain their normal training regime for the duration of the study (nine weeks) and not to begin any new recreational or training programs. Subjects were excluded from the study if they were injured or if they were using any medication that may have influenced their responses to the various tests. A full retrospective running history (Appendix B), which included best 10-km, 21.1-km, 42.2-km and Comrades marathon times (90-km) as well as total training distance per year (kilometers per year), was obtained from each subject. Subjects were also required to keep a detailed training logbook (Appendix C) for the duration of the study.

The subjects in the downhill and level group were matched according to age, training history and running performance.

3.1.2 STUDY DESIGN (TESTING SCHEDULE)

The study followed a quasi–experimental design, with repeated measures and correlational analysis. The study design (testing schedule), for the downhill and level group, has been explained on a time line (Appendix D).

3.1.2.1 Experimental protocol

(a) Preliminary testing

On the first visit to the laboratory, six weeks before the 30-km time trial, an anthropometrical assessment was conducted on each subject for the estimation of body composition. Each subject was asked to complete a training data and racing history data sheet. The subjects also received a training logbook to record their training for the duration of the study. Maximal oxygen consumption (VO_{2max}) and peak treadmill running speed (PTRS) were determined. Subjects also wore a heart rate monitor during the VO_{2max} test to determine maximum heart rate (HR_{max}).

Each subject also performed a 15 minute submaximal recovery run ($VO_{2submax}$) an hour after the VO_{2max} test. Oxygen consumption (VO_2), minute ventilation (V_i), carbon dioxide

production (VCO_2), respiratory exchange ratio (RER), heart rate (HR), rate of perceived exertion (RPE), stride frequency (SF) and stride length (SL) were measured during the submaximal run.

(b) Downhill (Eccentric) training protocol

A week after preliminary testing the subjects in the downhill group started performing repeated bouts of eccentric exercise (nine 40 minute downhill runs; -10% gradient) over a period of five weeks. Prior to each downhill run a blood sample was taken for the analysis of plasma creatine kinase (CK) activity and muscle soreness was evaluated both subjectively and objectively. Each subject also wore a heart rate monitor, for the duration of the 40 minute run, to record heart rate.

During the first (Week 1) and last week (Week 5) of downhill (eccentric) training, blood samples were also taken and muscular soreness was evaluated at 24, 48, and 72 hours after the 40 minute downhill run.

(c) 30-km Time trial

After the five weeks of downhill (eccentric) training all the subjects in the downhill group performed a 30-km time trial.

Prior to and immediately after the 30-km time trial blood samples were taken for the analysis of plasma creatine kinase (CK) activity and plasma glucose concentration. Body mass was recorded before and after the 30-km time trial.

Each subject wore a heart rate monitor for the duration of the 30-km time trial to record heart rate. Rate of perceived exertion, stride frequency and stride length were recorded during the 30-km time trial.

(d) Recovery testing

The last stage of testing consisted of four submaximal recovery runs (15 minute duration). These runs were performed on day 4, 7, 14 and 21 after the 30-km time trial. Prior to each submaximal recovery run ($VO_{2submax}$) a blood sample was taken for the analysis of plasma creatine kinase (CK) activity and muscular soreness was evaluated both subjectively and objectively. Oxygen consumption (VO_2), minute ventilation (V_i), carbon dioxide production (VCO_2), respiratory exchange ratio (RER), heart rate (HR), rate of perceived exertion (RPE), stride frequency (SF) and stride length (SL) were measured during each submaximal run.

In the week immediately after the 30-km time trial (Recovery Day 1, 2, 3, 5 and 6), blood samples were taken for the analysis of plasma creatine kinase activity and muscular soreness was evaluated.

3.1.2.2 Level training protocol (level group)

The testing protocol was similar for the subjects in the level group, except that these subjects performed nine 40 minute level training runs (0% gradient) rather than nine 40 minute downhill training runs (-10% gradient).

3.1.3 ANTHROPOMETRICAL ASSESSMENT

Anthropometrical composition was calculated according to the procedures described by Durnin and Womersley (1974) and Ross and Marfell–Jones (1991). The sum of four skinfolds (Durnin and Womersley, 1974) was used to describe body fat percentage. The calculation involved the measurement (to the nearest 0.1 mm) of the biceps, triceps, subscapular as well as the suprailiac. Body fat was also assessed by the sum of seven skinfolds. Skinfold thickness was measured to the nearest 0.1 mm at the triceps, biceps, suprailiac, subscapula, abdomen, calf and mid thigh as described by Ross and Marfell-Jones (1991). All measurements were taken on the right side of the body, except for the

abdominal measurement, which was recorded on the subjects left side, with a skinfold caliper (Holtain, Froud, Essex, U.K.)

Stature was recorded, to the nearest 0.1 cm, with subjects barefoot and with their arms hanging by their sides. Body mass was recorded on a calibrated scale, to the nearest 100 grams, with the subjects only wearing light clothing and without shoes. The age of each subject was recorded (Appendix E).

3.1.4 MAXIMAL OXYGEN CONSUMPTION TEST, PEAK TREADMILL RUNNING SPEED AND MAXIMUM HEART RATE

A maximal treadmill test was conducted in order to determine maximum oxygen consumption (VO_{2max}), peak treadmill running speed (PTRS) and maximum heart rate (HR_{max}) (Appendix F). Prior to the maximal test, subjects warmed up. The nature, duration and intensity of the warm–up was specific to each subject. The sequence and time course of each individual warm–up was documented and was maintained, by the subject, throughout the study.

The maximal test was performed on a motor driven treadmill (Powerjog, Ergoline EG30, Birmingham, England), using a continuous, incremental running protocol. The energy cost of treadmill running most closely represents that of outdoor running when the gradient of the treadmill is set at 1% (Jones and Doust, 1996). Therefore, a treadmill gradient of 1% was maintained during the maximal oxygen consumption test. Before each test, the gradient of the treadmill was calibrated using a spirit level.

Following the warm–up, subjects were fitted with a Polar Sport Tester heart rate monitor (Polar Vantage XL, Polar Electro, Kempele, Finland), which recorded heart rate at five second intervals during the maximal test.

The subjects then mounted the treadmill and the speed was increased to 10 $km.h^{-1}$. This speed was maintained for two minutes, after which it was increased by 0.5 $km.h^{-1}$ every 30 seconds until volitional fatigue. This protocol has previously been described as the

Noakes protocol. During the maximal test, subjects wore a mouth–piece and a nose clip. The expired air passed through an on–line computer system attached to an Oxycon 4 (Mijnhardt, Bunnik, Netherlands) automated gas analyzer using a Hans Rudolph non-rebreathing valve system (Wyandotte, Kansas City, USA) for the determination of oxygen consumption (VO_2). Before each test, the gas analyzer was calibrated using a gas of known composition (gas chromatograph, Afrox, Pty. Ltd.) The composition of the calibrated mixture resembled that of expired air (O_2: 16.34%; CO_2: 5.59%; N_2: 78.07%). Analyzer outputs were processed by a computer which calculated minute ventilation (V_i), oxygen consumption (VO_2), carbon dioxide production (VCO_2) and respiratory exchange ratio (RER), using conventional equations.

Each subject's VO_{2max} (ml $O_2.kg^{-1}.min^{-1}$) was defined as the VO_2 value that coincided with volitional fatigue. Peak treadmill running speed (PTRS) was defined as the highest speed that the runner could maintain for a complete 30 second increment prior to fatigue. Maximal heart rate (HR_{max}) was defined as the highest heart rate during the last 30 seconds of the treadmill test.

The peak treadmill running speed (PTRS) was used to determine the work rate for the subsequent 40 minute training sessions (downhill running for the downhill group and level running for the level group) as well as for the 15 minute submaximal recovery tests and the 30-km time trial. The work rate that was selected corresponded to 70% of the subject's PTRS and was kept constant throughout the experiment. Verbal encouragement was provided by the researcher to elicit a maximal effort from each subject.

3.1.5 DOWNHILL RUNNING (ECCENTRIC TRAINING)

The subjects in the downhill group participated in the downhill training protocol. Each subject completed nine 40 minute downhill training runs (Appendix G). The first downhill training run was conducted a week after preliminary testing. The following week each subject in the downhill group completed two x 40 minute downhill training runs, the week after all the subjects completed three x 40 minute downhill training runs. During the fourth

week each subject completed two x 40 minute downhill training runs and during the fifth week all the subjects completed one downhill training run.

The downhill training protocol was executed in the following manner:

TRAINING WEEK	NUMBER OF TRAINING RUNS
1	1 x 40 minute run
2	2 x 40 minute runs
3	3 x 40 minute runs
4	2 x 40 minute runs
5	1 x 40 minute run

Prior to the 40 minute downhill training runs, the subjects warmed up. The nature, duration and intensity of the warm–up was specific to each subject. The downhill runs were performed on a motor driven treadmill (Powerjog, Ergoline EG30, Birmingham, England) with the gradient set at –10%. Before each test, the gradient of the treadmill was calibrated using a spirit level. The subjects then mounted the treadmill and the speed was increased to the pre–determined work rate of 70% of PTRS for each subject. Each subject completed a 40 minute treadmill test at this intensity.

The time of day of testing for each subject was kept constant for the duration of the study. During all testing laboratory conditions were standardized at a temperature of approximately 21° C.

3.1.6 LEVEL RUNNING

The subjects in the level group participated in the level training protocol (0% gradient). Each level subject completed nine level training runs. The first level training run was conducted a week after preliminary testing. The following week each subject in the level group completed two x 40 minute level training runs, the week after all the subjects in the level group completed three x 40 minute level training runs. During the fourth week each

subject completed two x 40 minute level training runs and during the fifth week all the subjects in the level group completed their last level training run.

Prior to the level tests the subjects warmed up. The nature, duration and intensity of the warm–up was specific to each subject. The level tests were performed on a motor driven treadmill (Powerjog, Ergoline EG30, Birmingham, England) at 70% of peak treadmill running speed, with the gradient set at 0%. Before each test, the gradient of the treadmill was calibrated using a spirit level. The subjects then mounted the treadmill and the speed was increased to the pre–determined work rate for each subject. Each subject completed a 40 minute treadmill test at this intensity.

The time of day of testing for each subject was kept constant for the duration of the study. During all testing laboratory conditions were standardized at a temperature of approximately 21° C.

3.1.7 MEASUREMENT OF HEART RATE

Heart rate was measured during the five submaximal recovery runs and also during the 30-km time trial.

Before each submaximal recovery test, subjects were fitted with a Polar Sports Tester heart rate monitor (Polar Vantage XL, Polar Electro, Kempele, Finland), which recorded heart rate at five second intervals during the 15 minute submaximal recovery test. The heart rate values were later averaged at 3, 6, 9, 12 and 14.5 minutes for subsequent statistical analyses.

Prior to the 30-km time trial, subjects were fitted with a Polar Sports Tester heart rate monitor (Polar Vantage XL, Polar Electro, Kempele, Finland), which recorded heart rate at 15 second intervals. The heart rate values were later averaged every two kilometers (at 2, 4, 6, 8, 10, 12, 14, 16, 18, 20, 22, 24, 26, 28 and 30 kilometers) for subsequent statistical analyses.

3.1.8 DETERMINATION OF HEART RATE DRIFT

Before each 40 minute training test (downhill run for the downhill group and level run for the level group), the subjects were fitted with a Polar Sports Tester heart rate monitor (Polar Vantage XL, Polar Electro, Kempele, Finland), which recorded heart rate at five second intervals during the 40 minute test. All non-physiological heart rate values were filtered out, during the first few minutes of the recording, before calculating average heart rate and standard deviation (SD).

To determine heart rate drift, the average heart rate and SD were calculated from minute 5 to 6 and also from minute 38 to 39. Heart rate drift was defined as the delta change in heart rate (beats.min^{-1}) between minute 5 and minute 38. The correlation and slope was also calculated.

3.1.9 BLOOD SAMPLING AND ANALYSIS

Blood samples were taken to enable the quantification of plasma creatine kinase activity (U.l^{-1}) and plasma glucose concentration (mmol.l^{-1}). All blood samples were taken from veins of the antecubital fossa.

A 5 ml sample, for later analysis of plasma creatine kinase concentration, was collected into a pre–chilled tube containing lithium heparin. All samples were kept on ice until centrifugation. Samples were centrifuged at 3000 rpm for 15 minutes upon completion of each treadmill test (Hemle, Zeiss, Germany). A 1 ml plasma sample was pipetted into an Eppendorf reaction tube and stored at approximately –20° C for later analysis.

Plasma creatine kinase concentrations were measured by spectrophotometric (Beckman DU–62, Beckman Instruments, Fullerton, California, USA) enzymatic assays. Analysis of the enzyme activity was achieved using a CK NAC–activated Kit (Boehringer Mannheim, Meylan, France). The rate of conversion of NAD^+ to NADH at 30° C was determined by measuring change of absorbance at 340 nm with the spectrophotometer.

Blood samples for the measurement of plasma glucose concentration ($mmol.l^{-1}$) were taken prior to and 34.1 ± 8.9 seconds after the 30-km time trial. Approximately 2 ml of blood was collected into a test tube containing both sodium fluoride and potassium oxalate (these compounds function to inhibit glycolysis), for the subsequent analysis of plasma glucose concentration. The samples were put on ice and centrifuged at 3000 rpm for 15 minutes (Hemle, Zeiss, Germany). Plasma was pipetted into eppendorf reaction tubes and stored at approximately –20° C for later analysis.

Plasma glucose concentrations ($mmol.l^{-1}$) were measured by the glucose oxidase method in a glucose analyzer (Glucose Analyzer 2, Beckman Instruments, Fullerton, California, USA).

3.1.10 MEASUREMENT OF MUSCULAR PAIN

Muscle pain was assessed both objectively and subjectively (perceived pain).

3.1.10.1 Objective muscle pain

Objective pain was measured with a pressure probe. The pressure probe is a spring–loaded plunger, designed by the Department of Biomedical Engineering at the University of Cape Town. With the application of a vertical force downwards, the pressure probe delivers a force of 4 N to the muscle, for every 1 cm change in length. The end of the plunger is covered with a plastic cap, to minimize any potential discomfort that could result from the application of the pressure probe to the muscle.

The rectus femoris and the vastus medialis muscles of the quadriceps femoris muscle of the subject's right leg were used for the measurement of objective pain. Subjects were required to sit, with their legs hanging over the edge of a chair. The muscles were identified after an isometric contraction. The belly of each muscle was located by palpitation. A transparent grid was placed centrally on each muscle belly. The grid was marked out on a xerox transparency, with the area of each square on the grid being 1 cm by 1 cm. Holes were made in the transparency at the intersection of each line and on the

borders of the grid. The area of the grid marked out on both the rectus femoris muscle and the vastus medialis muscle was 2 cm by 2 cm, which corresponded to nine sites on each muscle. Subjects were instructed to relax their legs and the pressure probe was applied to each of the nine sites over each muscle.

Objective pain for each of the nine sites over each muscle was quantified using an arbitrary scale. A pain score of zero was assigned if the plunger was depressed by 4 cm (16 N) and the subject experienced no pain. A pain score of one was assigned if pain was felt with the plunger at 3 cm (12 N), a score of two if pain was felt with the plunger at 2 cm (8 N), a score of three if pain was felt with the plunger at 1 cm (4 N) and a maximal score of four if pain was felt with the plunger at 0 cm (0 N). The pain scores for each of the nine sites over each muscle were summed and from this objective pain scores for the rectus femoris and vastus medialis muscles were determined.

3.1.10.2 Subjective muscle pain (Perceived pain)

Two methods were used to quantify perceived muscular pain. Perceived pain was firstly quantified by using a perceived muscle soreness rating scale (Appendix H). Subjects were instructed to squat and to then rate the degree of discomfort they experienced in their leg extensor muscles. The scale was set from zero to ten, with a zero score comparable to no pain being experienced in the leg extensor muscles on squatting, while a score of ten was comparable to maximal pain being experienced in the leg extensor muscle on squatting. The subjects were not allowed to consult their ratings of pain on previous occasions.

The second method used to quantify perceived muscular soreness involved the use of two diagrams of the superficial skeletal muscles of the body's musculature. The first diagram was of the anterior view (Appendix I) and the second diagram was of the posterior view (Appendix J) of the body's musculature. The subjects were instructed to use a colored pen to label each diagram (Appendix K) to indicate exactly where they experienced soreness. Perceived muscular soreness could be indicated at 13 sites on the anterior diagram of the musculature. These sites included the rectus abdominis,

quadriceps femoris muscles (rectus femoris, vastus lateralis and vastus medialis), gastrocnemius and soleus, tibialis anterior and peroneus longus as well as extensor digitorum brevis and flexor digites brevis. Subjects could indicate perceived soreness at 13 sites on the posterior diagram of the musculature, these sites included latissimus dorsi, gluteal muscles (gluteus maximus), hamstrings (semitendinosus, biceps femoris and semimembranosus), the gastrocnemius muscles and the calcaneal (Achilles) tendon and flexor hallicus longus.

Perceived pain for each of the 13 sites on both the anterior and posterior diagram's was quantified by using an arbitrary scale. A score of zero was awarded if a subject did not indicate any perceived pain at a particular site and a score of one was awarded if the subject indicated that pain was experienced. The pain scores for each of the 13 sites were summed and from this objective pain scores for the anterior and posterior view of the superficial skeletal muscles of the body's musculature were determined.

3.1.11 SUBMAXIMAL RUNNING AND MEASUREMENT OF OXYGEN CONSUMPTION

The subjects in both the downhill and level group participated in the submaximal recovery tests. Each subject completed five submaximal recovery runs. The first run was conducted during preliminary testing and the remaining submaximal recovery runs were conducted on day 4, 7, 14 and 21 after the 30-km time trial (Appendix L).

Prior to each submaximal recovery test, subjects warmed up. The nature, duration and intensity of the warm–up was specific to each subject, as previously described.

The 15 minute submaximal recovery test was performed on a motor driven treadmill (Powerjog, Ergoline EG30, Birmingham, England). As soon as the subjects mounted the treadmill, the speed was increased to 10 $km.h^{-1}$. This speed was maintained for two minutes, after which it was increased to the pre–determined work rate of 70% of peak treadmill running speed (PTRS) for each subject. After the pre–determined work rate was reached the 15 minute submaximal recovery test started, with the gradient set at 0% for

the first five minutes. Thereafter the gradient was decreased to –10% for the next five minute period and for the last five minutes the gradient was increased again to 0%. Before each test, the different gradients of the treadmill were calibrated using a spirit level.

During the submaximal test, subjects wore a mouth–piece and a nose clip. The expired air passed through an on–line computer system attached to an Oxycon 4 (Mijnhardt, Bunnik, Netherlands) automated gas analyzer using a Hans Rudolph non-rebreathing valve system (Wyandotte, Kansas City, USA) for the determination of oxygen consumption (VO_2). Before each test, the gas analyzer was calibrated using a gas of known composition (gas chromatograph, Afrox, Pty. Ltd.) The composition of the calibrated mixture resembled that of expired air (O_2: 16.34%; CO_2: 5.59%; N_2: 78.07%). Analyzer outputs were processed by a computer which calculated minute ventilation (V_i), oxygen consumption (VO_2), carbon dioxide production (VCO_2) and respiratory exchange ratio (RER), using conventional equations.

These values were recorded every 15 seconds for the duration of the submaximal recovery test. The values were later averaged at 3, 6, 9, 12 and 14.5 minutes for subsequent statistical analyses.

3.1.12 DETERMINATION OF PERCEIVED EXERTION

During the submaximal recovery tests and the 30-km time trial, the subjects were required to indicated their rate of perceived exertion, using a modified Borg scale (Borg, 1982; Mc Ardle et al., 1994) (Appendix M). The perceived exertion scale ranged from 6 to 20, with a 7 score comparable to "*very, very light exertion*", while a score of 20 was comparable with "*maximal exertion*".

During submaximal recovery testing perceived exertion was measured immediately after starting the test and then at 3, 6, 12 and 14.5 minutes during the test. During the 30-km time trial perceived exertion was measured every two kilometers (at 2, 4, 6, 8, 10, 12, 14, 16, 18, 20, 22, 24, 26, 28 and 30 kilometer).

3.1.13 DETERMINATION OF STRIDE FREQUENCY AND STRIDE LENGTH

Stride frequency and stride length was determined during both the five 15 minute submaximal recovery runs and the 30-km time trial.

Stride frequency was recorded for 30 seconds at 4, 9 and 14 minutes during each submaximal recovery test. During the 30-km time trial stride frequency was recorded every two kilometers, at the 2, 4, 6, 8, 10, 12, 14, 16, 18, 20, 22, 24, 26, 28 and 30-kilometer mark. Stride length was derived from these measurements by calculating the distance run in 30 seconds and dividing this by the stride frequency.

3.1.14 30-KM TIME TRIAL

All the subjects, downhill and level group, performed a 30-km time trial after finishing either the downhill training protocol (downhill group) or the level training protocol (level group) (Appendix N).

The 30-km time trial was performed on a motor driven treadmill (Powerjog, Ergoline EG30, Birmingham, England). As soon as the subjects mounted the treadmill, the speed was increased to the pre-determined work rate of 70% of peak treadmill running speed (PTRS) for each subject. After the pre-determined work rate was reached the 30-km time trial started. The gradient of the treadmill was changed, between a 0% and a –10% gradient, throughout the time trial.

The gradient was changed in the following manner:

Kilometer	Treadmill Gradient	Level/Downhill Running
1 – 6	0%	Level
7, 8	-10%	Downhill
9 – 14	0%	Level
15, 16	-10%	Downhill
17 – 22	0%	Level
23, 24	-10%	Downhill
25 – 30	0%	Level

The subjects were allowed to stop for two minutes after completing 10 kilometers and again after completing 20 kilometers of the 30-km time trial. Before and after the 30-km time trial body mass was recorded on a calibrated scale, to the nearest 100 grams, with the subjects only wearing light clothing and without shoes. Food and fluid intake was recorded for the duration of the 30-km time trial. The subjects were instructed to bring their own food and drinks for the 30-km time trial.

3.2 STATISTICAL ANALYSIS

All data are represented by the mean ± standard deviation (SD). An independent t-test was used to compare the general characteristics of subjects in the downhill and level group. A two way analysis of variance (ANOVA) with repeated measures was used to analyze differences within or between the groups for heart rate, plasma creatine kinase activity, perceived pain, objective pain as well as stride length and rate of perceived exertion, during the 30-km time trial. A three way analysis of variance (ANOVA) with repeated measures was used to analyze differences within or between the groups for rate of perceived exertion, stride length, ventilation, oxygen consumption, carbon dioxide consumption, respiratory exchange ratio and heart rate (during the submaximal recovery runs. After the ANOVA identified a significant F ratio ($P < 0.05$) a least significant difference (LSD) post hoc test was used to identify specific differences between groups. The level of statistical significance for all analyzes was accepted as $P < 0.05$.

3.3 RESULTS

The results are presented in the following order. First the characteristics of the subjects are shown. Then the data are presented for the training phase of the study where the subjects ran either downhill or level on a treadmill for nine 40 minute training runs (70% of PTRS). Next the data collected during the 30-km time trial are shown, followed by the data collected during the five 15 minute submaximal recovery runs. The first submaximal recovery run was performed before the start of the training phase and again on four occasions after the 30-km time trial (Appendix D).

3.3.1 SUBJECT CHARACTERISTICS

Age, mass, stature, sum of seven skinfolds, percentage body fat, maximal oxygen consumption (VO_{2max}), respiratory exchange ratio, maximum heart rate, peak treadmill running speed and 70% of peak treadmill running speed for the downhill and level group are shown in Table 1. There were no differences between the two groups.

Table 1: General characteristics of the downhill (n=9) and level group (n=7). Values are expressed as mean ± SD.

Variables	Downhill group (n=9)	Level group (n=7)
Age (years)	34.1 ± 4.4	33.4 ± 7.7
Mass (kg)	70.0 ± 6.5	74.6 ± 7.9
Stature (cm)	176.6 ± 4.4	176.7 ± 4.9
Sum of 7 skinfolds (mm)	62.3 ± 19.6	69.8 ± 23.9
% Body fat	15.7 ± 3.3	15.5 ± 3.3
VO_2max (ml $O_2kg^{-1}.min^{-1}$)	53.5 ± 4.7	52.2 ± 5.5
Respiratory exchange ratio (RER) at VO_{2max}	1.1 ± 0.1	1.1 ± 0.0
Maximum heart rate (beats.min^{-1})	187 ± 7	192 ± 7
Peak treadmill running speed (PTRS) (km.hr^{-1})	17.4 ± 0.3	17.2 ± 0.5
70% of PTRS (km.hr^{-1})	12.2 ± 0.2	12.1 ± 0.3

There were no differences in the personal best (PB) times achieved by both groups for 10-km, 21.1-km, 42.2-km and Comrades marathon (90-km) as well as average training distance per week (for the duration of the study) (Table 2).

Table 2: Personal best times and training distance during the study of the downhill (n=9) and level group (n=7). Values are expressed as mean ± SD.

Variables	Downhill group (n=9)	Level group (n=7)
10 km Personal Best (PB) (min)	37.7 ± 2.0	39.9 ± 4.6
21.1 km PB (min)	85.3 ± 13.2	87.7 ± 9.3
42.2 km PB (min)	191.2 ± 30.7	193.1 ± 22.8
Comrades marathon PB (min)	484.9 ± 78.5	551.9 ± 48.1
Average training distance ($km.week^{-1}$)	74.9 ± 25.4	68.7 ± 14.1

3.3.2 DOWNHILL / LEVEL TRAINING

3.3.2.1 Heart rate

(a) Heart rate drift

Figure 1 represents changes in heart rate ($beats.min^{-1}$) for the downhill group, between the start (minute 5) and the end (minute 38) of each of the nine 40 minute downhill training runs. A significant interaction was found between the downhill training runs and pre-run (minute 5) and post-run (minute 38) ($P < 0.0002$). There was also a significant difference between pre-run (minute 5) and post-run (minute 38) for the downhill group ($P < 0.002$). A significant difference was found for the downhill group between training run 1 and training run 3, run 4, run 5, run 6, run 7, run 8 and run 9 ($P < 0.003$). There was also a significant difference between downhill training run 2 and downhill training run 7, run 8 and run 9 ($P < 0.02$). Downhill training run 3 was also significantly different from downhill training run 7 and 8 ($P < 0.04$) and downhill training run 5 was different to training run 8 ($P < 0.02$) (Table 3).

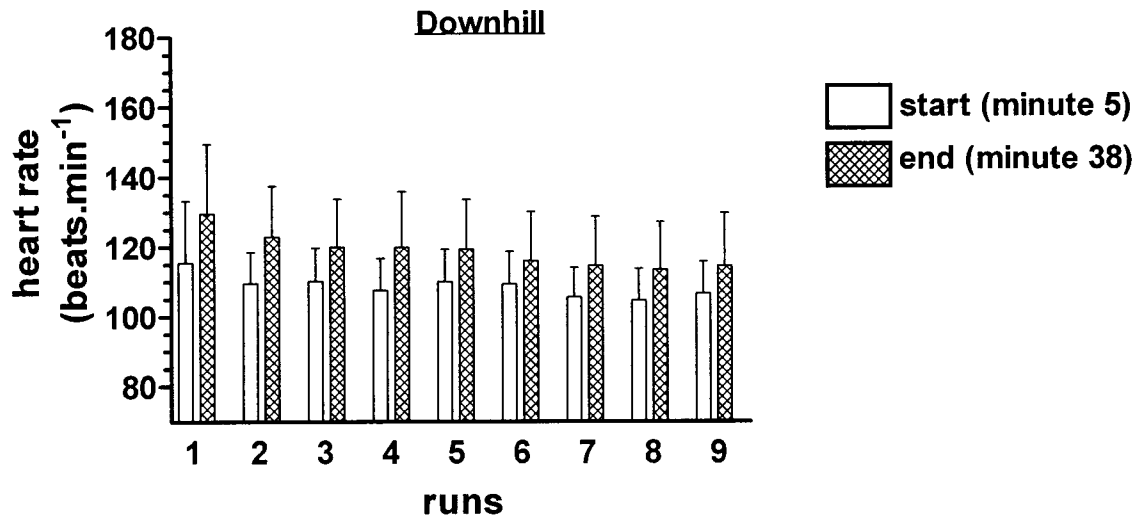

Figure 1: Changes in heart rate (beats.min^{-1}) for the downhill group, between the start (minute 5) and end (minute 38) of each of the nine x 40 minute training runs. The downhill group (n=9) ran at 70% of PTRS down a 10% gradient.

Interaction: Runs vs. Pre-run, Post-run
** $P < 0.0002$

Main effect: Pre-run vs. Post-run
** $P < 0.002$

Main effect: Runs
** $P < 0.003$ Run 1 vs. Run 3, 4, 5, 6, 7, 8 and 9

Figure 2 shows changes in heart rate (beats.min^{-1}) for the level group, between the start (minute 5) and end (minute 38) of each of the nine x 40 minute level training runs. A significant difference was found for the level group between pre-run (minute 5) and post-run (minute 38) ($P < 0.00003$) (Table 3). In contrast to the downhill group there was no significant interaction (runs vs. pre-run:post-run) or difference between the nine 40 minute training runs for the level group.

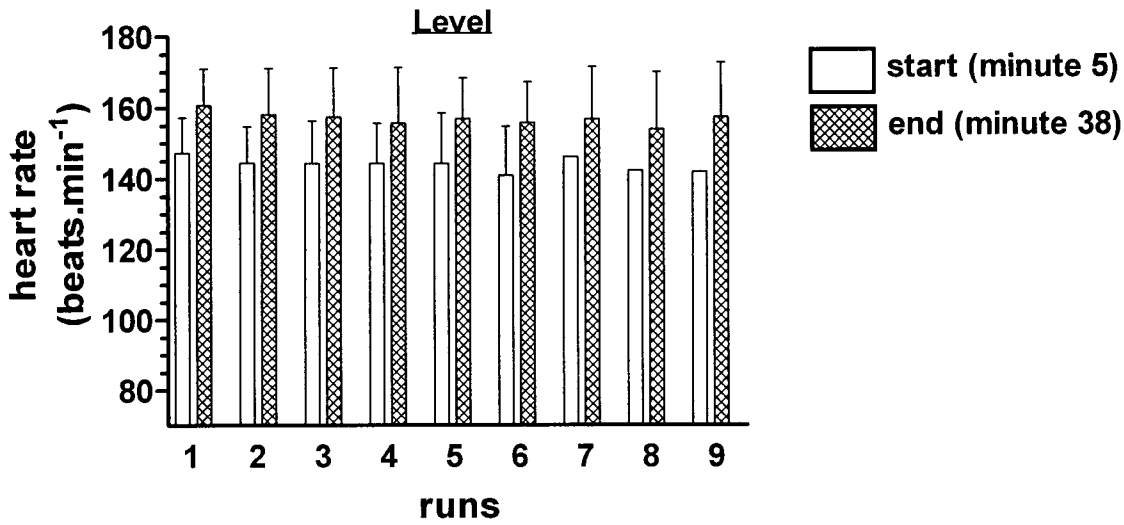

Figure 2: Changes in heart rate (beats.min^{-1}) for the level group, between the start (minute 5) and end (minute 38) of each of the nine x 40 minute training runs. The level group (n=7) ran at 70% of PTRS at a 0% gradient.

Main effect: Pre-run vs. Post-run
** $P < 0.00003$

Figure 3 and Table 3 shows changes in heart rate (beats.min^{-1}) between the start (minute 5) and end (minute 38) of each of the nine x 40 minute training runs for the downhill and level groups. There was a significant difference between the downhill and level group ($P < 0.00006$), with the downhill group having a consistently lower heart rate both at the start and end of the 40 minute training runs (Table 3).

Figure 3 shows changes in heart rate (beats.min^{-1}) between the start (minute 5) and end (minute 38) of each of the nine x 40 minute training runs.

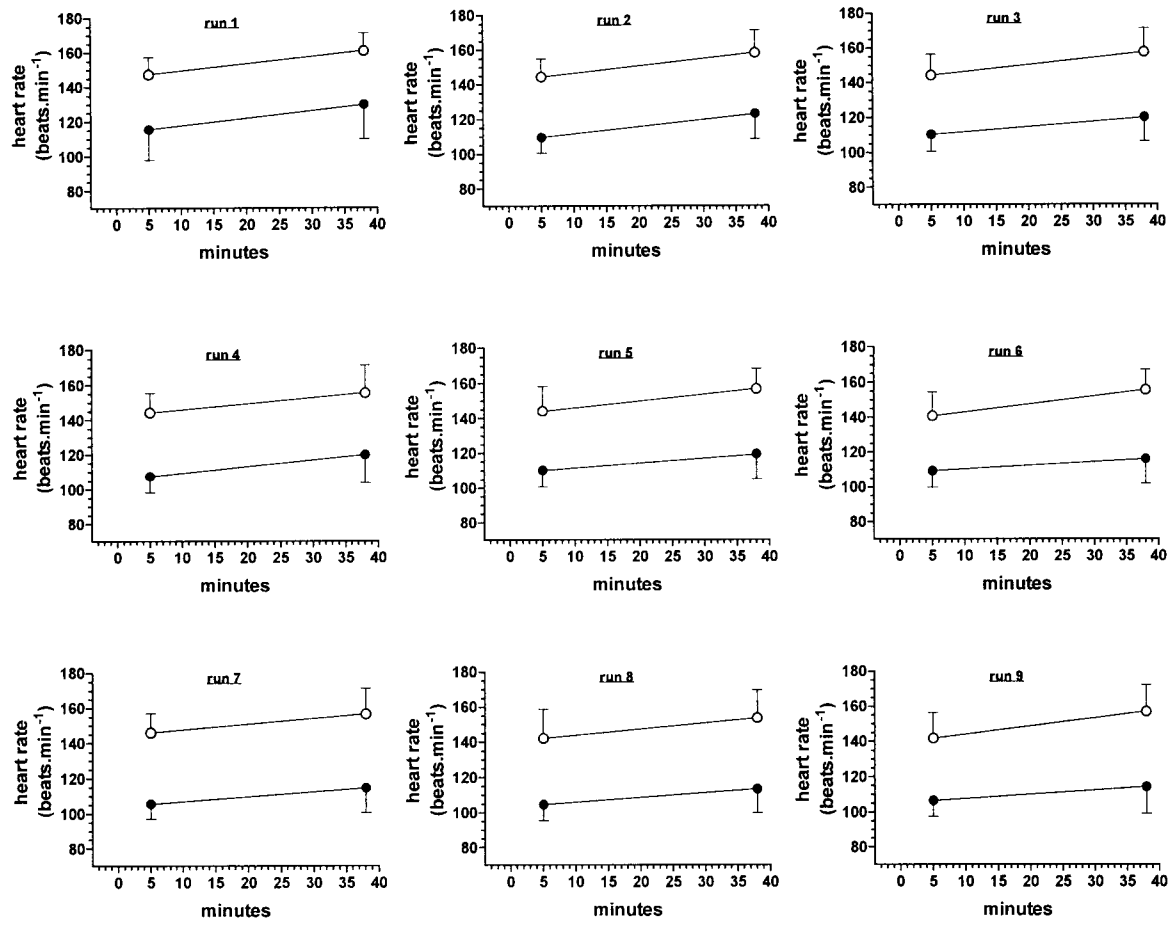

Figure 3: Changes in heart rate (beats.min^{-1}) between the start (minute 5) and end (minute 38) of each of the nine x 40 minute training runs. The downhill group (● n=9) ran at 70% of PTRS down a 10% grade and the level group (O n=7) ran at a 0% gradient.

Main effect: Group
** P < 0.00006

Table 3: Changes in heart rate (beats.min^{-1}) between the start (minute 5) and end (minute 38) of each of the nine x 40 minute training runs. Downhill (n=9) group ran down a 10% grade (70% PTRS) and the level group (n=7) ran at a 0% gradient. Values are expressed as mean ± SD.

Day	Training run	Time (minutes)	Downhill group	Level group
			Heart rate (beats.min^{-1})	Heart rate (beats.min^{-1})
1	Run 1	5	115.5 ± 17.7	147.2 ± 10.0
		38	129.6 ± 19.9	160.6 ± 10.3
8	Run 2	5	109.6 ± 9.0	144.4 ± 10.3
		38	122.9 ± 14.5	157.9 ± 13.1
11	Run 3	5	110.2 ± 9.6	144.2 ± 12.0
		38	119.9 ± 13.7	157.4 ± 13.7
15	Run 4	5	107.5 ± 9.2	144.2 ± 11.3
		38	119.8 ± 16.0	155.4 ± 15.7
17	Run 5	5	110.0 ± 9.2	144.2 ± 14.2
		38	119.3 ± 14.2	156.8 ± 11.4
19	Run 6	5	109.2 ± 9.4	140.7 ± 13.9
		38	116.0 ± 14.1	155.6 ± 11.5
22	Run 7	5	105.5 ± 8.6	146.0 ± 10.9
		38	114.6 ± 13.9	156.6 ± 14.7
25	Run 8	5	104.6 ± 9.1	142.1 ± 16.6
		38	113.4 ± 13.7	153.6 ± 16.1
29	Run 9	5	106.5 ± 9.2	141.8 ± 14.6
		38	114.4 ± 15.2	157.0 ± 15.3

Main effect: Group
** $P < 0.00006$

(b) Heart rate difference

Figure 4 shows the difference in heart rate ($HR_{difference}$) (beats.min^{-1}) between the start (minute 5) and end (minute 38) of each of the nine x 40 minute training runs. Analysis of $HR_{difference}$ showed a significant interaction between the two groups over the nine x 40 minute training runs ($P < 0.02$), suggesting that the heart rate drift was different between the two groups over the training phase of the experiment. Figure 4 shows that the heart rate drift was between 10 and 16 beats.min^{-1} during the training phase in the level group. In contrast in the downhill group the heart rate drift started at about 14 beats.min^{-1} and steadily decreased to about 9 beats.min^{-1} during the 9th 40 minute training run.

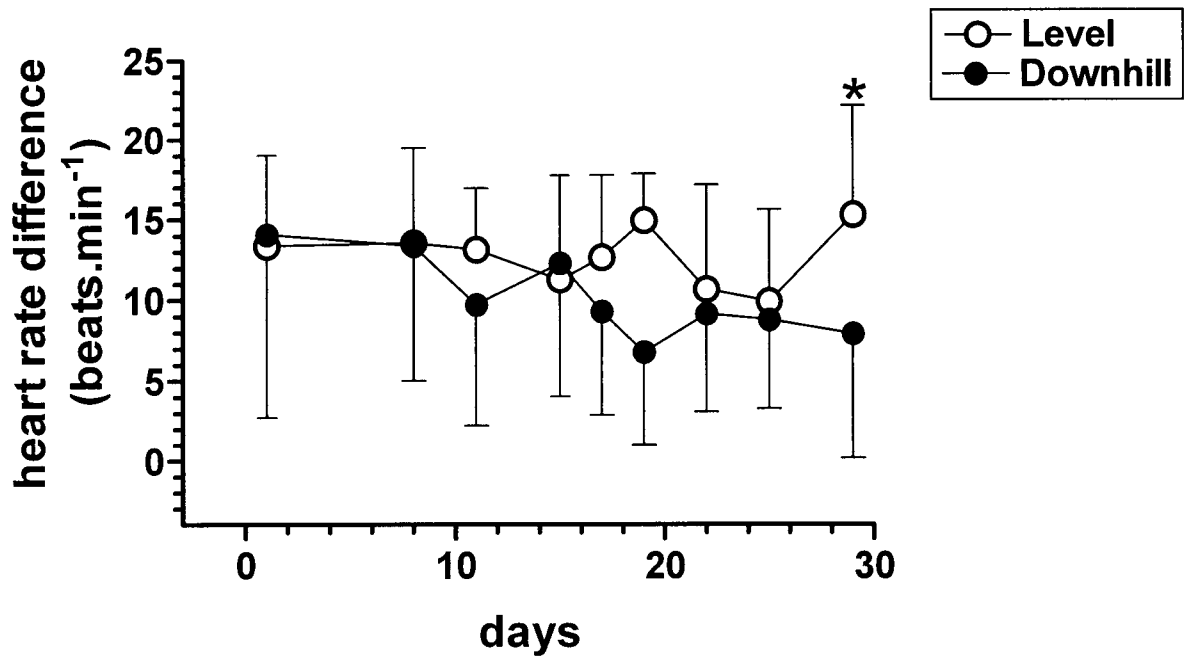

Figure 4: The difference in heart rate ($HR_{difference}$) between the start (minute 5) and end (minute 38) of each of the nine x 40 minute training runs. The downhill group (n=9) ran at 70% of PTRS down a 10% grade and the level group (n=7) ran at a 0% gradient.

Interaction: Group vs. Runs
* $P < 0.02$

3.3.2.2 Measurement of muscle pain

The units for all the pain measurements are arbitrary units. Therefore all the pain data are just expressed as a score.

(a) Objective muscle pain

(i) Objective pain score in the rectus femoris muscle

The training phase objective pain scores of the rectus femoris muscle for the two groups are shown in Table 4. The data are also shown graphically in Figure 18, in combination with the data from the recovery phase of the experiment.

There was a significant interaction of group vs. days ($P < 0.0000001$). Furthermore, the main effects of group ($P < 0.0004$) and days ($P < 0.0000001$) were significant.

When the two groups were analyzed independently by an analysis of variance with repeated measures there were significant differences between days for the downhill group, whereas there was no differences in objective pain in the rectus femoris throughout the training phase of the experiment for the level group. The specific differences are shown in Table 4.

Table 4: Objective pain scores during the training phase obtained by the downhill (n=9) and the level group (n=7) for the rectus femoris muscle. Values are expressed as mean ± SD.

Day	Trial	Downhill group Rectus femoris	Level group Rectus femoris
1	Training run 1	0.0 ± 0.0	0.1 ± 0.4
2	24 hours	7.4 ± 2.9 **	0.0 ± 0.0
3	48 hours	10. 9 ± 5.1 **	0.0 ± 0.0
4	72 hours	8.0 ± 4.1 **	0.0 ± 0.0
8	Training run 2	1.7 ± 2.1	0.4 ± 0.8
11	Training run 3	1.0 ± 2.6	0.4 ± 0.5
15	Training run 4	1.3 ± 4.0	0.1 ± 0.4
17	Training run 5	0.9 ± 1.8	0.1 ± 0.4
19	Training run 6	0.7 ± 1.4	0.4 ± 0.8
22	Training run 7	0.0 ± 0.0	0.4 ± 0.8
25	Training run 8	0.0 ± 0.0	0.6 ± 1.5
29	Training run 9	0.0 ± 0.0	0.1 ± 0.4
30	24 hours	0.0 ± 0.0	0.1 ± 0.4
31	48 hours	0.0 ± 0.0	0.1 ± 0.4
32	72 hours	0.0 ± 0.0	0.0 ± 0.0
34	30-km Time Trial		

Interaction: Group vs. Days
** $P < 0.0000001$

Main effect: Group
** $P < 0.0004$

Main effect: Days
** $P < 0.00000001$ 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)

(ii) Objective pain score in the vastus medialis muscle

The training phase objective pain scores of the vastus medialis muscle for the two groups are shown in Table 5 and Figure 19.

The pain scores were similar to the rectus femoris scores as there was a significant interaction between the two groups over days ($P < 0.0000001$) and the main effects of group and days were significant ($P < 0.002$ and $P < 0.000004$, respectively).

When the two groups were analyzed independently with an analysis of variance, with repeated measures, there were significant differences between days for the downhill group. The level group showed no significant differences in objective pain in the vastus medialis muscle throughout the training phase of the experiment. The specific differences are shown in Table 5.

Table 5: Objective pain scores during the training phase obtained by the downhill (n=9) and the level group (n=7) for the vastus medialis muscle. Values are expressed as mean ± SD.

Day	Trial	Downhill group Vastus medialis	Level group Vastus medialis
1	Training run 1	0.0 ± 0.0	0.0 ± 0.0
2	24 hours	6.9 ± 5.7 **	0.0 ± 0.0
3	48 hours	11.8 ± 7.2 **	0.0 ± 0.0
4	72 hours	7.8 ± 4.5 **	0.0 ± 0.0
8	Training run 2	1.6 ± 2.0	0.9 ± 1.5
11	Training run 3	1.3 ± 2.4	1.0 ± 1.2
15	Training run 4	0.3 ± 1.0	0.1 ± 0.4
17	Training run 5	0.4 ± 1.0	0.7 ± 1.0
19	Training run 6	0.7 ± 1.4	0.6 ± 1.0
22	Training run 7	0.0 ± 0.0	0.6 ± 0.8
25	Training run 8	0.4 ± 1.3	0.4 ± 0.8
29	Training run 9	0.0 ± 0.0	0.1 ± 0.4
30	24 hours	0.0 ± 0.0	0.1 ± 0.4
31	48 hours	0.0 ± 0.0	0.1 ± 0.4
32	72 hours	0.0 ± 0.0	0.0 ± 0.0
34	30-km Time Trial		

Interaction: Group vs. Days
** $P < 0.0000001$

Main effect: Group
** $P < 0.002$

Main effect: Days
** $P < 0.000004$ 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)

(b) Perceived muscle pain

(i) Muscle soreness rating scale

Table 6 shows the perceived pain score in both the downhill and level group obtained for the training phase of the trial. The data are also displayed in Figure 20 in combination with the perceived pain scores from the recovery phase of the experiment.

A significant interaction, during training, was found between the two groups over days ($P < 0.0000001$). Furthermore a significant difference was found between the downhill and level group ($P < 0.00007$).

When the two groups were analyzed independently with an analysis of variance with repeated measures there were significant differences between days for the downhill group, whereas there was no differences in perceived pain throughout the training phase of the experiment for the level group. The specific differences are shown in Table 6.

Table 6: Perceived pain of the downhill (n=9) and the level group (n=7) for the training phase of the trial. Perceived pain was quantified by using a perceived muscle soreness rating scale. Values are expressed as mean ± SD.

Day	Trial	Downhill group Perceived pain	Level group Perceived pain
1	Training run 1	0.0 ± 0.0	0.0 ± 0.0
2	24 hours	3.7 ± 1.8 **	0.0 ± 0.0
3	48 hours	5.3 ± 1.4 **	0.0 ± 0.0
4	72 hours	3.7 ± 2.3 **	0.0 ± 0.0
8	Training run 2	0.9 ± 1.3	0.1 ± 0.4
11	Training run 3	1.1 ± 1.7 *	0.1 ± 0.4
15	Training run 4	0.4 ± 1.3	0.0 ± 0.0
17	Training run 5	0.4 ± 1.3	0.0 ± 0.0
19	Training run 6	0.2 ± 0.4	0.0 ± 0.0
22	Training run 7	0.0 ± 0.0	0.0 ± 0.0
25	Training run 8	0.4 ± 1.3	0.4 ± 1.1
29	Training run 9	0.0 ± 0.0	0.1 ± 0.4
30	24 hours	0.0 ± 0.0	0.1 ± 0.4
31	48 hours	0.0 ± 0.0	0.0 ± 0.0
32	72 hours	0.0 ± 0.0	0.0 ± 0.0
34	30-km Time Trial		

Interaction: Group vs. Days
** $P < 0.0000001$

Main effect: Group
** $P < 0.00007$

Main effect: Days
* $P < 0.04$ training run 3 vs. baseline (training run 1)
** $P < 0.0000001$ 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)

(ii) Diagrammatic presentation of superficial skeletal muscles

Diagrammatic presentation of anterior view of the superficial skeletal muscles

The training phase perceived pain scores derived from the diagrammatic presentation of the anterior view of the superficial skeletal muscles of the downhill and level groups are shown in Table 7. Figure 21 represents the training phase perceived pain scores derived from the diagrammatic presentation of the anterior view of the superficial skeletal muscles for both groups in combination with the recovery phase of the experiment.

There was a significant interaction, between the two groups over days ($P < 0.0000001$) and the main effect of group was also different ($P < 0.0000001$).

An analysis of variance with repeated measures revealed significant differences between days for the downhill group, whereas there were no differences in perceived pain throughout the training phase of the study for the level group. The specific differences are shown in Table 7.

Table 7: Perceived pain scores during the training phase obtained by the downhill (n=9) and the level group (n=7) for the diagrammatic presentation of the anterior view of the superficial skeletal muscles. Values are expressed as mean ± SD.

Day	Trial	Downhill group Anterior diagram	Level group Anterior diagram
1	Training run 1	0.0 ± 0.0	0.0 ± 0.0
2	24 hours	5.1 ± 2.0 **	0.0 ± 0.0
3	48 hours	5.0 ± 1.4 **	0.0 ± 0.0
4	72 hours	3.8 ± 2.1 **	0.0 ± 0.0
8	Training run 2	0.2 ± 0.7	0.0 ± 0.0
11	Training run 3	0.4 ± 0.9	0.0 ± 0.0
15	Training run 4	0.4 ± 1.3	0.0 ± 0.0
17	Training run 5	0.3 ± 0.7	0.0 ± 0.0
19	Training run 6	0.0 ± 0.0	0.0 ± 0.0
22	Training run 7	0.0 ± 0.0	0.0 ± 0.0
25	Training run 8	0.1 ± 0.3	0.0 ± 0.0
29	Training run 9	0.0 ± 0.0	0.0 ± 0.0
30	24 hours	0.0 ± 0.0	0.0 ± 0.0
31	48 hours	0.0 ± 0.0	0.0 ± 0.0
32	72 hours	0.0 ± 0.0	0.0 ± 0.0
34	30-km Time Trial		

Interaction: Group vs. Days
** $P < 0.0000001$

Main effect: Group
** $P < 0.0000001$

Main effect: Days
** $P < 0.0000001$ 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)

Diagrammatic presentation of posterior view of the superficial skeletal muscles

The training phase perceived pain scores derived from the diagrammatic presentation of the posterior view of the superficial skeletal muscles of the downhill and level groups are shown in Table 8. Figure 22 represents the training phase perceived pain scores derived from the diagrammatic presentation of the posterior view of the superficial skeletal muscles for both groups in combination with the recovery phase of the experiment.

There was a significant interaction for perceived pain between the two groups over days ($P < 0.0000001$) and the main effect of group was also different ($P < 0.0002$).

When the two groups were analyzed independently with an analysis of variance with repeated measures there were significant differences between days for the downhill group, whereas there was no differences in perceived pain throughout the training phase of the experiment for the level group. The specific differences are shown in Table 8.

Table 8: Perceived pain scores during the training phase obtained by the downhill (n=9) and the level group (n=7) for the diagrammatic presentation of the posterior view of the superficial skeletal muscles. Values are expressed as mean ± SD.

Day	Trial	Downhill group Posterior diagram	Level group Posterior diagram
1	Training run 1	0.2 ± 0.7	0.0 ± 0.0
2	24 hours	6.4 ± 3.2 **	0.0 ± 0.0
3	48 hours	5.1 ± 2.0 **	0.0 ± 0.0
4	72 hours	3.8 ± 1.9 **	0.0 ± 0.0
8	Training run 2	1.4 ± 1.9 *	0.6 ± 1.0
11	Training run 3	1.2 ± 2.3	0.1 ± 0.4
15	Training run 4	0.9 ± 2.7	0.0 ± 0.0
17	Training run 5	0.2 ± 0.7	0.1 ± 0.4
19	Training run 6	0.0 ± 0.0	0.0 ± 0.0
22	Training run 7	0.0 ± 0.0	0.0 ± 0.0
25	Training run 8	0.3 ± 1.0	0.3 ± 0.8
29	Training run 9	0.2 ± 0.7	0.6 ± 1.0
30	24 hours	0.0 ± 0.0	0.0 ± 0.0
31	48 hours	0.0 ± 0.0	0.0 ± 0.0
32	72 hours	0.0 ± 0.0	0.0 ± 0.0
34	30-km Time Trial		

Interaction: Group vs. Days
** $P < 0.0000001$

Main effect: Group
** $P < 0.0002$

Main effect: Days
* $P < 0.03$ training run 2 vs. baseline (training run 1)
** $P < 0.00002$ 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)

3.3.2.3 Plasma creatine kinase activity

The changes in plasma CK activity in both the downhill and level group for the duration of the training phase of the study are shown in Table 9. These data are also shown in combination with the plasma CK activity during recovery in Figure 23.

A statistically significant interaction was found between the two groups over days for plasma CK activity ($P < 0.009$).

The differences in plasma CK activity for the downhill group are shown in the legend beneath Table 9. Plasma CK activity did not change significantly in the level group during the training phase of the study.

Table 9: Changes in plasma creatine kinase activity (CK) ($U.l^{-1}$) between the downhill (n=9) and the level group (n=7) for the training phase of the trial. Values are expressed as mean ± SD.

Day	Trial	Downhill group CK ($U.l^{-1}$)	Level group CK ($U.l^{-1}$)
1	Training run 1	94.7 ± 35.5	90.9 ± 32.7
2	24 hours	152.3 ± 46.9 **	82.5 ± 24.9
3	48 hours	205.4 ± 80.6 **	82.7 ± 17.0
4	72 hours	140.8 ± 59.5 **	75.2 ± 16.0
8	Training run 2	94.7 ± 38.0	87.5 ± 43.0
11	Training run 3	86.1 ± 28.6	96.5 ± 33.5
15	Training run 4	99.4 ± 34.7	80.8 ± 27.1
17	Training run 5	111.1 ± 36.3	87.2 ± 19.9
19	Training run 6	93.3 ± 31.9	92.6 ± 16.8
22	Training run 7	77.4 ± 36.2	88.0 ± 25.2
25	Training run 8	85.3 ± 32.0	66.6 ± 22.3
29	Training run 9	77.6 ± 34.5	78.9 ± 26.5
30	24 hours	94.5 ± 32.0	90.7 ± 19.7
31	48 hours	89.5 ± 37.6	72.2 ± 35.3
32	72 hours	82.6 ± 32.0	81.2 ± 29.0
34	30-km Time Trial		

Interaction: Group vs. Days
** $P < 0.009$

Main effect: Group
* $P < 0.03$ difference between groups, during recovery

Main effect: Days
** $P < 0.009$ 24 hours after training run 1 vs. 48 hours after training run 1, training run 8, 9 and 48, 72 hours after training run 9
** $P < 0.003$ 48 hours after training run 1 vs. all the other time points
** $P < 0.003$ 72 hours after training run 1 vs. training run 8, 9 and 48, 72 hours

3.3.2.4 Summary of the training phase of the study

The heart rate drift during the 40 minute training sessions decreased up to the 8th training session in the downhill group whereas it remained relatively constant in the level group throughout the training phase of the study.

Both the objective and perceived measurements of muscle pain showed that muscle pain peaked at 72 hours after the first downhill training run. Thereafter the downhill training runs did not elicit any pain. Throughout the nine training sessions the level group did not report any muscle pain.

Plasma CK activity increased after the first downhill training run, peaking at 72 hours and thereafter did not increase again after a training session. Plasma CK activity did not increase after any training sessions in the level group.

Collectively these data suggest that the first downhill training run caused rather severe delayed onset muscle soreness, which subsided with subsequent training sessions.

3.3.3 30-KM TIME TRIAL

All subjects performed a 30-km time trial in the laboratory after finishing either the nine 40 minute downhill or level training protocol. During the 30-km time trial the gradient of the treadmill was changed, between 0% (1-6 km, 17-22 km and 25-30 km) and a –10% gradient (7-8 km, 15-16 km and 23-24 km), throughout the 30-km time trial. Four subjects (two subjects in each group) could not complete the entire time trial, due to the fast pace maintained throughout the duration of the 30-km time trial. However, all these subjects were able to complete more than 26 kilometers of the 30-km time trial and therefore their data are included in the analyses.

3.3.3.1 Heart rate

Figure 5 and Table 10 shows the heart rate of the downhill and level group during the 30-km time trial.

There was a significant interaction for heart rate (beats.min^{-1}) between the two groups over time ($P < 0.02$) (Table 10). This suggests that the heart rate of the downhill group responded differently to the heart rate of the level group during the 30-km time trial.

As expected the heart rate in both groups decreased during the downhill phase of the 30-km time trial (kilometer 8, 16 and 24 vs. all other time points, $P < 0.00005$) (Table 10). Furthermore, Figure 5 shows that heart rate generally increased in both groups as the duration of the 30-km time trial progressed. Although not significant, there was a tendency for the heart rate of the downhill group to be lower than the level group throughout the experiment.

Table 10: Heart rate (beats.min^{-1}) of the downhill (n=9) and the level group (n=7) during the 30-km time trial. For the duration of the time trial both groups ran at 70% of PTRS. The gradient of the treadmill was changed, between 0% (1–6 km, 17-22 km and 25-30 km) and a –10% gradient (7-8 km, 15-16 km and 23-24 km), throughout the time trial. Values are expressed as mean ± SD.

Time Trial (km)	Downhill group	Level group
	HR (beats.min^{-1})	HR (beats.min^{-1})
2	138.1 ± 10.2	141.4 ± 13.4
4	142.4 ± 10.8	147.5 ± 12.0
6	145.3 ± 11.3	149.1 ± 11.1
8	121.6 ± 14.4	131.7 ± 18.0 **
10	145.4 ± 12.5	146.7 ± 13.1
12	145.9 ± 13.4	151.2 ± 12.0
14	146.8 ± 13.6	152.5 ± 12.4
16	123.1 ± 15.3	133.6 ± 17.5 **
18	147.5 ± 12.8	155.5 ± 12.6
20	149.5 ± 13.3	155.0 ± 14.8
22	149.1 ± 10.3	161.1 ± 12.2
24	125.3 ± 11.6	142.7 ± 16.7 **
26	151.7 ± 9.7	164.2 ± 13.0
28	152.4 ± 11.8	169.0 ± 14.1
30	154.4 ± 11.1	166.2 ± 9.0

Interaction: Group vs. Time
* $P < 0.02$

Main effect: Time
** $P < 0.00005$ km 8, 16, 24 vs. all the other time points

Figure 5 shows heart rate (beats.min^{-1}) of the downhill and level group during the 30-km time trial.

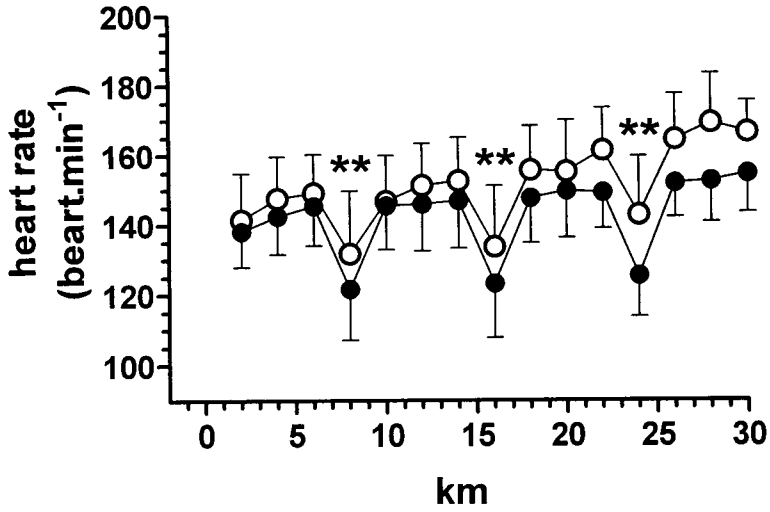

Figure 5: Heart rate (beats.min^{-1}) of the downhill (● n=9) and level group (O n=7) during the 30-km time trial. Both groups ran at 70% of PTRS for the duration of the time trial. The gradient of the treadmill was changed, between 0% (1-6 km, 17-22 km and 25-30 km) and a –10% gradient (7-8 km, 15-16 km and 23-24 km), throughout the time trial.

Interaction: Group vs. Time
* $P < 0.02$

Main effect: Time
** $P < 0.00005$ km 8, 16, 24 vs. all the other time points

3.3.3.2 Rate of perceived exertion

The rate of perceived exertion (RPE) between the downhill and level group during the 30-km time trial are shown in Table 11 and Figure 6.

There was a significant interaction for RPE between the two groups over time ($P < 0.003$) suggesting that the RPE changed differently in the downhill and level group during the 30-km time trial.

The RPE decreased, in both groups coinciding with each stage of the 30-km time trial when the gradient of the treadmill was decreased. The specific statistical differences are shown in the legend beneath Table 11 and Figure 6.

From about 15-km in the time trial there was a tendency for the RPE in the downhill group to be lower than the level group. This difference between groups was, however, not significant.

Table 11: Rate of perceived exertion (RPE) of the downhill (n=9) and the level group (n=7) during the 30-km time trial. For the duration of the time trial both groups ran at 70% of PTRS. The gradient of the treadmill was changed, between 0% (1–6 km, 17-22 km and 25-30 km) and a –10% gradient (7-8 km, 15-16 km and 23-24 km), throughout the time trial. Values are expressed as mean ± SD.

Time Trial (km)	Downhill group	Level group
	RPE	RPE
2	10.2 ± 2.5	8.4 ± 2.1
4	10.3 ± 2.6	8.7 ± 2.4
6	11.0 ± 3.1	9.7 ± 2.7
8	9.1 ± 2.2	7.9 ± 1.7 **
10	10.8 ± 3.3	9.4 ± 2.6
12	11.2 ± 3.2	10.3 ± 2.8
14	11.9 ± 3.8	11.3 ± 2.8
16	10.1 ± 3.2	10.1 ± 2.1 **
18	11.9 ± 3.8	12.4 ± 2.6
20	12.3 ± 4.0	13.0 ± 3.1
22	11.9 ± 3.9	13.4 ± 3.4
24	10.6 ± 2.9	12.7 ± 2.9 **
26	12.9 ± 3.6	14.2 ± 3.4
28	12.1 ± 2.7	14.8 ± 3.8
30	12.3 ± 2.8	14.6 ± 3.8

Interaction: Group vs. Time
** $P < 0.003$

Main effect: Time
** $P < 0.002$ km 8 vs. all other time points
** $P < 0.001$ km 16 vs. km 8, 14, 18, 20, 22, 24, 26, 28, 30
** $P < 0.001$ km 24 vs. km 2, 4, 8, 16, 26, 28, 30

Figure 6 represents changes in rate of perceived exertion (RPE) between the downhill and level group during the 30-km time trial.

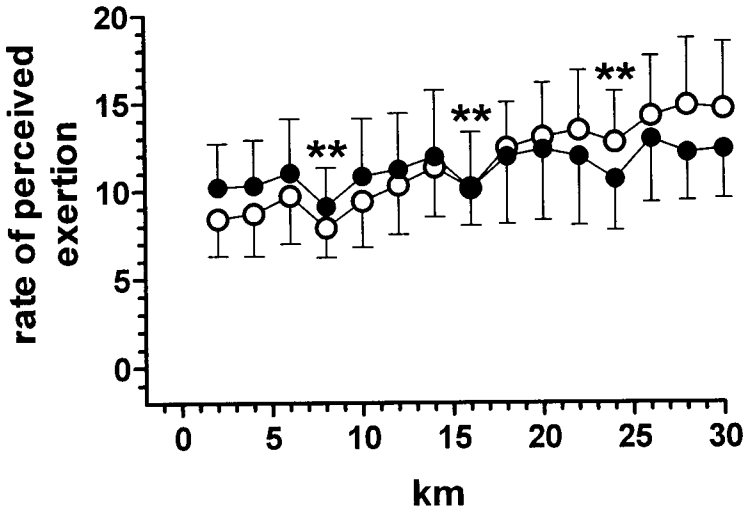

Figure 6: Rate of perceived exertion (RPE) of the downhill (● n=9) and level group (O n=7) during the 30-km time trial. Both groups ran at 70% of PTRS for the duration of the time trial. The gradient of the treadmill was changed, between 0% (1-6 km, 17-22 km and 25-30 km) and a –10% gradient (7-8 km, 15-16 km and 23-24 km), throughout the time trial.

Interaction: Group vs. Time
** $P < 0.003$

Main effect: Time
** $P < 0.002$ km 8 vs. all other time points
** $P < 0.001$ km 16 vs. km 8, 14, 18, 20, 22, 24, 26, 28, 30
** $P < 0.001$ km 24 vs. km 2, 4, 8, 16, 26, 28, 30

3.3.3.3 Stride length

Table 12 and Figure 7 shows changes in stride length between the two groups during the 30-km time trial.

There was no significant interaction between the groups over time and there was no significant difference for the main effect of group. However, the main effect of time was different. Generally stride length increased in both groups coinciding with the downhill phase of the treadmill protocol. Detailed statistical comparisons are shown in Table 12 and Figure 7.

Table 12: Stride length of the downhill (n=9) and the level group (n=7) during the 30-km time trial. For the duration of the time trial both groups ran at 70% of PTRS. The gradient of the treadmill was changed, between 0% (1–6 km, 17-22 km and 25-30 km) and a –10% gradient (7-8 km, 15-16 km and 23-24 km), throughout the time trial. Values are expressed as mean ± SD.

Time Trial (km)	Downhill group	Level group
	SL (m)	SL (m)
2	1.25 ± 0.05	1.22 ± 0.03
4	1.24 ± 0.05	1.20 ± 0.02
6	1.25 ± 0.06	1.20 ± 0.04
8	1.31 ± 0.07	1.26 ± 0.03 **
10	1.25 ± 0.06	1.21 ± 0.04
12	1.24 ± 0.04	1.22 ± 0.04
14	1.23 ± 0.05	1.23 ± 0.05
16	1.30 ± 0.06	1.27 ± 0.03 **
18	1.24 ± 0.03	1.22 ± 0.05
20	1.23 ± 0.04	1.21 ± 0.04
22	1.23 ± 0.04	1.22 ± 0.03
24	1.29 ± 0.07	1.25 ± 0.06 **
26	1.23 ± 0.04	1.20 ± 0.03
28	1.22 ± 0.04	1.19 ± 0.04 *
30	1.22 ± 0.05	1.22 ± 0.06

Main effect: Time
* $P < 0.04$ km 28 vs. km 2, 6, 10, 14 and 18
** $P < 0.003$ km 8, 16 and 24 vs. all other time points

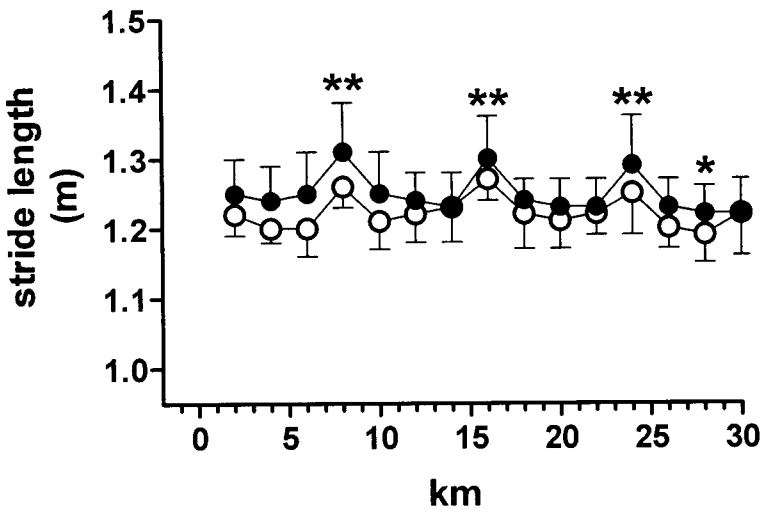

Figure 7: Stride length (SL) of the downhill (● n=9) and level group (O n=7) during the 30-km time trial. Both groups ran at 70% of PTRS for the duration of the time trial. The gradient of the treadmill was changed, between 0% (1-6 km, 17-22 km and 25-30 km) and a –10% gradient (7-8 km, 15-16 km and 23-24 km), throughout the time trial.

Main effect: Time
* $P < 0.04$ km 28 vs. km 2, 6, 10, 14 and 18
** $P < 0.003$ km 8, 16 and 24 vs. all other time points

3.3.3.4 Body mass

Body mass decreased similarly in both groups after the 30-km time trial (downhill group 71.4 ± 7.0 kg vs. 69.7 ± 6.7 kg pre vs. post and level group 75.0 ± 7.7 kg vs. 73.2 ± 7.5 kg pre vs. post) ($P < 0.0000001$) (Figure 8).

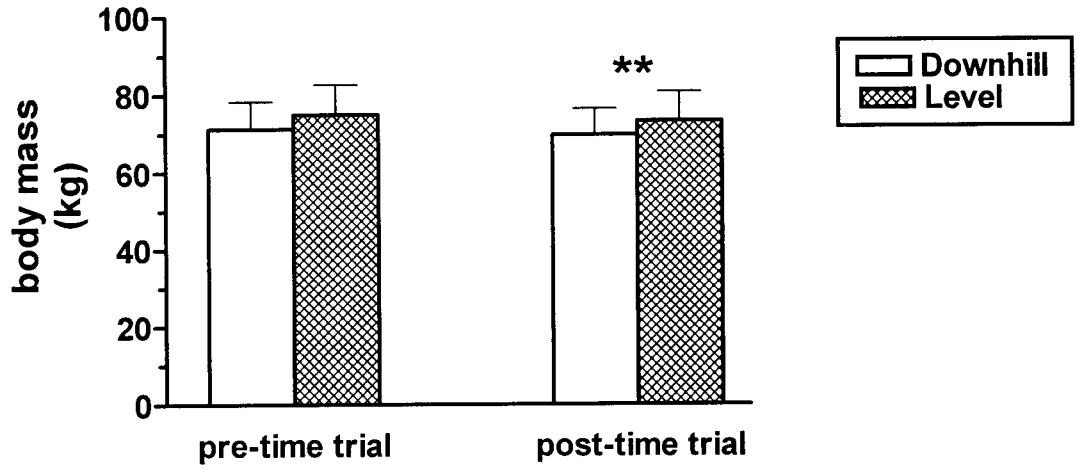

Figure 8: Changes in body mass between the downhill (n=9) and level group (n=7) during the 30-km time trial. Both groups ran at 70% of PTRS for the duration of the time trial. The gradient of the treadmill was changed, between 0% (1-6 km, 17-22 km and 25-30 km) and a –10% gradient (7-8 km, 15-16 km and 23-24 km), throughout the time trial.

** $P < 0.000001$ Pre-time trial vs. Post-time trial

3.3.3.5 Plasma glucose concentration

Figure 9 shows changes in plasma glucose concentration ($mmol.l^{-1}$) between the downhill and level group during the 30-km time trial. No significant difference was found for plasma glucose concentration prior to the 30-km time trial (downhill group 4.5 ± 1.2 $mmol.l^{-1}$; level group 4.9 ± 0.7 $mmol.l^{-1}$) and after the time trial (downhill group 4.9 ± 0.6 $mmol.l^{-1}$; level group 4.8 ± 0.4 $mmol.l^{-1}$). The average sampling period after the completion of the 30-km time trial was 34.1 ± 8.9 seconds.

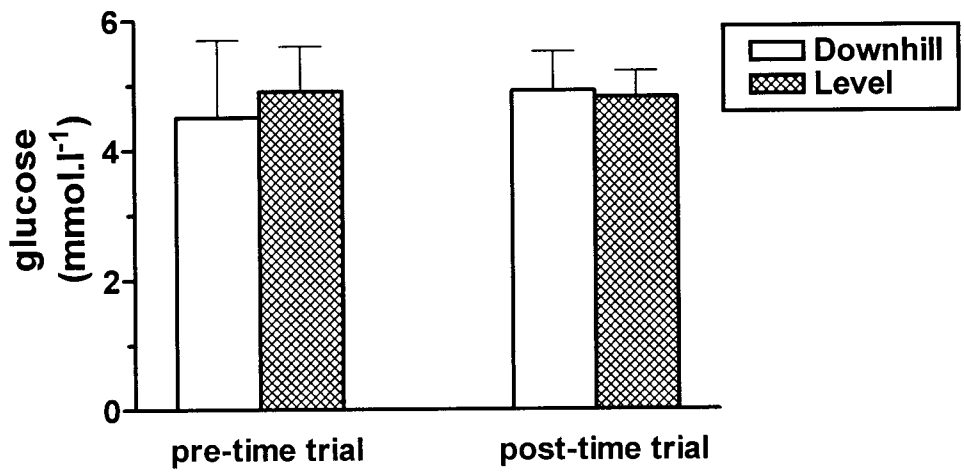

Figure 9: Changes in plasma glucose concentration between the downhill (n=9) and level group (n=7) during the 30-km time trial. Both groups ran at 70% of PTRS for the duration of the time trial. The gradient of the treadmill was changed, between 0% (1-6 km, 17-22 km and 25-30 km) and a –10% gradient (7-8 km, 15-16 km and 23-24 km), throughout the time trial.

3.3.3.6 Plasma creatine kinase activity

Plasma CK activity increased similarly in both groups in the plasma sample collected immediately after the 30-km time trial ($P < 0.00008$). There were no differences between groups prior to the 30-km time trial (76.4 ± 33.5 vs. 102.7 ± 31.7 $U.l^{-1}$; downhill vs. level) or after (126.3 ± 51.7 vs. 143.3 ± 49.2 $U.l^{-1}$; downhill vs. level) the 30-km time trial (Figure 10).

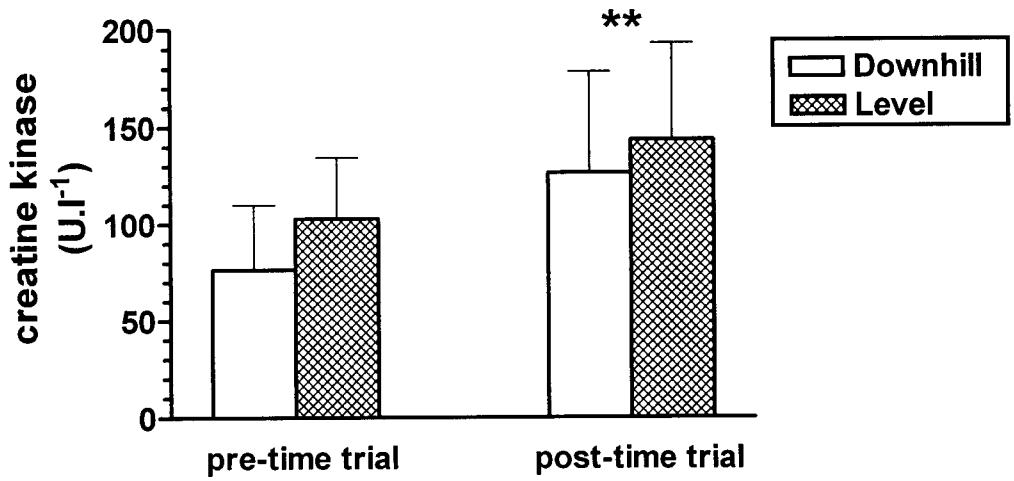

Figure 10: Changes in plasma creatine kinase activity between the downhill (n=9) and level group (n=7) during the 30-km time trial. Both groups ran at 70% of PTRS for the duration of the time trial. The gradient of the treadmill was changed, between 0% (1-6 km, 17-22 km and 25-30 km) and a –10% gradient (7-8 km, 15-16 km and 23-24 km), throughout the time trial.

** $P < 0.000008$ Pre-time trial vs. Post-time trial

3.3.3.7 Summary of the 30-km time trial phase of the study

The heart rate and the ratings of perceived exertion of the downhill group responded differently than that of the level group over the 30-km time trial. There was a tendency for the heart rate of the downhill group to be lower than the level group throughout the 30-km time trial and for the ratings of perceived exertion of the downhill group to be lower towards the end of the time trial. Stride length increased similarly in both groups when the subjects ran downhill in the 30-km time trial and the changes in body mass, plasma glucose concentration and plasma creatine kinase activity were similar in both groups.

Based on the heart rate and rating of perceived exertion there is evidence to suggest that the downhill group tolerated the 30-km time trial better than the level group.

3.3.4 RECOVERY TESTING

During the recovery phase of the study, the subjects in both the downhill and level group performed five 15 minute submaximal recovery runs on the treadmill. The first 15 minute recovery run was performed prior (pre) to the training phase of the study and again on four occasions after the 30-km time trial. Both groups ran at 70% of PRTS at a 0% grade for the first five minutes, thereafter the gradient was decreased to –10% for the next five minutes and for the last five minutes the grade was increased again to 0%.

3.3.4.1 Heart rate

Figure 11 shows changes in heart rate (beats.min^{-1}) during the 15 minute submaximal recovery runs for the downhill and level group prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial. Table 13 also represents changes in heart rate during the 15 minute submaximal recovery runs.

A significant interaction was found for heart rate between the 15 minute submaximal recovery runs and the duration (minutes) of the recovery runs ($P < 0.005$). This suggests that heart rate changed differently during the submaximal runs over the course of the recovery phase of the experiment.

When the main effect of runs was analyzed there was a statistically significant difference for heart rate, between the 15 minute submaximal recovery run prior to training (pre) and submaximal recovery run 1, run 2, run 3 and run 4 ($P < 0.0000001$).

Heart rate changed during the submaximal recovery runs in accordance with the changes in treadmill gradient. The details of these changes are shown below the legend of Figure 11. Although Figure 11 shows a tendency for heart rate to be lower in the downhill group compared to the level group throughout the recovery phase of the experiment, this was not significant.

Table 13: Changes in heart rate (HR) (beats.min^{-1}) between the downhill (n=9) and the level (n=7) group during five x 15 minute submaximal recovery runs. Both groups ran at 70% of PTRS at a 0% grade for the first 5 minutes, thereafter the gradient was decreased to –10% for the next 5 minutes and for the last 5 minutes the grade was increased again to 0%. Values are expressed as mean ± SD.

Day	Recovery Run	Time (minutes)	Downhill group	Level group
			Heart rate (beats.min^{-1})	Heart rate (beats.min^{-1})
pre	Run pre	3	146.8 ± 8.6	152.8 ± 11.4
		6	134.7 ± 10.7	140.6 ± 14.0
		9	130.2 ± 9.9	135.8 ± 12.8
		12	150.3 ± 9.4	156.8 ± 10.9
		14	151.6 ± 10.1	159.0 ± 11.5
			142.7 ± 12.8	**149.0 ± 14.7**
4	Run 1	3	135.7 ± 11.2	145.8 ± 8.9
		6	117.0 ± 11.4	130.4 ± 6.7
		9	115.2 ± 11.1	126.8 ± 2.9
		12	140.3 ± 12.6	148.9 ± 7.9
		14	143.3 ± 12.8	151.4 ± 8.5
			130.3 ± 16.5	**143.1 ± 11.8**
7	Run 2	3	133.0 ± 11.2	141.0 ± 10.1
		6	116.4 ± 12.8	128.4 ± 12.6
		9	113.6 ± 13.4	125.8 ± 12.1
		12	137.9 ± 13.8	147.8 ± 12.2
		14	138.6 ± 15.0	150.8 ± 15.1
			127.9 ± 16.7	**138.8 ± 15.6**
14	Run 3	3	132.8 ± 8.7	140.8 ± 10.9
		6	115.6 ± 10.2	129.4 ± 12.9
		9	112.6 ± 9.6	125.2 ± 12.8
		12	138.4 ± 12.1	147.3 ± 12.9
		14	139.7 ± 11.7	150.0 ± 13.3
			127.8 ± 15.4	**138.5 ± 15.4**
21	Run 4	3	134.0 ± 9.9	142.8 ± 7.9
		6	115.9 ± 13.2	131.0 ± 10.2
		9	112.7 ± 11.5	127.2 ± 9.6
		12	138.9 ± 11.6	148.6 ± 8.1
		14	140.8 ± 13.3	151.7 ± 8.5
			128.5 ± 16.5	**140.2 ± 12.9**

Interaction: Runs vs. Minutes
** $P < 0.005$

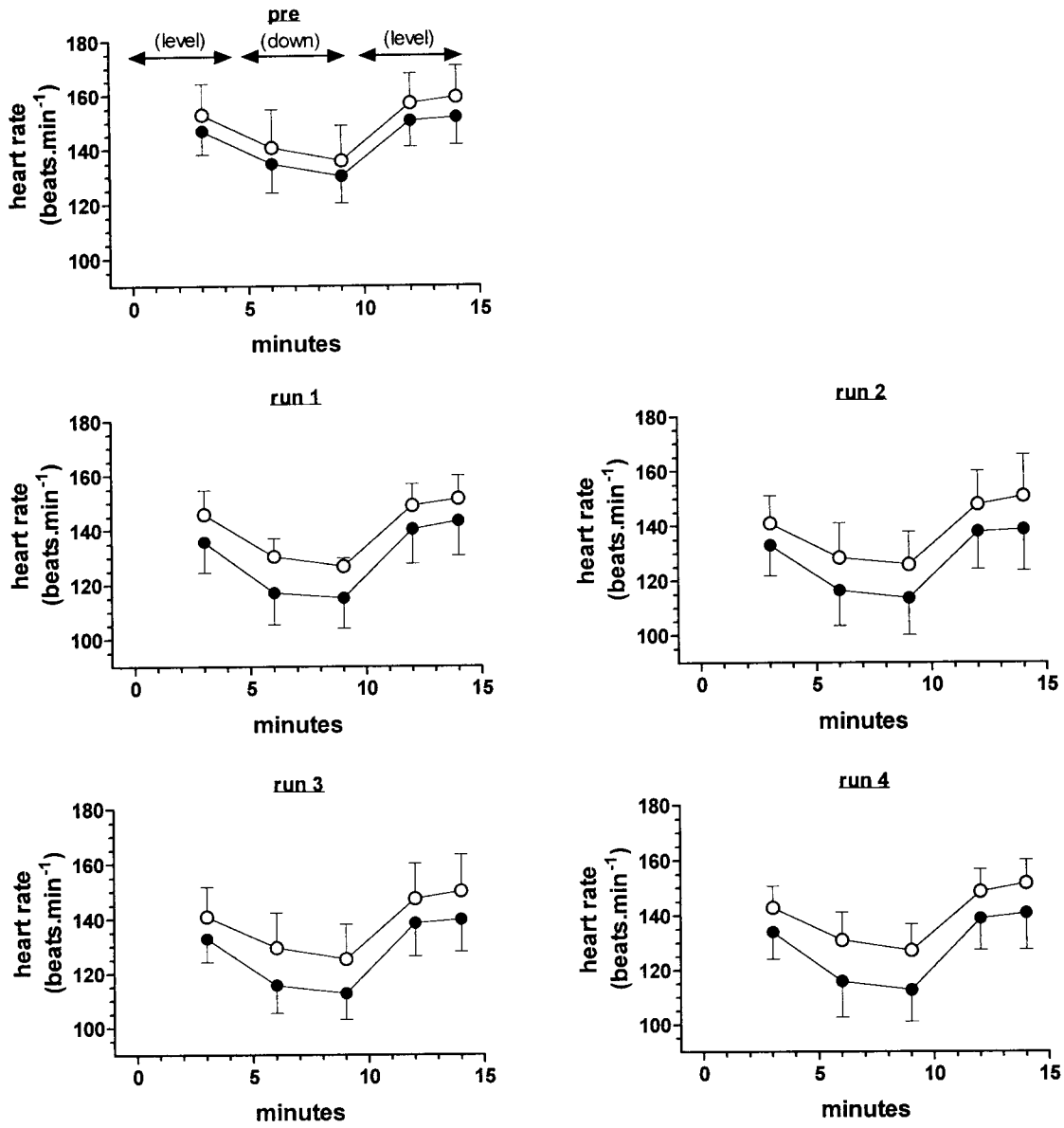

Figure 11: Changes in heart rate (HR) (beats.min^{-1}) during the 15 minute submaximal recovery runs for the downhill (● n=9) and level group (O n=7) prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial.

Interaction: Runs vs. Minutes
** $P < 0.005$

Main effect: Minutes
** $P < 0.0000001$ minute 3 vs. minute 6, 9, 12, 14
** $P < 0.01$ minute 6 vs. minute 9, 12, 14
** $P < 0.0000001$ minute 9 vs. minute 12, 14

3.3.4.2 Rate of perceived exertion

The rate of perceived exertion (RPE) during the 15 minute submaximal recovery runs for the downhill and level group prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial are shown in Table 14 and Figure 12.

There was a significant difference for RPE, between the 15 minute submaximal recovery run prior to training (pre) and submaximal recovery run 1, run 2, run 3 and run 4 after the 30-km time trial ($P < 0.001$). The average RPE, in both groups, for the submaximal recovery run prior to training (pre) was 8.4 ± 2.5 and on day 4 (run 1) after the time trial it was 7.4 ± 1.5. Thereafter, on day 7 (run 2), 14 (run 3) and 21 (run 4) the combined RPE was 7.0 ± 1.6, 7.0 ± 1.6 and 7.0 ± 1.6 respectively. The average RPE for each group during each run is shown in Table 14. Although there was a tendency for the downhill group to have a lower RPE after the 30-km time trial, this was not significant.

There was a general increase in RPE over the duration of the 15 minute submaximal recovery runs. The details of these changes are shown below the legend of Figure 12. Both groups responded similarly and there was no decrease in RPE coinciding with the downhill phase of the protocol, as occurred with heart rate (Figure 12).

Table 14: Changes in rate of perceived exertion (RPE) between the downhill (n=9) and the level (n=7) group during the five x 15 minute submaximal recovery runs. Both groups ran at 70% of PTRS at a 0% grade for the first 5 minutes, thereafter the gradient was decreased to –10% for the next 5 minutes and for the last 5 minutes the grade was increased again to 0%. Values are expressed as mean ± SD.

Day	Recovery Run	Time (minutes)	Downhill group	Level group
			RPE	RPE
pre	Run pre	0	7.8 ± 2.0	7.4 ± 2.4
		3	8.8 ± 2.5	8.1 ± 2.3
		6	8.0 ± 2.2	8.0 ± 2.4
		9	8.1 ± 2.4	8.0 ± 2.5
		12	8.9 ± 2.6	9.4 ± 3.3
		14	9.1 ± 2.4	9.7 ± 3.5
			8.4 ± 2.3	**8.5 ± 2.7**
4	Run 1	0	7.0 ± 1.2	6.1 ± 0.4
		3	7.6 ± 1.7	7.1 ± 1.6
		6	6.6 ± 0.7	7.0 ± 1.0
		9	6.8 ± 1.1	7.3 ± 1.3
		12	7.9 ± 2.0	8.6 ± 1.9
		14	7.9 ± 1.6	8.7 ± 2.0
			7.3 ± 1.8	**7.5 ± 1.6**
7	Run 2	0	6.2 ± 0.4	7.1 ± 1.7
		3	6.7 ± 1.1	7.7 ± 2.2
		6	6.3 ± 0.7	6.6 ± 1.0
		9	6.4 ± 1.0	6.9 ± 1.1
		12	7.2 ± 1.6	8.1 ± 2.4
		14	7.4 ± 1.9	8.3 ± 2.5
			6.7 ± 1.3	**7.5 ± 1.9**
14	Run 3	0	6.6 ± 1.1	6.7 ± 1.1
		3	6.3 ± 0.7	7.6 ± 1.9
		6	6.2 ± 0.4	6.6 ± 0.8
		9	6.4 ± 1.0	7.0 ± 1.9
		12	7.3 ± 1.6	8.3 ± 2.6
		14	7.1 ± 1.6	8.3 ± 2.6
			6.7 ± 1.2	**7.4 ± 2.0**
21	Run 4	0	6.3 ± 1.0	6.7 ± 1.0
		3	6.4 ± 1.3	7.4 ± 2.2
		6	6.3 ± 1.0	7.1 ± 1.3
		9	6.2 ± 0.7	7.1 ± 1.3
		12	7.0 ± 1.6	8.4 ± 2.5
		14	6.9 ± 1.3	8.3 ± 2.1
			6.5 ± 1.2	**7.5 ± 1.8**

Main effect: Runs
** $P < 0.001$ pre vs. day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial

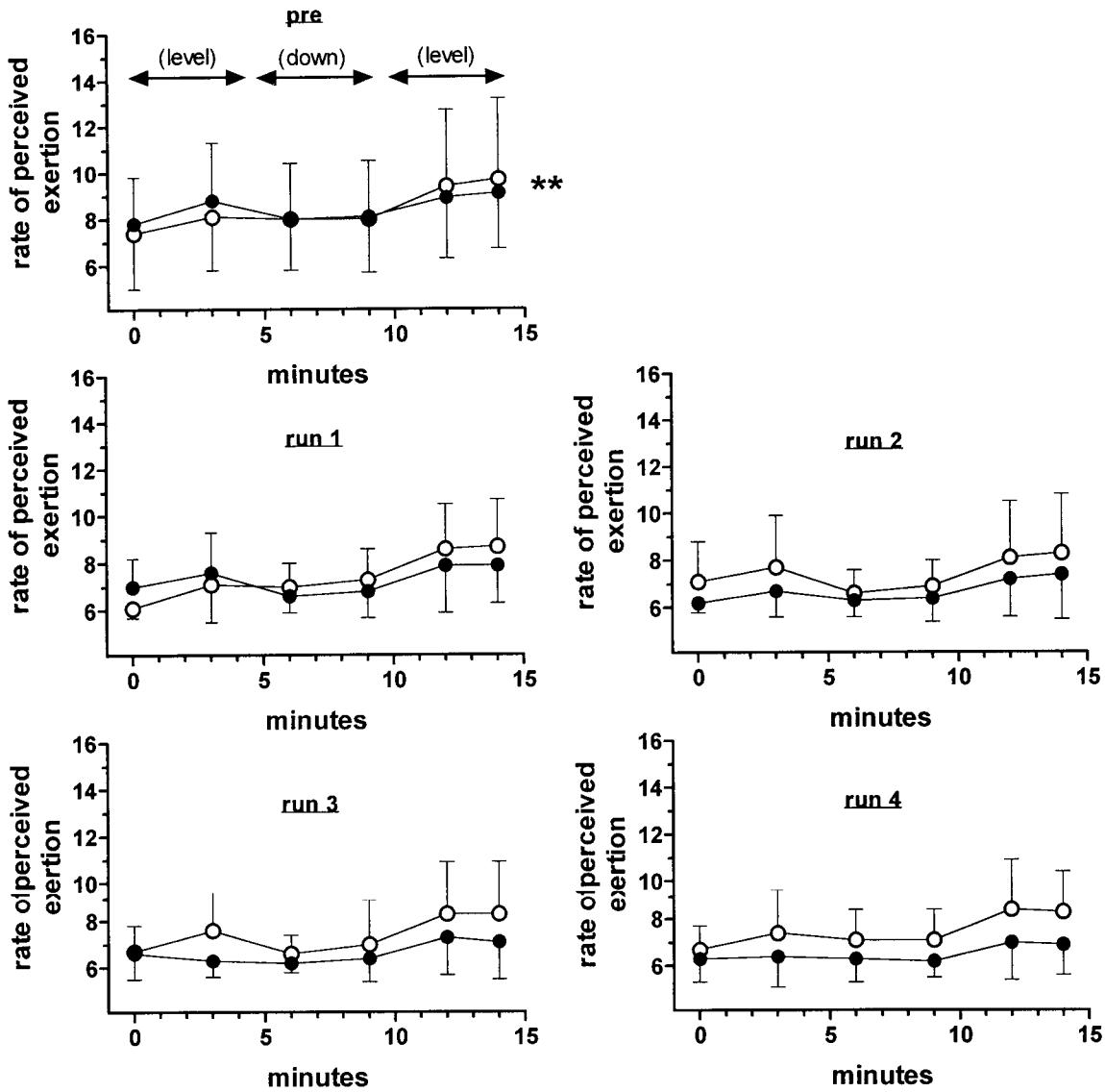

Figure 12: Rate of perceived exertion during the 15 minute submaximal recovery runs for the downhill (● n=9) and level group (O n=7) prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial.

Main effect: Runs
** P < 0.001 pre vs. day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4)

Main effect: Minutes
** P < 0.004 immediately vs. minute 3, 12, 14
** P < 0.01 minute 3 vs. minute 6, 12, 14
** P < 0.0000001 minute 6 vs. minute 12, 14
** P < 0.0000001 minute 9 vs. minute 12, 14

3.3.4.3 Stride length

Stride length during the 15 minute submaximal recovery runs for both groups prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial are shown in Table 15 and Figure 13.

There was a significant difference for stride length, between the 15 minute submaximal recovery run prior to training (pre) and submaximal recovery run 1, run 2, run 3 and run 4 after the 30-km time trial ($P < 0.04$). The average stride length for both groups before the 30-km time trial was 1.24 ± 0.07 m. Thereafter, the average stride length was 1.26 ± 0.05; 1.25 ± 0.04; 1.26 ± 0.05 and 1.27 ± 0.06 m for days 4, 7, 14 and 21 after the 30-km time trial.

Analysis showed a significant difference over time between stride length determined at minute 4 and minute 9, and between minute 9 and minute 14 of the submaximal recovery runs ($P < 0.0000001$).

There were no significant differences between groups at any of the time points. However, on day 4 (run 1) there was a tendency for the stride length of the level group to stay constant during the downhill phase of the protocol (minute 9), in contrast to a slight increase at this stage on the other testing days. This trend however was not statistically different.

Table 15: Changes in stride length (SL) between the downhill (n=9) and the level (n=7) group during five x 15 minute submaximal recovery runs. Both groups ran at 70% of PTRS at a 0% grade for the first 5 minutes, thereafter the gradient was decreased to –10% for the next 5 minutes and for the last 5 minutes the grade was increased again to 0%. Values are expressed as mean ± SD.

Day	Recovery Run	Time (minutes)	Downhill group	Level group
			SL (m)	SL (m)
pre	Run pre	4	1.23 ± 0.06	1.19 ± 0.04
		9	1.29 ± 0.09	1.24 ± 0.04
		14	1.25 ± 0.06	1.20 ± 0.06
			1.26 ± 0.07	**1.21 ± 0.05**
4	Run 1	4	1.25 ± 0.05	1.24 ± 0.04
		9	1.32 ± 0.05	1.25 ± 0.05
		14	1.25 ± 0.05	1.24 ± 0.04
			1.27 ± 0.06	**1.24 ± 0.04**
7	Run 2	4	1.26 ± 0.04	1.23 ± 0.03
		9	1.30 ± 0.04	1.27 ± 0.03
		14	1.24 ± 0.03	1.23 ± 0.05
			1.26 ± 0.04	**1.24 ± 0.04**
14	Run 3	4	1.25 ± 0.03	1.24 ± 0.04
		9	1.30 ± 0.04	1.28 ± 0.06
		14	1.26 ± 0.04	1.23 ± 0.05
			1.27 ± 0.04	**1.25 ± 0.05**
21	Run 4	4	1.25 ± 0.04	1.25 ± 0.07
		9	1.31 ± 0.05	1.29 ± 0.07
		14	1.25 ± 0.03	1.24 ± 0.06
			1.27 ± 0.05	**1.26 ± 0.07**

Main effect: Runs
* $P < 0.04$ pre vs. day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial

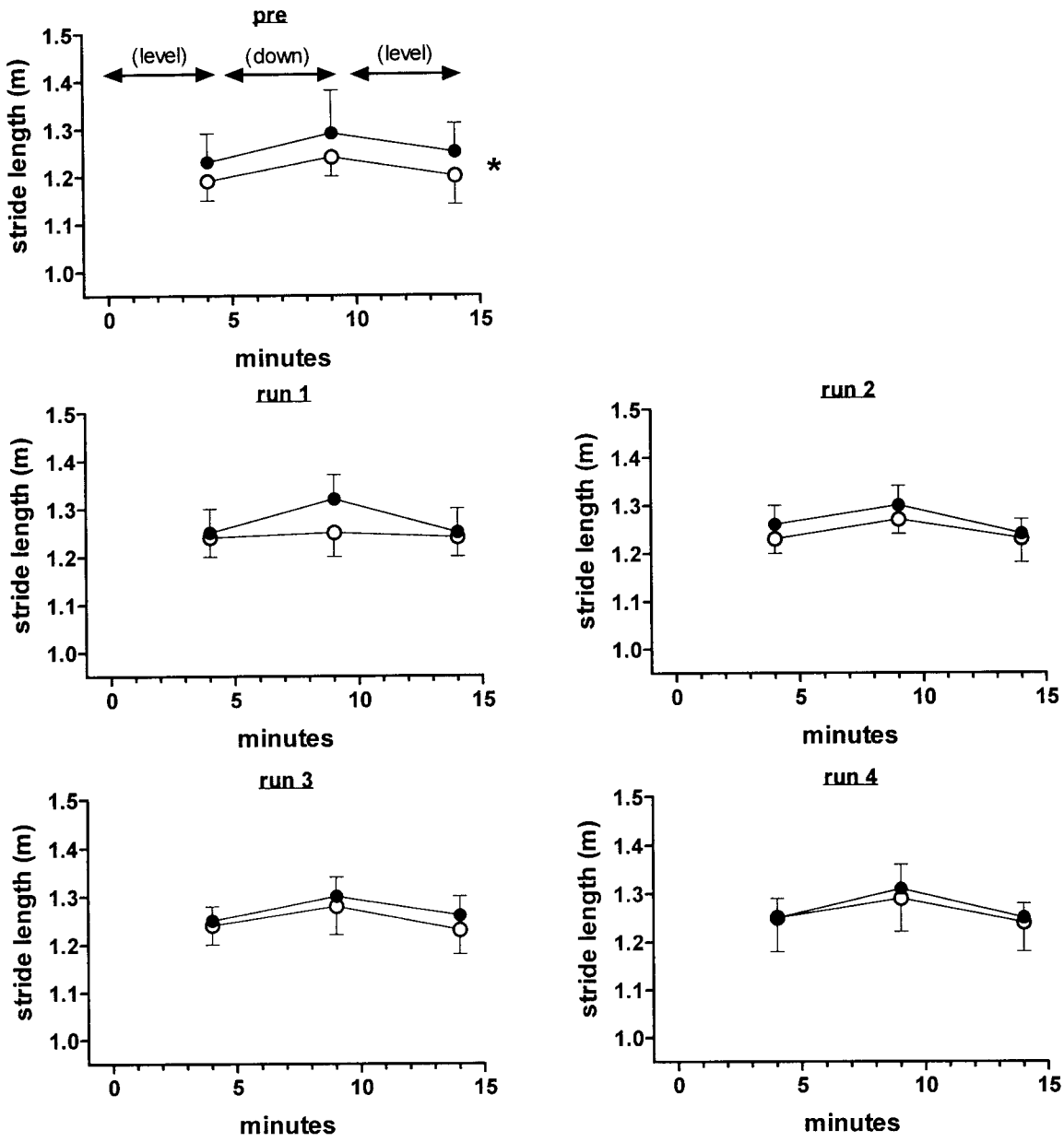

Figure 13: Stride length during the 15 minute submaximal recovery runs for the downhill (● n=9) and level group (O n=7) prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial.

Main effect: Runs
* $P < 0.04$ pre vs. day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4)

Main effect: Minutes
** $P < 0.0000001$ minute 4 vs. minute 9
** $P < 0.0000001$ minute 9 vs. minute 14

3.3.4.4 Ventilation

The ventilation rate during the 15 minute submaximal recovery runs for the downhill and level group prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial are shown in Table 16 and Figure 14.

There was a significant difference between the ventilation rate of the 15 minute submaximal recovery run prior to training (pre) (76.2 ± 13.0 $l.min^{-1}$) compared to submaximal recovery run 1 (72.8 ± 10.6 $l.min^{-1}$), run 2 (72.3 ± 13.1 $l.min^{-1}$), run 3 (72.3 ± 11.6 $l.min^{-1}$) and run 4 (73.0 ± 11.4 $l.min^{-1}$) after the 30-km time trial ($P < 0.01$).

The ventilation rate generally decreased during the downhill phase of the 15 minute recovery protocol (minute 9). Both groups responded similarly throughout this phase of the experiment. The details of these changes are shown below the legend of Figure 14.

Table 16: Changes in ventilation (Vi) between the downhill (n=9) and the level (n=7) group during five x 15 minute submaximal recovery runs. Both groups ran at 70% of PTRS at a 0% grade for the first 5 minutes, thereafter the gradient was decreased to –10% for the next 5 minutes and for the last 5 minutes the grade was increased again to 0%. Values are expressed as mean ± SD.

Day	Recovery Run	Time (minutes)	Downhill group	Level group
			Vi ($l.min^{-1}$)	Vi ($l.min^{-1}$)
pre	Run pre	3	78.8 ± 10.7	80.0 ± 9.7
		6	76.7 ± 14.2	75.4 ± 10.0
		9	61.8 ± 11.0	60.8 ± 10.9
		12	78.1 ± 9.7	80.2 ± 7.7
		14	84.8 ± 11.3	85.8 ± 10.7
			76.1 ± 13.4	**76.4 ± 12.7**
4	Run 1	3	74.5 ± 7.1	77.0 ± 5.0
		6	68.2 ± 6.5	73.0 ± 4.9
		9	55.6 ± 6.9	60.5 ± 4.4
		12	76.3 ± 7.8	79.4 ± 7.2
		14	79.4 ± 8.7	81.8 ± 7.8
			70.8 ± 11.1	**75.9 ± 9.0**
7	Run 2	3	75.1 ± 7.1	80.8 ± 10.9
		6	68.6 ± 7.5	66.3 ± 12.4
		9	57.6 ± 7.3	56.0 ± 9.0
		12	74.3 ± 8.6	83.2 ± 11.8
		14	78.4 ± 8.8	85.1 ± 13.3
			70.8 ± 10.5	**74.3 ± 15.8**
14	Run 3	3	77.3 ± 8.0	78.2 ± 7.4
		6	68.7 ± 6.6	71.0 ± 10.3
		9	56.1 ± 6.8	57.4 ± 7.1
		12	76.6 ± 8.2	77.8 ± 9.2
		14	79.6 ± 8.4	81.0 ± 9.5
			71.7 ± 11.3	**73.1 ± 11.9**
21	Run 4	3	76.7 ± 9.3	77.6 ± 5.0
		6	69.6 ± 9.1	72.0 ± 8.8
		9	57.2 ± 9.2	60.6 ± 8.7
		12	77.6 ± 11.0	77.4 ± 5.2
		14	79.4 ± 10.0	82.0 ± 6.0
			72.1 ± 12.4	**73.9 ± 10.0**

Main effect: Runs
** $P < 0.01$ pre vs. day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial

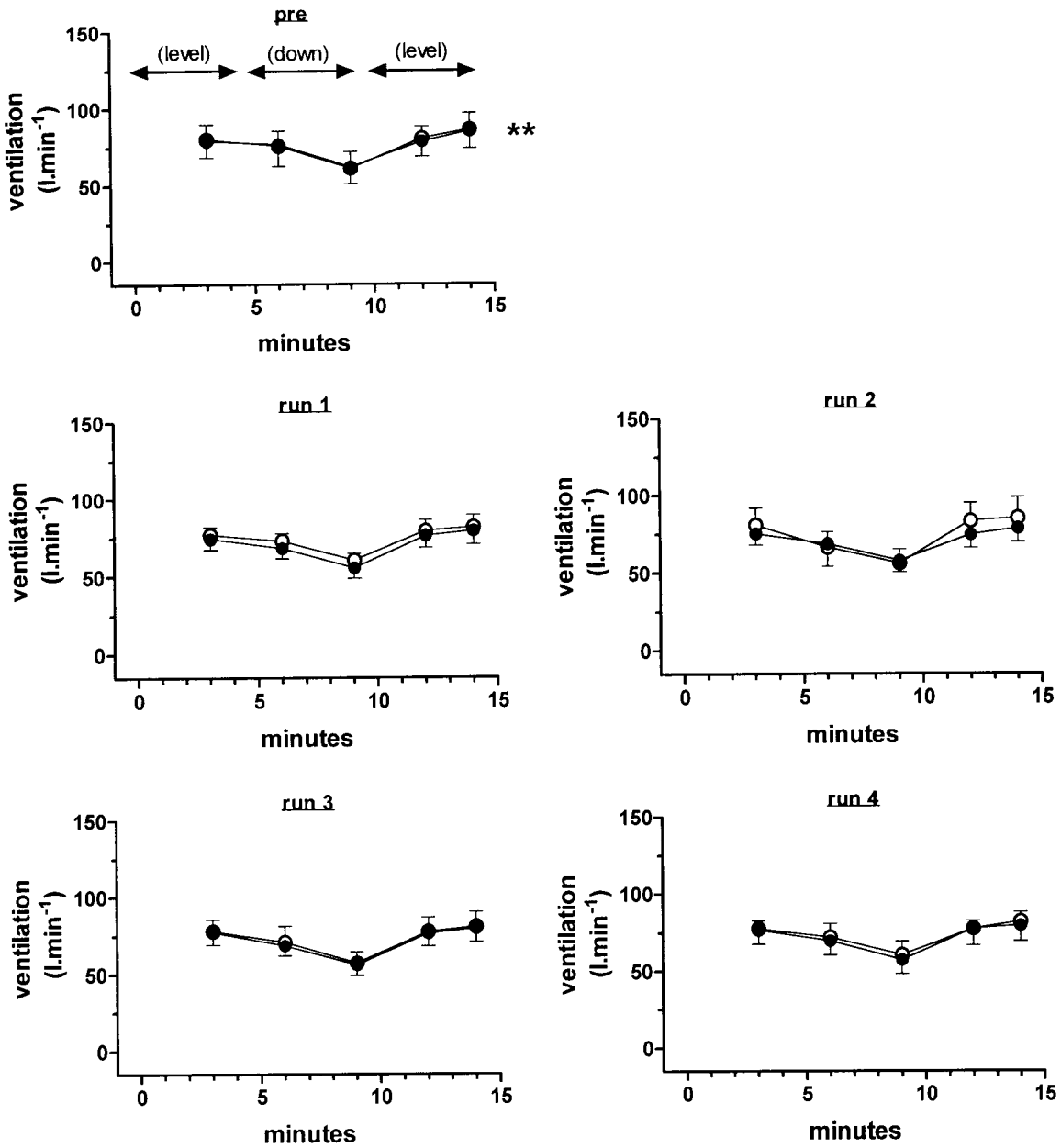

Figure 14: Ventilation (Vi) during the 15 minute submaximal recovery runs for the downhill (● n=9) and level group (O n=7) prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial. Both groups ran at 70% of PTRS at a 0% grade for the first 5 minutes, thereafter the gradient was decreased to −10% for the next 5 minutes and for the last 5 minutes the grade was increased again to 0%.

Main effect: Runs
** P < 0.01 pre vs. day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4)

Main effect: Minutes
** P < 0.001 minute 3 vs. minute 6, 9, 14
** P < 0.0000001 minute 6 vs. minute 9, 12, 14
** P < 0.0000001 minute 9 vs. minute 12, 14
** P < 0.0004 minute 12 vs. minute 14

3.3.4.5 Submaximal oxygen consumption

Oxygen consumption (ml $kg^{-1}.min^{-1}$) during the 15 minute submaximal recovery runs for both groups prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial are shown in Table 17 and Figure 15.

There was a significant difference in VO_2 (ml $kg^{-1}.min^{-1}$), between the 15 minute submaximal recovery run prior to training (pre) (downhill: 35.9 ± 5.9 ml $kg^{-1}.min^{-1}$; level: 34.3 ± 5.6 ml $kg^{-1}.min^{-1}$) and submaximal recovery run 1 (downhill: 33.9 ± 5.5 ml $kg^{-1}.min^{-1}$; level: 33.7 ± 4.9 ml $kg^{-1}.min^{-1}$), run 2 (downhill: 33.1 ± 5.0 ml $kg^{-1}.min^{-1}$; level: 32.9 ± 6.1 ml $kg^{-1}.min^{-1}$), run 3 (downhill: 33.7 ± 5.7 ml $kg^{-1}.min^{-1}$; level: 32.8 ± 5.0 ml $kg^{-1}.min^{-1}$) and run 4 (downhill: 33.4 ± 5.1 ml $kg^{-1}.min^{-1}$; level: 33.0 ± 5.2 ml $kg^{-1}.min^{-1}$) ($P < 0.003$).

The VO_2 generally decreased during the downhill phase of the protocol and then returned to the values measured at the start of the 15 minute recovery test when the treadmill gradient was increased to 0%. Both groups responded similarly throughout the experiment. The details of these changes are shown below the legend of Figure 15.

Table 17: Changes in oxygen consumption (VO_2) (ml $O_2kg^{-1}.min^{-1}$) between the downhill (n=9) and the level (n=7) group during the five x 15 minute submaximal recovery runs. Both groups ran at 70% of PTRS at a 0% grade for the first 5 minutes, thereafter the gradient was decreased to –10% for the next 5 minutes and for the last 5 minutes the grade was increased again to 0%. Values are expressed as mean ± SD.

Day	Recovery Run	Time (minutes)	Downhill group	Level group
			VO_2	VO_2
pre	Run pre	3	39.0 ± 3.5	37.3 ± 3.8
		6	34.3 ± 3.3	32.8 ± 2.9
		9	28.0 ± 4.1	26.4 ± 2.7
		12	38.7 ± 3.6	37.5 ± 4.3
		14	39.8 ± 5.2	37.8 ± 4.2
			35.9 ± 5.9	**34.3 ± 5.6**
4	Run 1	3	36.8 ± 2.9	35.3 ± 2.7
		6	31.7 ± 2.8	31.2 ± 2.4
		9	25.5 ± 2.6	24.5 ± 2.7
		12	37.6 ± 2.2	36.0 ± 3.1
		14	38.0 ± 2.7	36.3 ± 3.4
			33.9 ± 5.5	**33.7 ± 4.9**
7	Run 2	3	36.0 ± 2.1	36.1 ± 2.6
		6	31.1 ± 2.7	28.8 ± 3.7
		9	25.4 ± 2.7	24.4 ± 1.9
		12	36.3 ± 2.2	37.4 ± 3.3
		14	36.7 ± 2.3	37.6 ± 3.1
			33.1 ± 5.0	**32.9 ± 6.1**
14	Run 3	3	37.1 ± 2.9	35.9 ± 2.8
		6	31.1 ± 2.6	30.6 ± 2.1
		9	24.9 ± 2.6	25.2 ± 2.2
		12	37.3 ± 2.7	35.9 ± 3.1
		14	38.2 ± 2.6	36.5 ± 2.3
			33.7 ± 5.7	**32.8 ± 5.0**
21	Run 4	3	36.2 ± 2.0	36.0 ± 2.7
		6	31.4 ± 2.5	31.4 ± 2.9
		9	25.5 ± 2.6	25.2 ± 2.4
		12	36.6 ± 2.5	35.8 ± 3.7
		14	37.4 ± 2.6	36.7 ± 3.0
			33.4 ± 5.1	**33.0 ± 5.2**

Main effect: Runs
** $P < 0.003$ pre vs. day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial

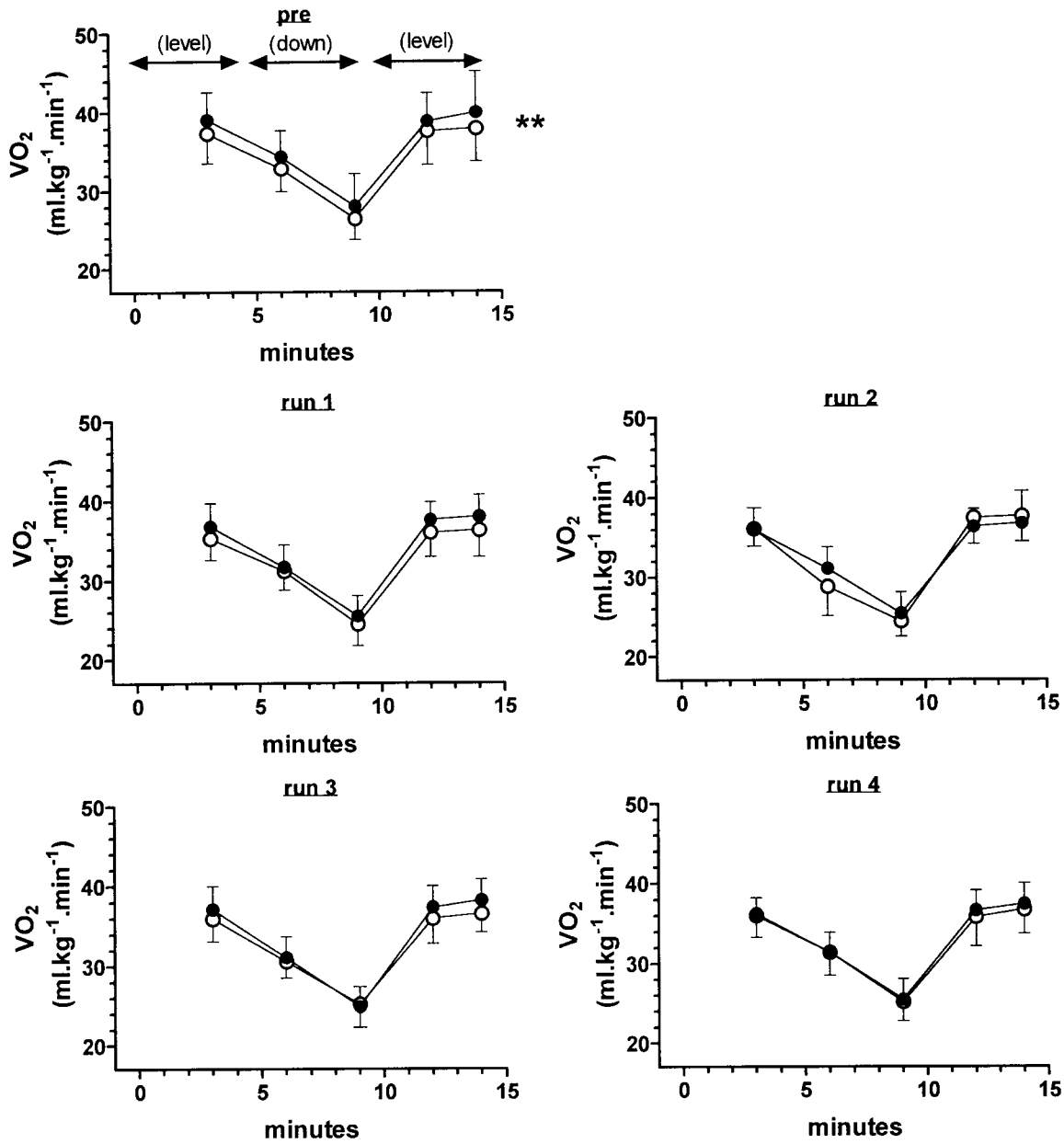

Figure 15: Oxygen consumption (VO_2) ($ml.kg^{-1}.min^{-1}$) during the 15 minute submaximal recovery runs for the downhill (● n=9) and level group (O n=7) prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial.

Main effect: Runs
** $P < 0.003$ pre vs. day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4)

Main effect: Minutes
** $P < 0.007$ minute 3 vs. minute 6, 9, 14
** $P < 0.0000001$ minute 6 vs. minute 9, 12, 14
** $P < 0.0000001$ minute 9 vs. minute 12, 14

3.3.4.6 Submaximal carbon dioxide production

Carbon dioxide production (VCO_2) (ml $kg^{-1}.min^{-1}$) during the 15 minute submaximal recovery runs for the downhill and level group prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial are shown in Table 18 and Figure 16.

Carbon dioxide production stayed relatively constant during the phase of the protocol when the treadmill gradient was 0% and decreased during the downhill phase (-10% grade) of the protocol. This response was similar before and after the 30-km time trial and was also similar between groups. The details of these changes are shown below the legend of Figure 16.

Table 18: Changes in carbon dioxide production (VCO_2) (ml $CO_2kg^{-1}.min^{-1}$) between the downhill (n=9) and the level (n=7) group during five x 15 minute submaximal recovery runs. Both groups ran at 70% of PTRS at a 0% grade for the first 5 minutes, thereafter the gradient was decreased to –10% for the next 5 minutes and for the last 5 minutes the grade was increased again to 0%. Values are expressed as mean ± SD.

Day	Recovery Run	Time (minutes)	Downhill group	Level group
			VCO_2	VCO_2
pre	Run pre	3	2.2 ± 0.2	2.3 ± 0.3
		6	2.1 ± 0.3	2.2 ± 0.4
		9	1.6 ± 0.2	1.6 ± 0.3
		12	2.3 ± 0.2	2.4 ± 0.3
		14	2.5 ± 0.3	2.5 ± 0.3
			2.1 ± 0.4	**2.2 ± 0.4**
4	Run 1	3	2.3 ± 0.2	2.4 ± 0.1
		6	2.1 ± 0.2	2.1 ± 0.2
		9	1.6 ± 0.2	1.6 ± 0.1
		12	2.4 ± 0.2	2.4 ± 0.2
		14	2.4 ± 0.3	2.4 ± 0.2
			2.2 ± 0.4	**2.3 ± 0.3**
7	Run 2	3	2.3 ± 0.2	2.4 ± 0.3
		6	2.0 ± 0.3	1.9 ± 0.3
		9	1.6 ± 0.2	1.6 ± 0.2
		12	2.3 ± 0.2	2.5 ± 0.4
		14	2.4 ± 0.3	2.6 ± 0.4
			2.1 ± 0.4	**2.2 ± 0.5**
14	Run 3	3	2.3 ± 0.3	2.3 ± 0.3
		6	2.0 ± 0.3	2.1 ± 0.4
		9	1.5 ± 0.2	1.6 ± 0.3
		12	2.3 ± 0.2	2.3 ± 0.2
		14	2.4 ± 0.3	2.4 ± 0.3
			2.1 ± 0.4	**2.1 ± 0.4**
21	Run 4	3	2.2 ± 0.3	2.4 ± 0.3
		6	2.0 ± 0.3	2.2 ± 0.4
		9	1.5 ± 0.2	1.7 ± 0.2
		12	2.3 ± 0.2	2.3 ± 0.2
		14	2.4 ± 0.3	2.5 ± 0.3
			2.1 ± 0.4	**2.2 ± 0.4**

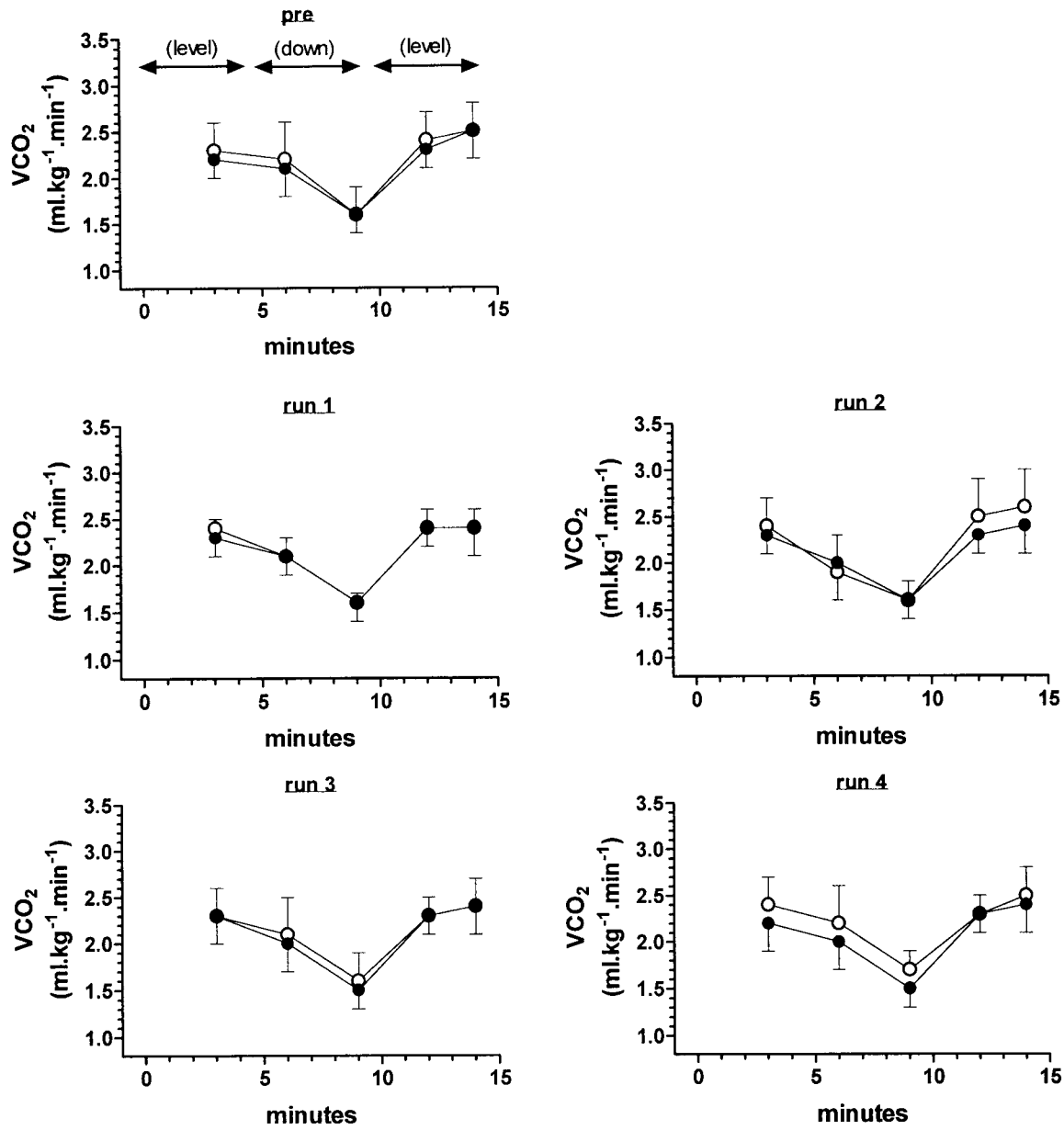

Figure 16: Carbon dioxide production (VCO_2) ($ml.kg^{-1}.min^{-1}$) during the 15 minute submaximal recovery runs for the downhill (● n=9) and level group (O n=7) prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial.

Main effect: Minutes
** $P < 0.000001$ minute 3 vs. minute 6, 9, 14
** $P < 0.0000001$ minute 6 vs. minute 9, 12, 14
** $P < 0.0000001$ minute 9 vs. minute 12, 14
** $P < 0.00003$ minute 12 vs. minute 14

3.3.4.7 Respiratory exchange ratio

Table 19 and Figure 17 shows changes in respiratory exchange ratio (RER) during the 15 minute submaximal recovery runs for the two groups prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial.

There was a significant interaction for RER between the five x 15 minute submaximal recovery runs and the duration (minutes) of the recovery runs, suggesting that the RER changed differently during the submaximal runs during the recovery period ($P < 0.0000001$). However, there were no differences between groups at any stage of the experiment.

The specific differences in RER at each time point are shown Figure 17 under the heading main effect minutes.

Table 19: Changes in respiratory exchange ratio (RER) between the downhill (n=9) and the level (n=7) group during five x 15 minute submaximal recovery runs. Both groups ran at 70% of PTRS at a 0% grade for the first 5 minutes, thereafter the gradient was decreased to –10% for the next 5 minutes and for the last 5 minutes the grade was increased again to 0%. Values are expressed as mean ± SD.

Day	Recovery Run	Time (minutes)	Downhill group	Level group
			RER	RER
pre	Run pre	3	0.82 ± 0.06	0.84 ± 0.06
		6	0.88 ± 0.04	0.89 ± 0.06
		9	0.84 ± 0.03	0.84 ± 0.05
		12	0.87 ± 0.04	0.87 ± 0.03
		14	0.91 ± 0.04	0.91 ± 0.02
			0.86 ± 0.05	**0.87 ± 0.05**
4	Run 1	3	0.88 ± 0.05	0.91 ± 0.04
		6	0.91 ± 0.03	0.92 ± 0.02
		9	0.86 ± 0.03	0.88 ± 0.01
		12	0.88 ± 0.03	0.89 ± 0.04
		14	0.90 ± 0.05	0.90 ± 0.02
			0.89 ± 0.04	**0.90 ± 0.03**
7	Run 2	3	0.88 ± 0.04	0.91 ± 0.06
		6	0.91 ± 0.03	0.91 ± 0.03
		9	0.87 ± 0.02	0.87 ± 0.03
		12	0.87 ± 0.02	0.91 ± 0.05
		14	0.90 ± 0.03	0.91 ± 0.03
			0.89 ± 0.03	**0.90 ± 0.04**
14	Run 3	3	0.89 ± 0.05	0.87 ± 0.08
		6	0.91 ± 0.04	0.90 ± 0.09
		9	0.87 ± 0.02	0.84 ± 0.09
		12	0.88 ± 0.03	0.86 ± 0.07
		14	0.90 ± 0.03	0.88 ± 0.08
			0.89 ± 0.04	**0.90 ± 0.08**
21	Run 4	3	0.86 ± 0.04	0.90 ± 0.03
		6	0.89 ± 0.04	0.93 ± 0.04
		9	0.85 ± 0.04	0.89 ± 0.02
		12	0.87 ± 0.02	0.88 ± 0.03
		14	0.88 ± 0.04	0.91 ± 0.03
			0.87 ± 0.04	**0.90 ± 0.03**

Interaction: Runs vs. Minutes
** $P < 0.0000001$

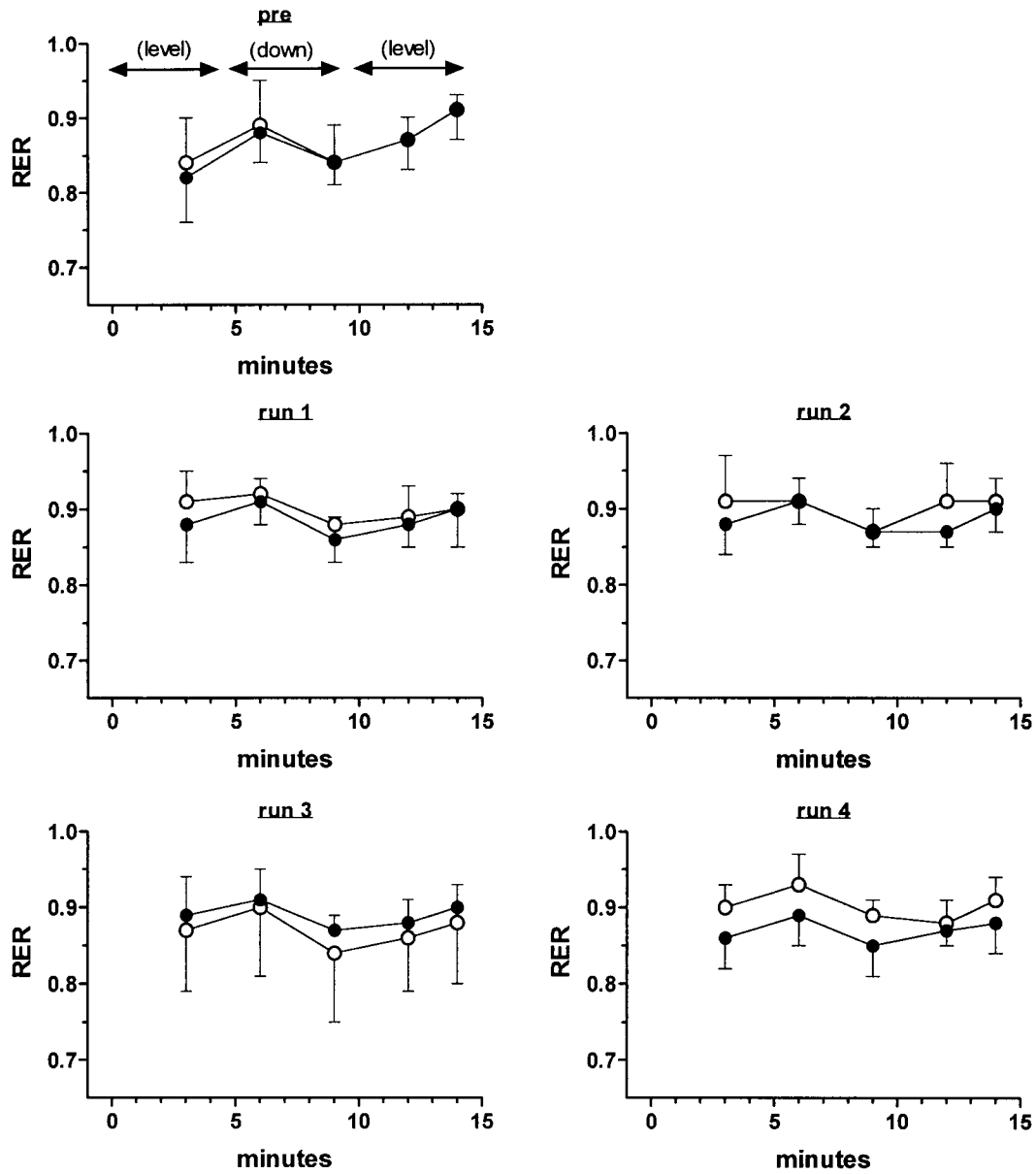

Figure 17: Respiratory exchange ratio (RER) during the 15 minute submaximal recovery runs for the downhill (● n=9) and level group (O n=7) prior to training (pre) and on day 4 (run 1), day 7 (run 2), day 14 (run 3) and day 21 (run 4) after the 30-km time trial.

Interaction: Runs vs. Minutes
** $P < 0.0000001$

Main effect: Minutes
** $P < 0.00002$ minute 3 vs. minute 6, 14
** $P < 0.0000001$ minute 6 vs. minute 9, 12
** $P < 0.0000001$ minute 9 vs. minute 14
** $P < 0.00002$ minute 12 vs. minute 14

3.3.4.8 Measurement of muscle pain

The units for all the pain measurements are arbitrary units. Thus all the pain data are just expressed as a score.

(a) Objective muscle pain

(i) Objective pain score in the rectus femoris muscle

The recovery phase objective pain scores for the rectus femoris muscle are shown in Table 20. Figure 18 shows the objective pain score obtained by the two groups for the rectus femoris muscle for the whole experiment (training and recovery).

A statistically significant interaction, during recovery testing, was found between the two groups over days ($P < 0.0000001$). A significant difference was found for objective pain in the rectus femoris muscle between the downhill and level group ($P < 0.0004$).

When the groups were analyzed independently, with an analysis of variance with repeated measures, there were significant differences between days for the level group. In contrast there was no difference in objective pain, in the rectus femoris muscle, throughout the recovery phase of the experiment for the downhill group. The specific differences are shown in Table 20 and Figure 18.

Table 20: Objective pain scores during the recovery phase obtained by the downhill (n=9) and the level group (n=7) for the rectus femoris muscle. Values are expressed as mean ± SD.

Day	Trial	Downhill group Rectus femoris	Level group Rectus femoris
34	30-km Time Trial		
35	Recovery day 1	0.0 ± 0.0	7.1 ± 2.0 **
36	Recovery day 2	0.0 ± 0.0	6.9 ± 3.1 **
37	Recovery day 3	0.0 ± 0.0	3.6 ± 2.5 **
38	Recovery day 4	0.0 ± 0.0	1.4 ± 1.5 *
39	Recovery day 5	0.0 ± 0.0	0.6 ± 0.8
40	Recovery day 6	0.0 ± 0.0	0.0 ± 0.0
41	Recovery day 7	0.0 ± 0.0	0.4 ± 1.1
48	Recovery day 14	0.0 ± 0.0	0.0 ± 0.0
55	Recovery day 21	0.0 ± 0.0	0.0 ± 0.0

Interaction: Group vs. Days
** $P < 0.0000001$

Main effect: Group
** $P < 0.0004$

Main effect: Days
* $P < 0.02$ recovery day 4 vs. recovery day 6, 14 and 21
** $P < 0.0000001$ recovery day 1 and 2 vs. recovery day 3, 4, 5, 6, 7, 14 and 21
** $P < 0.0005$ recovery 3 vs. recovery day 4, 5, 6, 7, 14 and 21

Figure 18 represents the objective pain score obtained by the two groups for the rectus femoris muscle.

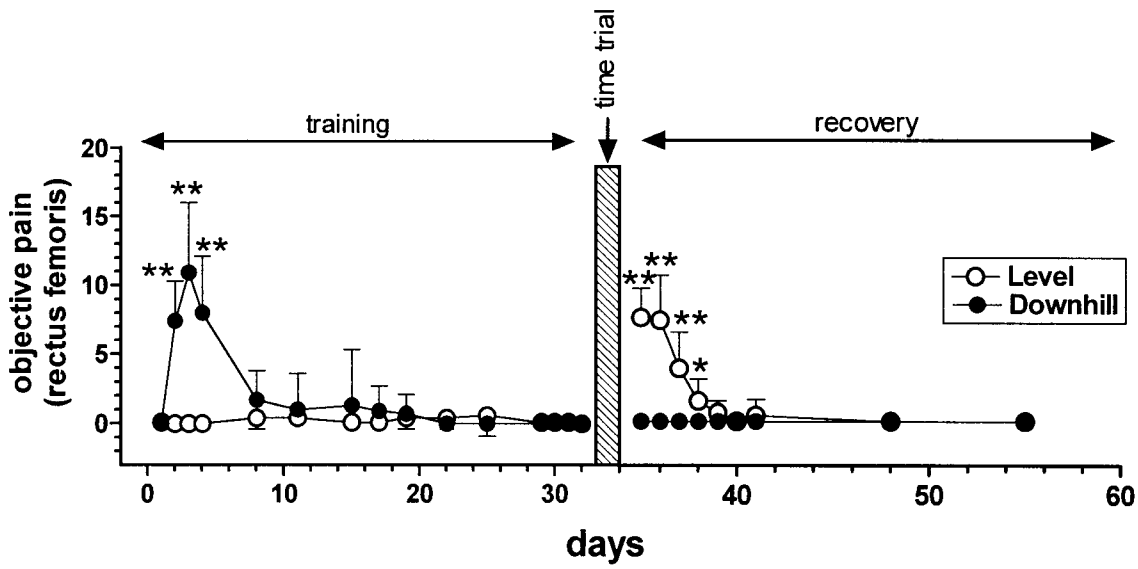

Figure 18: Objective pain score obtained by the downhill (● n=9) and level group (O n=7) for the rectus femoris muscle.

Interaction: Group vs. Days
** P < 0.0000001

Main effect: Group
** P < 0.0004

Main effect: Days
Training
** P < 0.00000001 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)
Recovery
* P < 0.02 recovery day 4 vs. recovery day 6, 14 and 21
** P < 0.0000001 recovery day 1 and 2 vs. recovery day 3, 4, 5, 6, 7, 14 and 21
** P < 0.0005 recovery 3 vs. recovery day 4, 5, 6, 7, 14 and 21

(ii) Objective pain score in the vastus medialis muscle

The recovery phase objective pain scores for the vastus medialis muscles are shown in Table 21. Figure 19 shows the objective pain score obtained by the downhill and level group for the vastus medialis muscle for the entire experiment (training and recovery).

A significant interaction, during recovery testing, was found between the two groups over days ($P < 0.0000001$). A statistically significant difference was found between the downhill and level group ($P < 0.002$).

When the groups were analyzed independently, with an analysis of variance with repeated measures, there were significant differences between days for the level group. In contrast there was no difference in objective pain, in the vastus medialis muscle, throughout the recovery phase of the experiment for the downhill group. The specific differences are shown in Table 21 and Figure 19.

Table 21: Objective pain scores during the recovery phase obtained by the downhill (n=9) and the level group (n=7) for the vastus medialis muscle. Values are expressed as mean ± SD.

Day	Trial	Downhill group Vastus medialis	Level group Vastus medialis
34	30-km Time Trial		
35	Recovery day 1	0.0 ± 0.0	10.6 ± 3.9 **
36	Recovery day 2	0.0 ± 0.0	9.6 ± 3.3 **
37	Recovery day 3	0.0 ± 0.0	5.1 ± 2.0 **
38	Recovery day 4	0.0 ± 0.0	3.6 ± 1.7 **
39	Recovery day 5	0.0 ± 0.0	1.9 ± 2.2 *
40	Recovery day 6	0.0 ± 0.0	0.6 ± 1.1
41	Recovery day 7	0.0 ± 0.0	0.3 ± 0.8
48	Recovery day 14	0.0 ± 0.0	0.0 ± 0.0
55	Recovery day 21	0.0 ± 0.0	0.0 ± 0.0

Interaction: Group vs. Days
** $P < 0.0000001$

Main effect: Group
** $P < 0.002$

Main effect: Days
* $P < 0.02$ recovery day 5 vs. recovery day 14 and 21
** $P < 0.0000001$ recovery day 1 and 2 vs. recovery day 3, 4, 5, 6, 7, 14 and 21
** $P < 0.0001$ recovery day 3 vs. from recovery day 5, 6, 7, 14 and 21
** $P < 0.0004$ recovery day 4 vs. recovery day 6, 7, 14 and 21

Figure 19 represents the objective pain score obtained by the two groups for the vastus medialis muscle.

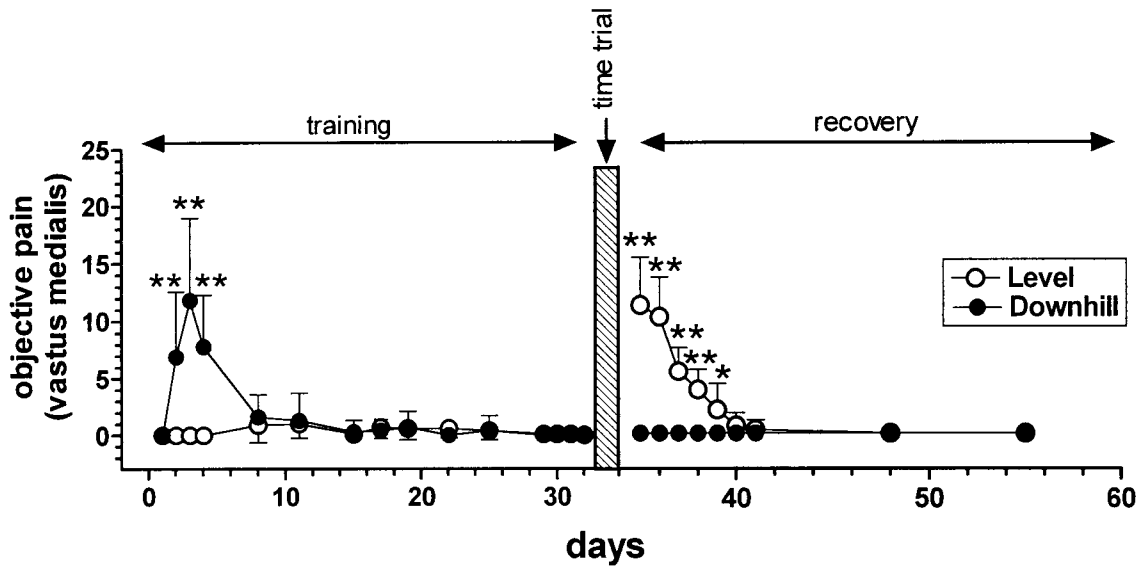

Figure 19: Objective pain score obtained by the downhill (● n=9) and level group (O n=7) for the vastus medialis muscle.

Interaction: Group vs. Days
** P < 0.0000001

Main effect: Group
** P < 0.002

Main effect: Days
Training
** P < 0.000004 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)
Recovery
* P < 0.02 recovery day 5 vs. recovery day 14 and 21
** P < 0.0000001 recovery day 1 and 2 vs. recovery day 3, 4, 5, 6, 7, 14 and 21
** P < 0.0001 recovery day 3 vs. from recovery day 5, 6, 7, 14 and 21
** P < 0.0004 recovery day 4 vs. recovery day 6, 7, 14 and 21

(b) Perceived muscle pain

(i) Muscle soreness rating scale

The recovery phase perceived pain scores are shown in Table 22. Figure 20 represents the perceived pain score in both the downhill and level group obtained for the duration of the trial (training and recovery).

A significant interaction, during recovery testing, was found between the two groups over days ($P < 0.0000001$). A statistically significant difference was found between the downhill and level group ($P < 0.00007$).

When the groups were analyzed independently, with an analysis of variance with repeated measures, there were significant differences between days for the level group. In contrast there was no difference in perceived pain, throughout the recovery phase of the experiment for the downhill group. The specific differences are shown in Table 22 and Figure 20.

Table 22: Perceived pain during the recovery phase of the downhill (n=9) and the level group (n=7). Perceived pain was quantified by using a perceived muscle soreness rating scale. Values are expressed as mean ± SD.

Day	Trial	Downhill group Perceived pain	Level group Perceived pain
34	30-km Time Trial		
35	Recovery day 1	0.0 ± 0.0	3.9 ± 0.7 **
36	Recovery day 2	0.0 ± 0.0	4.1 ± 1.9 **
37	Recovery day 3	0.0 ± 0.0	2.0 ± 1.4 **
38	Recovery day 4	0.0 ± 0.0	0.9 ± 0.9 *
39	Recovery day 5	0.0 ± 0.0	0.4 ± 0.5
40	Recovery day 6	0.0 ± 0.0	0.1 ± 0.4
41	Recovery day 7	0.0 ± 0.0	0.1 ± 0.4
48	Recovery day 14	0.0 ± 0.0	0.0 ± 0.0
55	Recovery day 21	0.0 ± 0.0	0.0 ± 0.0

Interaction: Group vs. Days
** $P < 0.0000001$

Main effect: Group
** $P < 0.00007$

Main effect: Days
* $P < 0.02$ recovery day 4 vs. recovery day 14 and 21
** $P < 0.0000001$ recovery day 1 and 2 vs. recovery day 3, 4, 5, 6, 7, 14 and 21
** $P < 0.003$ recovery day 3 vs. recovery day 4, 5, 6, 7, 14 and 21

Figure 20 shows the perceived pain score in both the downhill and level group obtained for the duration of the experiment.

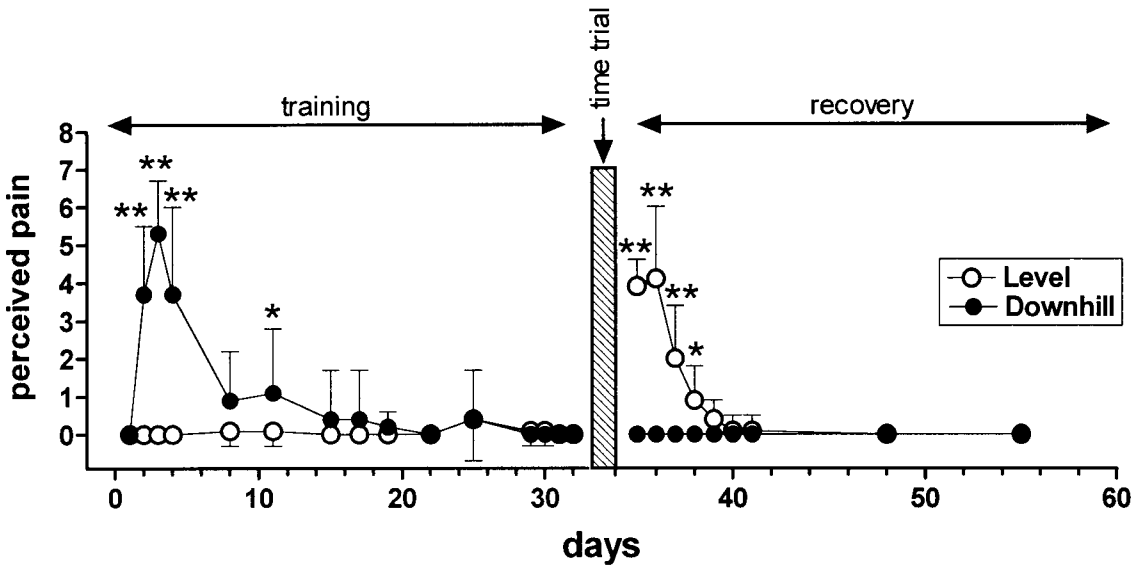

Figure 20: Perceived pain score in both the downhill (● n=9) and level group (O n=7) obtained for the duration of the trial. Perceived pain was quantified by using a perceived muscle soreness rating scale.

Interaction: Group vs. Days
** P < 0.0000001

Main effect: Group
** P < 0.00007

Main effect: Days
Training
* P < 0.04 training run 3 vs. baseline (training run 1)
** P < 0.0000001 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)
Recovery
* P < 0.02 recovery day 4 vs. recovery day 14 and 21
** P < 0.0000001 recovery day 1 and 2 vs. recovery day 3, 4, 5, 6, 7, 14 and 21
** P < 0.003 recovery day 3 vs. recovery day 4, 5, 6, 7, 14 and 21

(ii) Diagrammatic presentation of superficial skeletal muscles

Diagrammatic presentation of anterior view of the superficial skeletal muscles

Recovery phase perceived pain scores obtained by both groups using the diagrammatic presentation of the anterior view of the superficial muscles are shown in Table 23. Figure 21 shows perceived pain scores obtained by the downhill and level group for the diagrammatic presentation of the anterior view of the superficial skeletal muscles for the whole experiment (training and recovery).

A statistically significant interaction, during recovery testing, was found between the two groups over days ($P < 0.0000001$). A significant difference was found between the downhill and level group ($P < 0.0000001$).

When the groups were analyzed independently, with an analysis of variance with repeated measures, there were significant differences between days for the level group. In contrast there was no difference in perceived pain scores, obtained for the anterior superficial skeletal muscles, throughout the recovery phase of the experiment for the downhill group. The specific differences are shown in Table 23 and Figure 21.

Table 23: Perceived pain scores during the recovery phase obtained by the downhill (n=9) and the level group (n=7) for the diagrammatic presentation of the anterior view of the superficial skeletal muscles. Values are expressed as mean ± SD.

Day	Trial	Downhill group Anterior diagram	Level group Anterior diagram
34	30-km Time Trial		
35	Recovery day 1	0.0 ± 0.0	5.0 ± 1.5 **
36	Recovery day 2	0.0 ± 0.0	5.7 ± 1.4 **
37	Recovery day 3	0.0 ± 0.0	4.3 ± 1.8 **
38	Recovery day 4	0.0 ± 0.0	3.1 ± 1.6 **
39	Recovery day 5	0.0 ± 0.0	2.0 ± 1.6 **
40	Recovery day 6	0.0 ± 0.0	0.6 ± 1.5
41	Recovery day 7	0.0 ± 0.0	0.6 ± 1.5
48	Recovery day 14	0.0 ± 0.0	0.0 ± 0.0
55	Recovery day 21	0.0 ± 0.0	0.0 ± 0.0

Interaction: Group vs. Days
** $P < 0.0000001$

Main effect: Group
** $P < 0.0000001$

Main effect: Days
** $P < 0.004$ recovery day 1 and 2 vs. recovery day 4, 5, 6, 7, 14 and 21
** $P < 0.0004$ recovery day 3 vs. recovery day 5, 6, 7, 14 and 21
** $P < 0.00008$ recovery day 4 vs. recovery day 6, 7, 14 and 21
** $P < 0.001$ recovery day 5 vs. recovery day 14 and 21

Figure 21 shows perceived pain scores obtained by the two groups for the diagrammatic presentation of the anterior view of the superficial skeletal muscles.

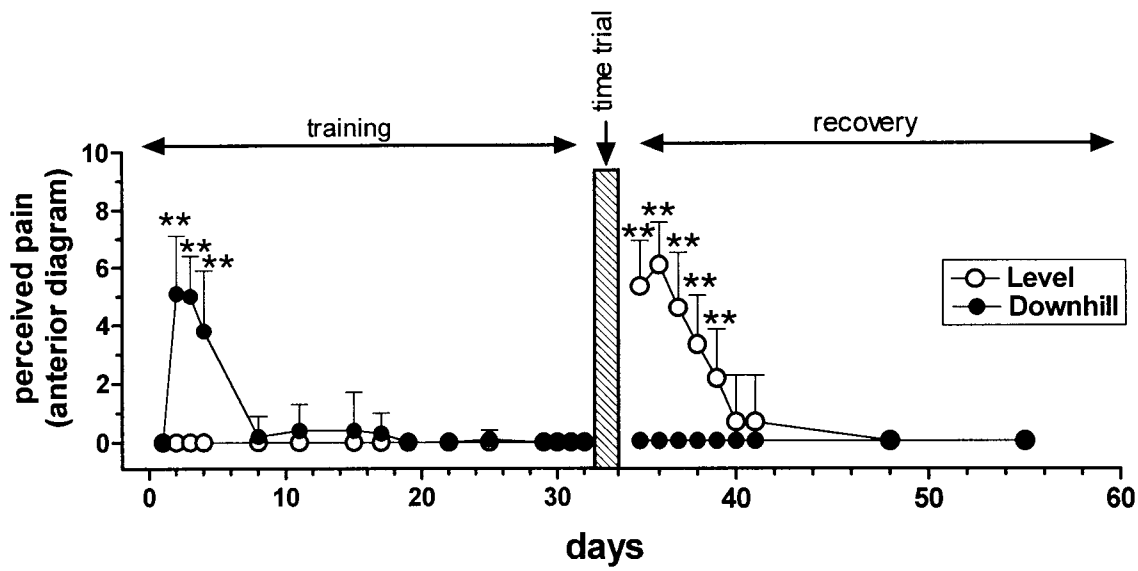

Figure 21: Perceived pain score obtained by the downhill (● n=9) and level group (O n=7) for the diagrammatic presentation of the anterior view of the superficial skeletal muscles.

Interaction: Group vs. Days
** P < 0.0000001

Main effect: Group
** P < 0.0000001

Main effect: Days
Training
** P < 0.0000001 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)
Recovery
** P < 0.004 recovery day 1 and 2 vs. recovery day 4, 5, 6, 7, 14 and 21
** P < 0.0004 recovery day 3 vs. recovery day 5, 6, 7, 14 and 21
** P < 0.00008 recovery day 4 vs. recovery day 6, 7, 14 and 21
** P < 0.001 recovery day 5 vs. recovery day 14 and 21

Diagrammatic presentation of posterior view of the superficial skeletal muscles

Recovery phase perceived pain scores obtained by both groups for the diagrammatic presentation of the posterior view of the superficial muscles are shown in Table 24. Figure 22 represents perceived pain scores obtained by the downhill and level group for the diagrammatic presentation of the posterior view of the superficial skeletal muscles for the whole experiment (training and recovery).

A significant interaction for perceived pain, during recovery testing, was found between the two groups over days ($P < 0.0000001$). A statistically significant difference was found between the downhill and level group ($P < 0.0002$).

When the groups were analyzed independently, with an analysis of variance with repeated measures, there were significant differences between days for the level group. In contrast there was no difference in perceived pain scores, obtained for the posterior superficial skeletal muscles, throughout the recovery phase of the experiment for the downhill group. The specific differences are shown in Table 24 and Figure 22.

Table 24: Perceived pain scores during the recovery phase obtained by the downhill (n=9) and the level group (n=7) for the diagrammatic presentation of the posterior view of the superficial skeletal muscles. Values are expressed as mean ± SD.

Day	Trial	Downhill group Posterior diagram	Level group Posterior diagram
34	30-km Time Trial		
35	Recovery day 1	0.0 ± 0.0	2.9 ± 1.1 **
36	Recovery day 2	0.0 ± 0.0	4.7 ± 2.8 **
37	Recovery day 3	0.0 ± 0.0	2.9 ± 2.8 **
38	Recovery day 4	0.0 ± 0.0	1.7 ± 1.4
39	Recovery day 5	0.0 ± 0.0	0.6 ± 1.0
40	Recovery day 6	0.0 ± 0.0	0.3 ± 0.8
41	Recovery day 7	0.0 ± 0.0	0.9 ± 2.3
48	Recovery day 14	0.0 ± 0.0	0.0 ± 0.0
55	Recovery day 21	0.0 ± 0.0	0.0 ± 0.0

Interaction: Group vs. Days
** $P < 0.0000001$

Main effect: Group
** $P < 0.0002$

Main effect: Days
* $P < 0.04$ recovery day 4 vs. recovery day 6, 14 and 21
** $P < 0.008$ recovery day 1 vs. recovery day 2, 5, 6, 7, 14 and 21
** $P < 0.008$ recovery day 2 vs. recovery day 3, 4, 5, 6, 7, 14 and 21
** $P < 0.004$ recovery day 3 vs. recovery day 5, 6, 7, 14 and 21

Figure 22 presents the perceived pain score obtained by two groups for the diagrammatic presentation of the posterior view of the superficial skeletal muscles.

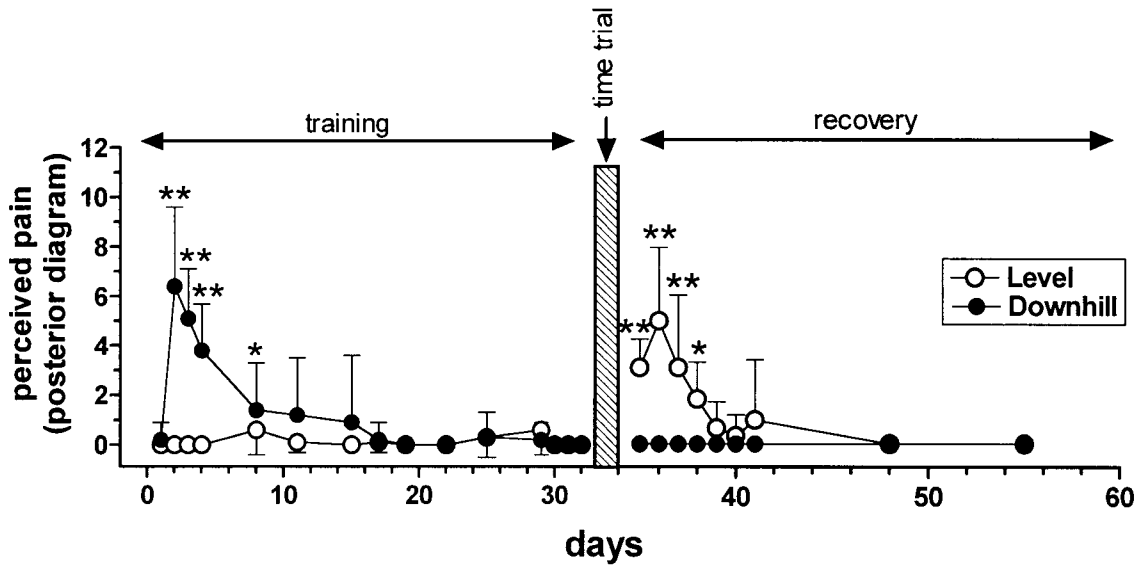

Figure 22: Perceived pain score obtained by the downhill (● n=9) and level group (O n=7) for the diagrammatic presentation of the posterior view of the superficial skeletal muscles.

Interaction: Group vs. Days
** P < 0.0000001

Main effect: Group
** P < 0.0002

Main effect: Days
Training
* P < 0.03 training run 2 vs. baseline (training run 1)
** P < 0.00002 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)
Recovery
* P < 0.04 recovery day 4 vs. recovery day 6, 14 and 21
** P < 0.008 recovery day 1 vs. recovery day 2, 5, 6, 7, 14 and 21
** P < 0.008 recovery day 2 vs. recovery day 3, 4, 5, 6, 7, 14 and 21
** P < 0.004 recovery day 3 vs. recovery day 5, 6, 7, 14 and 21

3.3.4.9 Plasma creatine kinase activity

The changes in plasma CK activity in both the downhill and level group for the duration of the recovery phase of the trial are shown in Table 25. Figure 23 shows the plasma CK activity for the entire protocol (training and recovery).

A statistically significant interaction was found between the two groups over days ($P < 0.009$). During recovery testing a significant difference was found between the downhill and level group ($P < 0.03$). The detailed comparisons are shown beneath the legend of Table 25 and Figure 23.

Table 25: Changes in plasma creatine kinase activity (CK) ($U.l^{-1}$) between the downhill (n=9) and the level group (n=7) for the duration of the recovery phase of the trial. Values are expressed as mean ± SD.

Day	Trial	Downhill group CK ($U.l^{-1}$)	Level group CK ($U.l^{-1}$)
34	30-km Time Trial		
35	Recovery day 1	162.9 ± 63.6	342.3 ± 216.7 **
36	Recovery day 2	109.4 ± 41.0	269.5 ± 218.5 **
37	Recovery day 3	87.7 ± 36.2	169.1 ± 70.2 *
38	Recovery day 4	74.1 ± 19.2	120.6 ± 28.3
39	Recovery day 5	77.7 ± 23.9	100.5 ± 30.3
40	Recovery day 6	76.6 ± 22.2	82.1 ± 21.9
41	Recovery day 7	84.7 ± 42.9	78.0 ± 45.7
48	Recovery day 14	76.6 ± 34.4	93.1 ± 32.0
55	Recovery day 21	73.1 ± 25.1	73.4 ± 18.8

Interaction: Group vs. Days
** $P < 0.009$

Main effect: Group
* $P < 0.03$ difference between groups, during recovery

Main effect: Days
* $P < 0.04$ recovery day 3 vs. recovery day 21
** $P < 0.00005$ recovery day 1 vs. recovery day 3, 4, 5, 6, 7, 14 and 21
** $P < 0.001$ recovery day 2 vs. recovery day 4, 5, 6, 7, 14 and 21

Figure 23 represents the changes in plasma CK activity in the two groups for the duration of the trial.

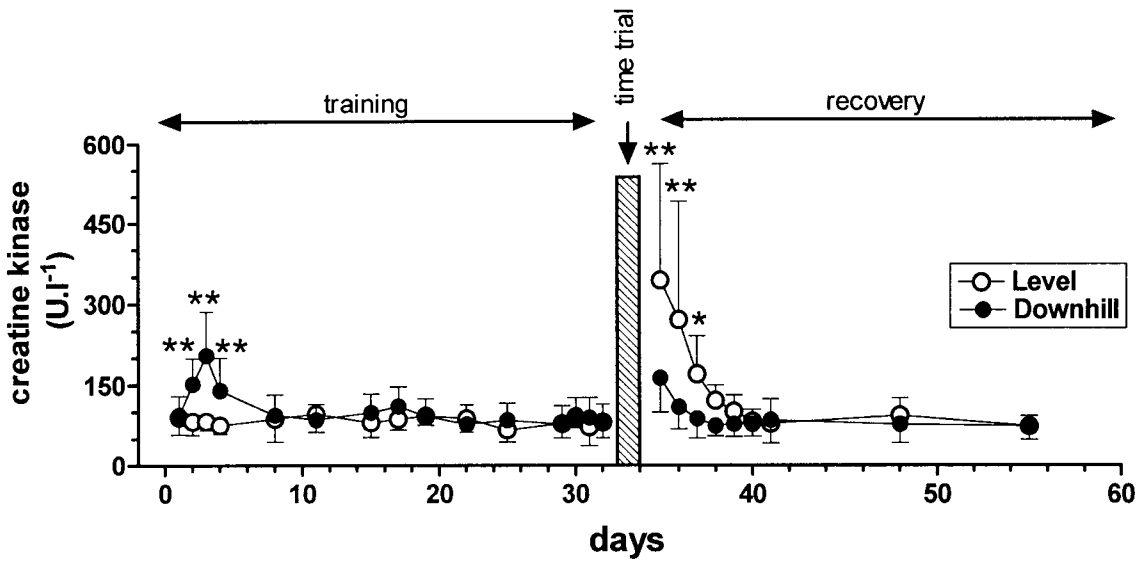

Figure 23: Changes in plasma creatine kinase activity (CK) ($U.l^{-1}$) in both the downhill (● n=9) and level group (O n=7) for the duration of the trial.

Interaction: Group vs. Days
** P < 0.009

Main effect: Group
* P < 0.03 difference between groups, during recovery

Main effect: Days
Training
** P < 0.009 24 hours after training run 1 vs. 48 hours after training run 1, training run 8, 9 and 48, 72 hours after training run 9
** P < 0.003 48 hours after training run 1 vs. all the other time points
** P < 0.003 72 hours after training run 1 vs. training run 8, 9 and 48, 72 hours
Recovery
* P < 0.04 recovery day 3 vs. recovery day 21
** P < 0.00005 recovery day 1 vs. recovery day 3, 4, 5, 6, 7, 14 and 21
** P < 0.001 recovery day 2 vs. recovery day 4, 5, 6, 7, 14 and 21

3.3.4.10 Summary of the recovery phase of the study

The plasma creatine kinase activity and muscle pain was significantly lower in the downhill group compared to the level group after the 30-km time trial. Although heart rate and rate of perceived exertion tended to be lower in the downhill group compared to the level group, these findings were not significant. There were no differences between groups for stride length, oxygen consumption, carbon dioxide production, ventilation rate or respiratory exchange ratio during the 15 minute submaximal recovery runs.

Collectively these results suggest that the physiological stress of the 30-km time trial was less in the downhill group compared to the level group.

3.4 DISCUSSION

The discussion will be presented in the following order. First data from the training phase of the study, where the subjects ran either nine downhill (-10% grade) or level (0% grade) training runs, are discussed. Next, the data collected during the 30-km time trial are discussed, followed by the data collected during the recovery phase of the study. Finally, the interaction of the training phase, the 30-km time trial and the recovery phase after the 30-km time trial are discussed with the overall goal of answering the main research question of this thesis:

"Does downhill training (running at 70% of peak treadmill running speed for nine x 40 minute training sessions on a –10% slope) cause adaptations which make the muscles more resistant to fatigue and damage in a 30-km time trial designed to mimic a road race?"

3.4.1 DOWNHILL/LEVEL TRAINING

3.4.1.1 Heart rate

(a) Heart rate drift

The first important finding was that heart rate increased in both groups during each of the nine 40 minute training runs. The downhill group's heart rate showed an average drift of 10 beats.min^{-1} for the nine downhill training runs, while the level group's heart rate showed an average drift of 13 beats.min^{-1} for the nine level runs. Cardiovascular drift has been defined as a gradual decrease in stroke volume and an increase in heart rate, which occurs after the first few minutes of exercise at a constant work rate. These responses, as well as a progressive reduction of arterial, pulmonary arterial and right ventricular end-diastolic pressures, are the salient components of the general phenomenon of cardiovascular instability during prolonged exercise (Nielsen et al., 1984; Hamilton et al., 1991; Montain and Coyle, 1992; Grant et al., 1997). The results of this study confirm the results found in numerous previous studies (Schwane et al., 1983b; Westerlind et al.,

1992; Schutte et al., 1998) that heart rate drift occurs during both steady state downhill and level running. However, little data are available describing how heart rate drift changes with repeated bouts of downhill and level running. Therefore one of the aims of this investigation was to examine changes in heart rate during repeated bouts of downhill and level running.

Heart rate drift for each of the nine 40 minute level training runs were similar (Training run one to nine: 13, 14, 13, 11, 15, 11, 12 and 15 beats.min^{-1}), suggesting that there was no adaptation, certainly with regards to heart rate, for repeated bouts of level running (Figure 2). In contrast to the findings of the level group, the heart rate drift of the downhill group tended to decrease during the nine downhill training runs (Training run one to nine: 14, 13, 10, 12, 9, 7, 9, 9 and 8 beats.min^{-1}). Furthermore, an analysis of the downhill heart rate data (Figure 1) showed an interaction between the nine training runs and the change in heart rate suggesting that the heart rate drift was different between training sessions. Accordingly, it can be concluded from the heart rate data that there was a training effect for heart rate in the downhill group, particularly after the 5th downhill training session.

In an attempt to explain this training effect it is necessary to firstly explain the mechanism for heart rate drift during exercise. Heart rate drift appears to be caused partially by hypovoleamia (Heaps et al., 1994). Montain and Coyle (1992) examined fluid ingestion during exercise and increases in skin blood flow. This study showed that for every 1% loss in body weight due to dehydration, heart rate increased by 7 beats.min^{-1}. However, this is unlikely to have been the explanation in this study as the exercise was submaximal and of relatively short duration and therefore unlikely to have caused dehydration.

Nielsen et al. (1984) concluded that the drift in heart rate cannot be due only to a reduction in plasma volume caused by a gradual dehydration due to sweating, since plasma volume decreased by 5-6% within the first minutes of exercise, but thereafter remained unchanged. Thus, heart rate drift cannot be caused by low-pressure baroreceptor reflexes from the atria or pulmonary circulation responding to reductions in filling pressure. The upward drift in heart rate has also been attributed to a fall in central

blood volume, which may cause a decreased filling pressure of the heart and thereby a decreased stroke volume. A decreased central blood volume may be caused either by a redistribution of blood to the periphery or by an increase in skin blood flow (Saltin and Steenberg, 1964; Rowell et al., 1969; Sawaka et al., 1979; Hamilton et al., 1991). Nielsen et al. (1984) excluded increases in core temperature as a possible mechanism for heart rate drift.

Schutte et al. (1998) investigated the relationship between heart rate drift and muscle damage during a bout of downhill running. The study showed that subjects with the most severe delayed onset muscle soreness after the downhill run, were also those subjects who had the greatest heart rate drift. These authors suggested a possible feedforward mechanism between muscle damage and the control of heart rate. Should this interpretation be correct then perhaps there was an adaptation in the muscle after downhill training, which protected the muscles against damage and therefore the heart rate drift was lower. The exact nature of the training effect for heart rate during repeated bouts of downhill running needs to be explored and warrants further investigation.

3.4.1.2 Measurement of muscle pain

The main finding in this phase of the study was that objective and perceived muscle pain, in the downhill group, increased significantly 24, 48 and 72 hours after the initial downhill training run and then gradually returned to baseline. The level group on the other hand did not experience any muscle pain after the first 40 minute level training run (Figure 18, 19, 20, 21 and 22).

These results are consistent with those of other investigators who reported that delayed onset muscle soreness is experienced after unaccustomed eccentric muscle action (Byrnes et al., 1985; Costill et al., 1990; Clarkson et al., 1992; Balnave and Thompson, 1993; Gleeson et al., 1995; Pizza et al., 1995; Schutte et al. 1998; Semark et al., 1999). The results also support the findings of Eston et al. (1996) who showed that muscle tenderness increased significantly from baseline values by 48 hours and decreased again by 96 hours and Mc Ardle et al. (1994) who reported that the major difference in muscle

soreness rating occurred at 25 hours post eccentric exercise. When comparing the sensations of muscle soreness following level and downhill running, Schwane et al. (1983b) also found that subjects reported muscle soreness at 24, 48 and 72 hours after downhill running.

In the present study when the downhill group performed the second downhill training run a week later, there was no measured sensation of either objective or perceived muscle pain. The same results were found for the remaining seven downhill training runs. This suggests that the downhill group adapted to the eccentric stress associated with running downhill after the first 40 minute training session making the muscles resistant to further damage. This phenomenon of adapting after a single exposure is known as the "repeated bout effect" and has been well documented (Newham et al., 1987; Clarkson et al., 1992; Westerlind et al, 1992; Mair et al., 1995; Hyatt and Clarkson, 1998; Smith et al., 1998).

The possible mechanism for the "repeated bout effect" is not known (Mair et al., 1995). Armstrong et al. (1983) suggested that an initial bout of novel exercise results in a temporary reduction in the pool of stress-susceptible or degenerating fibers. Although this hypothesis is plausible, there are data to suggest that fibers are not destroyed but strengthened (Clarkson and Tremblay, 1988). If the "repeated bout effect" was the consequence of the elimination of fragile fibers then injury to this population should be evident after a single initial bout of exercise. Schwane and Armstrong (1983) found that after 30 minutes of training in rats, the training protected the muscles from damage following a second bout performed three days later. However, there was no indication of damage after the first session suggesting that necrosis did not occur and lethal damage was not a prerequisite for muscle adaptation.

Newham et al. (1987) proposed that the mechanism responsible for the "repeated bout effect" could be associated with connective tissue and not muscle fibers per se. They concluded that damage caused by the first bout of exercise might act as a stimulus for new collagen synthesis. Thus, the collagen structure would be strengthened and protected from further damage. Golden and Dudley (1992) suggested that adaptation that takes place following eccentric exercise is due to a more efficient recruitment of motor

units. These neural adaptations would serve a "protective" function to set a limit for excessive force generation or better distribute the workload among the fibers.

The results of experimental studies (Maier et al., 1986; Wernig et al., 1990) suggest that the observed muscle fiber injury after exercise may be part of an adaptational degenerating-regenerating repair mechanism that involves changes in fiber type composition, contractile and other protein gene expressions in the muscle. Eston et al. (1996) concluded that it is likely that a combination of cellular and neurological factors is involved in the adaptation response which up to now cannot be explained.

3.4.1.3 Plasma creatine kinase activity

The plasma CK activity for the downhill group during the initial downhill run started increasing at 24 hours after the run, peaked at 48 hours and was still significantly increased 72 hours after the first downhill run ($P < 0.003$). The significant time effect for plasma CK activity seen in the downhill group is similar to what has been reported previously (Armstrong, 1986; Clarkson and Tremblay, 1988; Ebbeling and Clarkson, 1989; Ebbeling and Clarkson, 1990; Schutte et al., 1998), with CK peaking several days after the eccentric bout of exercise and then gradually returning to baseline (Figure 23).

Plasma CK activity in the level group did not increase significantly during any of the level training runs. This result was expected because the level group did not perform any unaccustomed strenuous eccentric exercise (Figure 23). Several previous studies have reported consistently less muscle damage following predominantly concentric exercise than eccentric exercise (Newham et al., 1983b; Schwane et al., 1983b; Dick and Cavanagh, 1987; Westerlind et al., 1994; Gleeson et al., 1995).

When the downhill group performed the second downhill run the plasma CK response was blunted compared to after the initial downhill run. Plasma CK activity did not increase again during any of the remaining downhill training runs. This phenomenon, as described earlier in the section on muscle pain, is know as the "repeated bout effect" and has been shown in numerous studies (Clarkson and Tremblay, 1988; Clarkson et al., 1992; Kuipers,

1994). Triffletti et al. (1988) studied the plasma CK response to a repeated bout using knee extension isometric exercise. Subjects did four bouts of exercise each separated by one week. They concluded that although there was a significant difference in plasma CK responses between bouts one and two, there were no additional reductions with subsequent bouts. Clarkson et al. (1985) investigated the serum CK response to a repeated bout of forearm flexion isometric exercise. Compared to a repeated bout, a substantial reduction in the serum CK response was found following the second bout that was performed one week later. In contrast, Balnave and Thompson (1993) who examined the effect of eccentric walking down a 25% gradient on a treadmill once a week for eight weeks showed a significant rise in serum CK every week blood was analyzed. They concluded that these results could be contributed to by the great individual variability in CK (Balnave and Thopmson, 1993) (Figure 23). The large intersubject variability in the CK response to exercise has been well documented (Noakes, 1987; Newham et al., 1987). However there is no clear explanation for the CK variability. Many factors have been suggested to influence the inter-subject variability, including age, gender, body composition and ethnic group (Balnave and Thompson, 1993). The low CK response for some subjects may be a consequence of a similar exercise performed prior to the laboratory exercise (Clarkson and Tremblay, 1988).

It is not known what causes this dramatic "repeated bout effect" in plasma CK activity. The reduced plasma CK response has been attributed to an adaptation effect in the exercised muscles (Clarkson et al., 1985). Nosaka and Clarkson (1994) documented a blunted response in plasma CK activity even when a second bout was performed with the opposite arm. Although it was expected that plasma CK levels after the second bout would surpass peak plasma CK activity observed from the first bout, the plasma CK response never exceeded the first peak. These authors suggested that accelerated clearance might have influenced plasma CK levels. A study by Hyatt and Clarkson (1998) investigated CK release and clearance, using MM variants, following repeated bouts of eccentric exercise. The authors noted that analysis of CK-MM isoforms following two bouts of eccentric exercise suggest that clearance of CK is enhanced after the first exercise bout. This acceleration would contribute to the dramatic blunting of total CK if a damaging exercise is performed within days of the first bout.

3.4.2 30-KM TIME TRIAL

The 30-km time trial was designed to mimic the demands of an endurance race. It would have been preferable to measure performance directly, by having subjects complete the 30-km time trial as quickly as possible. However, the protocol used in this study was chosen for the following reasons:

1.) All subjects were participating in ultra-endurance races within weeks of completion of the study and therefore would not have been willing to do a maximal exertion at this stage of their training. Had this been forced upon them the data would have been inaccurate and uninterpretable.
2.) To interpret the heart rate and perception of effort data, it was important to control workload.
3.) It is reasonable to assume that a decrease in heart rate and perception of effort at the same relative workload, translate into an improved maximal performance.

3.4.2.1 Heart rate

A main finding of the 30-km time trial was that the heart rates of the two groups responded differently. More specifically, the heart rate of the downhill group, towards the end of the 30-km time trial, did not increase to the same extent as the heart rate of the level group. Another interpretation is that the level group had a greater heart rate drift, than the downhill group (Figure 5). An explanation for this is in accordance with the discussion on proposed mechanisms for heart rate drift discussed earlier.

It can be hypothesized that the downhill group was exposed to damage induced by downhill training. This damage resulted in adaptations in the muscles, which made them more resistant to the demands of the 30-km time trial. Therefore, it is possible that the downhill group were able to perform better in the 30-km time trial as evidenced by heart rate, which did not drift to the same extent as the subjects in the level group who were not exposed to eccentric training.

3.4.2.2 Rate of perceived exertion

Ratings of perceived exertion (RPE), under most conditions, are an accurate marker of work intensity (Borg, 1974; Dunbar et al., 1992). The RPE of the downhill and level group changed differently over time ($P < 0.003$). More specifically the RPE score of the downhill group, towards the end of the 30-km time trial, did not increase to the same extent as the RPE score recorded for the level group. Therefore it can be hypothesized that the downhill training runs in the weeks prior to the 30-km time trial caused certain adaptations which made the subjects more resistant to fatigue towards the end of the time trial (Figure 6).

Previous research examining the perception of effort has shown that a multitude of variables, both cardiopulmonary and peripheral, could influence perceived exertion (Hampson et al., 2001). Though researchers have attempted to identify which variables are most important, it appears likely that perception of effort involves an integration of multiple variables. Hot environmental conditions have been used to elevate heart rate and examine RPE. In the investigations of Kamon et al. (1974) and Pandolf et al. (1972), elevations in heart rate associated with environmental conditions of 44 °C or 54 °C were not associated with proportional elevations in RPE at the different temperatures. However, this disagrees with the findings of Skinner et al. (1973), who found that RPE was related to heart rate in both neutral and hot conditions. In the present study differences in temperature did not influence RPE because during all the testing, laboratory conditions were standardized at a temperature of approximately 21 °C. Banister (1979) found that when increasing amounts of force were applied to the quadriceps and adductor pollicis muscles, perception of effort increased exponentially. In the current study because both groups were exposed to the same 30-km time trial and submaximal recovery protocol during which they ran at the same relative intensity, the differences in RPE found in these phases of the study, could not be attributed to differences in workload between the two groups.

According to Hassmen (1990) training status and exercise modality may also alter the relationship between heart rate and RPE. Highly trained runners, sedentary individuals

and general fitness-trained subjects associate a higher RPE at any given heart rate during cycling exercise compared to treadmill running. In contrast, trained cyclists do not show these differences in RPE ratings between the two activities. Again the current study protocol compensated for these factors that could influence RPE, by using runners with the similar training status and running experience. Other variables that could have affected the differences in RPE between the downhill and the level group such as humidity, training status, exercise intensity and environment was kept constant for the duration of the experiment in both groups.

3.4.2.3 Stride length

It is known from the results of previous studies (Cavanagh and Williams, 1982; Messier et al., 1986) that experienced runners choose a near optimal stride length. Furthermore, experienced runners possess stride lengths that differ from novice runners. The process by which an experienced runner adapts or alters this stride length is unknown, although it may be related to reducing running economy or reducing the risk of muscle damage (Cavanagh and Williams, 1982). A study has also shown that the stride length of novice runners remained variable following seven weeks of treadmill training (Bailey and Messier, 1991). They also concluded that stride length variations had no significant effect on running economy during this phase of training. They also stated that stride length manipulation during the first seven weeks of treadmill training, would most likely not hasten improvements in running economy.

In the present study stride length was not different between the downhill and the level group during the 30-km time trial (Figure 7). Therefore, it may be concluded that the downhill training before the 30-km time trial did not induce any adaptations which manifested as an altered stride length during the time trial.

However, stride length in both groups, showed a significant difference between downhill (-10% grade) and level running (0% grade) during the 30-km time trial ($P < 0.003$). During the downhill phases of the 30-km time trial there was an increase in stride length when compared to the level phases of the 30-km time trial (Figure 7). From these results it

could be hypothesized that while running downhill it was more economical for the runners to decrease stride frequency and therefore develop a longer stride length.

3.4.2.4 Body mass

Body mass decreased by about 1.8% in both groups after the 30-km time trial. This was expected and was similar to the decrease in body mass reported in other studies (Montain and Coyle, 1992; Heaps et al., 1994). It is reasonable to assume that the change in body mass can be attributed mostly to fluid loss during the 30-km time trial (Figure 8).

3.4.2.5 Plasma glucose concentration

There was no change in plasma glucose concentration measured prior to and immediately after the 30-km time trial. These results can be explained by the fact that the subjects in both groups were instructed to maintain their normal pre race eating habits and fluid consumption during the 30-km time trial, which would have minimized the risk of the developing hypoglycemia (Figure 9).

3.4.2.6 Plasma creatine kinase activity

The plasma CK activity increased similarly in both groups immediately after the trial (Figure 10). Although Byrnes et al. (1985) reported that after downhill running plasma CK activity increased three to six hours after exercise, most other studies report an increase in CK activity after a longer delay (Clarkson and Tremblay, 1988; Ebbeling and Clarkson, 1989; Ebbeling and Clarkson, 1990; Schutte et al., 1998).

Several mechanisms have been proposed for the release of CK in the blood after unaccustomed exercise. CK is released into the blood when the cell membrane is damaged or when there is a change in membrane permeability (Armstrong, 1984). Increases in intracellular calcium concentration may influence these changes in membrane integrity (Howell et al., 1993) through an increase in calcium influx through stretch-activated channels in the sarcolemma. Calcium then moves into the cell down the

concentration gradient (Franco and Lansman, 1990; Armstrong et al., 1993). Alternatively calcium influx can occur through ruptures or lesions in the sarcolemma (Duncan and Jackson, 1987).

While transient changes in calcium concentration are essential for muscle excitation-contraction coupling, sustained increases may result in the activation of calcium sensitive proteases and phospholipases (Belcastro et al., 1998). This is deleterious to cell membrane and sarcoplasmic reticulum integrity causing a change in membrane permeability (Jackson et al., 1987; Armstrong, 1990). Another consequence of a sustained elevation of intracellular calcium concentration is the activation of non-lysosomal cysteine proteases, such as calpain (Belcastro et al., 1998). Calpain cleaves a variety of protein substrates including cytoskeletal and myofibrillar proteins. Calpain mediated degradation of these proteins is thought to contribute to changes in muscle structure (Fridén et al., 1981). These morphological changes in the contractile machinery of the muscle may underlie the reduced muscle function of DOMS. In summary, interference with mechanisms of calcium homeostasis might explain the mechanism of CK release into the blood, as seen in both groups after the 30-km time trial.

3.4.3 RECOVERY TESTING

3.4.3.1 Heart rate

The main finding for heart rate during the recovery phase of the experiment was that there was a tendency for the heart rate of the downhill group to be lower during the submaximal recovery runs compared to the level group. This trend is clearly shown in Figure 11. This persisted for 21 days after the 30-km time trial.

Schutte et al. (1998) suggested a possible relationship between heart rate drift and muscle soreness. Muscle soreness has been attributed to muscle fiber damage produced by eccentric action (Fridén et al., 1983). A relationship between muscle soreness, an indicator of muscle damage, and heart rate drift could explain the increased heart rate drift in the level group during the recovery phase of the study. In accordance with this it could

be hypothesized that the muscles of the downhill group were better adapted and were less painful, which would therefore have effected heart rate during the recovery phase. However, the present study showed a dissociation between heart rate and muscle pain as the increased heart rate in the level group persisted for 21 days after the 30-km time trial, by which time muscle soreness had subsided in the level group. It remains unclear on the mechanism, which would be responsible for a long lasting heart rate effect in the level group during the recovery phase of the experiment. However, according to Clarkson et al. (1992) a neurological explanation would be consistent with a long lasting adaptation, since motor skills are known to be "stored" for long periods of time. Clarkson et al. (1992) concluded that it is likely that a combination of cellular and neurological factors is involved in the adaptation response. Future research should be directed towards investigating the mechanism, independent of muscle damage, involved in an increased submaximal heart rate after prolonged submaximal running.

3.4.3.2 Rate of perceived exertion

The interpretation of the RPE data are in accordance with the interpretation based on the heart rate data. This is not surprising as there is a strong relationship between RPE and heart rate (Edwards et al., 1972; Borg, 1974; Sargeant and Davies, 1973; Skinner et al., 1973; Stamford and Noble, 1976) (Figure 12). In analyzing the RPE data in more detail there was no difference during the submaximal recovery runs, between groups before the training phase of the experiment. When the subjects performed the first submaximal recovery run after the 30-km time trial, a small difference in RPE between the two groups could be seen towards the end of the 15 minute submaximal recovery run where the rate of perceived exertion recorded for the level group was higher than that of the downhill group. This difference in RPE between the two groups showed a more pronounced increase in the remaining submaximal runs performed after the 30-km time trial. Therefore, even 21 days after participating in the 30-km time trial there was a trend for the perception of effort of the downhill group to be less than the level group (Figure 12). However, these results must be interpreted with caution because they were not statistically significant.

3.4.3.3 Stride length

Although average stride length during the submaximal recovery runs increased slightly after the training phase of the study and the 30-km time trial, there were no significant differences between the two groups (Figure 13). Stride length, in both groups, tended to increase during the downhill phase of the protocol, possibly to resist the eccentric stress of downhill running.

Based on Figure 13 it is tempting to speculate that during the first submaximal recovery run after the 30-km time trial (day 4) that stride length did not change during the downhill phase of the protocol in the level group. Whether or not this inability to adapt stride length was related to damaged painful muscles will have to be examined in future studies.

3.4.3.4 Cardiorespiratory variables

Analysis revealed a significant difference for ventilation, determined during submaximal recovery testing, over time ($P < 0.001$). While running downhill there was a decrease in ventilation and when the grade of the treadmill was returned to a 0% gradient, ventilation increased again. This pattern was similar in both the downhill and the level group (Figure 14). The average ventilation rate was higher at the start of the experiment (pre), before the training phase and the 30-km time trial, compared to the recovery phase after the 30-km time trial, but this pattern was the same in both groups. These findings are in contrast to the results of a study done by Gleeson et al. (1995). Gleeson et al. (1995) investigated the effects of prolonged eccentric and concentric exercise on muscle soreness and physiological responses to cycling exercise. This study showed an increased ventilation rate during 15 minutes of eccentrically braked cycling (80% of VO_{2max}), performed 48 hours after a bout of eccentric exercise (bench stepping), compared with concentric exercise (uphill walking). Although the results of the two experiments seem contradictory the training protocols for the two experiments differed sufficiently that one could not draw parallels between the two studies.

As expected the oxygen consumption (Figure 15) and carbon dioxide production (Figure 16) decreased, during with the downhill phase of the 15 minute submaximal recovery protocol throughout the experiment. This is in accordance with the study of Davies et al. (1980) who showed that for each 1% decrease in gradient there was a reduction in oxygen cost of about 1.5 ml $O_2.kg^{-1}.min^{-1}$.

With respect to oxygen consumption and carbon dioxide production both groups responded similarly throughout the experiment. The higher average oxygen consumption in both groups at the start of the experiment (pre), compared to after the training phase and the 30-km time trial, can perhaps be attributed to familiarization with treadmill running and therefore an improved running economy.

Based on the results of Gleeson et al. (1995) it was expected that the oxygen cost of running in the level group would have been higher co-inciding with the 15 minute submaximal recovery run that occurred when their muscles were painful (day 4). As mentioned previously the experimental protocol used for the current study and that used in the Gleeson et al. (1995) study was very different and thus comparisons must be made with caution. By the time that the recovery phase of the experiment started the subjects in both the downhill and the level group had run a total of about nine hours on the treadmill. This prolonged period of time on the treadmill may have induced a further degree of familiarization, which resulted in improved running economy and reduced submaximal oxygen consumption.

Analysis showed a significant difference in RER during the 15 minute submaximal recovery runs over time ($P < 0.00002$) (Figure 17). The results revealed that RER changed over time, RER increased continuously from the start of the 15 minute submaximal recovery run to the end of the run. Analysis also showed that while running downhill, there was a slight decrease in RER when compared to running level. Based on the assumption that RER reflects prevailing fuel utilization, it may be assumed that the subjects used slightly less carbohydrate as fuel because RER decreased during the downhill phase of the 15 minute recovery runs. However, valid interpretation of cardiorespiratory variables derived from indirect calorimetry assumes that the metabolism

of the subjects were in steady state. This, however, can not be assumed in the present study, with the changing gradient, therefore these data should be interpreted with caution.

3.4.3.5 Measurement of muscle pain

The main finding of this phase of the experiment was that objective (Figure 18 and 19) and perceived muscle pain (Figure 20, 21 and 22) in the level group, were increased on recovery day 1, 2, 3, 4 and 5 after the 30-km time trial ($P < 0.02$), thereafter muscle pain gradually returned to baseline. In contrast, the downhill group did not experience any muscle pain after participating in the 30-km time trial. These results clearly show that the downhill group was better adapted to the stress of the 30-km time trial than the level group.

These results suggest that the prior bouts of eccentric training that the downhill group was exposed to in the weeks before the 30-km time trial provided a degree of protection against further muscle soreness, as experienced by the level group who was not exposed to eccentic training before the 30-km time trial.

It cannot be determined from the design of the experiment whether nine 40 minute downhill training runs were the optimum exposure or whether the results during the recovery phase would have been the same with fewer exposures. The duration of the training run as well as the slope of the treadmill is other variables, which can also be manipulated during training. The different permutations of number of training runs vs. duration of training runs vs. the gradient of the treadmill and the effect this has on subsequent muscle damage and pain is an important question in determining the appropriate amount of training. The reason this question is so important is because downhill training is associated with an increased risk of injury (Fridén, 1984). Therefore, ideally the minimum training load, which induces the adaptations resulting in an increased resistance to fatigue and protection against muscle damage, should be prescribed. However, it is interesting to note that no injuries occurred during the total downhill training protocol, which included approximately 54 hours of downhill running.

3.4.3.6 Plasma creatine kinase activity

Analysis of plasma CK activity showed an interaction between the two groups over days, during the recovery phase of the study (Figure 23).

After the 30-km time trial plasma CK activity in the downhill group showed a small increase. In contrast, the level group who was subjected to the same strain during the 30-km time trial showed a markedly higher increase in plasma CK activity compared with the downhill group. These data support the muscle pain, heart rate and RPE data which showed that the downhill group were able to resist fatigue and had less muscle damage compared to the level group. In interpreting the CK data it is important to note that when measuring CK in the blood, total CK concentration represent both the efflux and clearance of the enzyme and it must also be kept in mind that although exercise induced CK elevation may indicate muscle damage, it does not provide an index of the magnitude of damage. Thus, interpretation of peak changes in CK should be done with caution (Ebbeling and Clarkson, 1989).

3.4.4 SUMMARY AND CONCLUSION

The study was designed to examine the effect of repeated bouts of either downhill or level running on running performance in, and recovery from, a 30-km time trial, which included phases of downhill running in an attempt to mimic the demands of a road race. In the running community there is a large body of anecdotal evidence suggesting that it is beneficial for runners to include downhill training into their training program, while preparing for an endurance event. The aim of this study was to scientifically test this hypothesis.

The first important finding was that the heart rate decreased in the downhill group during the training phase of the study, suggesting a training effect. The level group did not show any heart rate training effect. Measurement of objective and perceived muscle pain as well as the analysis of plasma CK activity revealed a significant increase in muscle damage after the first downhill training run. However, a "repeated bout effect" was

evident after the initial increase in these indicators of muscle damage, as they did not show any further increases for the duration of the training phase. Thus, it can be concluded that the performance of a single bout of 40 minutes of downhill running (10% grade, 70% PTRS) resulted in adaptations in the muscles to the extent that those muscles were more resistant to the effects of the subsequent bouts of downhill running. Towards the end of the 30-km time trial the level group, showed a significant greater heart rate drift as well as an increased perception of effort when compared with the downhill group, suggesting that they were not able to resist fatigue to the same extent as the downhill group.

Heart rate and RPE recorded during the recovery phase of the study suggested that the downhill group showed a better recovery after the 30-km time trial than the level group. It is important to note that only a trend could be seen and these results must be interpreted with caution. Measurement of objective and perceived pain during the recovery phase after the 30-km time trial showed that the downhill group experienced no significant increase in muscle pain after performing the 30-km time trial. Thus, the adaptation to the muscles of the subjects in the downhill group during the training phase provided protection to those muscles during the time trial and therefore decreased the amount of muscle damage after the 30-km time trial. Plasma CK activity, a marker of muscle damage, was blunted after the 30-km time trial in the downhill group in contrast to the level group who had a significant increase in plasma CK activity. This suggests that the downhill group who performed downhill training showed a degree of protection against muscle damage after the time trial.

In conclusion, the results of the investigation support the hypothesis that the inclusion of downhill training into a training program cause changes, which can be interpreted as enhancing performance during an endurance event and recovery after the event.

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APPENDICES

Appendix I

Table 26: The difference in heart rate ($HR_{difference}$) between the start (minute 5) and end (minute 38) of each of the nine x 40 minute training runs. The downhill group (n=9) ran at 70% of peak treadmill running speed down a 10% grade and the level group ran at a 0% gradient. Values are expressed as mean ± SD.

Day	Training run	Downhill group	Level group
		$HR_{difference}$	$HR_{difference}$
1	Run 1	14.1 ± 11.3	13.4 ± 5.7
8	Run 2	13.3 ± 8.3	13.5 ± 5.9
11	Run 3	9.7 ± 7.5	13.1 ± 3.8
15	Run 4	12.3 ± 8.3	11.3 ± 6.4
17	Run 5	9.3 ± 6.4	12.6 ± 5.1
19	Run 6	6.8 ± 5.8	14.9 ± 2.9
22	Run 7	9.1 ± 6.1	10.7 ± 6.5
25	Run 8	8.8 ± 5.5	9.9 ± 5.7
29	Run 9	7.9 ± 7.7	15.3 ± 6.8

Interaction: Group vs. Runs
* $P < 0.02$

Appendix II

Table 27: Objective pain scores obtained by the downhill (n=9) and the level group (n=7) for the rectus femoris muscle. Values are expressed as mean ± SD.

Day	Trial	Downhill group Rectus femoris	Level group Rectus femoris
1	Training run 1	0.0 ± 0.0	0.1 ± 0.4
2	24 hours	7.4 ± 2.9 **	0.0 ± 0.0
3	48 hours	10. 9 ± 5.1 **	0.0 ± 0.0
4	72 hours	8.0 ± 4.1 **	0.0 ± 0.0
8	Training run 2	1.7 ± 2.1	0.4 ± 0.8
11	Training run 3	1.0 ± 2.6	0.4 ± 0.5
15	Training run 4	1.3 ± 4.0	0.1 ± 0.4
17	Training run 5	0.9 ± 1.8	0.1 ± 0.4
19	Training run 6	0.7 ± 1.4	0.4 ± 0.8
22	Training run 7	0.0 ± 0.0	0.4 ± 0.8
25	Training run 8	0.0 ± 0.0	0.6 ± 1.5
29	Training run 9	0.0 ± 0.0	0.1 ± 0.4
30	24 hours	0.0 ± 0.0	0.1 ± 0.4
31	48 hours	0.0 ± 0.0	0.1 ± 0.4
32	72 hours	0.0 ± 0.0	0.0 ± 0.0
34	30-km Time Trial		
35	Recovery day 1	0.0 ± 0.0	7.1 ± 2.0 **
36	Recovery day 2	0.0 ± 0.0	6.9 ± 3.1 **
37	Recovery day 3	0.0 ± 0.0	3.6 ± 2.5 **
38	Recovery day 4	0.0 ± 0.0	1.4 ± 1.5 *
39	Recovery day 5	0.0 ± 0.0	0.6 ± 0.8
40	Recovery day 6	0.0 ± 0.0	0.0 ± 0.0
41	Recovery day 7	0.0 ± 0.0	0.4 ± 1.1
48	Recovery day 14	0.0 ± 0.0	0.0 ± 0.0
55	Recovery day 21	0.0 ± 0.0	0.0 ± 0.0

Interaction: Group vs. Days
** $P < 0.0000001$

Main effect: Group
** $P < 0.0004$

Main effect: Days
Training
** $P < 0.00000001$ 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)
Recovery
* $P < 0.02$ recovery day 4 vs. recovery day 6, 14 and 21
** $P < 0.0000001$ recovery day 1 and 2 vs. recovery day 3, 4, 5, 6, 7, 14 and 21
** $P < 0.0005$ recovery 3 vs. recovery day 4, 5, 6, 7, 14 and 21

Appendix III

Table 28: Objective pain scores obtained by the downhill (n=9) and the level group (n=7) for the vastus medialis muscle. Values are expressed as mean ± SD.

Day	Trial	Downhill group Vastus medialis	Level group Vastus medialis
1	Training run 1	0.0 ± 0.0	0.0 ± 0.0
2	24 hours	6.9 ± 5.7 **	0.0 ± 0.0
3	48 hours	11.8 ± 7.2 **	0.0 ± 0.0
4	72 hours	7.8 ± 4.5 **	0.0 ± 0.0
8	Training run 2	1.6 ± 2.0	0.9 ± 1.5
11	Training run 3	1.3 ± 2.4	1.0 ± 1.2
15	Training run 4	0.3 ± 1.0	0.1 ± 0.4
17	Training run 5	0.4 ± 1.0	0.7 ± 1.0
19	Training run 6	0.7 ± 1.4	0.6 ± 1.0
22	Training run 7	0.0 ± 0.0	0.6 ± 0.8
25	Training run 8	0.4 ± 1.3	0.4 ± 0.8
29	Training run 9	0.0 ± 0.0	0.1 ± 0.4
30	24 hours	0.0 ± 0.0	0.1 ± 0.4
31	48 hours	0.0 ± 0.0	0.1 ± 0.4
32	72 hours	0.0 ± 0.0	0.0 ± 0.0
34	30-km Time Trial		
35	Recovery day 1	0.0 ± 0.0	10.6 ± 3.9 **
36	Recovery day 2	0.0 ± 0.0	9.6 ± 3.3 **
37	Recovery day 3	0.0 ± 0.0	5.1 ± 2.0 **
38	Recovery day 4	0.0 ± 0.0	3.6 ± 1.7 **
39	Recovery day 5	0.0 ± 0.0	1.9 ± 2.2 *
40	Recovery day 6	0.0 ± 0.0	0.6 ± 1.1
41	Recovery day 7	0.0 ± 0.0	0.3 ± 0.8
48	Recovery day 14	0.0 ± 0.0	0.0 ± 0.0
55	Recovery day 21	0.0 ± 0.0	0.0 ± 0.0

Interaction: Group vs. Days
** $P < 0.0000001$

Main effect: Group
** $P < 0.002$

Main effect: Days
Training
** $P < 0.000004$ 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)
Recovery
* $P < 0.02$ recovery day 5 vs. recovery day 14 and 21
** $P < 0.0000001$ recovery day 1 and 2 vs. recovery day 3, 4, 5, 6, 7, 14 and 21
** $P < 0.0001$ recovery day 3 vs. from recovery day 5, 6, 7, 14 and 21
** $P < 0.0004$ recovery day 4 vs. recovery day 6, 7, 14 and 21

Appendix IV

Table 29: Perceived pain of the downhill (n=9) and the level group (n=7) for the duration of the trial. Perceived pain was quantified by using a perceived muscle soreness rating scale. Values are expressed as mean ± SD.

Day	Trial	Downhill group Perceived pain	Level group Perceived pain
1	Training run 1	0.0 ± 0.0	0.0 ± 0.0
2	24 hours	3.7 ± 1.8 **	0.0 ± 0.0
3	48 hours	5.3 ± 1.4 **	0.0 ± 0.0
4	72 hours	3.7 ± 2.3 **	0.0 ± 0.0
8	Training run 2	0.9 ± 1.3	0.1 ± 0.4
11	Training run 3	1.1 ± 1.7 *	0.1 ± 0.4
15	Training run 4	0.4 ± 1.3	0.0 ± 0.0
17	Training run 5	0.4 ± 1.3	0.0 ± 0.0
19	Training run 6	0.2 ± 0.4	0.0 ± 0.0
22	Training run 7	0.0 ± 0.0	0.0 ± 0.0
25	Training run 8	0.4 ± 1.3	0.4 ± 1.1
29	Training run 9	0.0 ± 0.0	0.1 ± 0.4
30	24 hours	0.0 ± 0.0	0.1 ± 0.4
31	48 hours	0.0 ± 0.0	0.0 ± 0.0
32	72 hours	0.0 ± 0.0	0.0 ± 0.0
34	30-km Time Trial		
35	Recovery day 1	0.0 ± 0.0	3.9 ± 0.7 **
36	Recovery day 2	0.0 ± 0.0	4.1 ± 1.9 **
37	Recovery day 3	0.0 ± 0.0	2.0 ± 1.4 **
38	Recovery day 4	0.0 ± 0.0	0.9 ± 0.9 *
39	Recovery day 5	0.0 ± 0.0	0.4 ± 0.5
40	Recovery day 6	0.0 ± 0.0	0.1 ± 0.4
41	Recovery day 7	0.0 ± 0.0	0.1 ± 0.4
48	Recovery day 14	0.0 ± 0.0	0.0 ± 0.0
55	Recovery day 21	0.0 ± 0.0	0.0 ± 0.0

Interaction: Group vs. Days ** $P < 0.0000001$

Main effect: Group ** $P < 0.00007$

Main effect: Days

Training

* $P < 0.04$ training run 3 vs. baseline (training run 1)

** $P < 0.0000001$ 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)

Recovery

* $P < 0.02$ recovery day 4 vs. recovery day 14 and 21

** $P < 0.0000001$ recovery day 1 and 2 vs. recovery day 3, 4, 5, 6, 7, 14 and 21

** $P < 0.003$ recovery day 3 vs. recovery day 4, 5, 6, 7, 14 and 21

Appendix V

Table 30: Perceived pain scores obtained by the downhill (n=9) and the level group (n=7) for the diagrammatic presentation of the anterior view of the superficial skeletal muscles. Values are expressed as mean ± SD.

Day	Trial	Downhill group Anterior diagram	Level group Anterior diagram
1	Training run 1	0.0 ± 0.0	0.0 ± 0.0
2	24 hours	5.1 ± 2.0 **	0.0 ± 0.0
3	48 hours	5.0 ± 1.4 **	0.0 ± 0.0
4	72 hours	3.8 ± 2.1 **	0.0 ± 0.0
8	Training run 2	0.2 ± 0.7	0.0 ± 0.0
11	Training run 3	0.4 ± 0.9	0.0 ± 0.0
15	Training run 4	0.4 ± 1.3	0.0 ± 0.0
17	Training run 5	0.3 ± 0.7	0.0 ± 0.0
19	Training run 6	0.0 ± 0.0	0.0 ± 0.0
22	Training run 7	0.0 ± 0.0	0.0 ± 0.0
25	Training run 8	0.1 ± 0.3	0.0 ± 0.0
29	Training run 9	0.0 ± 0.0	0.0 ± 0.0
30	24 hours	0.0 ± 0.0	0.0 ± 0.0
31	48 hours	0.0 ± 0.0	0.0 ± 0.0
32	72 hours	0.0 ± 0.0	0.0 ± 0.0
34	30-km Time Trial		
35	Recovery day 1	0.0 ± 0.0	5.0 ± 1.5 **
36	Recovery day 2	0.0 ± 0.0	5.7 ± 1.4 **
37	Recovery day 3	0.0 ± 0.0	4.3 ± 1.8 **
38	Recovery day 4	0.0 ± 0.0	3.1 ± 1.6 **
39	Recovery day 5	0.0 ± 0.0	2.0 ± 1.6 **
40	Recovery day 6	0.0 ± 0.0	0.6 ± 1.5
41	Recovery day 7	0.0 ± 0.0	0.6 ± 1.5
48	Recovery day 14	0.0 ± 0.0	0.0 ± 0.0
55	Recovery day 21	0.0 ± 0.0	0.0 ± 0.0

Interaction: Group vs. Days ** $P < 0.0000001$

Main effect: Group ** $P < 0.0000001$

Main effect: Days

Training

** $P < 0.0000001$ 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)

Recovery

** $P < 0.004$ recovery day 1 and 2 vs. recovery day 4, 5, 6, 7, 14 and 21

** $P < 0.0004$ recovery day 3 vs. recovery day 5, 6, 7, 14 and 21

** $P < 0.00008$ recovery day 4 vs. recovery day 6, 7, 14 and 21

** $P < 0.001$ recovery day 5 vs. recovery day 14 and 21

Appendix VI

Table 31: Perceived pain scores obtained by the downhill (n=9) and the level group (n=7) for the diagrammatic presentation of the posterior view of the superficial skeletal muscles. Values are expressed as mean ± SD.

Day	Trial	Downhill group Posterior diagram	Level group Posterior diagram
1	Training run 1	0.2 ± 0.7	0.0 ± 0.0
2	24 hours	6.4 ± 3.2 **	0.0 ± 0.0
3	48 hours	5.1 ± 2.0 **	0.0 ± 0.0
4	72 hours	3.8 ± 1.9 **	0.0 ± 0.0
8	Training run 2	1.4 ± 1.9 *	0.6 ± 1.0
11	Training run 3	1.2 ± 2.3	0.1 ± 0.4
15	Training run 4	0.9 ± 2.7	0.0 ± 0.0
17	Training run 5	0.2 ± 0.7	0.1 ± 0.4
19	Training run 6	0.0 ± 0.0	0.0 ± 0.0
22	Training run 7	0.0 ± 0.0	0.0 ± 0.0
25	Training run 8	0.3 ± 1.0	0.3 ± 0.8
29	Training run 9	0.2 ± 0.7	0.6 ± 1.0
30	24 hours	0.0 ± 0.0	0.0 ± 0.0
31	48 hours	0.0 ± 0.0	0.0 ± 0.0
32	72 hours	0.0 ± 0.0	0.0 ± 0.0
34	30-km Time Trial		
35	Recovery day 1	0.0 ± 0.0	2.9 ± 1.1 **
36	Recovery day 2	0.0 ± 0.0	4.7 ± 2.8 **
37	Recovery day 3	0.0 ± 0.0	2.9 ± 2.8 **
38	Recovery day 4	0.0 ± 0.0	1.7 ± 1.4
39	Recovery day 5	0.0 ± 0.0	0.6 ± 1.0
40	Recovery day 6	0.0 ± 0.0	0.3 ± 0.8
41	Recovery day 7	0.0 ± 0.0	0.9 ± 2.3
48	Recovery day 14	0.0 ± 0.0	0.0 ± 0.0
55	Recovery day 21	0.0 ± 0.0	0.0 ± 0.0

Interaction: Group vs. Days ** $P < 0.0000001$

Main effect: Group ** $P < 0.0002$

Main effect: Days

Training

* $P < 0.03$ training run 2 vs. baseline (training run 1)

** $P < 0.00002$ 24, 48 and 72 hours after training run 1 vs. baseline (training run 1)

Recovery

* $P < 0.04$ recovery day 4 vs. recovery day 6, 14 and 21

** $P < 0.008$ recovery day 1 vs. recovery day 2, 5, 6, 7, 14 and 21

** $P < 0.008$ recovery day 2 vs. recovery day 3, 4, 5, 6, 7, 14 and 21

** $P < 0.004$ recovery day 3 vs. recovery day 5, 6, 7, 14 and 21

Appendix VII

Table 32: Changes in plasma creatine kinase activity (CK) ($U.l^{-1}$) between the downhill (n=9) and the level group (n=7) for the duration of the trial. Values are expressed as mean ± SD.

Day	Trial	Downhill group CK ($U.l^{-1}$)	Level group CK ($U.l^{-1}$)
1	Training run 1	94.7 ± 35.5	90.9 ± 32.7
2	24 hours	152.3 ± 46.9 **	82.5 ± 24.9
3	48 hours	205.4 ± 80.6 **	82.7 ± 17.0
4	72 hours	140.8 ± 59.5 **	75.2 ± 16.0
8	Training run 2	94.7 ± 38.0	87.5 ± 43.0
11	Training run 3	86.1 ± 28.6	96.5 ± 33.5
15	Training run 4	99.4 ± 34.7	80.8 ± 27.1
17	Training run 5	111.1 ± 36.3	87.2 ± 19.9
19	Training run 6	93.3 ± 31.9	92.6 ± 16.8
22	Training run 7	77.4 ± 36.2	88.0 ± 25.2
25	Training run 8	85.3 ± 32.0	66.6 ± 22.3
29	Training run 9	77.6 ± 34.5	78.9 ± 26.5
30	24 hours	94.5 ± 32.0	90.7 ± 19.7
31	48 hours	89.5 ± 37.6	72.2 ± 35.3
32	72 hours	82.6 ± 32.0	81.2 ± 29.0
34	30-km Time Trial		
35	Recovery day 1	162.9 ± 63.6	342.3 ± 216.7 **
36	Recovery day 2	109.4 ± 41.0	269.5 ± 218.5 **
37	Recovery day 3	87.7 ± 36.2	169.1 ± 70.2 *
38	Recovery day 4	74.1 ± 19.2	120.6 ± 28.3
39	Recovery day 5	77.7 ± 23.9	100.5 ± 30.3
40	Recovery day 6	76.6 ± 22.2	82.1 ± 21.9
41	Recovery day 7	84.7 ± 42.9	78.0 ± 45.7
48	Recovery day 14	76.6 ± 34.4	93.1 ± 32.0
55	Recovery day 21	73.1 ± 25.1	73.4 ± 18.8

Interaction: Group vs. Days ** $P < 0.009$

Main effect: Group * $P < 0.03$ difference between groups, during recovery

Main effect: Days

Training

** $P < 0.009$ 24 hours after training run 1 vs. 48 hours after training run 1, training run 8, 9 and 48, 72 hours after training run 9

** $P < 0.003$ 48 hours after training run 1 vs. all the other time points

** $P < 0.003$ 72 hours after training run 1 vs. training run 8, 9 and 48, 72 hours

Recovery

* $P < 0.04$ recovery day 3 vs. recovery day 21

** $P < 0.00005$ recovery day 1 vs. recovery day 3, 4, 5, 6, 7, 14 and 21

** $P < 0.001$ recovery day 2 vs. recovery day 4, 5, 6, 7, 14 and 21

Appendix VIII

Table 33: Changes in body mass (kg), plasma creatine kinase activity (CK) ($U.l^{-1}$) and plasma glucose concentration ($mmol.l^{-1}$) prior to and after the 30-km time trial. For the duration of the time trial both groups ran at 70% of PTRS. The gradient of the treadmill was changed, between 0% (1–6 km, 17-22 km and 25-30 km) and a –10% gradient (7-8 km, 15-16 km and 23-24 km), throughout the time trial. Values are expressed as mean ± SD.

Variables	Time Trial	Downhill group	Level group
Glucose ($mmol.l^{-1}$)	Before	4.5 ± 1.2	4.9 ± 0.7
	After	4.9 ± 0.6	4.8 ± 0.4
Body mass (kg)	Before	71.4 ± 7.0	75.0 ± 7.7
	After	69.7 ± 6.7	73.2 ± 7.5 **
Creatine kinase ($U.l^{-1}$)	Before	76.4 ± 33.5	102.7 ± 31.7
	After	126.3 ± 51.7	143.3 ± 49.2 **

** $P < 0.000008$ Pre-time trial vs. Post-time trial

APPENDIX A

MRC/UCT Research Unit for Exercise Science and Sports Medicine, Department of Human Movement Studies (RAU) and Department of Physiology (WITS)

INFORMED CONSENT FORM

I, _____ agree voluntary to participate in a research project of the MRC/UCT Research Unit for Exercise Science and Sports Medicine, Sport Science Institute, in conjunction with The Department of Human Movement Studies at the Rand Afrikaans University and The Department of Physiology at the University of the Witwatersrand, that involves the following procedures which have been explained to me in full:

1. Height and weight measurements.
2. Anthropometric assessment of body composition.
3. Maximal oxygen consumption test (VO_{2max} test) and a peak treadmill running speed test (PTRS).
4. Five submaximal runs ($VO_{2submax}$ test), the duration of each run will be 15 minutes, at 70% of PTRS.
5. Nine, 40 minute, runs on the treadmill at 70% of PTRS (Downhill group: -10% gradient; Level group: 0% gradient).
6. All subjects must participate in 30-km time trial on the treadmill in the laboratory.
7. Blood samples will be collected from an antecubital vein to determine plasma creatine kinase (an indicator of muscle damage) concentrations. This is the only invasive technique used in this study and standard medical practice and sterile procedures will be strictly adhered to.

8. Heart rate will be measured during every submaximal and 40 minute run (Downhill group: -10% gradient; Level group: 0% gradient) and during the 30-km time trial.
9. Muscle soreness will be measured objectively using a pressure probe and subjectively according to a "rating of perceived pain scale" and by labeling a diagram of the body's musculature to indicate where soreness is experienced.
10. Perceived exertion and stride frequency will be measured during every 15 minute submaximal run and during the 30-km time trial.
11. Subjects must complete a training data and a racing history data sheet.
12. All training activities for the duration of the study must be reported in a logbook.

I confirm that the nature, purpose, testing procedures and the likely duration of the project have been fully explained to me. I understand that I may ask questions at any time during testing procedures. I agree to comply with any instructions given during the study and to co-operate with the investigator. I realize that I am free to withdraw from the study without prejudice at any time, should I choose to do so. I have been informed that the personal information required by the researchers will be held in strict confidentiality. In addition, I know that the information derived from testing procedures will remain confidential and will be revealed only as a number in statistical analysis. I understand that this study has been reviewed by both the Ethics and Research Committee of the Faculty of Health Sciences, University of Cape Town and the Committee for research on human subjects of the University of the Witwatersrand.

I have carefully read this form. I understand the nature, purpose and procedure of this study. I agree to participate in this research project of the MRC/UCT Research Unit for Exercise Science and Sports Medicine, in conjunction with The Department of Human Movement Studies at the Rand Afrikaans University and The Department of Physiology at the University of the Witwatersrand.

Name (in full) of volunteer: _____

Signature of volunteer: _____

Name (in full) of witness : _____

Signature of witness: _____

APPENDIX B

MRC/UCT RESEARCH UNIT FOR EXERCISE SCIENCE AND SPORTS MEDICINE: TRAINING HISTORY

Personal Details

Name: _____

Postal address: _____

_____

_____

Phone number: Home _____

Cell _____

E-mail: _____

Date of birth: _____ Age: _____

Health Status

When was your last medical check up?

6 months ago ☐ > 6 months ago ☐

Family history of heart attack?

Yes ☐ No ☐

Are you on any medication?

Yes ☐ No ☐

If yes, mention the medication and the reason.

_____

_____

Activity History

Please complete the following table. List any sport / activity in which you have regularly participated and estimate for how long and how often you participated.

Sport / Activity	Year started	No. of years participated	Months / year	Hrs / week	Level *	HIT # (yes/no)

* competitive or social

\# high-intensity training or interval training included (yes/no)

Examples of sporting activities include:

- swimming
- cycling
- walking
- rugby
- football / soccer
- hockey
- tennis
- badminton
- volleyball
- squash
- canoeing
- golf
- dancing
- skating
- hiking
- rock climbing
- martial arts
- strength / resistance training

Retrospective Running History

Please complete the relevant sections of the table as accurately as possible. All information will be treated confidentially and used only for research purposes.

Year	Best 10 km	Best 21.1 km	Best 42.2 km	Best Comrades	Total training distance per year (km / year)
	min : ss	h : min : ss	h : min : ss	h : min : ss	
2000					
1999					
1998					
1997					
1996					
1995					
1994					
1993					
1992					
1991					
1990					
1989					
1988					
1987					
1986					
1985					
1984					
1983					
1982					
1981					
1980					

APPENDIX C

MRC/UCT RESEARCH UNIT FOR EXERCISE SCIENCE AND SPORTS MEDICINE: TRAINING DIARY

Name: _____ **Subject code:** _____

Perception of effort during training

Score	English	Afrikaans	Xhosa	Zulu
0	Rest	Rus	Ukuphumla	Phumula
0.5				
1	Really easy	Baie maklik	Ilula kakhulu	Kulula kakhulu
2	Easy	Maklik	(I)lula	Kulula
3	Moderate	Matig	Phakathi	Kulula kahle
4	Sort of hard	Effens moeilik	Inobonzima	Kululi-khuninje !
5	Hard	Moeilik	Inzima	
6	HARD !	MOEILIK	INZIMA	KULIKHUNI KABI
7	VERY HARD !	BAIE MOEILIK	INZIMA KAKHULU	KULIKHUNI KAKHULU
8	The coach tried to kill us !	Die afrigter wou ons dood maak !	Umqeqeshi ubezama ukusibulala	Umqeqeshi ubezama ukusibulala
9	I feel like death warmed over !	Ek voel of 'n trein my getrap het !	Ndiva ngathi ukufa kunifikele	Ngizwa sengathi ngiyafa
10	OH !	Ai !		Ngiyafa manje

Use the tables to document daily training

Week number: ____________

Day and Date	Sleep (hrs)	Training time (h : min: s)	Distance (km)	Perception of effort score	Comments

Week number: ____________

Day and Date	Sleep (hrs)	Training time (h : min: s)	Distance (km)	Perception of effort score	Comments

Week number: ____________

Day and Date	Sleep (hrs)	Training time (h : min: s)	Distance (km)	Perception of effort score	Comments

APPENDIX D: TIME LINE FOR THE DOWNHILL AND LEVEL GROUP

Pre-test (VO_{2max} test)
- anthropometrical measurements
- maximal oxygen consumption
- maximum heart rate
- peak treadmill running speed (PTRS)
- subjects complete training data and racing history data sheet
- subjects receive training logbook

Pre-test (Submax test)
15 minute duration
- oxygen consumption (VO_2)
- heart rate
- perceived exertion
- stride frequency

Training protocol
Week 1
1 × 40 minute downhill / level run
- blood sample (analysis of CK)
- heart rate
- muscle soreness

24,48,72 hours
after first 40 minute run
- blood sample (analysis of CK)
- muscle soreness

Week 2
2 × 40 minute downhill / level runs
- blood sample (analysis of CK)
- heart rate
- muscle soreness

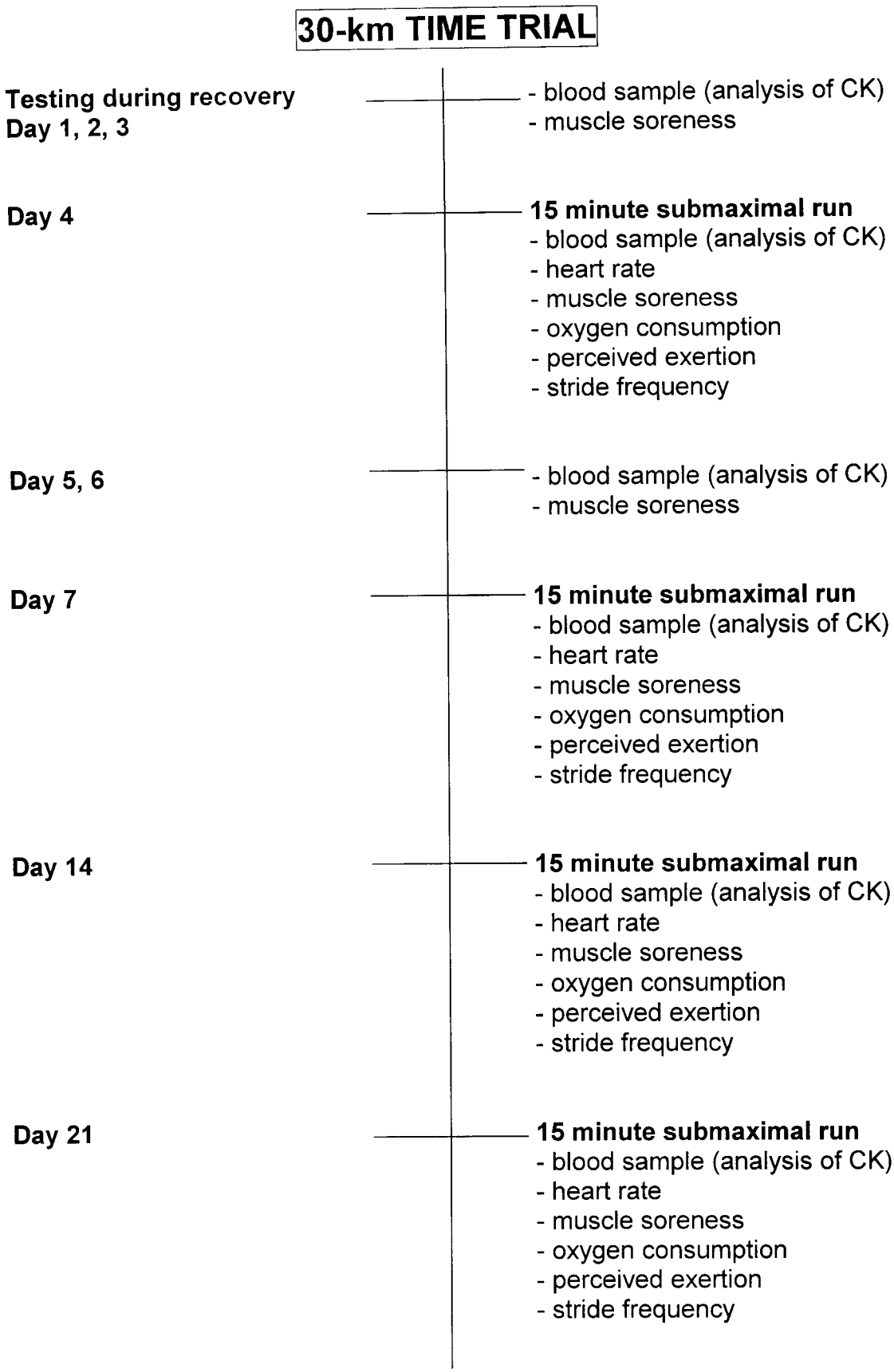
Week 3
3 × 40 minute downhill / level runs
- blood sample (analysis of CK)
- heart rate
- muscle soreness

Week 4
2 × 40 minute downhill / level runs
- blood sample (analysis of CK)
- heart rate
- muscle soreness

Week 5
1 × 40 minute downhill / level run
- blood sample (analysis of CK)
- heart rate
- muscle soreness

24,48,72 hours
after the last 40 minute run
- blood sample (analysis of CK)
- muscle soreness

30-km TIME TRIAL


30-km TIME TRIAL
Testing during recovery
Day 1, 2, 3
- blood sample (analysis of CK)
- muscle soreness
Day 4
15 minute submaximal run
- blood sample (analysis of CK)
- heart rate
- muscle soreness
- oxygen consumption
- perceived exertion
- stride frequency
Day 5, 6
- blood sample (analysis of CK)
- muscle soreness
Day 7
15 minute submaximal run
- blood sample (analysis of CK)
- heart rate
- muscle soreness
- oxygen consumption
- perceived exertion
- stride frequency
Day 14
15 minute submaximal run
- blood sample (analysis of CK)
- heart rate
- muscle soreness
- oxygen consumption
- perceived exertion
- stride frequency
Day 21
15 minute submaximal run
- blood sample (analysis of CK)
- heart rate
- muscle soreness
- oxygen consumption
- perceived exertion
- stride frequency

APPENDIX E

ANTHROPOMETRIC DATA SHEET

Name: _____ Date: _____

Code: _____ Age: _____

Body mass: _____ (kg) Stature: _____ (m)

SKINFOLDS (mm)

*Triceps _____

*Biceps _____

*Subscapular _____

*Suprailiac _____

Calf _____

Mid thigh _____

Abdominal _____

*Sum of 4 skinfolds (mm): _____

Sum of 7 skinfolds (mm): _____ % Fat: _____

APPENDIX F

VO_{2max} Test and Peak Treadmill Running Speed

Name: _____ **Date:** _____

Code: _____

Time (min)	Treadmill speed (km/h)	Heart Rate (bpm)	VO_2	RER

VO_{2max}: _____ (ml/kg/min) **PTRS:** _____ (km/h)

HR_{max}: _____ (beats/min) **70% PTRS:** _____ (km/h)

RER at VO_{2max}: _____

APPENDIX G

40 MINUTE DOWNHILL / LEVEL TRAINING RUNS

Name: _____ Run no: _____

Code: _____ Treadmill speed: _____ (km/h)

Time (min)	Heart rate (bpm)	Time (min)	Heart rate (bpm)
1		21	
2		22	
3		23	
4		24	
5		25	
6		26	
7		27	
8		28	
9		29	
10		30	
11		31	
12		32	
13		33	
14		34	
15		35	
16		36	
17		37	
18		38	
19		39	
20		40	

APPENDIX H

PERCEIVED MUSCLE SORENESS

0 - No pain at all

1 - Very, very slight

2 - Very slight

3 - Slight

4 - Mild

5 - Moderate

6 - Moderate to severe

7 - Severe

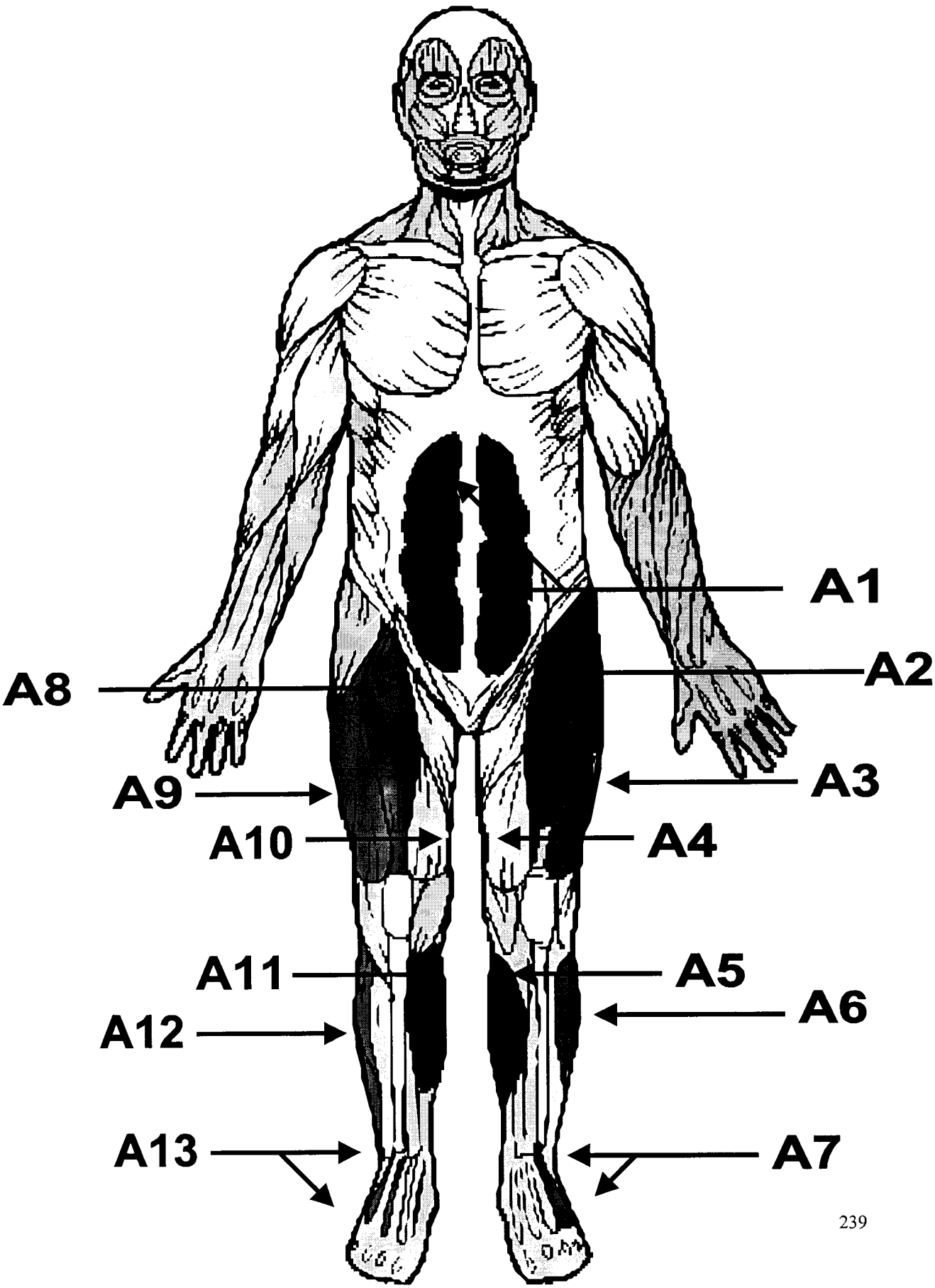
8 - Very severe

9 - Very, very severe

10 - Extreme / maximum pain

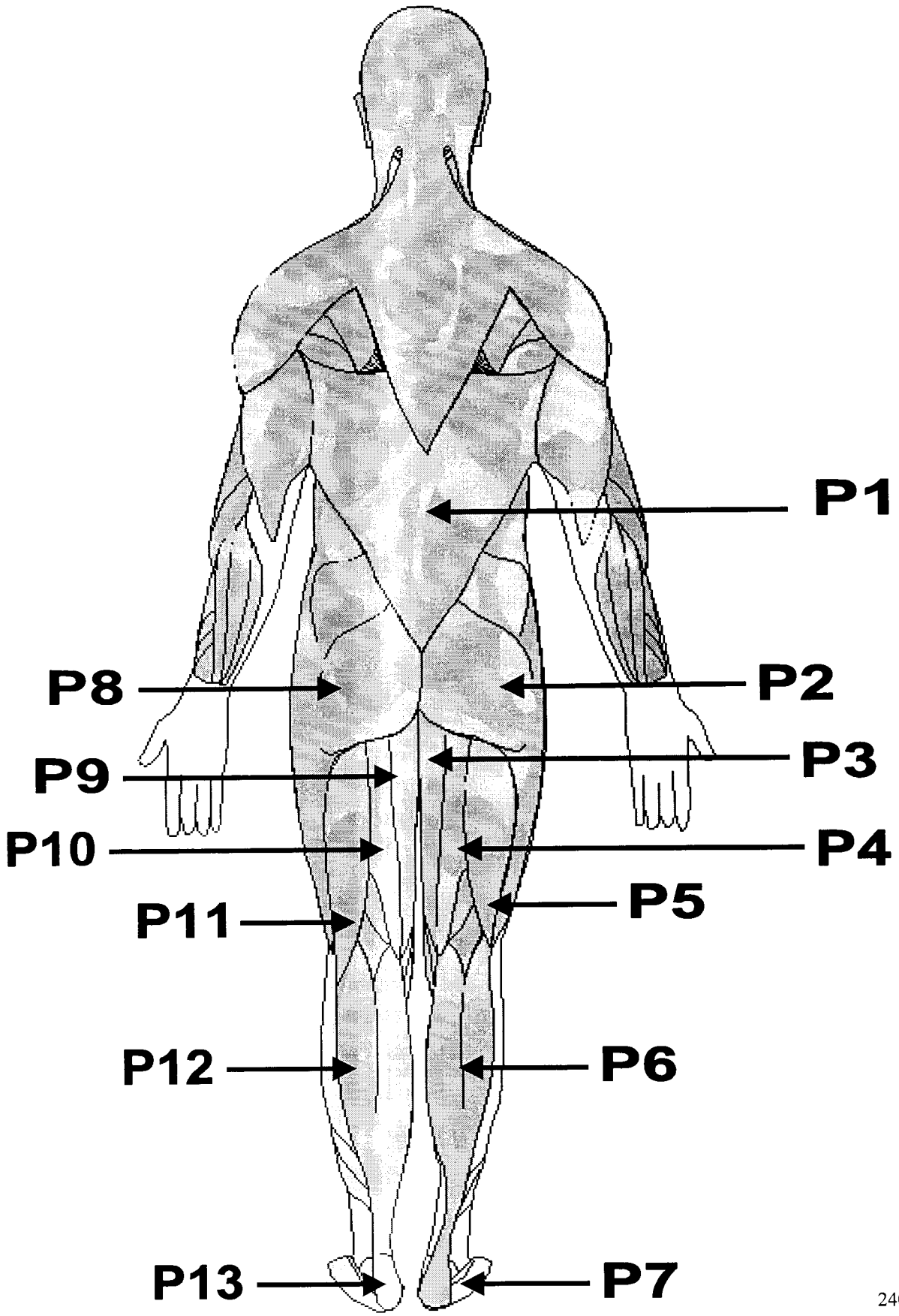
APPENDIX I

Perceived muscle soreness: Anterior view



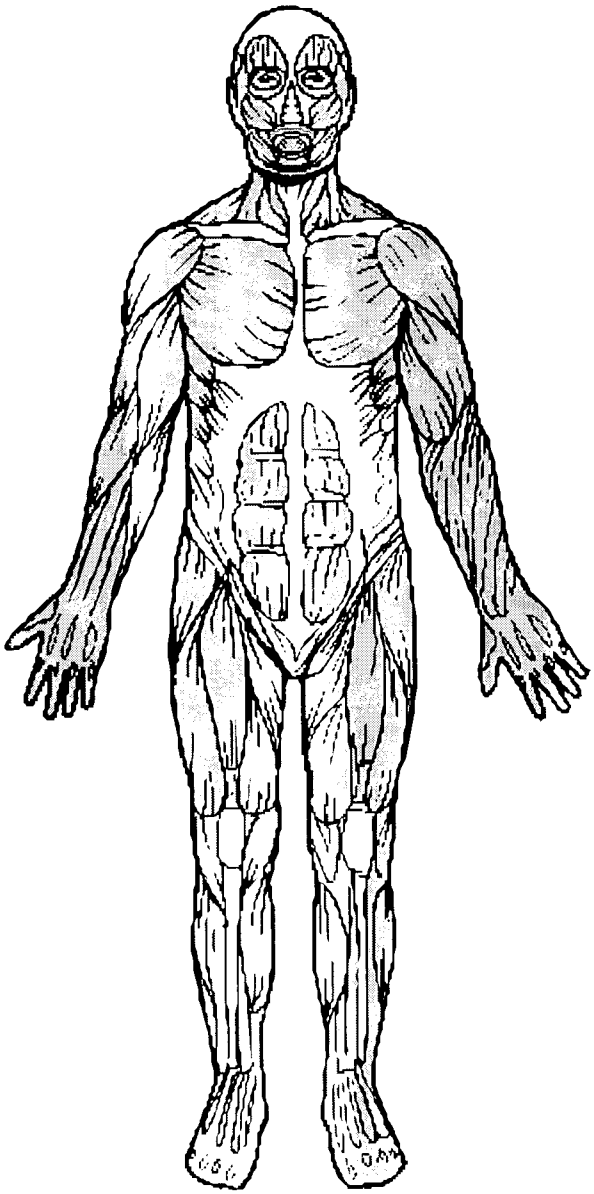
APPENDIX J

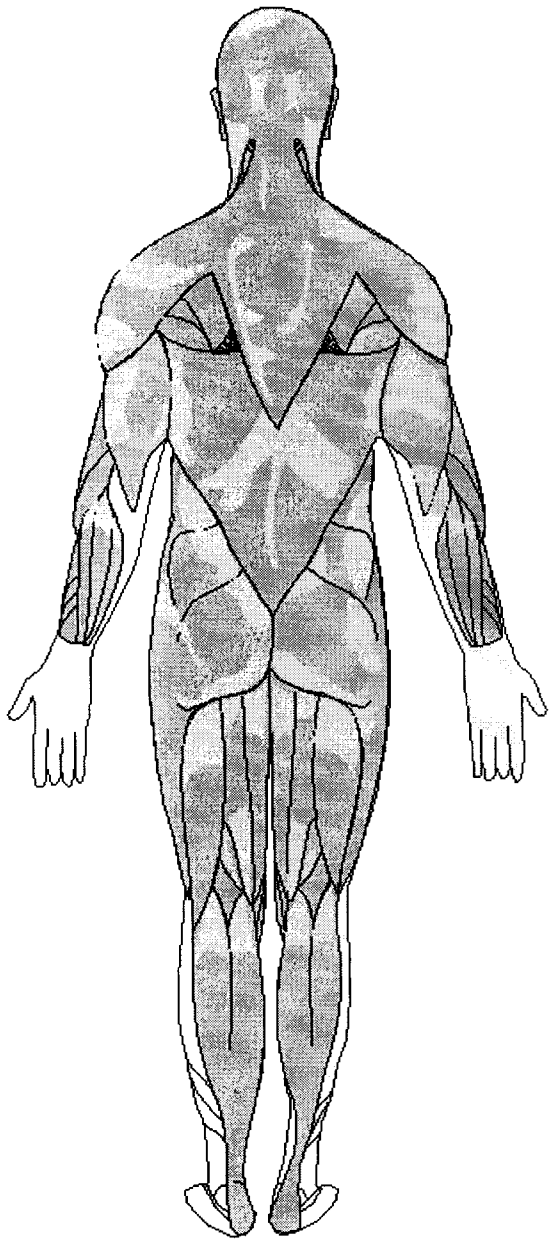
Perceived muscle soreness: Posterior view



APPENDIX K

Perceived muscle soreness





Subject name: _____ **Visit number:** _____

Date: _____ **Time:** _____

APPENDIX L

15 MINUTE SUBMAXIMAL RECOVERY RUNS

Name: _____ Run no: _____

Code: _____ Date: _____

Treadmill speed: _____ (km/h)

Time (min)	Heart rate (bpm)	Perceived exertion	Stride frequency	Stride length
Immediately	■		■	■
1		■	■	■
2		■	■	■
3			■	■
4		■		
5		■	■	■
6			■	■
7		■	■	■
8		■	■	■
9				
10		■	■	■
11		■	■	■
12			■	■
13		■	■	■
14		■		
15		(14.5)	■	■

Time (min)	V_i	VO_2	VCO_2	RER
3				
6				
9				
12				
14.5				

APPENDIX M

RATE OF PERCEIVED EXERTION

6	
7	VERY, VERY LIGHT
8	
9	VERY LIGHT
10	
11	FAIRLY LIGHT
12	
13	SOMEWHAT HARD
14	
15	HARD
16	
17	VERY HARD
18	
19	VERY, VERY HARD
20	MAXIMAL EXERTION

APPENDIX

30–km Time Trial

Name: _____ Code: _____

Treadmill speed: _____ (km/h) Date: _____

Distance (km)	Treadmill	
0-6	Level	
7-8	Downhill	
9-14	Level	
15-16	Downhill	
17-22	Level	
23-24	Downhill	
25-30	Level	

STOP	TIME
10km	
20km	

Distance (km)	PE	SF	SL	Time @ km
2				
4				
6				
8				
10				
12				
14				
16				
18				
20				
22				
24				
26				
28				
30				

Body mass Before:_____ (kg) After:_____ (kg)

Glucose Time taken for post sample: _____ (sec)

Food & Fluid (Type)	Amount

APPENDIX C

OBJECTIVE MUSCLE SORENESS DATA SHEET:
RECTUS FEMORIS / VASTUS MEDIALIS

Name: _____ **Code:** _____

TESTING	Rectus femoris 1	2	3	4	5	6	7	8	9	Total
1										
2										
3										
4										
5										
6										
7										
8										
9										
10										
11										
12										
13										
14										
15										
16										
17										
18										
19										
20										
21										
22										
23										
24										

APPENDIX P

PERCEIVED MUSCLE SORENESS DATA SHEET

Name: _____ **Code:** _____

TESTING	Perceived Muscle Soreness Rating Scale
1	
2	
3	
4	
5	
6	
7	
8	
9	
10	
11	
12	
13	
14	
15	
16	
17	
18	
19	
20	
21	
22	
23	
24	

APPENDIX Q

MUSCLE SORENESS DATA SHEET:

DIAGRAM (ANTERIOR / POSTERIOR VIEW)

Name: _____ **Code:** _____

VISIT	A1	A2	A3	A4	A5	A6	A7	A8	A9	A10	A11	A12	A13	Tot.
1														
2														
3														
4														
5														
6														
7														
8														
9														
10														
11														
12														
13														
14														
15														
16														
17														
18														
19														
20														
21														
22														
23														
24														

'n Pragtige deel is vir my afgemeet,
ja, wat ek ontvang het,
is vir my mooi.

(Psalm 16:6)