

The effect of high intensity training on the angle-torque relationship of the quadriceps and hamstring muscles in a group of well-trained cyclists.

by

**Student: Christel Rösemann
Student number: RSMCHR001**

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**Supervisor : Professor Mike Lambert, Department of Human Biology,
University of Cape Town**

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**The effect of high intensity training on the angle-torque
relationship of the quadriceps and hamstring muscles
in a group of well-trained cyclists.**

**A dissertation prepared by Christel Rösemann (RSMCHR001) in partial
fulfilment of the requirements for the Master of Philosophy degree in
Sports Physiotherapy (MPhil Sports Physiotherapy) from the University of
Cape Town**

March 2008

Declaration

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Table of contents

| | Page |
|---|------|
| Declaration | 2 |
| Acknowledgements | 3 |
| Table of contents | 4 |
| List of tables | 5 |
| List of figures | 6 |
| List of abbreviations | 7 |
| Abstract | 8 |
| Chapter 1: Introduction | 10 |
| Chapter 2: Current concepts of muscle mechanical mechanisms and muscle adaptation in response to exercise. | 13 |
| Chapter 3: The effect of high intensity training on the angle-torque relationship of the quadriceps and hamstring muscles in a group of well-trained cyclists. | 47 |
| Chapter 4: Summary and conclusion | 64 |
| References | 69 |
| Appendices | 75 |

List of tables

| | Page |
|---|-------------|
| Chapter 3: The effect of high intensity training on the angle-torque relationship of the quadriceps and hamstring muscles in a group of well-trained cyclists. | |
| Table 3.1: General descriptive data and cycling experience of control and trained subjects. | 56 |
| Table 3.2: Measurements associated with cycling performance in control and trained groups, before and after 8 high intensity training sessions. | 57 |
| Table 3.3: Changes in power output and 40 km TT performance expressed as a % (pre/post). | 57 |
| Table 3.4: Peak torque in control and trained groups before and after 8 high intensity training sessions. | 59 |
| Table 3.5: Angle coinciding with peak torque in control and trained groups before and after 8 high intensity training sessions. | 59 |
| Appendix H: Calculated data vs. Biodex® data | |
| Table H1: Peak torque for flexion and extension and angle coinciding with peak torque for flexion and extension respectively. | 90 |

List of figures

| | Page |
|--|------|
| Chapter 3: The effect of high intensity training on the angle-torque relationship of the quadriceps and hamstring muscles in a group of well-trained cyclists. | |
| Figure 3.1: Changes in power output (PPO) and 40 km time trial (40 km TT) performance expressed as a % (pre/post). | 58 |
| Appendix G: Examples of graphs representing the angle-torque data for a single subject | |
| Figure G1: Angle-torque graph representing 7 contractions for flexion and extension respectively. | 89 |
| Figure G2: Fourth order polynomial nonlinear regression curve fit, representing an average peak torque value for the set of flexion and extension movements respectively. | 89 |
| Figure G3: Differentiated curve on an XY Plot. | 89 |
| Appendix H: Calculated data vs. Biodex® data | |
| Figure H1: Peak torque for flexion and extension and angle coinciding with peak torque for flexion and extension respectively. | 91 |

List of abbreviations

| | | | |
|------|--------------------------------|---------------------|-------------------------|
| Ext | extension | PT | peak torque |
| Flex | flexion | RM | repetition maximum |
| HIT | high intensity training | rpm | repetitions per minute |
| HR | heart rate | SD | standard deviation |
| H | hour | s | seconds |
| iEMG | integrated electromyography | TT | time trial |
| kg | kilograms | VO ₂ | oxygen consumption |
| km | kilometers | VO _{2peak} | peak oxygen consumption |
| l | litres | W | watts |
| lbs | pounds | W _{peak} | peak workload |
| m | meters | yr | years |
| min | minutes | | |
| ml | milliliters | | |
| PO | power output | | |
| PPO | peak power output | | |

Abstract

Background: Data are available on the physiological adaptations that occur following high intensity training in the well-trained athlete. However, the available literature appears to limit discussion to the parameters that reflect a muscle's ability to adapt to training as a result of metabolic mechanisms or changes in muscle architecture. The mechanisms that have been identified to be involved in the physiological adaptations following high intensity training do not fully account for performance enhancements. The angle-specific behaviour for peak torque generation in skeletal muscle is related to the mechanical force-length properties of the muscle tissue. Therefore, measuring training related changes in the angle-torque relationship of a muscle in response to high intensity training has the potential to add to the current understanding of muscle mechanical mechanisms.

Objective: To establish if changes in cycling performance after high intensity training are associated with changes in the angle-torque relationship of the quadriceps and hamstring muscles in a group of well trained cyclists.

Methods: Fourteen well trained male cyclists (mean \pm SD; age = 31.7 ± 4.8 yr; $VO_{2peak} = 55.9 \pm 5.0$ ml.kg⁻¹.min⁻¹, $W_{peak} = 359 \pm 35$ W) with a competitive cycling experience of 12.4 ± 9.4 years volunteered for the study.

All cyclists were randomly assigned to either the high intensity training or the control group. Training for the high intensity group consisted of 8 sessions conducted within 28 days. Each session consisted of cycling for 4 min at approximately 80% of their peak power. This 4 min bout was repeated on 8 occasions during each training session with a 90 s rest periods between repetitions. The control group performed a self-paced training ride consisting of a 40 km simulated undulating route performed at below 70% of their peak power. Each subject completed a 40 km familiarization time trial (TT), a VO_{2peak} and a repeat 40 km TT test before the start of the training and again after the 8 training sessions.

Isokinetic torque testing was used to measure training related changes in the angle-torque relationship, and was performed after the pre- and the post-training VO_{2peak} tests. The isokinetic testing protocol consisted of 7 successive maximum voluntary concentric knee extension and flexion movements of the dominant leg, and was performed on a Biodex® dynamometer at a speed of $60^{\circ} \cdot s^{-1}$. Each contraction was performed over a 90° knee angle range, starting with the lower leg hanging in a vertical position at 90° .

Results: There was a significant improvement in the trained group in absolute peak power output ($4.8 \pm 3.4\%$), relative peak power output ($5.5 \pm 3.6\%$) and 40 km TT performance ($2.5 \pm 1.3\%$) after high intensity training. VO_{2peak} did not change. Peak torque values did not change for either flexion or extension following the 4 weeks of high intensity training. There were no significant differences in the angle-torque relationship for flexion or extension, either between groups or before and after high intensity training.

Conclusion: Following high intensity training in well-trained cyclists, muscles improve their ability to generate peak power for athletic performance. However, improvements in absolute and relative peak power output and 40 km TT performance are not associated with a shift of the optimum angle for peak torque production. It follows that muscle adaptational mechanisms other than, or in addition to, changes in muscle mechanical properties need to be explored to be able to explain improvements in peak power output and 40 km time trial performance following high intensity training in already well trained cyclists.

Key words: angle-torque relationship, high intensity training, cycling, quadriceps, hamstrings

Chapter 1

Introduction

It is well known that muscle adaptations, resulting from regular exposure to exercise training, involve metabolic, mechanical and neural mechanisms. These adaptations result in changes in muscle function and morphology. For example, Goto et al. (2004) have shown that resistance training induces muscle hypertrophy (as reflected by changes in muscle cross-sectional area) and improvements in muscular strength, power and endurance. Muscle mitochondrial content and muscle oxidative capacity increase after endurance training, resulting in muscle fibres functioning more aerobically and relying less on anaerobic energy supply. As a result, fewer additional muscle fibres are recruited over time to replace the fatigued fibres, therefore prolonging the time to fatigue and decreasing end-exercise active muscle and VO_2 (Saunders et al., 2003).

Data are available on muscle metabolism and the physiological adaptations occurring in skeletal muscle tissue following high intensity training in well trained athletes. A recent study by Laursen et al. (2005) showed that improvements in VO_{2peak} and 40 km time trial performance (40 km TT), following high-intensity interval training in well trained cyclists, are related to peripheral aerobic variables such as ventilatory thresholds and VO_{2peak} , as well as anaerobic related variables, such as anaerobic capacity and lactate buffering ability. Furthermore, the higher relative work rates in 40 km TT performance in already well-trained cyclists, following a period of high intensity training (HIT), do not appear to be related to rates of carbohydrate oxidation (Westgarth-Taylor et al. 1997).

The limited research that has examined changes in muscle enzyme activity in highly trained athletes, following the exposure to HIT, has revealed no change in oxidative or glycolytic enzyme activity, despite significant improvements in endurance performance; but instead, increased skeletal muscle buffering capacity has been proposed as a mechanism involved in improved endurance performance (Laursen and Jenkins, 2002). In addition to studies discussing the metabolic adaptations following high intensity training, Chapman et al. (2007) provide recent data that demonstrate a relationship between neuromuscular adaptations and performance improvements after HIT.

Muscle architecture adaptations occur rapidly in response to training programs imposed on athletes, but the adaptations are training-specific and differ between high-velocity and resistance training. Furthermore, these adaptations do not appear to account for performance differences between high velocity training and resistance training, suggesting that factors other than, or in addition to, muscle size and architecture are responsible for mediating the performance changes (Blazevich et al., 2003).

However, it is unlikely that the adaptations described above account for all the mechanisms involved in performance enhancements of the highly trained athlete, following high intensity training. It follows, that different mechanisms, likely to contribute to these adaptations, need to be investigated.

Contractile element damage plays an important role in the changes of muscle mechanical properties in response to exercise, particularly exercise involving predominantly eccentric actions, as described by Proske and Morgan (2001). The presence of disrupted sarcomeres in myofibrils and damage to the excitation-contraction coupling system, appear to be two prominent signs of muscle cell damage, following the exposure to eccentric exercise. In the regions where the myofibrils are unable to resist the active lengthening, sarcomeres are stretched beyond myofilament overlap and some become disrupted, leading to an increase in the series compliance of the muscle. Furthermore, muscle fibres with disrupted sarcomeres in series with still-functioning sarcomeres show a shift in optimum length for tension in the direction of longer muscle lengths (Proske and Morgan, 2001). It has long been known that an optimum muscle length is required for force production and that muscle power output depends, in part, on the length-tension properties of skeletal muscle tissue (Josephson, 1999). Angle-specific torque production behaviour, as a function of the mechanical properties of skeletal muscle tissue, has been observed in several studies, which demonstrate a measurable effect on the length-tension relationship following maximal torque production during various types of voluntary exercise stimuli (Folland et al., 2000; Pincivero et al., 2004; Butterfield and Herzog, 2005; Prasartwuth, 2006; Whitehead et al., 1998).

Being the first researchers to have demonstrated a shift of the *optimal angle* for maximal torque production towards longer muscle lengths in human hamstring muscle,

Brockett et al. (2001) provide valuable insight into human muscle mechanical behaviour in response to an eccentric training stimulus. Such behaviour indicates that athletes exposed to exercise consisting of predominantly eccentric muscle actions, if not protected by training, may be exposed to an increased risk of injury.

Therefore, measuring training related changes in the angle-torque relationship may provide insight into muscle mechanical behaviour in response to exercise training, and may therefore provide a means to measure a muscle's ability to adapt to a specific strength training stimulus, or conversely, - to measure its susceptibility to muscular strain injury. This information could be used as a guide in the design of adequate training programs and as an objective and reliable musculoskeletal screening tool in the athletic population. Furthermore, understanding the specificity of training adaptation may also provide therapeutic targets for the treatment of acute and chronic skeletal muscle injury.

Our understanding of muscle mechanical mechanisms in response to high intensity training is limited, and scientific data to improve our current understanding of muscle mechanical adaptation in response to high intensity training is lacking. To the author's knowledge, there is no published literature on the effect of high intensity training on muscle mechanical mechanisms and adaptation. In particular there are no data to show whether high intensity training causes changes in the angle-torque relationship represented by shifts in the optimum angle for force production.

This thesis reviews the existing knowledge of skeletal muscle properties and muscle metabolic, neuromuscular and mechanical mechanisms, involved in muscle adaptation in response to exercise training, specifically high intensity training. The experimental phase of the thesis examines the effect of four weeks of high intensity training on the angle-torque relationship of the quadriceps and hamstring muscles in a group of well-trained cyclists. This study is designed to test the hypothesis that a shift of the optimal angle for maximum torque is partially associated with an improvement in performance after high intensity training.

Chapter 2

Literature Review:

Current concepts of muscle mechanical mechanisms and muscle adaptation in response to exercise

| Contents | Page |
|--|-------------|
| 2.1 Properties of skeletal muscle – An overview..... | 15 |
| 2.1.1 Determinants of force and work output..... | 15 |
| 2.1.2 Series elasticity..... | 16 |
| 2.1.3 Length-velocity-force characteristics..... | 17 |
| 2.2 Sarcomere behaviour, muscle fibre stability, sarcomerogenesis, length-tension characteristics and angle-torque relationship | 18 |
| 2.2.1 Sarcomere behaviour and muscle fibre stability..... | 18 |
| 2.2.2 Sarcomerogenesis..... | 19 |
| 2.2.3 Length-tension characteristics and angle-torque relationship..... | 21 |
| 2.2.4 Muscle geometry, length-tension characteristics and angle-torque relationship..... | 22 |
| 2.3 Contractile element damage in response to eccentric exercise, and changes in the length-tension characteristics and angle-torque relationship..... | 23 |
| 2.3.1 Skeletal muscle fibre damage..... | 23 |
| 2.3.2 Fibre type..... | 24 |
| 2.3.3 Shifts in optimum muscle length..... | 25 |
| 2.3.4 Changes in the length-tension relationship and associated factors..... | 26 |
| 2.4 Mechanisms involved in skeletal muscle adaptation in response to exercise training..... | 27 |
| 2.4.1 Mechanisms involved in performance improvements following exercise training..... | 28 |
| 2.4.1.1 Oxidative and glycolytic enzyme adaptation..... | 28 |
| 2.4.1.2 Glucose kinetics..... | 30 |
| 2.4.1.3 Capillary density..... | 31 |
| 2.4.1.4 Sarcoplasmic reticulum adaptations..... | 31 |
| 2.4.1.5 Potassium kinetics..... | 31 |
| 2.4.1.6 Lactate kinetics..... | 32 |
| 2.4.1.7 Neuromuscular adaptations..... | 32 |
| 2.4.1.8 Muscular adaptations..... | 33 |

| | |
|--|----|
| 2.4.2 Mechanisms involved in skeletal muscle adaptation in response to exercise training in endurance cyclists..... | 37 |
| 2.4.2.1 High intensity training, performance changes and possible adaptations..... | 38 |
| 2.4.3 Mechanisms involved in altered muscle mechanical properties and changes in the angle-torque relationship, following exposure to exercise training..... | 40 |
| 2.4.3.1 Skeletal muscle adaptation in response to exercise..... | 40 |
| 2.4.3.2 Skeletal muscle adaptation in response to exercise in the elderly..... | 40 |
| 2.4.3.3 Tendon stiffness..... | 41 |
| 2.4.3.4 Shifts in the length tension relationship as an indicator of muscular compliancy..... | 42 |
| 2.4.3.5 Passive tension..... | 43 |
| 2.4.3.6 Shift in optimum angle..... | 43 |
| 2.5 Conclusion..... | 45 |

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2. 1 Properties of skeletal muscle – An overview

A general statement on the function of muscle is that it has to contract to produce force. This means that the muscle has to deliver work externally by exerting force while changing length. The work that the muscle does as it contracts is determined by the properties that are related to the muscle material and architectural properties. According to Huijing (1998), muscular properties can be classified into contextual and intrinsic properties. Contextual properties are determined by the structure of the muscle, which determines the specific function of a particular muscle. For example, a particular muscle can either function as a joint flexor or extensor, and as a mono- or a bi-articular muscle. Intrinsic properties of muscle determine the functional capabilities of muscle to deliver external work. These properties are defined at the molecular, subsarcomeric or sarcomere level of intracellular mechanisms of the muscle fibre (Huijing, 1998). The capability of active muscle to produce movement is mainly determined by the maximum (optimal) force that can be generated by a particular muscle, the muscle length at which it generates optimal force (optimum muscle length), and the total muscle length range over which active force is generated (Huijing, 2005).

2.1.1 Determinants of force and work output

Josephson (1999) has described the determinants of muscle force during a contraction as primary and secondary determinants of force generation and work output. The primary determinants are the instantaneous muscle length (the length-tension relationship), the shortening or lengthening velocity (the force-velocity relationship) and the degree of muscle activation, which reflects when and in what pattern the muscle is stimulated. The secondary determinants are length-dependent changes in the kinetics of muscle activation and shortening deactivation, arising from interactions between the primary determinants of muscle force throughout a shortening-lengthening cycle (i.e. the effects of muscle length on the time course of muscle activation and deactivation associated with muscle shortening and work production). Furthermore, the force generated by the interaction of the cross-bridges (contractile components) is transmitted to the load through the myofilaments, Z-line and tendons or aponeuroses, all of which are partially compliant (collectively described as the series elastic component), and whose properties can affect the length and the velocity of the

contractile elements and therefore the work output. The presence of the series elasticity allows some internal sliding of filaments and shortening of sarcomeres when the muscle is held at a constant length in an isometric contraction (Josephson, 1999).

When studying muscle function, it is important to have an understanding of the behaviour of the muscle fascicles, as muscle performance is influenced by the type of activity performed. For example, force may decrease as a function of the shortening velocity during concentric muscle contractions, while force produced during eccentric actions may be stronger than the maximum isometric force. Furthermore, force production may be improved if a concentric contraction follows an eccentric action (Hoyt et al., 2005).

2.1.2 Series elasticity

To be able to understand human movement in terms of muscle function and adaptation, it is essential to understand the contributions of intrinsic properties of muscle at the level of the muscle as well as the muscle-tendon unit (Huijing, 1998). Length changes of the series elastic component correspond with length changes of the contractile elements. However, the change in muscle fascicle length may differ from the length change of the overall muscle-tendon unit, because of the compliance of the series elasticity. According to Josephson (1999), the length change and velocity of the contractile components may be greater than, or less than that of the muscle as a whole, depending on whether force is increasing and the series elastic component is becoming stretched, or force is decreasing and the series elastic component is shortening.

In addition to serving to attach muscles to bones, the series elastic components (tendons and aponeuroses) serve two other important functions: Firstly, elastic storage of energy and secondly, mechanical buffering (reducing eccentric activity by protecting against potentially damaging rapid stretch of muscle fibres to long lengths). Consequently, fascicles of joint extensor muscles may not undergo eccentric activity when the joint flexes. An equine study by Hoyt et al. (2005) investigated the relationship between length changes of the muscle-tendon unit (as estimated from the kinematics) and length changes in the muscle fascicles, and demonstrated that different joint extensor muscles in the same species exhibit similar activity, despite differences in

muscle architecture and kinematic patterns of the associated joints. However, the extent to which this occurs may vary with the compliance of the series elasticity, and may be different between species (Hoyt et al., 2005).

2.1.3 Length-velocity-force characteristics

The force generated by skeletal muscle varies with sarcomere length and velocity (Burkholder and Lieber, 2001), following which, it appears obvious that limitations of joint excursion, resulting from muscular deficiency or injury, can be caused by consequences of faulty regulation of either the optimum length or the length range of active force exertion. An understanding of the sarcomere length changes, which occur during movement, therefore provides insight into the physiological importance of the relationship between active force generation and optimum muscle length.

Length effects, velocity effects and the effects of degrees of activation and recruitment should not be considered as independent determinants of muscle functioning (Huijing, 1998). Furthermore, length-velocity-force characteristics should not be considered as unique properties of a muscle, as these characteristics are strongly influenced by the actual effects of recruitment, firing frequency, amount of shortening performed, velocity of shortening, as well as the short time history of these factors (Huijing, 1998). In addition, differences in muscle morphology, particularly those related to fibre mean sarcomere length, play an important role, as they affect the length-force characteristics of active muscle. Physiological individuality causes considerable variation between individuals (Huijing, 1998); however, the above properties have to be considered when interpreting joint angle-sarcomere length data.

Although studies are required to determine the exact nature and extent of the design principles that determine sarcomere length operating range, the musculoskeletal system seems to tailor itself to a particular functional task. For example, when power generation is required, systems are in place to maximize power generation; however, when joint stability or control is required, the musculoskeletal system rearranges itself to provide this stability even if it is at the cost of less power (Burkholder and Lieber, 2001).

2. 2 Sarcomere behaviour, muscle fibre stability, sarcomerogenesis, length-tension characteristics and angle-torque relationship

2.2.1 Sarcomere behaviour and muscle fibre stability

Optimal sarcomere length is defined as the length at which maximum tetanic tension is generated. Sarcomere number is highly plastic and tightly regulated; therefore sarcomere length remains very consistent in a given muscle among similar-sized individuals of the same species (Burkholder and Lieber, 2001). Since a muscle fibre contains a large number of sarcomeres in series, a number of potential problems arise which are related to sarcomere behaviour within a fibre, one of these problems being stability. Muscle fibre stability may be an inherent property of the active force producing process within a sarcomere; however, the exact regulation of stability has yet to be identified (Allinger et al., 1996). Allinger et al. (1999) have found that the mechanical conditions required for sarcomere and fibre stability could be associated with known properties of skeletal muscle fibres. They have demonstrated that a fibre consisting of a large number of sarcomeres can be stable, but still exhibit an apparently unstable (negatively sloped) descending limb of the length-tension curve. These results indicate that the active force producing process of skeletal muscle provides for stability over the entire range of sarcomeres (Allinger et al., 1999).

The term length-tension relationship represents the tension producing potential of a fibre or a sarcomere under isometric conditions (i.e. constant length of a fibre or constant length of a group of sarcomeres). According to Allinger et al. (1996), when measuring force-length properties of a muscle fibre, a decreasing force with increasing sarcomere lengths is observed for the 'descending limb' of the length-tension curve. Furthermore, this behaviour has been associated with instability of sarcomere lengths, where instability refers to a muscle fibre "pulling itself apart". The length-tension curve has been described by Allinger et al. (1996) to consist of four regions: an ascending limb, a plateau, a descending limb, and a passive limb. On the descending limb, any non-uniformities in the length of the sarcomeres along the fibre are associated with the short (strong) sarcomeres shortening at the expense of the long (weak) sarcomeres. This behaviour is believed to continue until all sarcomeres have achieved a stable force level (i.e. until the strong sarcomeres have shortened onto the active or 'ascending limb'

of the length-tension curve and the weak sarcomeres have stretched past the thin-thick myofilament overlap onto the 'passive limb' of the length-tension curve).

Instability of a muscle fibre over a large portion of its operating range would result in an unstable and suboptimal environment for muscle function, leading to problems in movement control, by:

- producing gross differences in sarcomere lengths along a fibre,
- limiting control of sarcomere extension to only the passive structures which are unable to regulate force production,
- an inability of the muscle fibres to regulate their lengths over extended contractions,
- large sarcomere length changes, which result in increased cross-bridge cycling (ATP splitting) and increased energy requirements, compared to a more stable behaviour,
- producing unstable angle-torque relations at the joints, caused by the compromised muscular output (Allinger et al., 1996).

2.2.2 Sarcomerogenesis

According to Proske and Morgan (2001), sarcomerogenesis serves as an adaptation to prevent operation on the 'plateau' and 'descending limb' of the sarcomere length-tension curve, thus preventing the onset of fibre instability, which they postulate to be the first stage in the process leading to damage from eccentric exercise. The addition of sarcomeres allows the sarcomeres of a given muscle-tendon unit to work at shorter lengths compared with the lengths preceding sarcomerogenesis, and therefore provides for a beneficial adaptation, which protects against fibre damage.

Because fibre lengths may vary within a muscle, directing sarcomerogenesis at the cellular level (the addition of sarcomeres in series within a fibre) would allow the maintenance of consistent sarcomere lengths between fibres. Increasing serial sarcomere number would be of benefit in a static contractile situation, and would improve muscle function by altering the length-tension relationship (Butterfield et al., 2005).

Lynn et al. (1998) showed that rats, which had been trained to walk downhill, acquired a greater serial sarcomere number in their quadriceps muscle (vastus intermedius) than rats which had been trained to walk uphill. These findings provide early evidence of the protective effect of eccentric exercise training against the changes that indicate muscle fibre damage, following initial exposure to eccentric activity (Proske and Morgan, 2001). Exercise predominated by eccentric muscle actions acts as a stimulus for sarcomerogenesis. However, the lengthening of active muscle at long lengths is distributed very non-uniformly, as the initial length changes in each myofibril result primarily from the sarcomeres that have become stretched to beyond myofilament overlap (Proske and Morgan, 2001). Because eccentric exercise facilitates sarcomerogenesis, resulting in a greater number of sarcomeres in series, one would assume that eccentric exercise training leads to greater optimum angles for peak torque production in any given muscle.

Similar to Lynn et al. (1998), Butterfield et al. (2005) also investigated the relationship between contraction type, exercise duration, and serial sarcomere number adaptation in the knee extensor muscles (vastus lateralis) of rats. The findings by Butterfield et al. (2005) indicate that differential muscular adaptations occurring after uphill and downhill walking are related to contraction type and fibre dynamics, which support the earlier results by Lynn et al. (1998). Muscle shortening during uphill concentric-biased exercise resulted in a loss of serial sarcomeres, and muscle lengthening during downhill eccentric-biased exercise resulted in a gain of serial sarcomeres. However, the lack of differences in serial sarcomere adaptation after 5 days of exercise training in the study by Butterfield et al. (2005) contradicts the earlier results by Lynn et al. (1998), which demonstrate significant differences in serial sarcomere numbers in the knee extensor muscles between the uphill and downhill trained rats that were exposed to exercise training of similar nature and over the same time period. Collectively, these studies indicate that differential adaptations occurring in knee extensor muscles after uphill and downhill walking in rats are related to contraction type. However, even though fibre dynamics of individual knee extensor muscles (i.e. vastus intermedius and vastus lateralis) are assumed to be similar, the sarcomere adaptations varied between the two extensor muscles used in the above studies. These differential adaptations may result from differences in individual muscle architecture and fibre lengths, which could influence the extent of the fibre strain or injury (Butterfield et al., 2005).

2.2.3 Length-tension characteristics and angle-torque relationship

Force output in skeletal muscle depends, in part, on the mechanical length-tension properties of skeletal muscle tissue (Pincivero et al., 2004). According to Pincivero et al. (2004), the generation of voluntary knee extensor torque is a function of knee angle, and peak values are attained within the midrange, at 70° knee flexion, of knee joint movement. Their observed variation in peak extensor torque at different knee joint angles was attributed to the difference in the force-generating capacity between the different portions of the quadriceps femoris (QF) muscle, which mainly exists due to angle-dependent changes in the muscle length. With the exception of the rectus femoris muscle, which is a bi-articular muscle, the vastii components of the QF muscle are at their shortest length during full knee extension, and force generating ability of the QF muscle therefore is lowest at full knee extension. Extending or flexing the knee and placing the QF muscle at a different length from its optimal length for peak torque generation therefore influences force output (Pincivero et al., 2004).

Angle-specific responses of knee extensor torque to maximal effort contractions have been observed in other studies which demonstrate a measurable effect on the length-tension relationship following various forms of exercise (Folland et al., 2001; Butterfield and Herzog, 2005; Prasartwuth et al., 2006; Whitehead et al., 1998).

The observed differences in the number of sarcomeres in series following exposure to eccentric contractions lead to changes in the length-tension relationship of a muscle (Lynn et al., 1998). Accordingly, the decrease in tension at constant length therefore does not reflect a real decrease in the tension-generating capability, but is partly due to a shift in the length-tension curve. It therefore becomes difficult to accurately measure a muscle's length and relate it to its physiological range. This problem can be overcome by leaving the muscle with its insertion and origin intact and instead measuring the torque generated as a function of the joint angle. The moment arm of the knee is relatively independent of joint angle; therefore the angle-torque curve should be very similar to a length-tension curve (Lynn et al., 1998). Consequently, measuring the shift in optimum muscle length for the production of peak active torque has the potential to be an accurate and reliable way to determine changes in the mechanical force-length properties of skeletal muscle tissue.

The measurement of the *shift* in optimum length for the production of peak active torque provides evidence for the first stage of the process that leads to histologically observable muscle damage (Proske and Morgan, 2001). The shift in optimum length has the advantage of being present immediately after the eccentric actions, and is mostly independent of fatigue (Morgan et al., 2004). As sarcomere length in humans is difficult to measure *in vivo*, a functional measure such as the joint angle at which peak torque occurs (termed optimum joint angle) is a useful measurement tool (Butterfield and Herzog, 2005). Assuming there is no change in the relationship between moment arm and joint angle, any change in optimum angle should reflect a change in the length-tension relationship of a muscle.

2.2.4 Muscle geometry, length-tension characteristics and angle-torque relationship

The relationship between joint angle, torque and pennation has been investigated in human brachialis muscle by Herbert and Gandevia (1995). The pennation angle of a muscle refers to the oblique alignment of the muscle fibres in relation to the long axis of the muscle, and assists in transmitting forces from muscles to tendons. Under physiological conditions, joint angle and joint torque have been observed to interact to produce large and non-linear increases in the pennation of the human brachialis muscle. The changes in pennation angle determine the magnitude of the changes in the fibre lengths (which accompany changes in muscle length), and therefore partly determine the force which the muscle fibres produce at any given muscle length or velocity. Collectively, the degree of pennation influences the size of the torque which a muscle can produce at a given joint.

Mademli and Arampatzis (2005) demonstrated a decrease in the fascicle length and an increase in the pennation angle of human gastrocnemius muscle following sustained and fatiguing submaximal isometric plantar flexions. These changes in muscle architecture have been shown to influence the length-tension characteristics of the muscle tendon unit, leading to an alteration in its contractile capacity.

2.3 Contractile element damage in response to eccentric exercise, and changes in the length-tension characteristics and torque-angle relationship

2.3.1 Skeletal muscle fibre damage

Exercise incorporating a predominance of eccentric actions is unique to other forms of exercise, in that it routinely produces skeletal muscle damage in untrained individuals (McHugh, 1999). The damage of the contractile elements, following exposure to unaccustomed eccentric exercise, plays an important role in the altered mechanical properties of a muscle (Proske and Morgan, 2001).

Skeletal muscle damage sustained after eccentric exercise is associated with characteristic signs, demonstrating amongst others, subjective discomfort 48 hours after the exercise, and a significant loss of strength, which is suggestive of structural damage of the muscle tissue (Folland et al., 2000).

Muscle fibres are vulnerable to mechanically induced stress, and disruptions of the muscle plasma membrane are an early form of structural damage to the muscle fibres of eccentrically exercised muscle (McNeil and Khakee, 1992). Proske and Morgan (2001) have reviewed the mechanisms and the mechanical signs of the described fibre damage, as well as the muscle's ability to rapidly adapt to the damage from eccentric exercise to prevent further damage. Accordingly, the presence of disrupted sarcomeres in myofibrils and damage to the excitation-contraction coupling system appear to be two prominent signs of muscle damage following eccentric exercise. In the regions where the myofibrils are unable to resist the active lengthening, sarcomeres are stretched beyond myofilament overlap and some become disrupted, leading to an increase in the series compliance.

Skeletal muscle damage sustained after eccentric exercise expresses itself by causing delayed onset of muscle soreness and changing the mechanical properties of muscle. Furthermore, fibre integrity is compromised when sarcomeres are actively lengthened beyond myofilament overlap, resulting in fibre necrosis and regeneration (Proske and

Morgan, 2001). While Proske and Morgan (2001) reviewed exercise-induced muscle damage with the focus mainly on the mechanical consequences of such exercise, Allen et al. (2005), in a more recent review, focused on the pathways which lead to the muscle damage. In particular, the role of ionic changes in triggering the damage such as changes in intracellular calcium, sodium and pH following eccentric actions, are discussed. It appears that changes in intracellular ions contribute to the reduced force production, increased protein breakdown and increased membrane permeability, but the pathways for damage are still poorly defined. However, there is evidence that Ca^{2+} entry into a muscle cell plays a role in the eccentric exercise-induced damage pathway in normal muscle, and that a muscle is capable of complete repair from such damage over a period of 1–2 weeks (Allen et al., 2005).

Morgan et al. (1996) excluded reduced myoplasmic Ca^{2+} concentration as the cause of the shift in optimum length, after exposing single muscle fibres to eccentric actions in a model involving frog muscle. It instead appears that the shift in optimum length and the associated mechanical changes are causing the disturbances of the intracellular Ca^{2+} dynamics in damaged muscle fibres (Morgan et al., 1996).

2.3.2 Fibre type

Exercise with a predominantly eccentric component causes muscle damage, which subsequently affects dynamic muscle function during intermittent maximal intensity exercise (Twist and Eston, 2005). Reduced peak power output, changes in fatigue resistance during maximal intensity cycling, and impaired sprint ability indicate that fast twitch fibres may be preferentially damaged during exercise with a high eccentric component. As a result the muscle becomes weaker, but appears less fatigable, suggesting a greater reliance on the lower threshold muscle fibres following muscle damaging exercise (Twist and Eston, 2005).

Previously, Jones et al. (1986) reported greater ultrastructural damage in fast twitch fibres, following eccentric exercise in human gastrocnemius muscle. However, it remains to be established whether this preferential involvement of the fast twitch fibres is due to different patterns of use during exercise, or due to a greater intrinsic susceptibility of these fibres to damage.

2.3.3 Shifts in optimum muscle length

A shift in a muscle's length-tension relationship, resulting from exposure to eccentric exercise, was for the first time described in human muscle by Brockett et al., (2001). They observed a shift in the direction of longer muscle lengths for the production of active peak force. This shift reflects a rightward shift of the '*peak torque - joint angle*' relationship.

Susceptibility for muscle damage from eccentric actions has shown to be independent of force levels and fatigue, provided that activation is sufficient to produce a descending limb on the length-tension curve. Morgan et al. (2004) used feline gastrocnemius muscle to study differences in the shift in optimum muscle length for peak torque production following eccentric and concentric muscle actions. They showed that the shift in optimum length is unique to muscle damage associated with eccentric exercise, and only a small number of eccentric actions are sufficient to cause such a shift. Concentric contractions which produced a similar decrease in active tension were not accompanied by a shift in optimum length (Morgan et al., 2004).

The consequences of a shift in optimum length for peak torque production after eccentric exercise have to be considered, when one wants to measure peak torque accurately. Once the fibre length has adjusted to a new, longer optimum length, the disruption of sarcomeres lying in series with still functioning sarcomeres, by itself, would not be expected to cause any decreases in active force production. The exact mechanisms underlying the decrease in active force production remain unclear, but factors such as fatigue due to metabolic exhaustion, structural damage to the contractile filaments, and failure of the excitation-contraction coupling process may be involved (Proske and Morgan, 2001).

Exposure to the effects of eccentric actions which cause muscle damage provides protection from future bouts of eccentric exercise. This phenomenon is referred to as the repeated-bout effect (McHugh et al., 1999). This beneficial training effect has not been found to exist in concentric contractions, or in muscles exposed to a training regimen of concentric exercise prior to a single eccentric exposure which causes injury (Whitehead et al., 1998; Gleeson et al., 2003). Concentrically-trained human calf

muscles showed larger damage from eccentric exercise than eccentrically-trained calf muscles after exposure to a bout of exercise which consisted predominantly of eccentric actions (Whitehead et al., 1998). Gleeson et al. (2003) demonstrated similar findings in an upper limb exercise model using the elbow flexor muscles.

2.3.4 Changes in the length-tension relationship and associated factors

Whitehead et al. (2003) investigated the effects of eccentric muscle actions, performed over different muscle lengths, on the active and passive length-tension relationship in mammalian muscle. In this study, a larger shift of the optimum length for active tension as well as a larger decrease in peak isometric contraction occurred at longer muscle lengths compared to shorter muscle lengths. Paschalis et al. (2005) extended the above study to a human exercise model, and investigated if the rectus femoris muscle in a group of healthy males is affected differently at different muscle lengths following eccentric exercise. Their results show that eccentric exercise causes greater muscle damage and peak torque declines at shorter muscle lengths (Paschalis et al., 2005), and are therefore in contrast to the earlier findings of Whitehead et al. (2003). There is no forthcoming explanation for these differences.

Lynn et al (1998) proposed that during recovery from eccentric exercise, extra sarcomeres are added to the myofibrils to increase optimal muscle length and protect against further eccentric damage. Two shifts in optimal length have been proposed:

- an acute shift due to disrupted sarcomeres,
- and a longer-term shift reflecting the longitudinal addition of sarcomeres.

The occurrence of changing sarcomere numbers raises the question of whether the tendon length is changing at the same time, to partially compensate for differences in the number of sarcomeres. If the tendon length did not change, then tension generation at short muscle lengths (i.e. towards full knee extension) would be compromised after eccentric exercise training. Lynn et al., (1998) suggest that some tendon adaptation does occur; however the magnitude of the change remains to be determined.

According to Proske and Morgan (2001), muscle fibres with disrupted sarcomeres in series with still-functioning sarcomeres show a shift in optimum length for tension generation in the direction of longer muscle lengths, thereby changing their mechanical

behaviour, whereas structurally, non-uniform sarcomere length distribution will be observed. The amount of evidence supporting the sarcomere non-uniformity hypothesis has grown considerably, and is able to explain certain behaviour of skeletal muscle and the observed differences in the effects from concentric and eccentric exercise (Proske and Morgan, 2001).

Both central and peripheral factors limit muscle performance after exercise-induced muscle damage. Muscle damage reduces maximal voluntary torque following eccentric exercise, but impaired voluntary activation and neural drive to the muscle also contribute to the early force loss after eccentric exercise, independent of the muscle soreness (Prasartwuth et al., 2005). Prasartwuth et al. (2006) showed that eccentric exercise causes a short-term shift in the optimal angle for maximum voluntary contractions, and produces a clear muscle length-dependent impairment in voluntary activation. The optimal angle shifts to longer lengths for maximum voluntary contractions, and voluntary activation is impaired, particularly at short muscle lengths. Furthermore, in their study, optimal angle recovered to pre-exercise values within one week, while both maximal voluntary torque and twitch evoked torque were not fully recovered, and voluntary activation at the shortest test length was still impaired after one week (Prasartwuth et al., 2006).

2. 4 Mechanisms involved in skeletal muscle adaptation in response to exercise training

Skeletal muscle is a dynamic, plastic tissue that is able to adapt in many different ways to an exercise stimulus, and various training approaches are used to facilitate muscle adaptation.

“Training programs are used to improve the strength and/or endurance capacities of athletes. Specific stimuli are either high tension in contracting myofibrils or high turnover rates in the oxidative metabolism of the muscle cell. Structural adaptations consist of the synthesis of additional myofibrillar material or increased volume density of both interfibrillar and subsarcolemmal mitochondria. Functional adaptations consist of higher activities of important enzymes in either anaerobic or aerobic energy metabolism and of larger intracellular stores of the respective substrates. All adaptations are highly specific

and reversible through detraining or immobilization of the muscle. Transformation of fibre types at the level of the molecular structure of myosin seems possible with high-intensity training of long duration" (Howald, 1985, p 365).

2.4.1 Mechanisms involved in performance improvements following exercise training

Adaptations of muscle to exercise training can be separated into morphological, metabolic and neuromuscular changes. Changes in muscle contractile characteristics observed in response to sprint training include changes in the speed of muscle contraction and muscle strength. These changes are related to morphological adaptations, which are determined largely by muscle fibre type, sarcoplasmic reticulum adaptation and muscle cross-sectional area (Ross and Leveritt, 2001). Metabolic adaptations in a muscle are associated with enhancing the ability of the muscle to produce energy. According to Ross and Leveritt (2001), this is achieved by one or more, of three different mechanisms. Firstly, key regulatory enzymes can increase their activity, thereby increasing the rate of energy production. Secondly, increased muscle stores of substrate amount can increase total energy production. Finally, the muscle can increase its intramuscular buffering capacity to prevent the accumulation of certain metabolites associated with fatigue.

Adaptations to exercise training include, but are not limited to, oxidative and glycolytic enzyme adaptation (Korzeniewski and Zoladz, 2003), adaptations in glucose kinetics (Bergman et al., 1999a) and capillary density (Jensen et al., 2004), sarcoplasmic reticulum adaptations (Holloway et al., 2005), adaptations in potassium kinetics (Nielsen et al., 2003) and lactate kinetics (Bergman et al., 1999b), neuromuscular adaptations (Häkkinen et al., 2003) and muscular adaptations (for example, Putman et al., 2004; Blazevich et al., 2003; Cameron Smith, 2002; Crameri et al., 2004).

2.4.1.1 Oxidative and glycolytic enzyme adaptation

The mechanisms facilitating the adaptation of oxidative phosphorylation in skeletal muscles in response to physical exercise are not fully understood. Korzeniewski and Zoladz (2003) have analysed existing experimental results, and conclude that an

increased amount of mitochondrial proteins together with an increased parallel activation of ATP demand and supply during exercise (i.e. increased direct stimulation of oxidative phosphorylation complexes accompanying the stimulation of ATP consumption) are partially responsible for the training-related acceleration in the oxygen-uptake kinetics at the onset of exercise. Furthermore, these adaptations are believed to be responsible for the training-related improvement of ATP/ADP homeostasis. Intensified parallel activation of ATP demand and supply during exercise not only accelerates oxygen-uptake kinetics and improves homeostasis of metabolite concentrations, but also increases the maximum ATP production rate of oxidative phosphorylation. The specific mechanism, causing parallel activation of the different components of the oxidative phosphorylation system during muscle contraction, is still unknown, but appears to be related to the behaviour of calcium ions (Korzeniewski and Zoladz, 2003).

Russel et al. (2003) showed that endurance training affects skeletal muscle UCP3 (mitochondrial uncoupling protein 3) expression. They demonstrated significant fibre-type related differences in UCP3 expression as a result of endurance running training, as compared to sprint running training. The endurance training caused a preferential fibre-type reduction of UCP3 protein, with the greatest reduction occurring in type I muscle fibres, a lesser reduction occurring in type IIa fibres, and the least reduction occurring in type IIb fibres. The differential fibre-type regulation of the UCP3 protein, after the endurance running training vs. the sprint running training, is a reflection of a differential fibre-type recruitment pattern, which is stimulated by the different fibre-type recruitment frequencies at different training intensities (Russel et al., 2003). Therefore, a reduced UCP3 content after training may be an adaptation that partially assists with an improvement in performance. This can be associated with an increased efficiency of oxidative phosphorylation resulting in performing submaximal exercise at a higher intensity (Russel et al., 2003). From a sprinting performance perspective the rate of phosphocreatine resynthesis would increase (Russel et al., 2003).

As previously established, exercise training induces certain metabolic and morphological responses and associated adaptations in skeletal muscle that function to minimize cellular disturbances during any subsequent training sessions. The adaptations of skeletal muscle to prolonged, intense endurance training have been

summarized by Hawley (2002). Chronic adaptations in skeletal muscle result from repeated bouts of exercise, whilst the progressive adaptations occurring with subsequent training sessions follow upon the initial exercise stimulus. Endurance training is associated with an increased activity of key enzymes of the mitochondrial electron transport chain, which result in an increased mitochondrial protein concentration. The activation of several mitogen-activated protein kinases (MAPK) may play a role in the regulation of exercise-induced adaptations in skeletal muscle. Furthermore, AMP-activated protein kinase appears to be involved in the regulation of the acute metabolic response to exercise and in the chronic adaptations in skeletal muscle to prolonged training. The above adaptations, together with an increased capillary network, enable trained muscle to become more reliant on fat as a fuel, thereby reducing the glycolytic flux and establishing a tighter regulation of the acid–base status. Altogether, these adaptations result in improved performance capacity (Hawley, 2002).

2.4.1.2 Glucose kinetics

Bergman et al. (1999a) investigated the effects of endurance training and exercise intensity on the relationships among exercising muscle, net glucose uptake and whole body glucose kinetics in previously untrained subjects. Their results showed that exercise that requires a high power output, regardless of the state of training of the subjects, depends on fuel derived from carbohydrate. Therefore, the relative exercise intensity is able to predict the pattern of substrate utilization. Furthermore, net glucose uptake, glycogenolysis, and whole body glucose disposal rate of exercising muscle decreased for a given submaximal power output after exercise training. These changes were accomplished in an environment with a constant concentration of circulating insulin. Therefore it is proposed that certain intracellular signals are responsible for the training effect, and they exert their effect by decreasing the working muscle's sensitivity to insulin during moderate exercise intensity. Results of the study further support the concept that blood glucose is re-directed from inactive tissues to active muscle during exercise at an intensity around and above 65% VO_{2peak} , independent of the training state (Bergman et al., 1999a). In contrast, Kristiansen et al. (2000) found glucose uptake to be higher in trained compared to untrained human skeletal muscle when working at similarly high relative workloads and with low glycogen concentrations. In

addition, training increased the muscle content of GLUT-4. The glucose uptake during exercise at the highest workload correlated significantly with the availability of GLUT-4 in the muscle. Therefore, the rapid increase in GLUT-4 expression occurs as part of the early adaptive response of muscle to an adequate exercise stimulus (Kristiansen et al., 2000). This adaptation enhances the ability to replenish muscle glycogen stores, and appears to be mediated by pre-translational mechanisms (Ren et al., 1994).

2.4.1.3 Capillary density

Intense intermittent endurance training induces capillary growth and proliferation of endothelial cells in previously untrained human skeletal muscle. These changes are equally distributed around type I and type II fibres, appear within 4 weeks, and appear to be transient. This is supported by a reduction in proliferating endothelial cells and no further capillarisation after 7 weeks of training (Jensen et al., 2004).

2.4.1.4 Sarcoplasmic reticulum adaptations

The sarcoplasmic reticulum (SR) influences muscle contraction by regulating both muscle rate of contraction and relaxation. A greater development of the SR allows for more efficient release and re-uptake of Ca^{2+} , hence the size and structure of the SR plays an important role in the rate of muscle contraction and relaxation (Ross and Leveritt, 2001). Holloway et al., (2005) investigated the repetition-dependent effects of heavy, intermittent cycle exercise on SR Ca^{2+} transport properties in muscle of untrained subjects, and concluded that SR Ca^{2+} handling properties adapt rapidly to repetitive exercise. Furthermore, these adaptations result in a decreased perturbation in Ca^{2+} uptake, which appears to be mediated by an improved maintenance of Ca^{2+} -ATPase enzyme activity. Collectively, these findings suggest that the cycling behaviour of Ca^{2+} improves in repetitively exercised muscle, which contributes to improved fatigue resistance (Holloway et al., 2004).

2.4.1.5 Potassium kinetics

Performance improvement and delayed fatigue during exercise has been associated with lower interstitial potassium accumulation in muscle after training in habitually active

individuals (Nielsen et al., 2003). Furthermore, intense intermittent training reduces accumulation of potassium in human skeletal muscle interstitium during exercise, possibly resulting from an increased potassium re-uptake due to a greater activity of the muscle Na^+ , K^+ -ATPase pumps.

2.4.1.6 Lactate kinetics

According to Bergman et al. (1999b), endurance training at moderate exercise intensity increases lactate clearance in active muscle and decreases whole body and active muscle lactate production. In contrast, endurance training at high relative exercise intensities increases whole body and active muscle lactate clearance, but does not influence lactate production. Therefore, the mechanism involved in the decreased arterial lactate concentration after endurance training varies depending on the exercise intensity (Bergman et al., 1999b).

2.4.1.7 Neuromuscular adaptations

Saunders et al. (2003) propose that the reduction in end-exercise VO_2 , observed following endurance training in cyclists, may be due to an alteration in muscle recruitment and attenuated muscle activity. Endurance training decreased muscle activity in the quadriceps muscle (a prime force producer during cycling), possibly as a result of the reduction in the oxygen demand required to maintain a bout of high-intensity submaximal work. However, the exact mechanism by which the use of end-exercise muscles during heavy constant-load exercise is attenuated, following such period of endurance training, remains to be established. One possibility is that the increased muscle mitochondrial content and oxidative capacity after training allows muscle fibres, used during training, to function more aerobically with less reliance on anaerobic energy supply, which in turn prolongs the time to fatigue of some of the muscle fibres. As a result, fewer additional muscle fibres will be recruited to replace the fatigued fibres, thus decreasing the end-exercise active muscle and VO_2 (Saunders et al., 2003).

Häkkinen et al. (2003) investigated the effects of combined strength and endurance training *versus* the effects of strength training alone on both functional and structural

adaptations of the neuromuscular system in men during a prolonged training period. Following the training period, similar improvements were observed for the 1RM load and maximal isometric force in both training groups. Similarly, maximum iEMG of the vastus lateralis, cross-sectional area of the quadriceps femoris throughout the predetermined muscle length, and mean fibre areas of fibre types I, IIa and IIb increased for both training groups. Importantly, the rate of force development increased only in the strength training group. Therefore, improved power development, following training-induced adaptations in trained muscles, appears to be partly mediated by more rapid voluntary neural activation of the trained muscles (Häkkinen et al., 2003).

2.4.1.8 Muscular adaptations

(a) Myofibre function

Changes in fibre-type distribution (i.e. fibre-type transitions), fibre cross-sectional area and myosin heavy chain isoform content of human vastus lateralis muscle are influenced by training mode and occur as distinctive adaptational processes (Putman et al., 2004). The unique findings from Harber et al. (2004) indicate that the contractile properties of myofibres from trained endurance athletes are sensitive to alterations in training intensity. More specifically, interval training was a potent stimulus for eliciting changes in the cellular function of myofibres. Furthermore, variations in training (running training in this study) altered the contractile function of slow-twitch fibres (type I fibres), and fast-twitch fibres (type IIa fibres). Trappe et al., (2006) also illustrated a high degree of muscle plasticity with long distance running in recreational individuals. During the course of a 13 week training program, type I and type IIa muscle fibres decreased in diameter, while maintaining their force-generating capacity, but increasing their peak power. Only type I fibres increased their speed with training. Following the taper, type I fibre contractile properties did not change significantly, while the strength and power of type IIa fibres increased. These data show that both fibre types are susceptible to training-induced alterations following periodized run training. In summary, this study shows that the contractile functions of slow- and fast-twitch muscle fibres undergo differential adaptations following the exposure to different training modes.

The mechanisms responsible for stimulating a change in muscle fibre contractile characteristics, for example fibre type I towards fibre type IIb (I→IIa→IIb), are uncertain, but likely to vary between individuals. Several studies have examined physiological mechanisms that are thought to influence these changes, and factors such as stretch and stimulation, stimulation intensity and recovery duration between bouts of stimulation, as well as general overloading of muscle, have been considered (Ross and Leveritt, 2001).

(b) Muscle architecture

Muscle architecture adaptations occur rapidly in response to training programs imposed on athletes, but do not appear to account for performance differences between high velocity training and resistance training, suggesting that factors other than, or in addition to, muscle size and muscle architecture are responsible for mediating performance changes (Blazevich et al., 2003). In a subsequent study, Blazevich et al. (2007) confirm previous findings of rapid muscle architectural adaptation in response to resistance training. In addition, they showed that the adaptations of fascicle angle and length are different. Changes in fascicle angle continued in a relatively linear way for the first few months of training, whereas fascicle length adapted rapidly and did not continue past the first few weeks. The fascicle lengths increased similarly in the two groups which did resistance training involving only concentric or eccentric training. This suggests that the type of contraction is probably not the predominant stimulus responsible for this adaptation. Therefore, in this study, adaptations in fascicle length were not influenced by the type of contraction (i.e. concentric vs. eccentric resistance training), but more likely by the range of motion during training (i.e. fascicle strain). The changes in fascicle length observed during training and detraining were closely associated with shifts in the angle-torque curve. The increases were similar in both training groups; therefore it is likely that the range of motion during training has the most influence. Whether the length range of force production or the absolute muscle length is the greater stimulus for these adaptations cannot be determined from their data (Blazevich et al., 2007).

Blazevich et al. (2007) have been able to confirm that the genetic or signalling basis for fascicle length adaptation is different from that for muscle hypertrophy and fascicle

angle adaptation in humans. The architectural arrangement of fascicles strongly influences a muscle's force generating properties. Knowing the highly plastic nature of muscle architecture, it is important to understand the mechanical stimuli that influence muscle adaptation, so as to be able to optimize muscle function and performance. In particular, fibre-, or fascicle length has a profound effect on muscle range of motion, maximum shortening velocity (muscle power), and the length-tension relationship. However, the primary mechanical signal required to stimulate the functional adaptation of muscle fascicle length has yet to be identified (Blazevich et al., 2007).

(c) Gene transcription and molecular adaptation

Skeletal muscle is a complex tissue capable of remarkable adaptation in response to exercise training. Myofibres are capable of hypertrophy, but this requires activation and myogenic differentiation of mononuclear satellite cells that fuse with the enlarging or repairing myofibre (Cameron Smith, 2002). Furthermore, only a single bout of exercise is able to activate the expression of many different groups of genes. Muscular hypertrophy, following resistance exercise, depends on the activation of satellite cells and their subsequent myogenic maturation. In comparison, endurance exercise requires the simultaneous activation of mitochondrial and nuclear genes to enable mitochondrial growth. However, the impact of repeated exercise bouts during exercise training on the expression of genes has yet to be investigated systematically (Cameron Smith, 2002).

Coffey and Hawley (2007) recently summarized the literature and describe the molecular responses to exercise training as follows: "*The process of exercise-induced adaptation in skeletal muscle involves a multitude of signalling mechanisms initiating replication of specific DNA genetic sequences, enabling subsequent translation of the genetic message and ultimately generating series of amino acids that form new proteins. The functional consequences of these adaptations are determined by training volume, intensity and frequency, and the half-life of the protein*" (p 737).

Results of Bickel et al. (2005) indicate that a single session of resistance training is sufficient to stimulate certain molecular responses, which are indicative of the initiation of myogenic processes in skeletal muscle. These observations indicate that the

mechanisms that regulate the adaptation of skeletal muscle to increased loading respond in a relatively short time.

The increased contractile activity associated with physical exercise plays a major role in the regulation of several metabolic functions in skeletal muscle, such as glucose transport and glycogen metabolism (Bergman et al., 1999a; Kristiansen et al., 2000). Additionally, exercise induces transient changes in gene transcription and can influence the rates of protein metabolism, both of which may be responsible for chronic adaptations in skeletal muscle in response to repeated bouts of exercise (Putman et al., 2004; McArdle et al., 2004; Cameron-Smith, 2002). The identification of the intracellular signalling mechanisms responsible for converting mechanical stress into chemical signals has been the focus of intensive research by Sakamoto et al. (2003). The different contractile and metabolic properties of slow-twitch and fast-twitch fibres translate distinct mechanical signals to the various muscles. Data suggest that Akt (protein kinase B) plays a major role in the signalling for cellular growth and metabolism in skeletal muscle, and that the level of contractile activity in the muscle is fundamental to this regulation. Furthermore, physiological exercise *in vivo* activates Akt in multiple skeletal muscle fibre types, and mechanical tension appears to be a part of the mechanism by which muscle contraction activates Akt in fast-twitch fibres (Sakamoto et al., 2003).

(d) Muscle growth and regeneration

The mechanism, by which mechanical loading of muscle induces the activation of signalling pathways and growth factors *in vivo*, remains poorly understood (Bickel et al., 2005). Furthermore, while *in vitro* models continue to investigate the signalling pathways, only *in vivo* investigations are able to examine the activation of these pathways with exercise. Crameri et al. (2004) showed that an increase in the number of satellite cells was possible with a single bout of unaccustomed voluntary high intensity exercise. However, terminal differentiation of these cells may require repeated bouts of exercise or myofibre lesions.

Folland et al., (2001) investigated the relationship between eccentric exercise-induced muscle damage (and the subsequent initiation of the regenerative processes) and

adaptations to strength training in human elbow flexor muscles. A major finding of this study was that muscle damage reduced strength performance and significantly inhibited the acquisition of training-induced strength gains for at least 5 weeks after the bout of maximal eccentric exercise. Furthermore, the muscle damage did not provide any benefits during the 9 weeks following the eccentric exercise. These results indicate that a bout of damaging eccentric exercise does not enhance the response to conventional strength training, and therefore suggest that recovery from eccentric muscle damage and the adaptations to strength training are two different processes. The outcome of this study may have been limited by the possibility that the muscle damage was insufficient to initiate significant strength gains. Alternatively, disruption of the contractile elements may have played a minor role and overcompensation of muscle material was required to obtain muscle hypertrophy and strength gains (Folland et al., 2001).

McNeil and Khakee (1992) propose that basic fibroblast growth factor (bFGF) is released through muscle fibre plasma membrane breaks, following mechanically induced stress. This mechanism could initiate the growth in muscle damaged by exercise and in muscle undergoing hypertrophy in response to exercise. It could even initiate the growth in undamaged muscle, in which the maintenance of normal muscle mass depends on regular muscle action.

2.4.2 Mechanisms involved in skeletal muscle adaptation in response to exercise training in endurance cyclists

Altogether, the above data present evidence of training-induced muscular adaptations in untrained or recreationally active individuals. However, evidence of muscular adaptations in already highly trained endurance athletes is limited due to the lack of scientific studies in this field.

The training-induced changes in skeletal muscle resulting from the high-volume, high-intensity training undertaken by elite cyclists are at least partly responsible for the observed improvements in their endurance performance (Hawley and Stepto, 2001). However, the scientific knowledge of the effects of specific training interventions on certain skeletal muscle adaptations in professional cyclists and their role in improving endurance performance is limited. This can be attributed to the fact that scientists have

found it difficult to persuade elite cyclists to experiment with their training regimens or to obtain muscle and blood samples from these athletes (Hawley and Stepto, 2001). Owing to the lack of scientific study, Hawley and Stepto (2001) present a theoretical model of some of the major training-induced adaptations in skeletal muscle that determine performance capacity in elite cyclists. This model includes, but is not limited to, 3 highly interdependent components: skeletal muscle morphology, acid-base status and fuel supply. Any training-induced adaptation within one component is likely to influence the muscle's overall adaptation and therefore its response to the training stimulus. In summary, the sum of adaptations within each individual component of the model ultimately influences the performance capacity of a rider at any given time (Hawley and Stepto, 2001).

2.4.2.1 High intensity training: Performance changes and possible adaptations

While submaximal exercise training is characterised by prolonged, continuous activity, high intensity training (HIT) is achieved through the use of intervals, and is broadly defined as repeated bouts of short to moderate duration exercise (i.e. 10 seconds to 5 minutes), which are completed at an intensity greater than the anaerobic threshold (Laursen and Jenkins, 2002). Exercise bouts are separated by brief periods of low-intensity work or inactivity that allow a partial recovery. Furthermore, HIT elicits significant enhancements in endurance performance in highly trained athletes (Laursen and Jenkins, 2002).

Gibala et al. (2006) observed that the initial molecular and cellular adaptations in the vastus lateralis muscle of healthy males are similar after low volume sprint-interval cycling training and high volume endurance cycling training. Specifically, training-induced increases in muscle oxidative capacity, muscle buffering capacity and glycogen content were all observed to be similar between the two training groups. Therefore, in recreationally active males, short-term sprint interval cycling training induces metabolic and performance adaptations which are comparable to traditional endurance cycling training (Gibala et al., 2006). According to Laursen and Jenkins (2002), little research has examined the adaptations of central or peripheral factors following HIT in highly trained athletes. Despite significant improvements in endurance performance, the available data do not indicate changes in oxidative or glycolytic enzyme activity

following HIT in highly trained athletes, but rather suggest increased skeletal muscle buffering capacity to be involved in the improved endurance performance (Laursen and Jenkins, 2002). Similarly, Westgarth-Taylor et al. (1997) maintain that improved 40 km TT performance, following a period of HIT in already well-trained cyclists, is not related to rates of carbohydrate oxidation.

Chapman et al. (2007) studied neuromuscular adaptations in response to HIT and proposed that improvements in performance after HIT occur through an additional recruitment of the motor units required to sustain exercise at, or close to, race pace. They studied the patterns and timing of muscle recruitment and co-activation, variation in the amplitude of muscle activity during the pedal stroke, population variance, individual variance, and the influence of cadence on leg muscle recruitment and co-activation during the cycling movement. Their findings suggest that muscle recruitment is highly skilled in highly trained cyclists and less refined in novice cyclists. As a result, performance improvement in highly trained cyclists after HIT can be partially explained by neuromuscular adaptations and more efficient muscle recruitment, due to the repeated performance of the cycling movement in training and competition (Chapman et al., 2007).

The physiological adaptations responsible for observed improvements in VO_{2peak} and 40 km TT performance, following high-intensity interval training in well trained cyclists, are related to peripheral aerobic variables such as ventilatory thresholds and VO_{2peak} , as well as anaerobic related variables, such as anaerobic capacity and lactate buffering ability (Laursen et al., 2005). It is therefore apparent that muscles adapt to high intensity interval training through improvements in aerobic energy metabolism and improved muscle buffering capacity, and that peripheral adaptations rather than central adaptations are involved in the improved performances observed in well-trained endurance athletes (Laursen et al., 2005).

2.4.3 Mechanisms involved in altered muscle mechanical properties and changes in the peak torque – angle relationship following exposure to exercise training

2.4.3.1 Skeletal muscle adaptation in response to exercise

It is well known that muscle adaptations which result from regular exposure to exercise training involve metabolic, mechanical and neural mechanisms, which cause changes in muscle function and morphology. Goto et al. (2004) showed that resistance training induces muscle hypertrophy (reflected by changes in muscle cross-sectional area) and improvements in muscular strength, power and endurance. Blazevich et al., (2003) demonstrated increased fascicle length in response to an athletic training intervention and showed that these changes are linked to the force or velocity characteristics of the muscle, rather than the movement patterns of the different training exercises. Furthermore, they established that changes in muscle architecture such as fibre hypertrophy, increases in fascicle or fibre angle (pennation), and increases in fascicle length result in increased muscle mass and influence a muscle's force generating capacity. Increases in muscle thickness are greatest at proximal muscle sites, which supports the view that muscle hypertrophy, in response to training, occurs inconsistently along the length of the muscle (Blazevich et al., 2003). To what extent mechanical properties of muscle such as angle-torque relationship are affected by selective hypertrophy remains to be determined.

2.4.3.2 Skeletal muscle adaptation in response to exercise in the elderly

Skeletal muscle retains its capacity for adaptation into old age (Reeves et al., 2004; Narici et al., 2006). The smaller pennation angles and fascicle lengths observed in muscle tissue in older individuals suggest that aging causes a loss in sarcomere number, both in parallel and in series, however, the exact mechanism responsible for this phenomenon is still unknown (Narici et al., 2006). Furthermore, the loss of muscle mass (also described as sarcopenia) associated with aging, is one of the main causes of muscle weakness and reduced locomotor ability in old age. However, sarcopenia alone is not fully responsible for the observed weakness, as decreases in force levels do not fully correspond with the decreases in muscle size. Narici et al. (2006) further

explain that the reduction in force generating capacity at whole muscle level results from a combination of changes in muscle architecture (reduced fascicle fibre length and reduced pennation angle), tendon mechanical properties, neural drive (reduced agonist and increased antagonist activity), and single-fibre-specific tension. Differences in muscle architecture attributed to ageing account for about half of the loss of muscle function in the elderly. The effect that these architectural alterations have on function also depends on the stiffness of the tendon (Narici et al., 2006).

Following a period of 14 weeks of resistance training, Reeves et al., (2004) demonstrated an increase in force production capability at whole muscle level (compared to single fibre level) of the vastus lateralis muscle in elderly subjects. The increase in the force producing capability at whole-muscle level after training was partly responsible for the strength gains observed in these elderly subjects. Ultrasound studies identified adaptations in muscle architecture and showed increases in muscle fascicle length and pennation angle, which suggest training-related increases in sarcomere numbers, both in series and in parallel. A greater number of sarcomeres in parallel enable muscles to generate higher maximum force levels. An increased number of sarcomeres in series enable muscles to follow a greater excursion range, however, *in vivo* this would be affected by the anatomical constraints that determine the range of motion about a joint (Reeves et al., 2004). Reeves et al., (2004) further demonstrated that the relative increase in isometric knee extension torque, following a period of resistance training, is not constant across the joint range, indicating that a training-induced shift in the optimal angle occurs. This finding is significant, because by obtaining isometric torque at a single joint angle, the actual training-induced strength gains may be inaccurate due to an under- or overestimation.

The force-velocity characteristics of muscle are not only influenced by muscle fibre composition, but also by the stiffness of the tendon, as this anatomical structure is located between muscle and bone and therefore is highly sensitive to mechanical load changes (Narici et al., 2006).

2.4.3.3 Tendon stiffness

According to Narici et al. (2006), an increase in tendon stiffness, in response to exercise training, occurs due to a change in the material properties of the tendon. These changes have certain functional implications. More specifically, they increase the rate of contractile force transmission, decrease the likelihood of tendon strain injury, and alter the force-length relationship of the muscle. The changes in the force-length relationship are related to the anatomical location of the tendon between muscle and bone. Therefore, the amount of tendon elongation directly affects the amount of shortening of the in-series muscle (Narici et al., 2006). Increased tendon stiffness following resistance training could therefore be expected to cause reduced shortening of muscle fibres, resulting in changes in the operating range of the muscle. However, Reeves et al. (2004) demonstrated that the operating range of the muscle remains unchanged after training. They subsequently attributed their finding to increases in sarcomere number and therefore fascicle length. These findings indicate that the muscle-tendon interaction is monitored by a sensory control mechanism that strives to maintain the operating range of the muscle.

2.4.3.4 Shifts in the length tension relationship as an indicator of muscular compliancy

A shift in the optimum muscle length for active force generation, in the direction of longer lengths, is an indirect indicator of post-exercise changes in muscle compliance (Proske and Morgan, 2001). Whitehead et al. (2003) proposed that a shift in the length-tension relationship and a rise in muscle passive tension are "*simple and reliable indicators*" (p 1233) for assessing contractile element damage following eccentric exercise. However, Butterfield and Herzog (2005) showed that eccentric, as well as isometric exercise (which is thought not to cause damage), are able to cause changes in the angle-torque relationship, the length-tension relationship and the peak force level after exercise. In addition, the reduced torque production after exercise was more evident at the short, compared to the long muscle lengths. These findings indicate that the shift in peak torque is caused by a combination of muscle damage and post-exercise fatigue (Butterfield and Herzog, 2005), and therefore a change in the angle-torque or length-tension relationship would not be a "simple and reliable indicator" of

muscle damage associated with eccentric exercise. However, according to Whitehead et al. (2003), the increase in whole muscle passive tension, immediately after a series of eccentric actions, occurs independent of fatigue, and therefore measurements of changes in whole muscle passive tension should be considered as a reliable indicator of muscle damage following eccentric muscle action (Whitehead et al., 2003).

2.4.3.5 Passive tension

Relatively little is known about the passive mechanical properties of muscle, but two factors have been considered to influence the level of passive tension in a muscle, following a series of eccentric actions. Firstly, injury contractures in damaged muscle fibres tend to increase passive tension. Secondly, the presence of disrupted sarcomeres in series with still-functioning sarcomeres tends to reduce passive tension (Whitehead et al., 2003). Furthermore, only some muscle fibres develop an injury contracture after eccentric action and the amount of the increase in passive tension is therefore length-dependent. Whitehead et al. (2003) have described contracture formation as a dynamic process: sarcomeres in damaged muscle regions shorten and consequently stretch the sarcomeres and muscle membranes in the adjacent areas, thereby spreading the contracture. The disruption of sarcomeres initiates the contracture formation, whereas the injured muscle fibres generate the increased level of passive tension over time. In short, muscle fibres develop several disrupted sarcomeres as a result of the eccentric actions and some disrupted areas progress to contracture formation, which, in turn raises the passive tension. However, the evidence of the factors determining the balance between contracture formation and disrupted sarcomeres has yet to be established (Whitehead et al., 2003).

2.4.3.6 Shift in optimum angle

The ability of muscle to generate power is of functional importance for physical activity and athletic performance. Therefore, alterations to the angle-torque relationship have important implications for muscle performance, particularly as they relate to functional impairment of muscle at maximal intensity exercise.

Reeves et al. (2004) show that there is a shift in optimal angle after resistance training in older adults. The displacement of the angle-torque data towards higher torque values was associated with a training-induced increase in agonist activation. The shift in optimal angle was associated with changes in the muscle-tendon properties. Furthermore, different mechanisms are involved in the changes occurring in the relationship between torque production and joint angle, including changes in the tendon moment arm length-angle relationship, changes in the length-tension relationship of agonist muscles, and changes in the activation level of agonist and antagonist muscles. The tendon moment arm length-angle relation cannot be altered by training, as the tendon moment arm is determined by the anatomical constraints of the skeleton. However, training can change the muscle length-tension relationship through causing changes in the sarcomere numbers in series, or in tendon stiffness. Therefore, *in vivo*, an interaction occurs between the contrasting effects resulting from changes in fascicle length and tendon stiffness. This complicates the interpretation of the resultant effects on the length-tension and angle-torque relationship (Reeves et al., 2004). Nevertheless, to be able to interpret training-related changes in the angle-torque relationship accurately, inter-angle differences in torque improvement have to be considered during the measurements following a period of strength training.

Brockett et al. (2001) demonstrated a shift in the optimal angle for maximal voluntary eccentric torque production in human hamstring muscle after eccentric exercise. This shift remained for ten days, although voluntary torque had recovered by the third day. This shift is consistent with the shift towards longer muscle lengths for peak torque generation that are reported in later studies (Whitehead et al., 2003), and is believed to occur due to the presence of disrupted sarcomeres in series with still functioning sarcomeres, which increases the series compliance of a muscle (Brockett et al., 2001).

The study by Reeves et al. (2004) has in part been discussed earlier, but considers the role of tendon in the changes of the angle-torque relationship in more detail in this section. In an attempt to better understand the origin of the changes in the angle-torque relationship following training, Reeves et al. (2004) investigated the effects of resistance training on muscle architecture (fascicle length and pennation angle) and the length-tension relationship of muscle fascicles. Collectively, the training-induced shift in optimal muscle length for peak torque generation has been attributed to length changes

of muscle fascicles (resulting from the addition of sarcomeres), and the mechanical properties of tendon. The observed changes in muscle architecture during contraction after training support the view that training results in tendon stiffening. Since a tendon functions in series with the muscle, it appears obvious that the amount of fascicle shortening during muscle contraction depends primarily on the amount of tendon elongation. In summary, the training-related shift in the length-tension relationship towards longer lengths can be explained by an increase in fascicle lengths, while the maintenance of the longer lengths upon contraction can be explained by increases in the stiffness of the tendon (Reeves et al., 2004).

2.5 Conclusion

It has been established that training-induced muscle adaptations affect muscle fibre contraction dynamics. Furthermore, it has been well documented that force output in skeletal muscle depends, in part, on the mechanical force-length properties of skeletal muscle tissue (Pincivero et al., 2004). Considerable data are available on the physiological adaptations that occur following exercise training. However, the literature has focused mainly on those parameters that reflect a muscle's ability to adapt to training as a result of metabolic mechanisms, or changes in muscle architecture.

Contractile element damage plays an important role in the observed changes in muscle mechanical properties following exposure to eccentric actions (Proske and Morgan, 2001). A consequence of the changes in the mechanical properties is that there is a shift in the optimal angle for peak torque production towards longer muscle lengths (Brockett et al., 2001).

Measuring training-related changes in isokinetic angle-torque relationship, as a function of a muscle's length-tension relationship, fascicle length, and the relationship between the muscle's moment arm and joint angle, provides a means of measuring the ability of a muscle to adapt to a specific exercise training stimulus (Butterfield and Herzog, 2005). Furthermore, measurement of the angle-torque relationship of a muscle has the potential to be used as a screening tool and as a guide for the design of appropriate training and rehabilitation programs in the athletic population.

Possible mechanisms for improvements in performance, following high intensity training, have been identified (Laursen et al., 2005; Hawley and Stepto, 2001), but data discussing the role of muscle mechanical adaptations in response to high intensity training are scarce. Therefore, investigating the relationship between changes in whole muscle power output and changes in the angle-torque relationship has the potential to provide further insight into the muscle mechanical mechanisms, which may account for the performance changes that have been observed following a period of high intensity training.

University of Cape Town

Chapter 3

The effect of high intensity training on the angle-torque relationship of the quadriceps and hamstring muscles in a group of well trained cyclists.

3.1 Introduction

The musculoskeletal system tailors itself for a particular functional task in response to different forms of exercise training. Various studies have examined the factors that determine the extent and nature of the design principles that determine certain behaviour of skeletal muscle (Brockett et al., 2001; Burkholder & Lieber, 2001; Proske & Morgan, 2001; Lynn et al., 1998; Whitehead et al., 1998).

It is well known that muscle adaptations resulting from regular exposure to exercise training involve metabolic, mechanical and neural mechanisms which mediate changes in muscle function and morphology. The physiological adaptations responsible for improvements in VO_{2peak} and 40 km TT performance, after high-intensity interval training in well-trained cyclists, are related to peripheral aerobic variables, including ventilatory thresholds and VO_{2peak} , and anaerobic related variables, including anaerobic capacity and lactate buffering ability (Laursen et al., 2005). Although it is well known that performance improves after high intensity training, the metabolic changes alone are not able to account for all the changes leading to the improvement in performance.

There is a lack of understanding about how the mechanical properties of muscle change in response to high intensity training. However, it is reasonable to assume that changes in muscle mechanical properties may also partially contribute to the improvement in performance after high intensity training. This assumption is supported by the fact that force output in skeletal muscle depends, in part, on the mechanical force-length properties of skeletal muscle (Pincivero et al., 2004). Furthermore, angle-specific responses of knee extensor torque to maximal-effort contractions have been

observed in various studies, which showed a measurable effect on the length-tension relationship following exercise training (Folland et al., 2000; Butterfield and Herzog, 2005; Prasartwuth et al., 2006; Whitehead et al., 1998).

A shift in the muscle's length-tension relationship (optimum length for active force production) has been described as an indicator of increased muscle compliance after exercise (Brockett et al. 2001). This shift reflects a rightward shift of the 'peak torque - joint angle relationship'. Determining the joint angle at which peak torque occurs (termed optimum joint angle) is a functional measure, which is able to describe the change in the length-tension relationship of a muscle (Butterfield and Herzog, 2005). It is not known whether the optimum joint angle changes after high intensity training, and furthermore, whether this is associated with changes in performance after high intensity training.

3.2 Aim

The aim of this study was to determine the effect of four weeks of high intensity training (HIT) on the angle-torque relationship of the quadriceps and hamstring muscles in a group of well trained cyclists. The hypothesis was that the angle-torque relationship changes in response to four weeks of high intensity training.

3.3 Methods

Subjects

Fifteen subjects volunteered for the study. Most of these subjects were recruited from the group of subjects participating in the high intensity training study by Swart (2007). One subject was unable to fully commit to the training times, and was excluded from further analysis. The final study sample therefore consisted of 14 well trained male cyclists (mean \pm SD; age = 31.7 ± 4.8 yr; $VO_{2peak} = 55.9 \pm 5.0$ ml.kg⁻¹.min⁻¹, $W_{peak} = 359 \pm 35$ W) with a competitive cycling experience of 12.4 ± 9.4 years. After being fully informed of the risks and stresses associated with the high intensity training and isokinetic testing study, all subjects completed a Physical Activity Readiness

Questionnaire (Appendix A), had their training logs analysed, had a personal interview about their cycling history (Appendix B), and received a subject information sheet (Appendices C and D). Each cyclist was verbally questioned about their injury history. Subjects presenting with a musculoskeletal condition, which could interfere with the trial performance, would be excluded from participation in the study. All subjects gave their written informed consent to participate in the studies (Appendices E and F). The experimental protocol was approved by the Ethics and Research Committee of the Faculty of Health Sciences of the University of Cape Town.

Cyclists were randomly assigned to one of two groups; a training group or a control group (Table 3.1). For purposes of the HIT study (Swart, 2007), the subjects in the high intensity training group were randomised to either power-based training or heart rate based training. This training however was similar and therefore subjects were grouped into a single high intensity training group.

Study Design

Each subject completed a 40 km familiarization time trial (TT), a peak aerobic capacity test (VO_{2peak}) and a repeat 40 km TT test at 7, 3 and 1 days prior to the start of the 28 day training period respectively.

Isokinetic torque testing was performed after the pre- and the post-training VO_{2peak} tests respectively. A rest period of 30 minutes was applied to allow for recovery between each VO_{2peak} and isokinetic torque test. A visit to the Biodex laboratory 7 days prior to the 28 day training protocol served to familiarize subjects with the isokinetic torque testing protocol.

Subjects reported to a laboratory with stable climatic conditions for all performance tests and training sessions. During each performance testing session, the subject was unaware of the power output, heart rate, speed and exercise time to prevent the adoption of pacing strategies or performance bias.

Subjects were asked to refrain from eating for at least 2 hours prior to each of the performance tests or supervised training sessions. Each subject was asked to refrain

from training for 24 hours prior to the VO_{2peak} test and to perform a 90 minute sub-maximal recovery ride (at an intensity below the “lactate turnpoint” determined during VO_{2peak} testing) 24 hours prior to the 40 km TT familiarization and 40 km TT tests. They were also asked to refrain from consuming any caffeine on the day of each performance test. Prior to each testing session subjects were questioned to confirm that they had adhered to these instructions.

Anthropometric measurements including height, body mass and sum of seven skinfolds (triceps, biceps, supra-iliac, sub-scapular, calf, thigh and abdomen) were taken immediately prior to the 40 km TT test, as described by Ross and Marfell Jones (1991).

Following the 28 day training period each subject completed a 10 day washout period of training during which time they were asked not to participate in any racing or prolonged high intensity exercise. Following the washout period, the VO_{2peak} test, isokinetic torque test, 40 km TT test and anthropometric measurements were repeated. Subjects were verbally questioned prior to the testing procedure to ensure that they had adhered to the training protocol.

Each subject was asked to keep a detailed training diary during participation in the study. The data from these diaries were used to confirm that subjects adhered to the prescribed training intensity during recovery sessions. Subjects were further asked to refrain from any lower limb weight training during the testing and training periods, and were questioned to confirm that they had adhered to these instructions.

Testing protocols

The VO_{2peak} and 40 km TT testing was performed at the cycling laboratory at the Sports Science Institute of South Africa in Cape Town. The isokinetic torque testing was performed at the Biodex[®] laboratory.

The VO_{2peak} and 40 km TT testing were performed on each subject's own road bicycle, which was mounted to an electronically braked cycle ergometer (Computrainer[™] Pro 3D, RacerMate, Seattle, USA). The subject's bicycle was attached to the ergometer by the rear axle quick release mechanism and supported under the front wheel by a plastic

support. Rear tyre pressure was inflated to 115 pounds per square inch prior to calibration. Calibration was performed automatically during the calibration mode by accelerating the bicycle to 40 km.h^{-1} and allowing the bicycle to coast until the rear wheel stopped. Calibration and load generator contact pressure was adjusted until the calibration value measured between 1.95-2.05 lbs (1.22 – 1.28 kg). Each subject completed a self-paced warm up for 15 minutes prior to each testing session.

(a) Progressive exercise test

The peak oxygen consumption ($\text{VO}_{2\text{peak}}$) and peak power output test (W_{peak}) was performed at a starting work rate of 2.50 W.kg^{-1} body mass and the load was increased incrementally at a rate of 20 W every 60 seconds until the subject could not sustain a cadence of greater than 70 rpm or was volitionally exhausted. During the progressive exercise test, ventilation volume (V_E), oxygen uptake (VO_2) and carbon dioxide production (VCO_2) were measured over 15-second intervals using an on-line breath-by-breath gas analyser and pneumotach (Oxycon, Viasis, Hoechberg, Germany). Calibration of this device was performed during the 15 minute self-paced warm-up according to the manufacturer's instructions. $\text{VO}_{2\text{peak}}$ was recorded as the highest VO_2 reading recorded for 30 seconds during the test. $\text{VO}_{2\text{peak}}$ was considered valid if the subjects attained 90% of age predicted HR_{peak} and respiratory exchange ratio (RER) exceeded 1.10. Peak power output was calculated by averaging the power output for the final minute of the $\text{VO}_{2\text{peak}}$ test.

(b) Isokinetic torque test

For the purpose of this study, an angle-torque curve is defined as the relationship between torque and joint angle, produced when the muscle is maximally activated during isovelocity shortening. The joint angle that coincides with maximal muscle torque is termed optimum angle.

This study used isokinetic testing to measure optimum knee angle for peak torque production in the quadriceps and hamstring muscles respectively. Angle-torque measurements were obtained before and after the four week training period for each subject (as described in the study design section). Shifts in the optimum angle were

determined by comparing differences in the angle-torque relationship between the pre- and post-training measurements amongst the training and control groups.

The isokinetic torque test was performed on the same dynamometer (Biodex® System 3 Shirley, NY, USA) for each test. Calibration of the Biodex® dynamometer was performed prior to every testing session according to the manufacturer's instructions.

Following the VO_{2peak} test and a 30 minute rest period, subjects completed a warm up, consisting of sub-maximal cycling on a cycle ergometer for 5 minutes. Subjects were requested to cycle at a self-selected cadence and resistance that they felt comfortable with, so as not to induce any significant muscle fatigue. Following the warm-up, subjects were positioned on the Biodex® dynamometer to start the testing. Each subject was instructed to perform the test with his self-selected dominant leg (defined as the leg he would choose to use to spontaneously kick a ball).

Subjects were seated in a comfortable, upright position on the Biodex® dynamometer accessory chair, with their hips at approximately 90° flexion. Subjects were secured using dual crossover straps for their upper bodies and a pelvic strap. A thigh strap was applied to the leg being tested. To minimize other body movements, particularly movement at the hip, subjects were asked to stabilize the other leg by bracing the knee at 90°. During the isokinetic torque measurements, subjects were required to keep their arms folded across their chests. The lateral femoral epicondyle was used as the bony surface landmark for matching the axis of rotation of the dynamometer resistance adaptor, and the chair position was adjusted accordingly. To ensure a standardized testing position, the subjects initial set-up position measurements were recorded and used for all subsequent testing.

Subjects were familiarised with the testing protocol by gradually building the contractions up to one maximal effort strength contraction. This was achieved by instructing each subject to perform 2 repetitions at 50%, 2 repetitions at 75% and 1 repetition at 100% of their maximal voluntary effort of the required movement. A rest interval of 2 minutes was allowed between the familiarisation and the testing. Based on a previous study (Brockett et al., 2001) and the National Olympic Committee of South

Africa guidelines (Durandt J, personal communication) for isokinetic testing velocities, it was decided to generate the angle-torque curves at a velocity of $60^{\circ}\cdot s^{-1}$.

The testing protocol for this study consisted of seven successive maximum voluntary concentric knee extension and flexion movements performed at a speed of $60^{\circ}\cdot s^{-1}$. Each contraction was performed over a 90° knee angle range, starting with the lower leg hanging in a vertical position at 90° . Subjects were instructed to kick their leg up and down as forcefully as they could, to ensure maximal effort of both the extension and flexion contractions. Subjects were required to produce maximum contractions with <10-15% variability between peak torques for all seven contractions. Verbal encouragement was given at the same level for every test. The testing procedure and verbal encouragement was administered by the same investigator for all the subjects.

(c) 40 km Time Trial Test

Subjects completed a self paced maximal 40 km time trial on a simulated flat 40 km route. As previously described, subjects were not given any feedback about their performance except for the completed distance. Subjects were allowed to consume water *ad libitum* throughout the test and were verbally encouraged to perform an effort that elicited the fastest possible 40 km TT time.

Training protocol

Following the testing protocols, each subject reported to the laboratory for a supervised training session on two occasions each week. High intensity and control training consisted of a 7 day cycle training protocol for a period of 28 days. Subjects performed the training sessions on their own bicycles mounted to a Computrainer™ ergometer (as described previously). Subjects were requested to complete a 15 minute self-paced warm up prior to the high intensity training sessions. Each high intensity training session consisted of 8 intervals of 4 minutes duration with a 90 second self paced recovery period between each interval. Each interval was performed at a load corresponding to approximately 80% of the subject's peak power output recorded during the VO_{2peak} test. [Refer to Swart (2007) for further detail regarding the determination of training load]. The control group performed a self-paced training ride

twice each week consisting of a 40 km simulated undulating route performed at below 70% of their peak power output recorded during the VO_{2peak} test. In addition, control subjects performed identical training to the HIT groups outside of the laboratory.

Between each laboratory training session, subjects were asked to alternately have a rest day, followed by a 90 minute recovery session (at intensity below the "lactate turnpoint" determined during VO_{2peak} testing) or a rest day, the 90 minute recovery session, followed by another rest day.

Data analysis

Outline

The respective torque values for extension and flexion obtained during testing were extracted and ordered according to knee angle. The graph of torque against angle was plotted. The optimum angle was defined as the knee angle for which the average torque in the quadriceps and hamstring muscles reached its respective peak. Shifts in the optimum angle for peak torque were obtained by calculating the difference between the joint angle at which peak torque was produced before and after training.

Specific details

The torque vs. knee angle data for the extension and flexion contractions on the Biodex[®] dynamometer (Biodex[®] System 3, Shirley NY) were selected and imported into Excel (Microsoft Excel Office 2003, Microsoft Corporation, USA). For the purpose of this study only negative torque and velocity values were accepted for a flexion curve, whereas only positive torque and velocity values were accepted for an extension curve. All remaining data were excluded. Any measurement errors from the Biodex[®] dynamometer were also excluded. The aim was to obtain cleaned and accurate data which could be used to produce a representative angle-torque graph from the values obtained from the flexion and extension contractions from each subject.

Each subject's data were then imported into Prism 3.0 (GraphPad Prism version 3.00 for Windows, GraphPad Software, San Diego California USA, www.graphpad.com) to

create an angle-torque graph for each of the 7 flexion and extension contractions respectively. Knee joint angles were represented on the x-axis (in degrees), whereas torque values were represented on the y-axis (in Nm) (Appendix G, Figure G1).

Outliers were defined as any contraction with a peak torque greater than within 10% of the contraction with the maximum peak torque for that specific testing set. A prerequisite for data analysis was a minimum requirement of 3 repetitions for each flexion and extension movement for each subject. If this could not be achieved after the first outlier determination (i.e. using the contraction with the maximum peak torque), the outliers were subsequently determined by using the peak torque second highest to the peak torque that was initially determined as the maximum peak torque. The final angle-torque graphs for each subject thus represented a minimum of 3 contractions within 10% for the respective flexion and extension movements, with any outliers being excluded.

Next, a fourth order polynomial nonlinear regression curve fit was used to obtain a single angle-torque curve representing an average peak torque value for each set of flexion and extension movements for every subject (with each set consisting of the 7 contractions) (Appendix G, Figure G2). From this curve a set of 5 curve parameters were calculated ($y = ax^4 + bx^3 + Cx^2 + dx$). By using these best-fit values in a fourth order formula in Excel, a set of x and y coordinates (degrees vs. torque) were calculated. These data were used to obtain a set of data points which could be used in Prism to produce a differentiated curve on an XY Plot, where knee joint angle and torque value were presented as x-coordinates and y-coordinates respectively. Of the resultant data set, the y-coordinates contained 2 regions, an ascending limb and a descending limb; and the turning point marked the maximum torque value for that curve (Appendix G, Figure G3). The x-coordinates represented the corresponding knee joint angles over the 90° movement range. The optimum angle for which the average torque in the quadriceps and hamstring muscles had reached its respective peak was then identified by determining the x-coordinate corresponding to the y-coordinate which had been identified as the turning point. The optimum angle for peak torque production was determined for flexion and extension respectively for each subject, and the resulting values were used for statistical analysis.

Two different sets of data were obtained for peak torque values and angles coinciding with peak torque, representing Biodex® data and calculated data respectively. However, the data obtained from the Biodex® were considered less accurate, as they did not exclude any contraction outliers, and could therefore not be considered for statistical analysis. The data representing the differences between the values obtained from the Biodex® and the calculated values are summarized in Appendix H, Table H1, while correlations are displayed graphically in Appendix H, Figure H1.

Statistical analysis

Data were analysed for statistical significance using STATISTICA version 7.0 (Stat-soft Inc., Tulsa, OK, USA). A one way analysis of variance (ANOVA) with repeated measures was used to examine differences in performance measures between the groups before and after HIT intervention or control training. A Tukey post-hoc test was used to determine differences in any of the significant interaction of group X time. The delta values were compared using an independent t-test. Relationships between variables were determined using a Pearson's product moment correlation. Statistical significance was accepted when $P < 0.05$. All data are expressed as means \pm standard deviation ($X \pm s$).

3.4 Results

The general descriptive data and cycling experience of subjects are shown in Table 3.1. None of these variables were significantly different between groups.

Table 3.1: General descriptive data and cycling experience of control (n = 5) and trained (n = 9) subjects.

| Variable | Control | Trained |
|--|-----------------|-----------------|
| Age (years) | 31.0 \pm 3.5 | 32.1 \pm 5.6 |
| Body mass (kg) | 79.6 \pm 7.5 | 77.3 \pm 7.0 |
| Stature (cm) | 183.2 \pm 5.4 | 180.3 \pm 6.8 |
| Body Fat % | 16.5 \pm 2.0 | 16.2 \pm 3.1 |
| Cycling experience (years) | 11.4 \pm 8.3 | 13.0 \pm 10.4 |
| Current cycling training (h.wk ⁻¹) | 7.0 \pm 5.5 | 8.1 \pm 4.7 |

There was a significant improvement in peak power output and 40 km TT performance after HIT in the trained group. VO_{2peak} and maximum heart rate did not change. These data are shown in more detail in Table 3.2.

Table 3.2: Measurements associated with cycling performance in control ($n = 5$) and trained ($n = 9$) groups, before and after 8 high intensity training sessions.

| <u>Variables</u> | <u>Control</u> | | <u>Trained</u> | | <u>P</u> |
|--|----------------|------------|----------------|------------|----------|
| | Pre | Post | Pre | Post | |
| VO_{2peak} ($ml \cdot kg^{-1} \cdot min^{-1}$) | 56.3 ± 7.1 | 56.8 ± 7.0 | 55.7 ± 3.9 | 58.0 ± 4.3 | 0.111 |
| PPO _{Absolute} (W) | 356 ± 34 | 357 ± 31 | 360 ± 38 | 377 ± 39 | 0.021 |
| PPO _{Relative} ($W \cdot kg^{-1}$) | 4.5 ± 0.7 | 4.5 ± 0.6 | 4.7 ± 0.5 | 4.9 ± 0.5 | 0.031 |
| Max HR ($b \cdot min^{-1}$) | 182 ± 11 | 182 ± 12 | 184 ± 10 | 184 ± 11 | 0.871 |
| 40 km TT (min) | 67.5 ± 1.7 | 67.6 ± 3.3 | 67.5 ± 2.3 | 65.8 ± 2.2 | 0.040 |
| HR 40 km TT ($b \cdot min^{-1}$) | 166 ± 9 | 164 ± 10 | 164 ± 9 | 169 ± 8 | 0.048 |
| PO 40 km TT (W) | 240 ± 13 | 241 ± 28 | 240 ± 23 | 257 ± 24 | 0.054 |

Where;

PPO = peak power output

Max HR = maximum heart rate during the VO_2 max test

TT = time trial

PO = power output

P = significance of the interaction of groups x time

The changes in performance were also expressed as a % change. These data are summarized in Table 3.3 and displayed graphically in Figure 3.1.

Table 3.3: Changes in power output and 40 km TT performance expressed as a % (pre/post).

| <u>Variables</u> | <u>Control</u> | <u>Trained</u> | <u>p</u> |
|---|----------------|----------------|----------|
| PPO _{Absolute} (W) % | -0.2 ± 2.7 | -4.8 ± 3.4 | 0.022 |
| PPO _{Relative} ($W \cdot kg^{-1}$) % | -1.0 ± 3.1 | -5.5 ± 3.6 | 0.038 |
| 40 km TT (min) % | 0.0 ± 3.0 | 2.5 ± 1.3 | 0.042 |
| PO 40 km TT (W) % | -0.3 ± 8.1 | -7.2 ± 3.8 | 0.050 |

Where;

PPO = peak power output

TT = time trial

PO = power output

P = significance of the interaction of groups x time

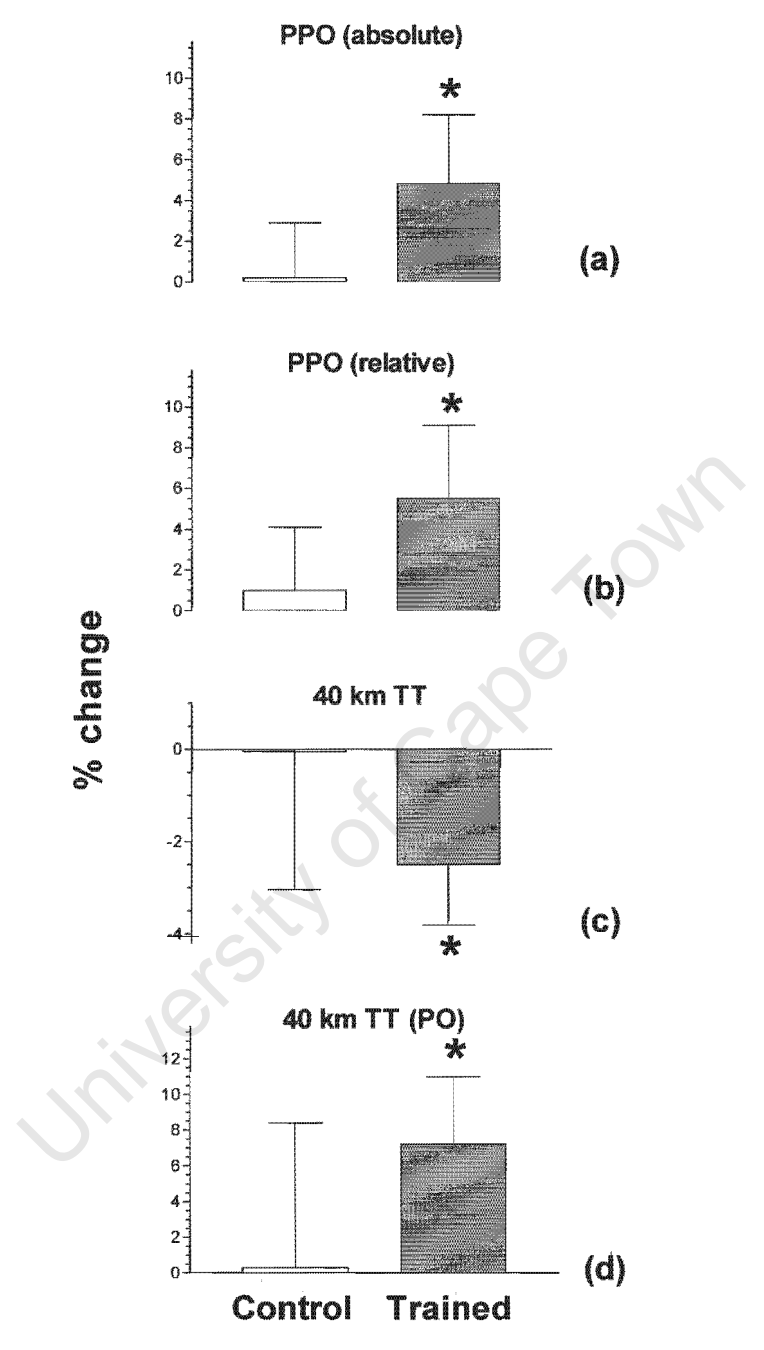


Figure 3.1: Changes in power output (PPO) and 40 km time trial (40 km TT) performance expressed as a % (pre/post).

Peak torque data are shown in Table 3.4. Peak torque values for flexion showed a group effect of $p=0.053$.

Table 3.4: Peak torque (Nm) in control ($n = 5$) and trained ($n = 9$) groups before and after 8 high intensity training sessions.

| <u>Variables</u> | <u>Control</u> | | <u>Trained</u> | | <u>P</u> |
|------------------|----------------|--------------|----------------|--------------|----------|
| | Pre | Post | Pre | Post | |
| PT Flex | 119.7 ± 10.1 | 117.3 ± 14.6 | 96.5 ± 18.9 | 102.9 ± 18.3 | n/s* |
| PT Ext | 186.2 ± 18.6 | 193.3 ± 9.9 | 170.5 ± 25.4 | 176.6 ± 20.8 | n/s |

Where;

PT = peak torque

Flex = Flexion

Ext = Extension

* $p= 0.053$ Group effect

Data describing the angle coinciding with peak torque are shown in Table 3.5. There were no significant differences for flexion or extension, either between groups or before and after training.

Table 3.5: Angle coinciding with peak torque in control ($n = 5$) and trained ($n = 9$) groups before and after 8 high intensity training sessions

| <u>Variables</u> | <u>Control</u> | | <u>Trained</u> | | <u>P</u> |
|------------------|----------------|-------------|----------------|-------------|----------|
| | Pre | Post | Pre | Post | |
| PT Angle Flex | 36.0 ± 13.4 | 34.6 ± 13.1 | 38.8 ± 15.2 | 36.7 ± 15.5 | n/s |
| PT Angle Ext | 59.8 ± 6.7 | 62.2 ± 3.3 | 62.4 ± 3.6 | 61.6 ± 5.6 | n/s |

Where;

PT = peak torque

Flex = Flexion

Ext = Extension

3.5 Discussion

The first finding of this study was that high intensity training elicits improvements in peak power output and 40 km TT performance in already well trained cyclists. The improvements in absolute peak power output ($4.8 \pm 3.4\%$), relative peak power output ($5.5 \pm 3.6\%$) and 40 km TT performance ($2.5 \pm 1.3\%$) are similar to changes previously reported following HIT in well-trained cyclists (Laursen and Jenkins, 2002; Laursen et

al., 2005). The next finding was that no statistically significant change was observed in the peak torque for flexion or extension following 4 weeks of high intensity cycling training. Most importantly, the angle coinciding with peak torque did not change for flexion or extension, either between groups or before and after 4 weeks of high intensity training.

In an attempt to interpret the findings it may be postulated that changes in the angle-torque relationship occur early in the 4 week training period, or conversely, that 4 weeks is insufficient time for changes in the angle-torque relationship, or that the angle-torque relationship does not change at all. If one considers eccentric exercise, the short-term shift in optimal angle for maximum voluntary contractions recovered to pre-exercise values within one week (Prasartwuth et al., 2006), while the maximal voluntary and twitch torque were not fully recovered, and voluntary activation at the shortest test length was still impaired after one week (Prasartwuth et al., 2006). These findings may not be directly extended to high intensity exercise or interval training, but may provide a possible explanation to the unchanged angle-torque relationship following the 4 weeks of training. In addition, following eccentric exercise, significant shifts in optimum angle for torque generation to longer muscle lengths have been observed immediately post-exercise, and up to 10 days beyond that (Brockett et al., 2001). Therefore, angle-torque measurements at more regular intervals during a 4 week training period may provide more information and perhaps better insight into the ongoing and progressive changes in the angle-torque relationship in response to high intensity training.

Whether the improvements in absolute peak power output ($4.8 \pm 3.4\%$) and relative peak power output ($5.5 \pm 3.6\%$) were facilitated by improvements in muscle cross-sectional area or by the rate of force development of the muscles, or both, cannot be determined from this study, as these parameters were not specifically measured.

Nevertheless, the angle coinciding with peak torque occurs at approximately 61.6 ± 5.6 for voluntary concentric knee extension and at 36.7 ± 15.5 degrees for voluntary concentric knee flexion (performed over a 90° knee angle range, starting with the lower leg hanging in a vertical position at 90°). The generation of voluntary knee extensor torque is well known to be a function of knee angle, with peak values being attained within the midrange of joint movement, at approximately 70° (Pincivero et al., 2004).

These data are supported by the observations from this study, and can partly be explained by the individual differences in the force-generating ability of the different portions of the quadriceps femoris muscle, which mainly exist as a result of changes in the whole muscle length due to changes in the knee angle (Pincivero et al., 2004). Therefore, either extending or flexing the knee and thereby placing the quadriceps femoris muscle in a shorter or longer length from its optimal length will result in a different force output at different knee angles.

With the exception of the rectus femoris muscle, which is a bi-articular muscle, the vastii components of the quadriceps femoris muscle are mono-articular muscles (Netter, 1989), and are at their shortest length during full knee extension. Force generating ability of the quadriceps therefore will be lowest when the knee is fully extended. Compared to only one bi-articular muscle component in the four-component quadriceps muscle, the hamstring muscle consists of three bi-articular muscle components (Netter, 1989). Interestingly, the data from this study indicate that the optimal whole muscle length for peak torque generation is different between the knee extensors and knee flexors, as peak torque generation does not occur around the midrange of joint movement for these muscles if one considers the whole muscle length.

In the current study, the bi-articular hamstring muscle appears to exhibit similar force generating ability compared to the semi bi-articular quadriceps muscle, by producing peak torque at the end of the first third of its movement range in the given 90° knee angle range. However, the normal range of motion at the knee occurs between 0° -135° (full extension to full flexion). Therefore, when extending the above findings to the full range of motion occurring at the knee joint, the quadriceps muscle's peak force generating ability occurs at around 75° of its extension range (and therefore midrange of extension movement at the knee joint, similar to the findings by Pincivero et al., 2004), compared to the hamstring muscle's peak force generating ability, which occurs at around 30° of its flexion range (and therefore the initial range of full flexion movement at the knee joint). Based on these findings, the hamstring muscle therefore generates peak torque at an even longer muscle length than the quadriceps muscle, despite being a fully bi-articular muscle and functioning in a partially lengthened position with the hip joint being statically flexed to 90° during the isokinetic testing position that was used in this study.

The above data support the existing understanding that optimal muscle length for peak torque generating ability occurs in the direction of longer muscle lengths (Pinivero et al., 2004), but at the same time indicate, that the optimal length for peak torque production is different between joint flexors and joint extensors, and between mono-and bi-articular muscles. This difference appears to exist independent of the exposure to a period of high intensity exercise, as the data remain unchanged after the training period. Such observations need to be further explored by studying the differences between the contextual properties of muscle (i.e. flexor/extensor muscle or mono-/bi-articular muscle) in relation to differences in muscle activation pattern, activation level between agonist and antagonist, tendon moment arm length-angle relation and tendon stiffness. Alternatively, the observed difference in these well-trained cyclists in optimal muscle length for peak torque generation between knee joint flexors and extensors may possibly be explained by an existing adaptation of the knee and other participating joints to the specific kinematic pattern of the cycling movement. Such differences may not be observed in runners or other athletic disciplines with a similar training history; however, studies are required to scientifically investigate this deduction.

3.6 Conclusion

Despite improvements in cycling performance, 4 weeks of high intensity training did not cause a displacement of the angle-torque relationship towards higher torque values, and similarly, did not cause a shift of the optimum joint angle for torque production in either the quadriceps or hamstring muscle.

Higher torque values are caused mainly due to a training-induced increase in agonist activation (Reeves et al., 2004), and therefore improved muscle recruitment. However, considering that the subjects in this study were already well-trained cyclists, 8 sessions of high intensity training may not be a sufficiently strong stimulus to significantly change the pre-training peak torque values in these subjects.

The measurements of the angle-torque relationship after training were similar to the measurements before training in the quadriceps and hamstring muscles, for both trained and control group. Therefore, 4 weeks of high intensity training did not alter the angle-torque relationship, and consecutively did not alter the length-tension relationship

in these muscles. However, angle-torque measurements at more regular intervals may provide better insight into the perhaps progressively changing angle-torque relationship in the exercised muscles, in response to a 4 week high intensity training period.

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Chapter 4

Summary and conclusion

This study investigated the relationship between performance changes after high intensity training and changes in the angle-torque relationship of the knee extensor and flexor muscles in well-trained cyclists, with the goal to improve our understanding of the adaptational mechanisms of muscle mechanics, in response to high intensity training. The results demonstrate improvements in absolute peak power output ($4.8 \pm 3.4\%$), relative peak power output ($5.5 \pm 3.6\%$), and in 40 km TT performance ($2.5 \pm 1.3\%$) after 4 weeks of high intensity training (HIT). Despite the performance improvements, the results do not indicate that high intensity training causes changes in the angle-torque relationship of the studied muscle groups.

The effects of specific training interventions, undertaken by well-trained cyclists, on skeletal muscle adaptation and on the specific factors that are involved in improving cycling endurance performance are not well understood (Hawley and Stepto, 2001). In fact, very little research has examined the adaptation of central and peripheral factors following HIT in highly trained athletes. Laursen et al. (2005) provide evidence that muscles adapt to high intensity interval training through improvements in aerobic energy metabolism and improved muscle buffering capacity, and propose that peripheral adaptations rather than central adaptations are likely to be responsible for the improved performances observed in well-trained endurance athletes.

A recent study (Chapman et al., 2007), however shows that neuromuscular adaptations appear to also contribute to improvements in performance after high intensity training in well-trained cyclists. Apart from the metabolic and neuromuscular adaptations that have been observed above, the muscle mechanical mechanisms that are involved in performance improvements in highly trained athletes, after high intensity training, are also not well understood (Hawley and Stepto, 2001; Laursen et al., 2005).

The relationship between exercise-induced muscle damage and shifts in optimum length for peak torque production towards longer muscle lengths has been well established (Brockett et al., 2001; Proske and Morgan, 2001; Prasartwuth et al., 2006).

Whitehead et al. (2003) have shown a shift towards longer muscle lengths for active tension generation and a larger decrease in peak torque towards longer muscle lengths, following eccentric exercise, while Paschalis et al. (2005) have shown that peak torque declines are greater at shorter muscle lengths, following the effects of eccentric exercise. Eccentric exercise is unique from other types of exercise, in that it routinely produces skeletal muscle damage and shifts in the angle-torque relationship in the muscles of untrained individuals (Brockett et al., 2001). It has been proposed that there are two shifts in optimal length for peak torque production after exposure to eccentric exercise (Lynn et al., 1998): (i) an acute shift due to disrupted sarcomeres, and (ii) a longer-term shift, reflecting the longitudinal addition of sarcomeres to the myofibrils. Both of these mechanisms increase the optimal muscle length for torque production, with the primary goal to protect against further damage from eccentric exercise. These adaptations naturally result in altered muscle mechanical properties, including changes in a muscle's length-tension relationship, as has already been discussed previously.

The damage of the contractile apparatus, following exposure to unaccustomed eccentric exercise, plays an important role in the altered mechanical properties of muscle tissue (Proske and Morgan, 2001). Muscle fibres with disrupted sarcomeres in series with still-functioning sarcomeres show a shift in optimum length for tension in the direction of longer muscle lengths. In this example the sarcomere length will be distributed non-uniformly along the lengths of the muscle fibres. The body of evidence in support of the sarcomere non-uniformity hypothesis can account for certain behaviour of skeletal muscle and for the observed differences in the effects from concentric and eccentric exercise (Proske and Morgan, 2001).

The ability of muscle to generate power is of functional importance to performance. Therefore, alterations to the torque-velocity or angle-torque relationship, following exercise-induced muscle damage, have implications for physical performance. According to Folland et al. (2001), recovery from eccentric muscle damage and the adaptations to strength training are two different processes. A post-training shift in optimal angle, as an adaptation to strength training, has been described in detail by Reeves et al. (2004). Their study provides data that indicate a post-training shift in optimal fascicle length for force generation towards longer resting fascicle lengths. In

addition, Reeves et al., (2004) explain that a muscle remains operating over the same portion of its sarcomere length-tension relationship, despite an increase in the fascicle lengths and the training-induced shift of the absolute fascicle length-tension relationship.

Mechanisms appear to be in place to closely regulate muscle-tendon interaction in response to different strain patterns; presumably to optimize the benefits of this function and to enhance the total power output of the muscle-tendon unit (Hoyt et al., 2005). According to Reeves, et al. (2004), the interaction between the contrasting effects, resulting from changes in fascicle length and tendon stiffness, play a role in the regulation of the angle-torque relationship in response to resistance training (Reeves, et al., 2004), but this phenomenon has yet to be investigated in more detail in response to high intensity training. However, changes in the interaction between muscle and tendon would be expected to further complicate the interpretation of changes in the length-tension and angle-torque relationships in response to a period of exercise training in general, and also in response to a period of high intensity training.

Skeletal muscle, when subjected to increased loading, has intrinsic strategies to ensure that maximal force is exerted over the same sarcomere length range (Burkholder and Lieber, 2001). This partly explains the results of this study, which indicate that the optimal angle for peak torque production does not change despite a period of high intensity training and significant improvements in athletic performance. However, identifying the exact mechanism that is responsible for these observations is beyond the scope of the study.

In this study, changes in the angle-torque relationship have been measured to investigate the mechanical properties of muscle in response to high intensity training. As previously mentioned, scientific data that explain the mechanisms for muscle adaptation in response to high intensity training in cyclists are very limited. However, mechanisms that are involved in muscle adaptations in runners have been investigated by Harber et al. (2004). They show that the contractile properties of myofibres from trained endurance runners are sensitive to alterations in training intensity. They further indicate that myofibre function is receptive to changes in run training, and interval training has been shown to be a potent stimulus for eliciting changes in cellular function

(Harber et al., 2004). More specifically, it appears that variations in run training alter the contractile function of slow-twitch fibres, whereas the fast-twitch fibres remain unchanged, despite changes in the intensity of the running training. The data therefore indicate that changes in training intensity cause changes in the physiology of the muscle cells (Harber et al., 2004), which in turn activate certain cellular mechanisms to facilitate adaptation to a change in the exercise environment.

Whilst it is possible to build a logical argument explaining why high intensity training induces changes in the angle-torque relationship of the muscles, and that this is associated with improved performance, the current study did not support this hypothesis. A limitation in the research design is that the angle-torque relationship was only measured before and after the training period, and was not measured at regular time intervals throughout the study. It is therefore questionable to what extent more frequent measurements of the angle torque-relationship during a period of high intensity training would have provided different results, and perhaps better insight into the muscle mechanical mechanisms that are associated with changes in performance after high intensity training.

Conclusively: Following high intensity training in well-trained cyclists, muscles improve their ability to generate peak power for cycling performance. Various mechanisms are involved in the performance improvements observed in previous exercise studies. However, there are no data available that explain the muscle mechanical mechanisms that are involved in performance improvements after high intensity training. Some mechanisms that are involved in the mechanical adaptation of muscle have been explained in association with the muscle damage that can be induced by eccentric exercise, but studies that investigate muscle mechanical adaptation in response to sport-specific training are limited to untrained or recreationally active subjects, and running studies. However, the results from this study do not support the hypothesis that the angle-torque relationship changes in response to 4 weeks of high intensity training in well-trained cyclists, and therefore indicate that additional mechanisms are involved. Existing data from running studies suggest that the myofibre contractile properties of trained endurance athletes are sensitive to alterations in training intensity (Harber et al., 2004). This phenomenon has so far only been observed in runners, but may provide a basis for conducting similar research in cyclists,

to provide more insight into the relationship between high intensity training and improved cycling performance in already well-trained cyclists.

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References

- Allen DG, Whitehead NP, Yeung EW 2005 Mechanisms of stretch-induced muscle damage in normal and dystrophic muscle: role of ionic changes. *Journal of Physiology* 567(3): 723-735
- Allinger TL, Epstein M, Herzog W 1996 Stability of muscle fibers on the descending limb of the force-length relation. A theoretical consideration. *Journal of Biomechanics* 29(5): 627-633
- Bergman BC, Butterfield GE, Wolfel EE, Lopaschuk GD, Casazza GA, Horning MA, Brooks GA 1999a Muscle net glucose uptake and glucose kinetics after endurance training in men. *American Journal of Physiology* 277:E81-E92
- Bergman BC, Wolfel EE, Butterfield GE, Gary D, Lopaschuk, Casazza GA, Horning MA, Brooks GA 1999b Active muscle and whole body lactate kinetics after endurance training in men. *Journal of Applied Physiology* 87(5): 1684-1696
- Bickel CS, Slade J, Mahoney E, Haddad F, Dudley GA, Adams GR 2005 Time course of molecular responses of human skeletal muscle to acute bouts of resistance exercise. *Journal of Applied Physiology* 98: 482-488
- Blazevich AJ, Gill ND, Bronks R, Newton RU 2003 Training-specific muscle architecture adaptation after 5-wk training in athletes. *Medicine and Science in Sports and Exercise* 35(12): 2013-2022
- Blazevich AJ, Cannavan D, Coleman DR, Horne S 2007 Influence of concentric and eccentric resistance training on architectural adaptation in human quadriceps muscles. *Journal of Applied Physiology* 103: 1565-1575
- Brockett C, Morgan DL, Proske U 2001 Human hamstring muscles adapt to eccentric exercise by changing optimum length. *Medicine and Science in Sports and Exercise* 33(5): 783-790
- Burkholder TJ, Lieber RL 2001 Sarcomere length operating range of vertebrate muscles during movement. *Journal of Experimental Biology* 204: 1529-1536
- Butterfield TA, Herzog W 2005 Is the force-length relationship a useful indicator of contractile element damage following eccentric exercise? *Journal of Biomechanics* 38: 1932-1937
- Butterfield TA, Leonard TR, Herzog W 2005 Differential serial sarcomere number adaptations in knee extensor muscles of rats is contraction type dependent. *Journal of*

Applied Physiology 99: 1352-1358

- Cameron-Smith D 2002 Exercise and skeletal muscle gene expression. *Clinical and Experimental Pharmacology and Physiology* 29: 209-213
- Chapman AR, Vicenzino B, Blanch P, Hodges PW 2007 Patterns of leg muscle recruitment vary between novice and highly trained cyclists. *Journal of Electromyography and Kinesiology* (in press)
- Coffey VG, Hawley JA 2007 The molecular bases of training adaptation. *Sports medicine* 37(9): 737-763
- Cramer RM, Langberg H, Magnusson P, Jensen CH, Schrøder HD, Olesen JL, Suetta C, Teisner B, Kjaer M 2004 Changes in satellite cells in human skeletal muscle after a single bout of high intensity exercise. *Journal of Physiology* 558(1): 333-340
- Folland JP, Chong J, Copeman EM, Jones DA 2001 Acute muscle damage as a stimulus for training-induced gains in strength. *Medicine and Science in Sports and Exercise* 33(7): 1200-1205
- Gibala MJ, Little JP, Van Essen M, Wilkin GP, Burgomaster KA, Safdar A, Raha S, Tarnapolsky MA 2006 Short-term sprint interval *versus* traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *Journal of Physiology* 575(3): 901-911
- Gleeson N, Eston R, Marginson V, McHugh M 2003 Effects of prior concentric training on eccentric exercise induced muscle damage. *British Journal of Sports Medicine* 37: 119-125
- Goto K, Nagasawa M, Yanagisawa O, Kizuka T, Ishii N, Takamatsu K 2004 Muscular adaptations to combinations of high-and low-intensity resistance exercises. *Journal of Strength and Conditioning Research* 18(4): 730-737
- Harber MP, Gallagher PM, Creer AR, Minchev KM, Trappe SW 2004 Single muscle fiber contractile properties during a competitive season in male runners. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology* 287: R1124-R1131
- Hawley JA, Stepto NK 2001 Adaptations to training in endurance cyclists. *Sports Medicine* 31(7): 511-520
- Hawley JA 2002 Adaptations of skeletal muscle to prolonged, intense endurance training. *Clinical and Experimental Pharmacology and Physiology* 29: 218-222
- Häkkinen K, Alen M, Kraemer WJ, Gorostiaga E, Izquierdo M, Rusko H, Mikkola J, Häkkinen A, Valkeinen H, Kaarakainen E, Romu S, Erola V, Ahtiainen J, Paavolainen

- L 2003 Neuromuscular adaptations during concurrent strength and endurance training versus strength training. *European Journal of Applied Physiology* 89: 42-52
- Herbert RD, Gandevia SC 1995 Changes in pennation with joint angle and muscle torque: *in vivo* measurements in human brachialis muscle. *Journal of Physiology* 482(2): 523-532
- Hoang PD, Gorman RB, Todd G Gandevia SC, Herbert RD 2005 A new method for measuring passive length-tension properties of human gastrocnemius muscle *in vivo*. *Journal of Biomechanics* 38: 1333-1341
- Holloway GP, Green HJ, Duhamel TA, Ferth S, Moule JW, Ouyang J, Tupling AR 2005 Muscle sarcoplasmic reticulum Ca²⁺ cycling adaptations during 16 h of heavy intermittent cycle exercise. *Journal of Applied Physiology* 99: 836-843
- Howald H 1985 Malleability of the motor system: Training for maximizing power output. *Journal of Experimental Biology* 115: 365-373
- Hoyt DF, Wickler SJ, Biewener AA, Cogger EA, De La Paz KL 2005 *In vivo* muscle function vs. speed I. Muscle strain in relation to length change of the muscle-tendon unit. *Journal of Experimental Biology* 208: 1175-1190
- Huijing PA 1998 Muscle, the motor of movement: properties in function, experiment and modeling. *Journal of Electromyography and Kinesiology* 8: 61-77
- Huijing PA, Jaspers RT 2005 Adaptation of muscle size and myofascial force transmission: a review and some new experimental results. *Scandinavian Journal of Medicine and Science in Sports* 15:349-380
- Jensen L, Bangsbo J, Hellsten Y 2004 Effect of high intensity training on capillarization and presence of angiogenic factors in human skeletal muscle. *Journal of Physiology* 557(2): 571-582
- Jones DA, Newham DJ, Round JM, Tolfree EJ 1986 Experimental human muscle damage: Morphological changes in relation to other indices of damage. *Journal of Physiology* 375: 435-448
- Josephson RK 1999 Dissecting muscle power output. *Journal of Experimental Biology* 202: 3369-3375
- Kristiansen S, Gade J, Wojtaszewski JFP, Kiens B, Richter EA 2000 Glukose uptake is increased in trained vs. untrained muscle during heavy exercise. *Journal of Applied Physiology* 89: 1151-1158
- Korzeniewski B, Zoladz JA 2003 Training-induced adaptation of oxidative phosphorylation in skeletal muscles. *Journal of Biochemistry* 374: 37-40

- Laursen PB, Jenkins DG 2002 The scientific basis for high-intensity interval training. *Sports Medicine* 32(1): 53-73
- Laursen PB, Shing CM, Peake JM, Coombes JS, Jenkins DG 2005 Influence of high-intensity interval training on adaptations in well-trained cyclists. *Journal of Strength and Conditioning Research* 19(3): 527-533
- Lynn R, Talbot JA, Morgan DL 1998 Differences in rat skeletal muscles after incline and decline running. *Journal of Applied Physiology* 85(1): 98-104
- Mademli L, Arampatzis A 2005 Behaviour of the human gastrocnemius muscle architecture during submaximal isometric fatigue. *European Journal of Applied Physiology* 94: 611-617
- McArdle F, Spiers S, Aldemir H, Vasilaki A, Beaver A, Iwanejko L, McArdle A, Jackson MJ 2004 Preconditioning of skeletal muscle against contraction-induced damage: The role of adaptations to oxidants in mice. *Journal of Physiology* 561(1): 233-244
- McHugh MP, Conolly DAJ, Eston RG, Gleim GW 1999 Exercise-induced muscle damage and potential mechanisms for the repeated bout effect. *Sports Medicine* 27(3):157-170
- McNeil PL, Khakee R 1992 Disruptions of muscle fiber plasma membranes. Role in exercise-induced damage. *American Journal of Pathology* 140(5): 1097-1109
- Morgan DL, Gregory JE, Proske U 2004 The influence of fatigue on damage from eccentric contractions in the gastrocnemius muscle of the cat. *Journal of Physiology* 561(3): 841-850
- Morgan DL, Claflin DR, Julian FJ 1996 The effects of repeated stretches on tension generation and myoplasmic calcium in frog single muscle fibres. *Journal of Physiology* 497(3): 665-674
- Narici MV, Maganaris CN 2006 Adaptability of elderly human muscles and tendons to increased loading. *Journal of Anatomy* 208: 433-443
- Netter FH Atlas of Human Anatomy USA: Ciba-Geigy Corporation, Section VII: plate 460-461;1989
- Nielsen JJ, Mohr M, Klarskov C, Kristensen M, Krstrup P, Juel C, Bangsbo J 2003 Effects of high-intensity intermittent training on potassium kinetics and performance in human skeletal muscle. *Journal of Physiology* 554(3) 857-870
- Paschalis V, Koutedakis Y, Baltzopoulos V, Mougios V, Jamurtas AZ, Giakas G 2005 Short vs. long length of rectus femoris during eccentric exercise in relation to muscle damage in healthy males. *Clinical Biomechanics* 20: 617-622

- Pincivero DM, Salfetnikov Y, Campy RM, Coelho AJ 2004 Angle- and gender-specific quadriceps femoris muscle recruitment and knee extensor torque. *Journal of Biomechanics* 37: 1689-1697
- Prasartwuth O, Taylor JL, Gandevia SC 2005 Maximal force, voluntary activation and muscle soreness after eccentric damage to human elbow flexor muscles. *Journal of Physiology* 567(1): 337-348
- Prasartwuth O, Allen TJ, Butler JE, Gandevia SC, Taylor JL 2006 Length-dependent changes in voluntary activation, maximum voluntary torque and twitch responses after eccentric damage in humans. *Journal of Physiology* 571(1): 243-252
- Proske U, Morgan DL 2001 Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. *Journal of Physiology* 537(2): 333-345
- Putman CT, Xu X, Gillies E, MacLean IM, Bell GJ 2004 Effects of strength, endurance and combined training on myosin heavy chain content and fibre-type distribution in humans. *European Journal of Applied Physiology* 92: 376-384
- Reeves ND, Narici MV, Maganaris CN 2004 In vivo human muscle structure and function: adaptations to resistance training in old age. *Experimental Physiology* 89(6): 675-689
- Ren J, Semenkovich CF, Gulve EA, Gao J, Holloszy JO 1994 Exercise induces rapid increases in GLUT 4 expression, glucose transport capacity, and insulin-stimulated glycogen storage in muscle. *Journal of Biological Chemistry* 269(20): 14396-14401
- Ross WD, Marfell-Jones MJ. Kinanthropometry. In: MacDougall, JD, Wenger HA and Green HJ. eds *Physiological Testing of the High-Performance Athlete*, Human Kinetics Books, Champaign, IL, USA, 1991, 223-308.
- Ross A, Leveritt 2001 Long-term metabolic and skeletal muscle adaptations to short-sprint training. *Sports Medicine* 31(15): 1063-1082
- Russel AP, Somm E, Praz M, Crettenand A, Hartley O, Melotti A, Giacobino J, Muzzin P, Gobelet C, Dériaz O 2003 UCP3 protein regulation in human skeletal muscle fibre types I, IIa and IIx is dependent on exercise intensity. *Journal of Physiology* 550(3): 855-861
- Sakamoto K, Aschanbach WG, Hirshman MF, Goodyear LJ 2003 Akt signaling in skeletal muscle: regulation by exercise and passive stretch. *American Journal of Physiology, Endocrinology and Metabolism* 285: E1081-E1088
- Saunders MJ, Evans EM, Arngrimsson SA, Allison JD, Cureton KJ 2003 Endurance

- training reduces end-exercise VO_2 and muscle use during submaximal cycling. *Medicine and Science in Sports and Exercise* 35(2): 257-262
- Swart J The efficacy of high intensity training prescribed using either heart rate or power on performance variables in well trained cyclists 2007, MPhil Sports Medicine thesis, University of Cape Town.
- Trappe S, Harber M, Creer A, Gallagher P, Slivka D, Minchev K, Whitsett D 2006 Single muscle fiber adaptations with marathon training. *Journal of Applied Physiology* 101: 721-727
- Twist C, Eston R 2005 The effects of exercise-induced muscle damage on maximal intensity intermittent exercise performance. *European Journal of Applied Physiology* 94: 652-658
- Westgarth-Taylor C, Hawley JA, Rickard S, Myburgh KH, Noakes TD, Dennis SC 1997 Metabolic and performance adaptations to interval training in endurance-trained cyclists. *European Journal of Applied Physiology* 75: 298-304
- Whitehead NP, Allen TJ, Morgan DL, Proske U 1998 Damage to human muscle from eccentric exercise after training with concentric exercise. *Journal of Physiology* 512: 615-620
- Whitehead NP, Morgan DL, Gregory JE, Proske U 2003 Rises in whole muscle passive tension of mammalian muscle after eccentric contractions at different lengths. *Journal of Applied Physiology* 95: 1224-1234

Appendices

Appendix A: Par Q Questionnaire

Human Biology, Faculty of Health Sciences

University Of Cape Town

UCT/MRC Research Unit of Exercise Science And Sports Medicine

Modified Physical Activity Readiness Questionnaire (PAR-Q)

| | | | |
|-------------|------------|-------------------|-------------------|
| Name | | Date | |
| DOB | Age | Home Phone | Work Phone |

Regular exercise associated with many health benefits, yet any change of activity may increase the risk of injury. Completion of this questionnaire is a first step when planning to increase the amount of physical activity in your life. Please read each question carefully and answer every question honestly:

| | | |
|------------|-----------|---|
| Yes | No | 1) Has a physician ever said you have a heart condition and you should only do physical activity recommended by a physician? |
| Yes | No | 2) When you do physical activity, do you feel pain in your chest? |
| Yes | No | 3) When you were not doing physical activity, have you had chest pain in the past month? |
| Yes | No | 4) Do you ever lose consciousness or do you lose your balance because of dizziness? |
| Yes | No | 5) Do you have a joint or bone problem that may be made worse by a change in your physical activity? |
| Yes | No | 6) Is a physician currently prescribing medications for your blood pressure or heart condition? |

| | | |
|------------|-----------|--|
| Yes | No | 7) Are you pregnant? |
| Yes | No | 8) Do you have insulin dependent diabetes? |
| Yes | No | 9) Are you 69 years of age or older? |
| Yes | No | 10) Do you know of any other reason you should not exercise or increase your physical activity? |

If you answered yes to any of the above questions, talk with your doctor BEFORE you become more physically active. Tell your doctor your intent to exercise and to which questions you answer yes.

If you honestly answered no to all questions, you can be reasonably positive that you can safely increase your level of physical activity gradually.

If your health changes so you then answer yes to any of the above questions, seek guidance from a physician.

| | |
|------------------------------|-------------|
| Participant Signature | Date |
|------------------------------|-------------|

Appendix B: Training Questionnaire

Department Of Human Biology, Faculty of Health Sciences
University Of Cape Town
UCT/MRC Research Unit of Exercise Science And Sports Medicine

**Physiological responses and training adaptations to fixed workloads and fixed
physiological stresses.**

Training Questionnaire

Name:

When did you first start cycling?

.....

Have you participated in any other forms of endurance sport? (If so, which
sport and when?)

.....

How many hours do you spend training each week?

Cycling

Other sports

At what level of competition have you participated?

.....

University of Cape Town

Appendix C: Subject information sheet (*Isokinetic torque testing*)

SUBJECT INFORMATION SHEET TO PARTICIPATION IN SCIENTIFIC RESEARCH PROJECT

The effect of high intensity training on the angle-torque relationship of the quadriceps and hamstring muscles in a group of elite cyclists

Dear Cyclist,

The MRC/UCT Research Unit for Exercise Science and Sports Medicine in conjunction with the University of Cape Town (U.C.T.) Postgraduate Division of Physiotherapy will be conducting a study to investigate muscular adaptations in the quadriceps and hamstring muscles in response to 4 weeks of high intensity cycling training. This study will be running concurrent with the high intensity cycling training study performed and supervised by Dr Jeroen Swart and Robert Lamberts.

This study will improve our understanding of muscle mechanical mechanisms in response to high intensity training. Results of this study may provide a measure of training effect after high intensity training and assist in developing an additional performance parameter for athletes. Such a measure would be an indicator of a muscle's ability to adapt to a strength training stimulus and of its susceptibility to muscular strain injury. It could therefore become a useful guide for the design of training programs.

Ethics approval for this study has been granted by the Ethics and Research Committee of the faculty of Health Sciences of the University of Cape Town.

The study will involve isokinetic testing on the Biodex dynamometer. All testing will be performed at the Biodex Laboratory at the Sports Science Institute in Newlands, Cape Town. The study will involve two testing session and a familiarization session, all of which will be performed on the same day and 30 minutes after your VO_{2peak} test on the Computrainer™.

Prior to the testing you will be required to perform a warm-up, consisting of sub-maximal cycling on the Computrainer™ for 5 minutes, where you will cycle at a self-selected cadence and resistance that you feel comfortable with, so as not to induce any significant muscle fatigue.

The testing will be performed on your self-selected dominant leg (i.e. the leg you would choose to use to kick a ball). The testing protocol will consist of seven successive maximum voluntary knee straightening and bending movements, which will be performed at a predetermined fixed speed. Each contraction will be performed over a 90° knee angle range, starting with the leg hanging in a vertical position at 90°. You will be instructed to kick your leg up and down as forcefully as you can, so as to ensure maximal effort of both, the upward and downward movements. It will be important to perform maximal effort contractions in both directions.

Important: You will be required not to engage in any weight training for your leg muscles during the testing and training periods.

The total time commitment for the study will be 3 sessions [familiarisation, before and after training] of a maximum duration of 45 minutes each.

Risks

Other than the risks associated with isokinetic muscle testing, this study will pose no other inherent risks. The risk related to with this testing is equivalent to the risk associated with normal gym training, primarily muscle injury. This risk will be minimized by thorough familiarization with the testing equipment and a standardized warm up before the testing procedures.

Benefits

Subjects will receive a summary of their results as well as the overall findings from this study. The individual results will include information regarding individual angle-torque curves and any training adaptations. Study results will be made available upon completion of the study.

Confidentiality

All records and results generated within this study will be stored in a computer database in a secure facility. All information will be kept confidential. A subject identification code will be

used for the purpose of processing statistical data. All participants will remain anonymous in any ensuing publication.

Voluntary participation

Participation in this study is voluntary. You are free to withdraw from the study at any time and without having to give reasons for your withdrawal. Please notify the investigator about your decision.

Informed consent

Subjects will be required to submit a completed informed consent form prior to participation in the study.

Questions

If you have questions relating to the study or the study procedure, or any other relevant concerns, feel free to contact the investigators:

Contact information

| Investigator Name | Telephone | Email |
|----------------------------------|-------------------------------|-------------------------------|
| Christel Rösemann | (021) 976 7365 / 083 494 7775 | christelrosemann@telkomsa.net |
| Associate Professor Mike Lambert | (021) 650 4567 | mlambert@sports.uct.ac.za |

Appendix D: Subject information sheet (*High Intensity Training*)

Department Of Human Biology, Faculty of Health Sciences
University Of Cape Town
UCT/MRC Research Unit of Exercise Science And Sports Medicine

Physiological responses and training adaptations to fixed workloads and fixed physiological stresses.

Subject information sheet

The MRC / UCT Research Unit for Exercise Science and Sports Medicine will be conducting a study to measure the effects of different training methods. This will help us determine the optimal training intensity.

In order to do this, we will require you to be familiarised with the protocol and equipment. You will be required to come into the Sports Science Institute of South Africa on 13 occasions in total over a period of 5 weeks.

On your first visit you will be asked to complete a questionnaire about the training you have done over the past 3 years. We will then take measurements of your body with skin callipers to determine body fat percentage and composition. You will then be asked to complete a 40 km time trial on an indoor device to which your own bicycle will be attached.

Four days after your first visit you will return to perform a VO_2 peak test. This test is done on the same device as the time trial test but will be a short duration test which will increase progressively until you can no longer continue. We will measure the air that you breathe in and out during this test by attaching a mask to your face.

Following the VO₂ peak test, you will perform a series of leg contractions on a Biodex dynamometer device. This is a machine which tests muscle strength and speed. You will do 7 maximum extensions and flexions of the knee joint of one of your legs. This test may cause some fatigue in the tested leg for a few minutes following the test. There is a minor risk of muscle strains, similar to that during normal exercise.

Six days after your first visit you will repeat the 40 km time trial.

One week after your first visit you will begin a four week training program.

During this training you will report to SSISA twice each week for a supervised training session which will be done on the Computrainer™ indoor trainer.

Between each of the supervised training sessions you will perform a training session of 90 minutes duration on one day (the intensity of which will be controlled by your heart rate) following which you will have a rest day.

After the four week training program, you will repeat the 40 km time trial and VO₂ peak test and biodex test.

It is important that you not consume any caffeine or any other stimulants on the day that the 40 km TT and VO₂ peak tests are performed.

You should consume a moderately sized meal about two hours before each of your visits to the institute. You should not drink any substances other than water in the two hours before coming to the institute.

Principal investigator: Dr Jeroen Swart

UCT/MRC Research Unit for Exercise Science and
Sports Medicine, PO Box 155, Newlands 7725.

Tel: 021-6504569

Co-investigators: Robert Lamberts

**UCT/MRC Research Unit for Exercise Science and
Sports Medicine, PO Box 155, Newlands 7725.**

Tel: 021-6504569

**Research and Ethics Committee of the Health
Sciences Faculty, University of Cape Town**

Mr. X. Fula

Tel: 021-4066492

University of Cape Town

Appendix E: Informed consent (*Isokinetic torque testing*)

MRC/UCT RESEARCH UNIT FOR EXERCISE SCIENCE AND SPORTS MEDICINE

INFORMED CONSENT FORM

The effect of high intensity training on the angle-torque relationship of the quadriceps and hamstring muscles in a group of elite cyclists.

Placing your signature in the space provided below serves as confirmation that:

- (a) You have been provided with a full and adequate oral and written explanation of the study and the testing procedures, including all possible risks involved in this study
- (b) you have had adequate time to read through and have understood the information sheet and the informed consent form, and that you are willing to voluntarily participate in this study
- (c) you understand the implications of your involvement in the study
- (d) you are aware of the possible risks involved in this study
- (e) you are aware of your right to withdraw from the study without reason or prejudice at any time, if you choose to do so
- (f) you are aware of the investigator's right to withdraw any subject from the study at any time
- (g) you may ask questions and discuss the study at any time
- (h) you are aware that all information recorded is confidential and anonymous
- (i) you participate in this study of your own accord and that you will not hold the investigators responsible for any loss or damage occurred during the study period.

| | | |
|------------------------------------|------------------------------|---------------|
| _____ Signature of Volunteer | _____ Name (Please print) | _____ Date |
| _____ Signature of Witness | _____ Name (Please print) | _____ Date |
| _____ Signature of Investigator | _____ Name (Please print) | _____ Date |

Appendix F: Informed consent (*High Intensity Training*)

Department Of Human Biology, Faculty of Health Sciences
University Of Cape Town
UCT/MRC Research Unit of Exercise Science And Sports Medicine

Physiological responses and training adaptations to fixed workloads and fixed physiological stresses.

Informed consent

Possible risks of participation

The methods used during this study pose no inherent risk to you, other than the risks of performing exercise.

Should any untoward events arise, effective treatment will be available in the form of on-site medical care. Thus, any potential risks will be minimized. Finally, the University of Cape Town has a no-fault insurance or public liability cover should some unforeseen event occur whilst you are participating in this study.

The study will be performed in accordance with the principles of the Declaration of Helsinki, ICH Good Clinical Practice (GCP) and the laws of South Africa.

Benefits

After the results have been analysed we will inform you of the significance of our findings.

We will also provide you with a comprehensive assessment of your performance tests and of the improvement during the training period.

You will also be entitled to a free 8 week training program which will be individualised and drawn up by Dr Swart.

Statement of understanding and consent:

I confirm that the exact procedure and techniques, and possible complications of the above tests have been thoroughly explained to me. I am free to withdraw from the study at any time, should I choose to do so. I understand that I may ask questions at any time during the testing procedure. I know that the personal information required by the researchers and derived from the testing procedure will remain strictly confidential and will only be revealed as a number in statistical analysis.

I have carefully read this form and understand the nature, purpose and procedures of this study. I agree to participate in this research project of the MRC / UCT Research Unit for Exercise Science and Sports Medicine.

Name of volunteer:

Signature:.....

Name of investigator:

Signature:

Date:

Principal investigator: Dr Jeroen Swart

UCT/MRC Research Unit for Exercise Science and Sports Medicine, PO Box 155, Newlands 7725.

Tel: 021-6504569

Co-investigators: Robert Lamberts

**UCT/MRC Research Unit for Exercise Science and
Sports Medicine, PO Box 155, Newlands 7725.**

Tel: 021-6504569

**Research and Ethics Committee of the Health
Sciences Faculty, University of Cape Town**

Prof. T. Zabouw or Mr. X. Fula

Tel: 021-4066492

University of Cape Town

Appendix G: Examples of graphs representing the angle-torque data for a single subject

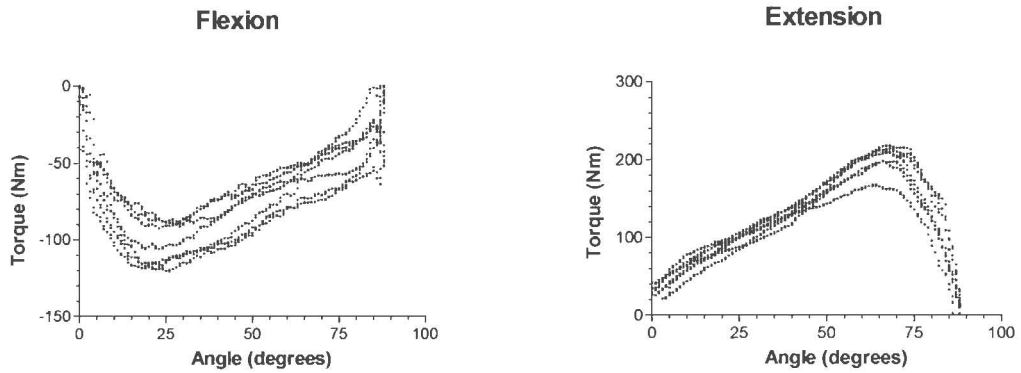


Figure G1: Angle-torque graph representing 7 contractions for flexion and extension respectively. Knee joint angles are represented on the x-axis, whereas torque values are represented on the y-axis.

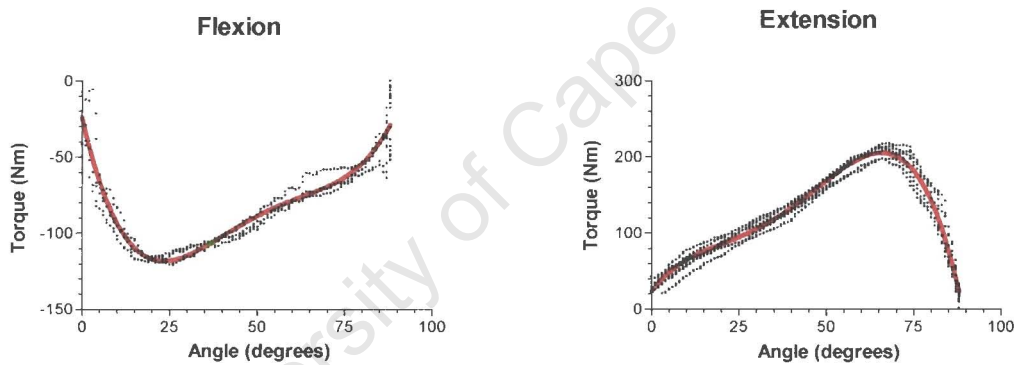


Figure G2: Fourth order polynomial nonlinear regression curve fit, representing an average peak torque value for the set of flexion and extension movements respectively. Outliers have been excluded.

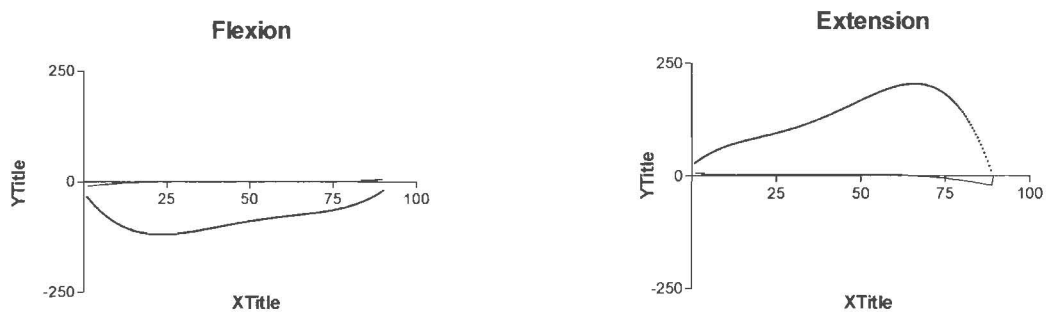


Figure G3: Differentiated curve on an XY Plot. The peak torque value is represented by the y-coordinate and the corresponding knee joint angle is represented by the x-coordinate. The maximum torque value is marked by the turning point of the curve.

Appendix H: Calculated data vs. Biodex data

Table H1: Peak torque for flexion and extension and angle coinciding with peak torque for flexion and extension respectively

| <u>Variables</u> | <u>Control</u> | | <u>Trained</u> | |
|----------------------------|----------------|--------------|----------------|--------------|
| | Pre | Post | Pre | Post |
| PT Flex (calculated) | 119.7 ± 10.1 | 117.3 ± 14.6 | 96.5 ± 18.9 | 102.9 ± 18.3 |
| PT Flex (Biodex) | 127.9 ± 11.8 | 124.6 ± 17.9 | 102.8 ± 19.1 | 110.0 ± 16.6 |
| PT Ext (calculated) | 186.2 ± 18.6 | 193.3 ± 9.9 | 170.5 ± 25.4 | 176.6 ± 20.8 |
| PT Ext (Biodex) | 199.0 ± 19.5 | 203.7 ± 12.6 | 180.1 ± 26.3 | 188.6 ± 21.4 |
| PT Angle Flex (calculated) | 36.0 ± 13.4 | 34.6 ± 13.1 | 38.8 ± 15.2 | 36.7 ± 15.5 |
| PT Angle Flex (Biodex) | 37.0 ± 9.9 | 30.2 ± 5.3 | 35.4 ± 13.2 | 33.9 ± 11.3 |
| PT Angle Ext (calculated) | 59.8 ± 6.7 | 62.2 ± 3.3 | 62.4 ± 3.6 | 61.6 ± 5.6 |
| PT Angle Ext (Biodex) | 62.4 ± 3.6 | 61.0 ± 6.3 | 60.9 ± 6.3 | 61.1 ± 6.3 |

Where;

PT = peak torque

Flex = Flexion

Ext = Extension

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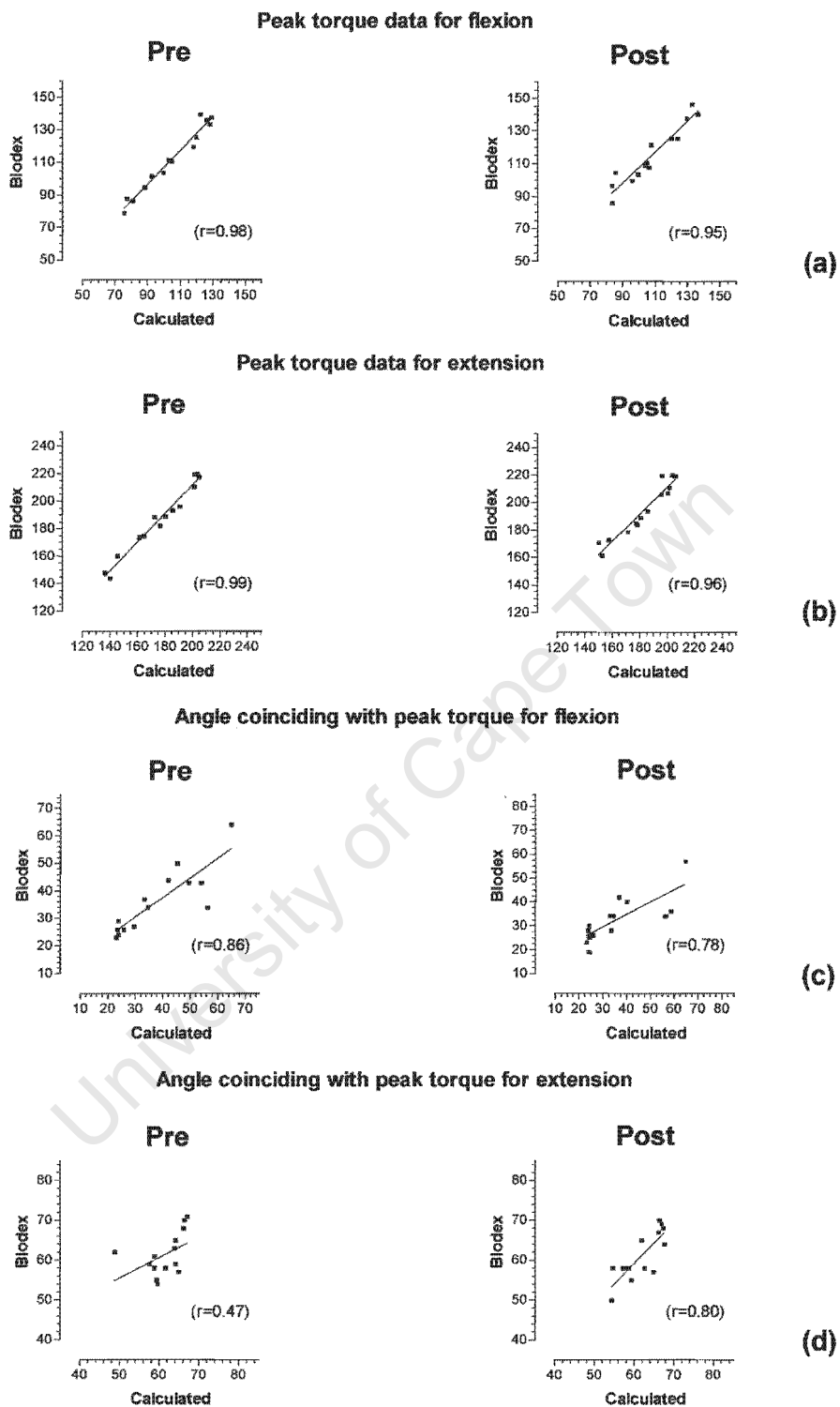


Figure H1: Graphs (a) and (b) represent peak torque for flexion and extension respectively, while graphs (c) and (d) represent angle coinciding with peak torque for flexion and extension respectively.