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# CONCENTRIC AND ECCENTRIC MUSCLE ACTIONS AND THEIR RELATIONSHIP WITH SPRINT PERFORMANCE.

by

**Martin N. Dowson**

M.Phil. Thesis

Submitted in partial fulfilment of the requirements for the award of Master of Philosophy of the Loughborough University of Technology

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## *ABSTRACT*

The aim of the studies described in this thesis was to investigate the differences in work and metabolism between concentric and eccentric muscle actions and to examine the relationship between these muscle actions and sprint performance.

The relationship between concentric and eccentric isokinetic muscle strength across three lower limb joints and sprinting performance, including the use of theoretical models was examined, in elite performers. Sprints were performed over 15 m and 35 m with times recorded over 0-15 m and 30-35 m. Using linear regression and expressing sprint performance as time, the strongest relationship found, amongst those joint actions and speeds tested, was between concentric knee extension at 240 deg.s$^{-1}$ and sprint performance (0-15 m times, r=-0.518, p<0.01, and 30-35 m times, r=-0.688 p<0.01 ). When 0-15 m performance was expressed as acceleration rather than time the correlation was improved slightly (r=0.590). However, when the data (0-15 m) was fitted to the allometric force model proposed by Gunther (1975), 77% of the variation in concentric knee extension torque at 240 deg.s$^{-1}$ could be explained by 0-15 m times, limb length (knee to buttocks) and body mass. The fitted parameters were similar to those from the theoretical model, demonstrating its validity. These findings suggest that the relationship between strength and sprint performance over 0-15 m (during the acceleration phase) is improved by taking an appropriate linear body dimension, as well as mass, into account.

The total work achieved during, and blood lactate response to, repeated knee extension and flexion at 120 deg.s$^{-1}$ were examined on two occasions, one performing eccentric actions for both movements and another performing concentric actions. Eccentric actions produced significantly greater average work (P<0.01) and lower blood lactate concentration (P<0.05) in comparison with concentric actions. Therefore despite producing greater work, less metabolic activity is required for eccentric actions than concentric during maximal voluntary actions. This may be due in part to the influence of neural inhibition which prevents the development of full muscle activation.

It has been postulated that during intermediate to fast muscle lengthening the actin-myosin cross-bridge is forcibly pulled apart without the breakdown of an ATP molecule. Thus it has been proposed that the energy cost of eccentric actions is only the amount necessary to maintain the cell in an activated state. However, following oral creatine supplementation, at a rate of 250 mg.kg bw$^{-1}$ per day, fatigue was reduced (p<0.05) and a lower concentration of blood lactate (P<0.05) was significantly found for repeated bouts of eccentric in comparison with concentric knee extension and flexion. Fatigue index was measured as 25.7 $\pm$ 14.9 % before and 16.0 $\pm$ 12.8 % after creatine supplementation. This finding indicates that eccentric actions may rely upon PCr to sustain work and delay the onset of fatigue which questions the current theory on energy utilisation during eccentric exercise. However it was postulated, to support the eccentric cross-bridge theory, that additional energy expenditure during repeated isokinetic eccentric actions may derive from either increased muscle damage and/or the involvement of antagonist activity.

The studies presented in this thesis have provided new methods for analysing the relationship between sprint performance and strength and identified the many different responses following concentric and eccentric actions. The established theories concerning energy utilising mechanisms for eccentric exercise have been questioned. The thesis concluded with recommendations on areas which need further investigation.

## Published papers

Dowson, M.N., Nevill, M.E., Lakomy, H.K.A., Nevill, A.M. and Hazeldine, R.J. (1995). Modelling the relationship between isokinetic muscle strength and sprint running performance. *J. Sport Sciences.*, (accepted subject to revision).

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# *CHAPTER 1. INTRODUCTION*

Skeletal muscle can produce force in either dynamic (concentric and eccentric) or static (isometric) contractions. Isometric forces are generated by a muscle action which permits no observable joint movement. Muscle actions are described as concentric and eccentric in nature when the force generated in the muscle is greater or less than respectively, the resisting movement of the limb, resulting in movement of that limb. The muscle actions examined were restricted to the concentric and eccentric actions only, rather than isometric actions. The review will follow Cavanagh's terminology by replacing muscle *'contraction'*, whether it follows, concentric or eccentric, with the term *'action'*, because the term does not accurately refer to all types of muscle actions (Cavanagh, 1988). Contraction medically is defined as 'shortening' (Medical Dictionary, 1987), however although there is some shortening of sarcomeres during both isometric and eccentric actions the muscle's length is fixed or lengthening respectively.

Generally in the scientific literature the mechanisms involved in concentric muscle actions have been well documented, whereas very little has been written on the mechanisms involved in eccentric actions. Since 1982 the introduction of isokinetic dynamometers have enabled examination of eccentric actions in a controlled clinical setting (Amundsen, 1990). This technical advance has inspired interest in the clinical use of eccentric testing and provided a reliable method of conducting research into the specific physiological responses to eccentric exercise.

An eccentric action is defined as a type of muscle action that involves an external force application with a resultant tension increase during physical lengthening of the musculotendinous unit (Albert, 1995). For this response to happen the external force has to be greater than the force the muscle attempts to develop by either an isometric or concentric action. Consequently the muscle lengthening continues until the external force decreases to a level equal or less than the force produced by the muscle during its attempt to shorten. Common examples of eccentric exercise include landing from a jump, losing an arm wrestle, walking downhill and returning a resistance to the starting position of a weight training exercise. Since the development of isokinetic dynamometers which can measure eccentric torque and work, there has been a renewed interest in how muscles act mechanically to resist

externally applied forces and in the physiological responses to eccentric exercise. However based on the current information in the literature it is still difficult to fully understand the actual mechanisms involved. One objective of this thesis was to contribute to a further understanding of the mechanical and functional differences between concentric and eccentric muscle actions.

The first study examines the relationship between each muscle action and motor performance. It is widely accepted that muscle strength is important for sprinting and numerous strength training studies have found that strength improvements did enhance performance (Chui, 1950; Peterson, 1975; Smith and Melton, 1981; Taiana et al., 1992). However there is ambiguity within the literature concerning which muscle actions are most strongly related to sprint performance. Therefore the first study examined the relationship between concentric and eccentric muscle strength and sprint running performance. The aim was to establish which actions relate strongly to sprint times, to find the best method to measure the relationship and to measure the torque differences between concentric and eccentric actions.

The remainder of the thesis is on the examination of the cross-bridge theory which has been postulated by numerous workers regarding how the contractile mechanisms and energetics function together during concentric and eccentric actions (Curtin and Davies, 1975; Huxley, 1969; Menard et al., 1991; Rall, 1985; Stauber, 1989). During concentric actions the actin and myosin filaments repeatedly bind and detach from each other (cross-bridge cycles) with the expense of one molecule of ATP broken down. However during eccentric actions it is postulated that myosin and actin are forced apart by the resistance acting on the lengthening muscle before ATP can be broken down (Curtin and Davies, 1975; Menard et al., 1991; Rall, 1985; Stauber, 1989). Investigators have based their theories on an examination of electrical stimulation of isolated animal muscle, measuring energy expenditure following sub-maximal exercise and on EMG measurements during maximal muscle actions in humans. The theory involving energy expenditure during eccentric actions is questioned by examining intact human muscle responses following maximal voluntary and eccentric muscle actions before and after creatine supplementation. If myosin and actin are forced apart before ATP hydrolysis in eccentric actions then creatine supplementation should be of less benefit than concentric actions.

The thesis is presented the seven chapters:

- The review of literature (Chapter 2), sets the conceptual background for the studies that follow.
- In the general methods (Chapter 3) the equipment, methods of analysis and general procedures followed during testing are presented.
- The first study (Chapter 4) examines the relationship between concentric and eccentric strength and sprint performance using both time and acceleration as the performance measure.
- The aim of the second study (Chapter 5) was to examine the work and blood lactate responses following repeated bouts of concentric and eccentric exercise.
- The purpose of the third study (Chapter 6) was to examine the effect of creatine supplementation on both concentric and eccentric repeated muscle actions.
- The final chapter (Chapter 7) integrates the findings of the three studies conducted for the thesis and presents possible theories involved in explaining the findings.

# *CHAPTER 2. REVIEW OF LITERATURE*

## 2.1 INTRODUCTION

Strength can be defined as the force or peak torque developed during a maximal voluntary action at a specific speed of movement (Sale and Norman, 1982). 'Torque' is usually defined as the force measured about a joint's axis of rotation and 'peak torque' is the point in the range of motion tested where the greatest torque was produced (Perrin, 1993).

During skeletal muscle activation there are three ways the muscle can act; concentric, eccentric and isometric. *Concentric* refers to a muscle action where the distance between the origin and insertion of the muscle becomes shorter whereas *eccentric* is where this distance increases. During an isotonic eccentric action the force developed by the active muscle produces less torque than the resisting movement of the limb but during concentric actions the force is greater by the active muscle. An *isometric* action is where the force developed by the active muscle and resisting movement is equal, hence movement does not occur. Isokinetic devices can measure torque from both concentric or eccentric actions as individuals exert force or angular movement against a force pad at a pre-determined velocity (Perrin, 1993).

The mechanisms involved and responses to high intensity concentric muscle actions have been well documented. However, only a few studies over the years have investigated and explained the mechanical changes during, and responses following, high intensity eccentric actions. This review will present the current research on strength in relation to the various differences that occur between concentric and eccentric muscle actions.

Firstly consideration will be given to the relationships between the two muscle actions and sprint performance. The need to investigate the different actions involved in strength is highlighted by illustrating the importance of both actions for sports performance. The current understanding of the determinants of muscle force is then reviewed with an explanation of the muscle's structure, the mechanisms involved during activation and the factors which influence force. The review will then consider energy supply during high intensity exercise and attempt to explain the possible mechanisms involved in fatigue, including the effect oral creatine supplementation has on high intensity exercise performance. To conclude, the review will attempt to

link the determinants of muscle force and energy supply by describing the differences in the excitation-coupling mechanism and metabolic responses between concentric and eccentric muscle actions.

## 2.2 THE RELATIONSHIP BETWEEN STRENGTH AND SPRINTING PERFORMANCE

Dynamic muscle strength is commonly considered an important component in sprint performance, for both maximal velocity and acceleration, by track sprinters, games players and coaches (Eckert, 1979; Radford, 1984).

Sprinting is defined as *running short distances at full speed* (Oxford Dictionary, Sixth Edition, 1978). This section refers to sprinting as running at maximal speed rather than slower or submaximal running. Relative to slower running the stride frequency is faster and stride length increased (Simonsen et al., 1985).

A sprint running cycle consists of two single strides, one for each leg. Each single cycle can be categorised into two phases, *ground* and *air*. Ground phase is the time the foot is in contact with the ground, whereas the air phase is the time period the foot is in the air. The ground phase can be sub-divided into two phases, *stabilisation* and *driving*, whereas air phase can be further categorised into 3 phases, *flight 1*, *swing* and *flight 2*. Flight 1 phase is the flight phase following the ground phase. The swing phase is where the opposite leg is in contact with the ground and the other leg is swinging like a pendulum. Flight 2 phase is the flight phase following the swing phase until the foot-strikes the ground (Simonsen et al., 1985). Figure 2.1 illustrates these phases of the running cycle and integrates the phases with other descriptions and joint movements stated by Elliot and Blanksby (1979).

During the ground phase hip extension, knee extension and ankle plantarflexion are predominately taking place, however at foot-strike the knee slightly flexes acting as a shock absorber. Hip extension continues mid-way into flight 1 and then flexes until the end of the swing phase, causing the high knee raise action. Once the other leg leaves the ground the hip begins to extend again. After toe-off the knee flexes bringing the heel close to the buttocks until the mid-swing phase where the knee extends until it prepares for the ground phase where slight flexion begins. Plantarflexion continues from the ground phase until the mid-swing phase. Dorsiflexion then takes place until immediately prior foot-strike to control the placement of the foot upon landing (Elliot and Blanksby, 1979; Mann, 1981; Simonsen et al., 1985).

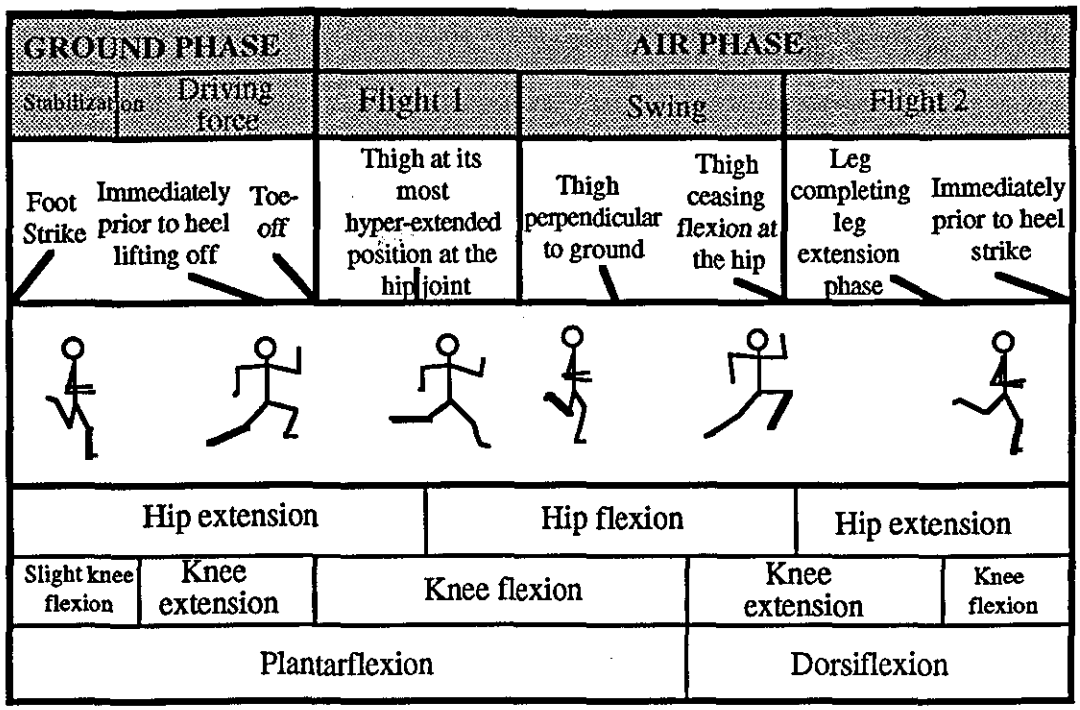



**FIGURE 2.1.** *Different stages in sprinting analysis (adapted from Elliot, B.C. and Blanksby, B.A., 1979; Mann, R.V., 1981; Simonsen, E.B., Thomsen, L., Klausen, K. 1985)*

Figure 2.2. summarises the muscle actions involved for each joint movement in each phase in a single leg cycle. The suggested joint movements and muscle actions have been illustrated based on measurements from synchronising muscle activity via EMG signals, muscle lengths observed via cinematography recordings and ground forces measured from a force platform or the free body segment method (Elliot and Blanksby, 1979; Mann, 1981; Simonsen et al., 1985). The action for each muscle or muscle group has been described as either concentric or eccentric during the different phases. The areas in the muscle action section in Figure 2.2 which are not shaded infer no significant muscle activity, suggesting the muscle is relatively relaxed during these periods. It is acknowledged that the muscle actions and limb movements may not be exact due to additional minor isometric actions from muscles, balanced muscular activity (static), small fluctuations in actions within a section and large individual variations (Mann, 1981). However, Figure 2.2. does summarise the major transitions that take place within each phase. Both concentric and eccentric actions take place for flexion and extension in the three lower limb joints except concentric ankle dorsiflexion.

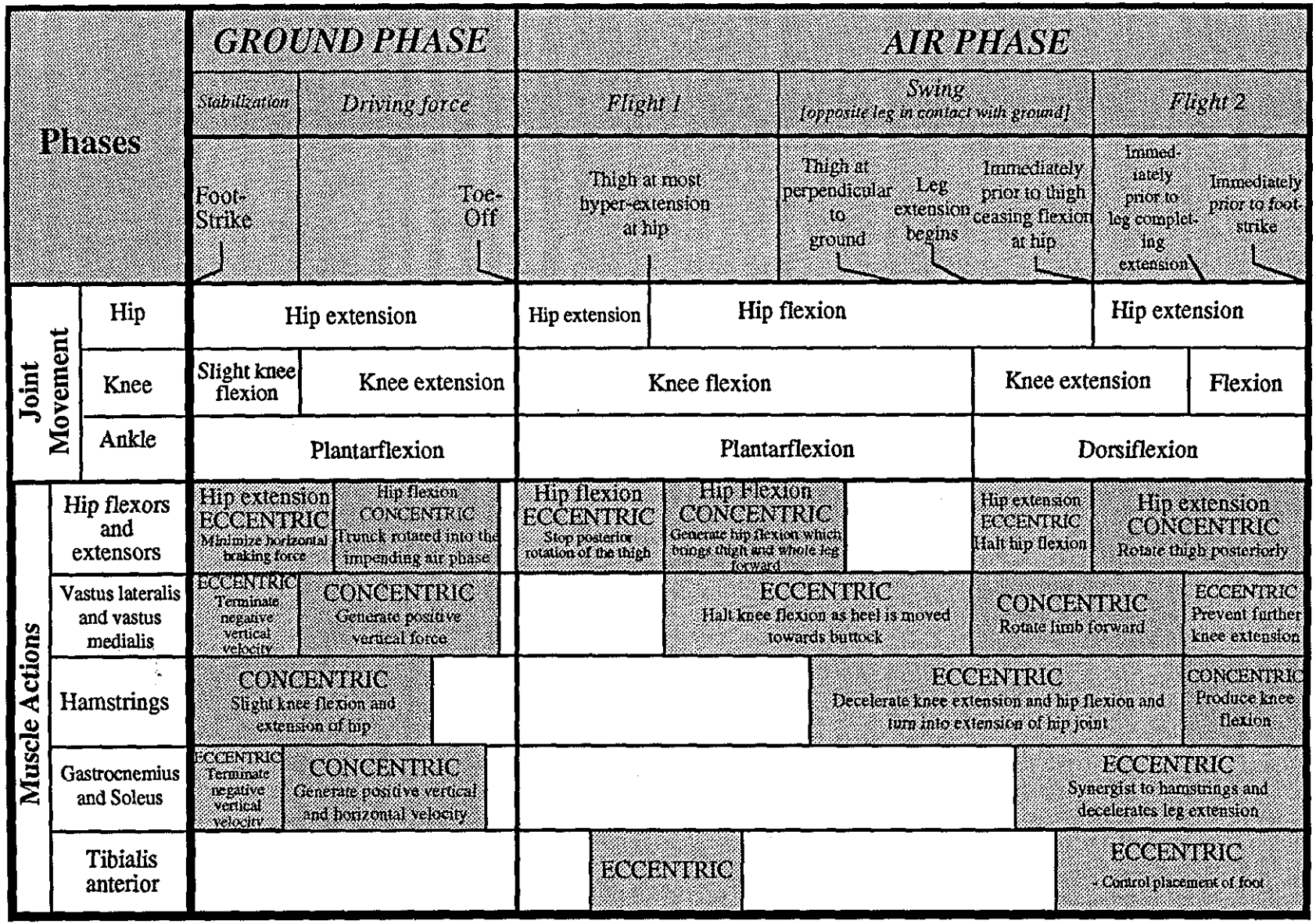



**FIGURE 2.2** *Joint movements and muscle actions during a single leg sprint cycle. (adapted from Elliot, B.C. and Blanksby, B.A., 1979; Mann, R.V., 1981; Simonsen, E.B., et. al., 1985 )*

Therefore the lower limb joints alternate between accelerating and decelerating the limbs during each movement cycle. The muscle moments described by Mann (1981) and Simonsen et al. (1985) show eccentric actions occur initially for the extensors for each joint during the ground phase following the foot-strike. The eccentric actions ensure total body stabilisation and act as 'shock absorbers' from the dominant force in preparation for the drive phase. Upon foot-strike the eccentric actions will also store kinetic energy in the series elastic components which can be partly re-used during the succeeding period of concentric work (Asmussen and Bonde-Petersen, 1974).

Alexander (1989) suggests that in theory, sprinting velocity is a direct result of the horizontal impulse (the product of force and time of contact) applied by the athlete during the ground phase. Various researchers have examined the body kinetic differences between elite and non-elite sprinters. For example, a significant difference was reported in the stride rate and support time for gold medal winners and place getters in the 1984 Olympic finals (Mann and Hernman, 1985). Sprinters have been compared with decathletes biomechanically and it was found the contact time of sprinters is very short and stride length and frequency larger (Kunz and Kaufmann, 1981). These findings suggest sprinters have a greater explosiveness during the ground phase, i.e., elite sprinters have the ability to generate a large force in a shorter period of time during the ground phase then non-elite sprinters. Therefore it appears that athletes who have greater strength in the responsible lower limb muscle groups would produce faster sprinting times if contact time remains the same. This theoretical viewpoint has lead many researchers into scientifically designed strength training programmes which are aimed specifically towards increasing sprinting speed.

### 2.2.1 The effect of strength training on sprint performance

Numerous authors have stated that strength training enhances sprint performance in short sprints of distances from 5-60 yards (4.6-54.9 metres) (Adams et al., 1991; Chui, 1950; Dintiman, 1964; Peterson, 1975; Schultz, 1967; Smith and Melton, 1981; Taiana et al., 1992). In each study which found a mean improvement in sprint times following strength training there appears to have been limitations such as, no statistical analysis (Chui, 1950; Peterson, 1975), no strength measurement implemented and no control over other training sessions, such as, sprint sessions (Taiana et al., 1992).

Other authors have reported that improvements in sprint performance are dependent on the type of strength training methods implemented. Smith and Melton (1981) detected sprint performance improvements, 10.1% over 40 yards (yd), only with the experimental group which performed fast velocity isokinetic training. In the slow velocity training group, performance decreased by -4.1% over 40 yards, over the six week training study. Another study employed strength training of all the major muscle groups and discovered that it was only the group that included the toe flexors that improved their performance in the acceleration (5-20 yards) and velocity (20-40 yards) tests (Adams et al., 1991).

In contrast some workers have postulated that strength training alone has no effect on sprint performance (Hoffman et al., 1990; 1991). For a 60 yard sprint, weight training alone did not significantly enhance performance, however when it was combined with sprint training there was a great improvement (Schultz, 1967). Dintiman (1964) found that sprint training in association with flexibility and strength training was the best combination to improve sprint performances at distances of 50 yards.

Ambiguity can therefore, be seen to exist within the literature, concerning the use of strength training to enhance sprinting performance. These findings suggest that non-specific strength training alone does not have a direct effect on sprinting performance. However benefits appear possible if strength training is performed on sprinting specific muscle groups at fast velocities in combination with flexibility and/or sprint training.

Therefore it is important to examine different muscle groups and find their relationship with sprinting performance to obtain a greater knowledge of the importance of strength for sprinting performance. This knowledge will aid more specific training prescription for sprint related athletes. However the literature regarding significant relationships between muscle strength in any of the lower limb movements at various velocities with sprinting performance is extremely limited.

**2.2.2 Relationships between strength and sprint performance**

Various studies have attempted to measure the relationship between muscle strength and sprint performance but most studies have documented only low

correlations (Anderson et al., 1991; Berg et al., 1986; Berger and Blaschke, 1967; Costill et al., 1968; Farrar and Thorland, 1987; Liba, 1967; Manning et al., 1988; Osinski, 1988). These studies have been described in Table 2.1. The first studies to identify a relationship between strength and sprint times were Berger and Blaschke (1967) and Liba (1967). Berger and Blaschke (1967) examined the correlation between one repetition maximum for a chin up and dip exercise and a 50 yd sprint but reported a poor relationship (r=-0.06). Similarly with female subjects Liba (1967) found a low correlation, r=0.41, between knee extension on a leg extension resistance device and 50 yd sprint performance.

From the mid 1980's strength was measured using isokinetic dynamometers. Various studies used this technique and examined the relationship between sprint times and isokinetic torque in the knee, hip and ankle joints using active college students (Anderson et al., 1991; Berg et al., 1986; Farrar and Thorland, 1987; Manning et al., 1988). However no significant correlations were found in any of these studies. For example, Anderson et al. (1991) reported that for university athletes the best predictor of 40 yard sprint times was knee flexion at 60 $deg.s^{-1}$ ($R^2$=0.33) using stepwise regression. These poor relationships may be partly due to the use of inappropriate strength and speed tests, the use of subject groups with varying sprint techniques and the examination of only one joint action.

Relationships have been reported, using elite sprinters, which measured between the sprinter's personal best 100 m times and isokinetic torque produced by the hip, knee and ankle joints (Alexander, 1989). This study was first to measure concentric and eccentric actions for each joint at different velocities. Significant correlations were found between strength during concentric knee extensor actions at 230 $deg.s^{-1}$ (r=-0.71) and eccentric dorsiflexion actions at 30 $deg.s^{-1}$ (r=-0.53) and 100 m sprint times. Concentric knee extension is an important joint movement in sprinting, especially during the ground phase by generating positive vertical force (Mann, 1981; Williams, 1985). However Alexander (1989) could not fully explain the relationship found between eccentric dorsiflexors and sprinting performance.

**TABLE 2.1.** *Previous literature measuring the relationship between strength measurements and sprint times*

| Authors | Subjects | Sprint distance | Strength Measurement | Statistical Analysis | Relationships |
|---|---|---|---|---|---|
| **Berger and Blaschke (1967)** | Male, n=38, college students | 50 yards ( 45.7 m) | 1 Repetition maximum for chin up and dip exercise | Correlation co-efficient | r= -0.06 |
| **Liba (1967)** | Female, n=52, College students | 50 yards ( 45.7 m) | Dynamometric measurement of leg extension | Pearson Product-Moment (P P-M) Correlation co-efficient | r=0.41 |
| **Costill et al. (1968)** | Male, n=76, P.E. University Students | 40 yards (36.6 m) | 1 Repetition maximum for Leg squat | Correlation co-efficient | r=0.20 |
| **Berg et al. (1986)** | Male, n=14, 10-11 year old | 30 m | Isokinetic torque for knee extension and flexion at 30 and 300 deg.s$^{-1}$ | (P P-M) Correlation Co-efficient | Best: Knee extension at 30 deg.s$^{-1}$, r=-0.374 |
| **Farrar and Thorland (1987)** | Male, n=52, College students | 40 and 100 yards (36.6 and 91.4 m) | Isokinetic torque measurements for the knee and hip at 60 and 300 deg.s$^{-1}$ | (P P-M) Correlation co-efficient | Best: Hip extension at 60 deg.s$^{-1}$ and 40 yd, r=-0.22 |
| **Osinski (1988)** | Male, n=211 and female, n=158, P.E. College students | 100 m | Isometric force measured with a foot-back indictive dynamometer | Correlation co-efficient | r=0.05 (male) r=0.04 (female) |
| **Manning et al. (1988)** | Male, n=31, College students | 40 yards (36.6 m) | Isokinetic torque for knee and ankle at 180 and 240 deg.s$^{-1}$ | Correlation co-efficient | Best: Knee extension at 180 and 240 deg.s$^{-1}$, r=-0.06 |
| **Anderson et al. (1991)** | Male, n=39, University Athletes | 40 yards (36.6 m) | Isokinetic torque for knee joint | Stepwise regression | Knee flexion at 60 deg.s$^{-1}$ $R^2$=0.33 |

Furthermore this study used a stepwise multiple regression for strength predictors of sprinting speed over 100m. Two variables were found to contribute significantly, concentric knee extension and eccentric hip flexion, both at fast velocities ($R^2$=0.67). Another study using college athletes have found significant correlations between isokinetic torque measurements and a 40 yd sprint time reporting relationships with hip flexion at 60 deg.$s^{-1}$ (r=-0.57) and hip extension at 60 deg.$s^{-1}$ (r=-0.56) and 240 deg.$s^{-1}$ (r=-0.41) (Guskiewicz et al., 1993).

Therefore it appears that relationships do exist between particular lower limb movements and sprinting performance if athletic subjects are used in the study and strength is measured across different lower limb joints. However it is important to investigate, not just which joint movements strongly relate to sprint performance but also which angular velocities, slow or fast, and which muscle action, concentric or eccentric. Each muscle action has a function in the sprint cycle but further understanding is required to determine which muscle action in which joints are best related.

Additionally sprint performance can be classified into three stages, acceleration, maintenance of top speed and deceleration. It is possible that the relationship between strength and sprint performance may differ between the acceleration stages of a sprint and at top speed. No study has examined the relationship between strength and sprint performance broken down into these stages.

In summary, further investigations are necessary to gain a more complete understanding of the relationship between strength during eccentric and concentric muscle action and sprint performance.

## 2.3 DETERMINANTS OF MUSCLE TENSION

To understand how a muscle functions and the differences between concentric and eccentric muscle actions it is first necessary to understand the structure and innervation of skeletal muscle and the action mechanism involved.

The following information in this section unless otherwise cited has been extracted from either Astrand and Rodahl (1986), Huxley (1969), Lakomy (1994), McArdle et al. (1991) and/or Vander et al. (1987).

### 2.3.1 Structure of skeletal muscle

There are more than 430 skeletal or voluntary muscles in the human body and each muscle consists of thousands of parallel cylindric shaped muscle cells called *muscle fibres*. Up to 150 muscle fibres are bundled together to form a *fascicle* (McArdle et al., 1991). Figure 2.3 shows the structure of skeletal muscle through cross-sections and the microscopic organisations.

Each part of skeletal muscle is wrapped and separated by layers of fibrous connective tissue:

- *Tendons* connects both ends of muscle to the outermost covering of the skeleton, the *periosteum*.
- *Epimysium* surrounds the entire muscle and blends into and joins intramuscular tissue sheaths to form the tendons.
- *Perimysium* surrounds the fascicle.
- *Endomysium* wraps each fibre which separates it from neighbouring fibres.

Force is transmitted directly through the connective tissue harness which results in a muscle action.

Each muscle fibre is enclosed within a thin plasma membrane called the *sarcolemma* which contains thousands of *myofibrils*, which are rod shaped structures parallel to each other. Each myofibril consists of several thousand *sarcomeres* which are made up of a series of repeating units. A sarcomere constitutes the functional unit of the muscle cell (Lakomy, 1994). The muscle cell is made up of two protein filaments, thin *(actin)* and thick *(myosin)* (Figure 2.3b). The thin filaments are made up of two chains polymerised into strands which are twisted together to form a helix (Figure 2.3ci). Two other

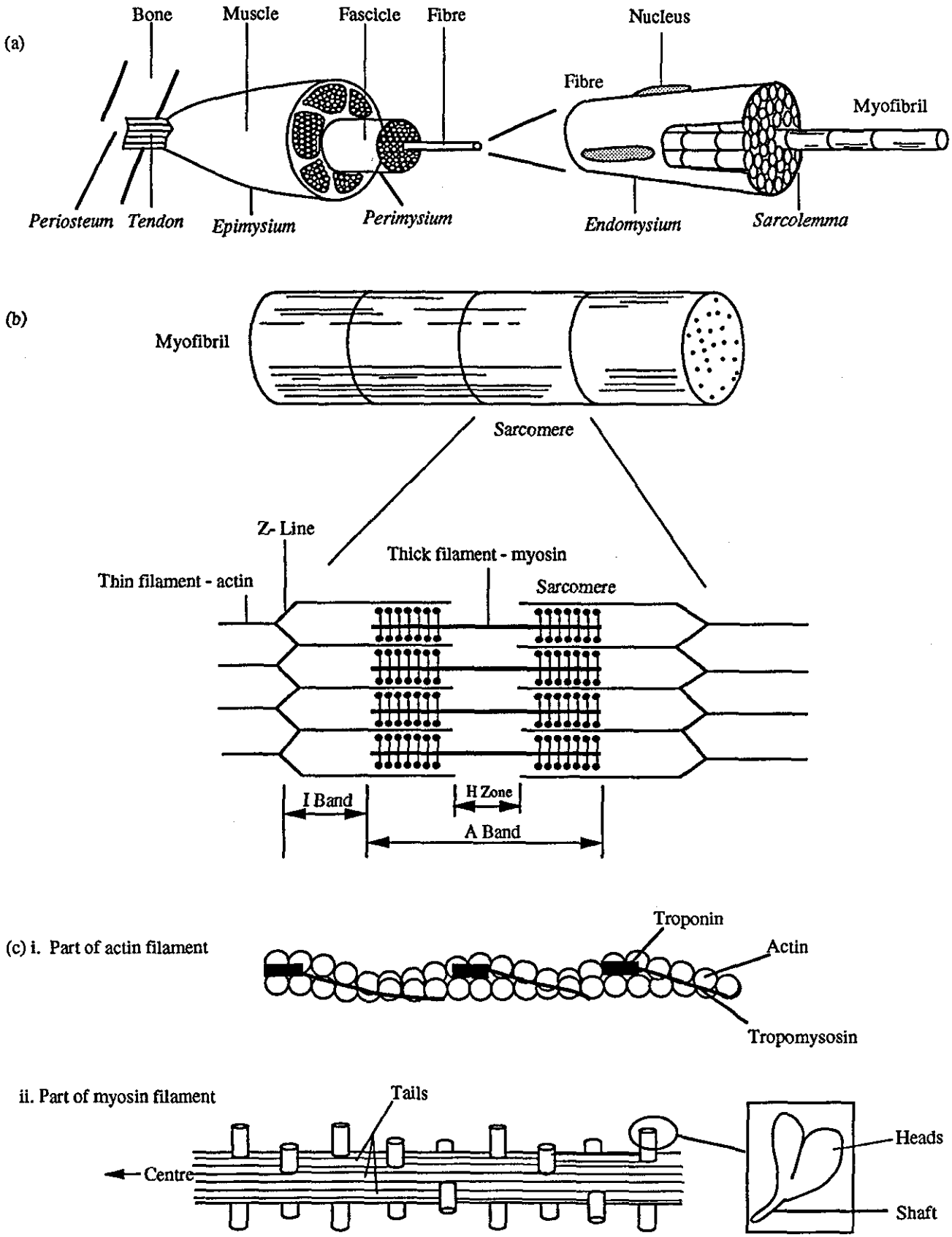



**FIGURE 2.3.** *Structure of skeletal muscle*
*2.3a shows the cross section of a muscle and the arrangements of connective tissue wrappings. 2.3b shows the microscopic organization of skeletal muscle of a sarcomere and 2.3c shows the protein filaments, actin (i) and myosin (ii) which make up the sarcomere. (adapted from Huxley, H.E., 1969 and Lakomy, H.K.A., 1994).*

important constituents of the actin helix are *troponin* and *tropomyosin*. Tropomyosin is a rod like structure distributed along the length of the actin filament in a twisted arrangement, bounded at regular intervals by a troponin complex. Troponin has a high affinity for calcium ions ($Ca^{2+}$) but during the absence of $Ca^{2+}$ it holds tropomyosin in a position which covers the binding sites on the actin filament. One end of the actin filaments attach together to form a narrow membrane, the *Z-line*, whereas the other ends are unattached. The thick myosin filament is made up of approximately 200 myosin molecules comprising of a tail with two heads at one end (Figure 2.3cii). These molecules are arranged into a bundle so the tails point to the centre of the filament and the heads protrude out towards the end (Lakomy, 1994).

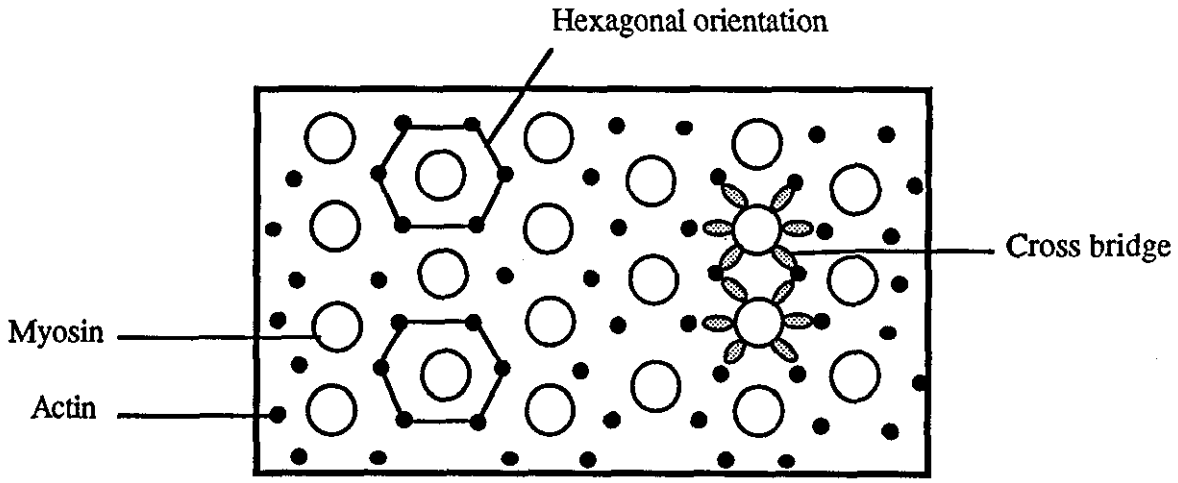



**FIGURE 2.4.** *Cross-section of a myofibril.*
*Note the hexagonal orientation of the action and myosin filaments as well as the cross-bridges. (adapted from Huxley, H.E., 1969 and McArdle, W.D., Katch, F.I. and Katch, V.L., 1991)*

The position of the thin and thick filaments in the sarcomere results in the overlap of the two filaments. This configuration allows the heads of the myosin to interact with the binding sites on the actin filaments. Figure 2.4 illustrates the hexagonal arrangement of actin and myosin filaments and the possibilities this arrangement gives for a myosin head to bind to an active site on one of six actin filaments. In a single muscle fibre, 100 μm in diameter and 1 cm long, there are about 8000 myofibrils and each myofibril consists of approximately 4500 sarcomeres. This means that a single fibre may have approximately 16 billion thick and 64 billion thin filaments (Vander et al., 1987).

The filaments are embedded in a fluid called *sarcoplasm* which contains soluble proteins, glycogen droplets, phosphate compounds and ions. Part of the sarcoplasm is called the *sarcoplasmic reticulum* which consists of tiny vesicles and channels between myofibrils. Located close, to but separated from, the sarcoplasmic reticulum is a tubular system, the *transverse tubular system* (T-Tubules). The T-Tubules are invaginated and start as an inward extension of the sarcolemma but do not open into the cytoplasm, surrounding the cell.

**2.3.2 Neuromuscular function**

A skeletal muscle action takes place when innervation occurs via a *motor neuron* which consists of a cell body (*soma*), numerous short dendrites which carry impulses into the soma and an axon which carries impulses away to skeletal muscle fibres. The end of the axon is located on a muscle fibre. This site is called the *neuromuscular junction* as shown in Figure 2.5a. The motor neuron and all the muscle fibres it innervates are collectively termed a *motor unit*. The greater the number of muscle fibres in a motor unit the greater the force it will produce. The number of muscle fibres within a motor unit varies in different muscles ranging from 10 to more than 1000 depending on the degree of fine motor control required.

A nervous impulse proceeds down an axon in the form of electrical energy. The axon is covered by a *myelin sheath* which is laid in segments with small spaces in-between known as *nodes of ranvier*. The myelin sheath insulates the axon from impulses from other motor neurons.

During rest the inside of the neuron has a net negative charge compared with the outside as shown in Figure 2.5b. Sodium ions ($Na^+$) and potassium ions ($K^+$) are predominantly located on the outside and inside respectively. In addition, the cell contains a high concentration on protein anions, which attract cations. The cell membrane is less permeable to $Na^+$ than $K^+$, thus the tendency for $K^+$ to diffuse out of the neuron is counteracted by the attraction from the anions. Therefore, there are more $Na^+$ outside the cell than there are $K^+$ inside which gives the inside a less positive or a net *negative charge*. When an impulse is transmitted down the axon the cell membrane becomes very permeable and both $Na^+$ and $K^+$ ions move from areas where they are highly concentrated to areas where they are less concentrated, i.e. $Na^+$ moves in and $K^+$ moves out. This causes a reversal in polarity which is termed an *action*

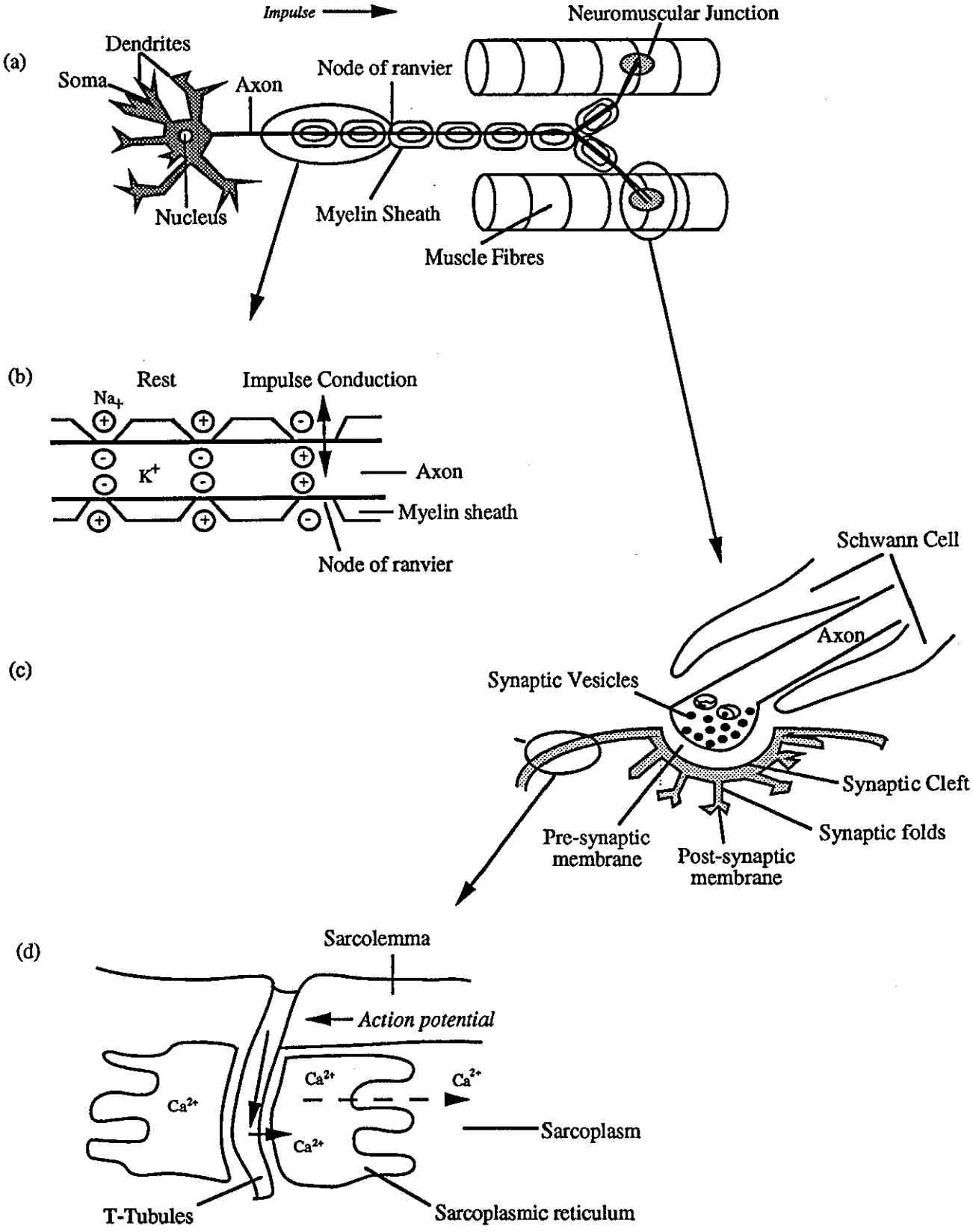



**FIGURE 2.5.** *The pathway of a nervous impulse transmission. 2.4a shows a m otor neurone and the muscle fibres it innervates. 2.4b shows the movement of Na +and K + during an impulse conduction. 2.4c shows the neuromuscular junction. 2.4d shows the pathway of an action potential in the muscle cell. (adapted from Fleck, S.J. and Kraemer, W.J., 1987; Lakomy, H.K.A., 1994)*

*potential*. This charge lasts for a few milliseconds and the membrane quickly becomes impermeable again and the charge is restored to its original state again. The nodes of ranvier allow action potentials to jump from one node to the next.

When the impulse reaches the neuromuscular junction it causes a release of a transmitter, acetylcholine, which is stored within the synaptic vesicles at the end of the axon, as shown in Figure 2.5c. Acetylcholine diffuses through the space between the two membranes, from the pre-synaptic membrane and into the post-synaptic membrane. Acetylcholine binds to a receptor and causes a change in permeability which if sufficient, causes an action potential to be created on the surface of the muscle, the sarcolemma. The electrical signal passes down the T-Tubules to the sarcoplasmic reticulum which causes the release of $Ca^{2+}$ illustrated in Figure 2.5d. It is currently unknown how depolarization of T-Tubules causes a release of $Ca^{2+}$ from the sarcoplasmic reticulum into the myofibrillar space (Grinnell and Brazier, 1981).

### 2.3.3 Sliding filament theory

The mechanism of a muscle action is not completely understood, however it is generally agreed that the changes in length of skeletal muscles (striated voluntary muscle) occur predominantly due to the sliding movements of actin and myosin filaments. During muscle shortening the filament length remains unchanged, but the actin moves inward into the arrays of myosin. This overlap causes the Z-lines to become closer and consequently the sarcomere shortens. This is referred as the '*Sliding filament theory*' and was first described by Huxley (1957).

At rest, tropomyosin blocks the binding sites on the actin which prevents the myosin heads from attaching to the actin. During activation an action potential passes through the T-Tubules and stimulates a muscle action via an *excitation-coupling cycle*, which can be broken down into seven steps as illustrated in Figure 2.6:

(1) $Ca^{2+}$ is released from the sarcoplasmic reticulum into the fluid surrounding the myofibrils, the sarcoplasm. At rest the sarcoplasm is mostly free from $Ca^{2+}$.

(2) $Ca^{2+}$ binds to the receptor sites on the troponin molecule.

(3) A change occurs in the troponin-tropomyosin complex which removes the tropomyosin rods from blocking positions and allows the head of the myosin to react with the binding sites on the actin filament.

(4) Myosin heads bind to the adjacent exposed site on actin molecules creating a *cross-bridge* energised by an ATP molecule which is bound to the myosin head.

(5) The cross-bridge undergoes an energy yielding conformational change which causes movement as actin is pulled along the myosin filaments. Tension is created by this conformational change.

(6) The myosin head 'reloads' with a fresh ATP which immediately dissociates the actin from myosin.

(7) The enzyme *myosin ATPase* becomes activated and the ATP located on the myosin head is broken down. The splitting of ATP occurs before the myosin reattaches to actin, however the ADP and inorganic phosphate remains bound to the myosin head.

At this point the cycle can continue from step (4) if $Ca^{2+}$ remains present on the binding sites. However if $Ca^{2+}$ is released from troponin then the binding sites become covered again and cross-bridge attachments are prevented and switch back to their original conformation. The $Ca^{2+}$ is transported from the troponin and the myofibrillar space across the membrane and back to the sarcoplasmic reticulum. This transport mechanism is generated by the *calcium pump* which is energy consuming, i.e. one ATP is hydrolysed for the transport of two $Ca^{2+}$.

**2.3.4 Determinants of force**

Unless otherwise cited the following information presented has been extracted from Lakomy (1994).

The degree of force developed in a muscle depends on the number of myofibrils activated in parallel with each other. The more myofibrils activated, the greater the force, therefore the overall force created by the muscle depends on the number of attached cross-bridges during a unit of time. Hence there are two aspects to consider, the total number of

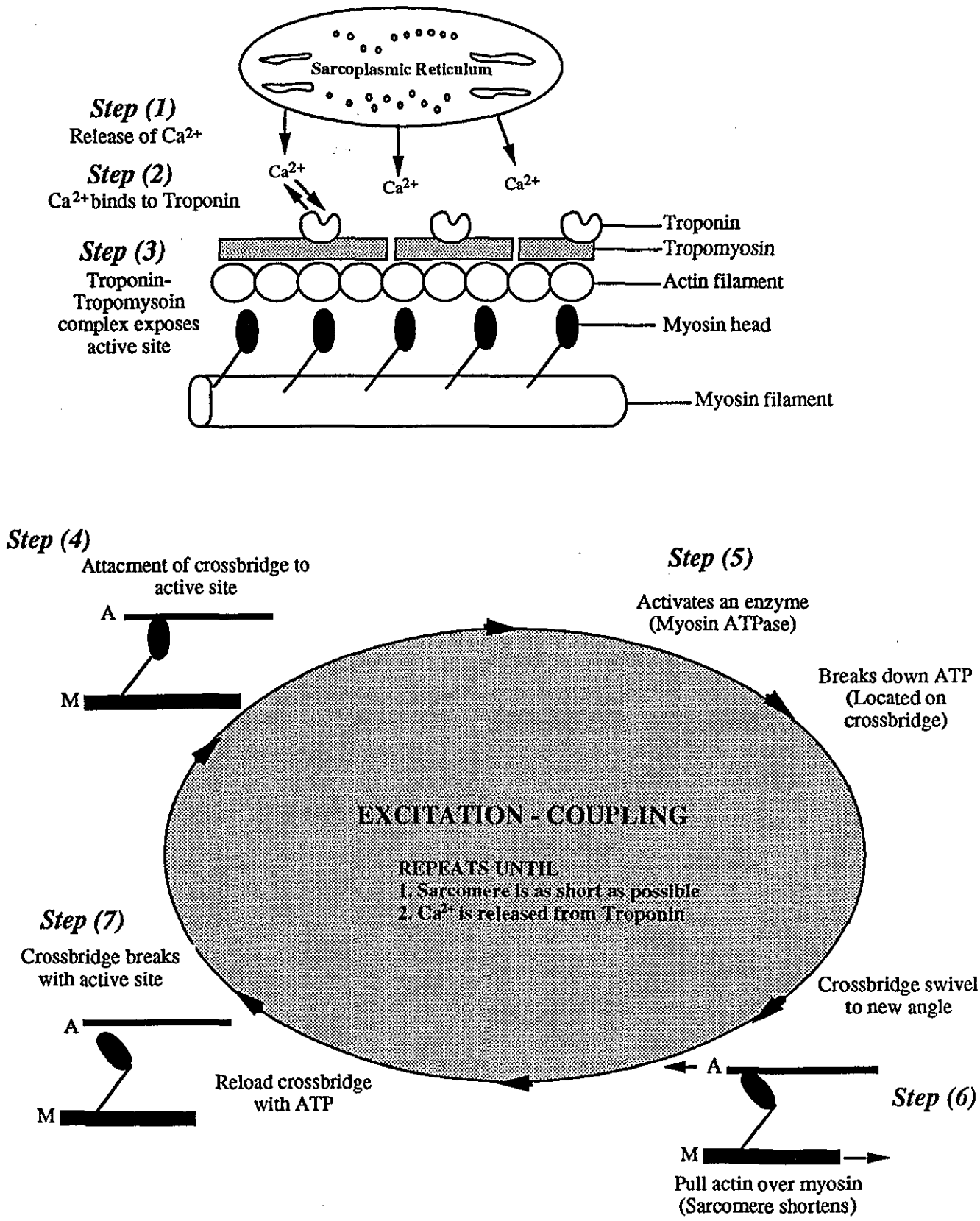



**FIGURE 2.6.** *Excitation-Coupling Cycle*
*The excitation-coupling cycle is broken down into seven steps from the release of $Ca^{2+}$ to the breakage of the crossbridge.*

cross-bridges being created and the duration of each cross-bridge attachment. There are a number of factors which influence these aspects and the amount of force generated. The following sections are an attempt to group these factors into those which influence the number of cross-bridge attachments and those which influence the duration.

**2.3.4.1 Factors influencing the total number of cross-bridges being created**

There are three main factors which influence the total number of cross-bridge attachments which are; length of sarcomere, number of motor units activated and angle of pennation.

**Length of sarcomere**

The extent of the overlap between the actin and myosin filaments affects the number of cross-bridges that can be formed. The overlap of the filaments is near optimal for cross-bridge attachments at the natural resting length of the muscle. If the muscle shortens, the actin filaments start to overlap each other and if the muscle is lengthened, then the overlap decreases, as shown in Figure 2.7. In both instances the number of binding sites available for the myosin heads decreases, thus the number of cross-bridges that can be created is reduced.

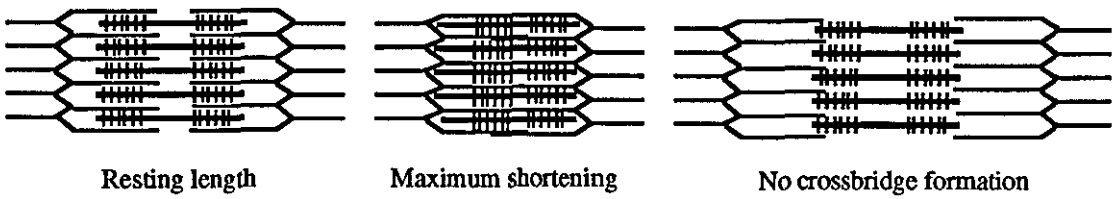



**FIGURE 2.7.** *Structural differences in sarcomere length (adapted from Lakomy, H.K.A. 1994)*

However force can also be generated from the contribution from the elastic structures. As the muscle is stretched beyond its resting length, muscle tissues are stretched which creates additional force. This contribution is referred as the *passive component* and becomes greater as the sarcomere length increases. Thus the overall force that a muscle action can develop is the sum of the force generated from cross-bridge attachments (active) and the passive component, as shown in Figure 2.8.

**Number of motor units activated**

As described in section 2.3.2, a motor unit comprises of a motor neuron and all the muscle fibres it innervates. The motor neuron will activate all or none of the muscle fibres belonging to that motor unit. Thus the more motor units activated the more myofibrils and subsequently the more cross-bridges are activated which all reflect the resultant force of the muscle. Each activated motor unit will make a different contribution to the force created in the muscle depending on the number of fibres within each motor unit and the total cross sectional area of each fibre. The greater the cross-sectional area of each fibre the greater the number of parallel myofibrils and sarcomeres. Thus a fibre with a large cross-sectional area will generate more force than one with less.

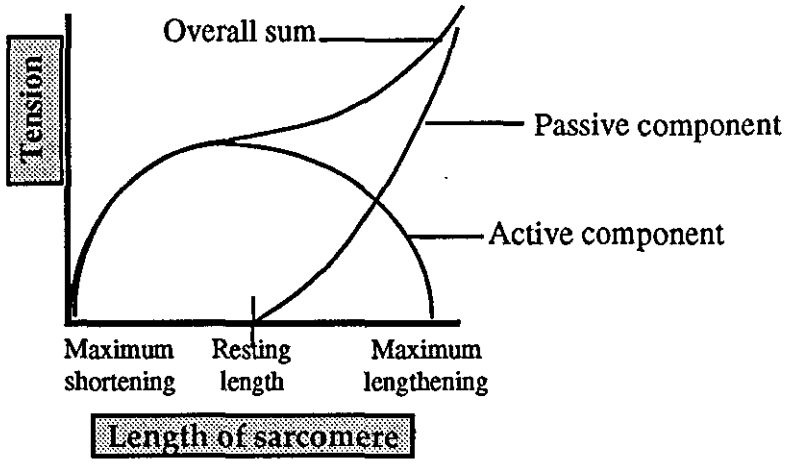



**FIGURE 2.8.** *The relationship between the overall force and the active and passive components during different sarcomere lengths.*

**Angle of pennation**

Fibres in some muscles lie parallel to the long axis of the muscle, whereas others fibres are arranged parallel together at an angle relative to the line of pull of the muscle. The latter are called pennated muscles and the angle between the muscle fibres and line of pull is called the *angle of pennation*. This is illustrated in Figure 2.9. The force produced is along the line of each fibre, hence the greater the angle of pennation the smaller the proportion of the force is produced along the long axis of the muscle. However the greater the angle of pennation the more fibres there are per unit of volume, which results in a greater number of cross-bridge attachments.

Therefore there is a trade-off between the number of cross-bridge attachments and the angle of pennation of the muscle. The optimal angle to transmit force is around 30-45°. However, although the maximum force occurs as the angle increases up to 45°, the range that a muscle can shorten decreases. Therefore the angle of pennation depends on the function of the muscles, e.g. some muscles require a large range of movement without high force output whereas others require large force with a small range of movement.

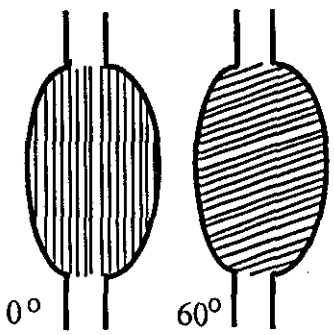



**FIGURE 2.9.** *Different angle of pennation of a muscle. (adapted from Lakomy, H.K.A. 1994)*

**2.3.4.2 Factors influencing the duration of cross-bridge attachments**

There are three possible factors which can influence the duration of a cross-bridge attachment, frequency of stimulation, fibre types and the velocity of movement.

**Frequency of stimulation**

A factor which influences the force developed for each motor unit is the frequency of stimulation, i.e. the way a motor unit is stimulated affects the magnitude of the force it develops and the duration of the activation of muscle fibres. A low force production results from a 'one off' stimulation which is called a *muscle twitch*. However if the stimulus is quickly repeated the muscle twitch overlaps and a *summation* occurs which creates greater force. Finally if the twitches are repeated sufficiently quickly a complete *tetanus* is developed which results in optimum force. The frequency of firing required to produce a complete tetanus for a motor unit stimulated individually appears to be approximately 50 Hz for human limb muscles (Enoka and Fulgevand, 1993).

**Individual fibre types**

The biochemical and physical characteristics of muscle fibres have been classified into different types, ranging from slow twitch (type I) to fast twitch

(type II). Type II fibres are suited to high intensity, short duration bouts whereas type I fibres are suited to endurance activity. Type II fibres develop high levels of force quickly which is due to a high activity level of the enzyme, myosin ATPase, that catalyses the hydrolysis of ATP releasing free energy for the fibre to shorten or lengthen. Therefore, type II fibres are able to act at a higher speed. Type I fibres develop force more slowly and resulting in a slower maximum speed of shortening.

Type II fibres motor units have very large motor neurons in comparison with type I fibres, hence the larger the motor neuron the harder it is to initiate a propagated action potential. The order in which motor units are recruited during an activity is relatively constant (Desmedt and Gadaint 1977). Therefore type II motor units are the last motor unit to be activated in a muscle action, e.g. if a light resistance is moved, type I motor units are predominately recruited, but if the resistance is increased, type II motor units are recruited.

**Velocity of muscle action**

An equation has been derived based on experimental work on isolated muscles, to examine the relationship between maximum muscle force and muscle velocity during concentric muscle actions (Hill, 1938). The equation predicted a curved inverse relationship between force and velocity, i.e., maximum velocity was produced when the load was zero and the maximum force the muscle produced was at a velocity of zero. In vivo this curve found for concentric actions is not exactly the same as that predicted by Hill (1938) but it does have similar characteristics, i.e. as the velocity increases the maximum force produced decreases (Dern et al., 1947; Wilkie, 1950). A more recent investigation on frog muscles have found the force-velocity relationship exhibits a biphasic shape rather than the monophasic hyperbolic course (Edman, 1988). The breakpoint which separates the regions of different curvature occurs about the point of 78 % of the isometric force. The curve was found to become shallower between 78-100 % of the isometric force.

A significant proportion of the change in force with changing velocity is caused by the duration of the cross-bridge attachment which decreases as the velocity increases. The time taken for the conformational change in a myosin head during the attachment phase decreases from the increased relative

velocities of the actin and myosin filaments. Thus on average less cross-bridges can attach per unit of time, resulting in less tension.

### 2.3.4.3 Summary

The factors that influence muscle force are summarised in Figure 2.10. Some of these factors will be expanded upon later in the review, Section 2.7, when the differences between concentric and eccentric muscle actions are discussed.

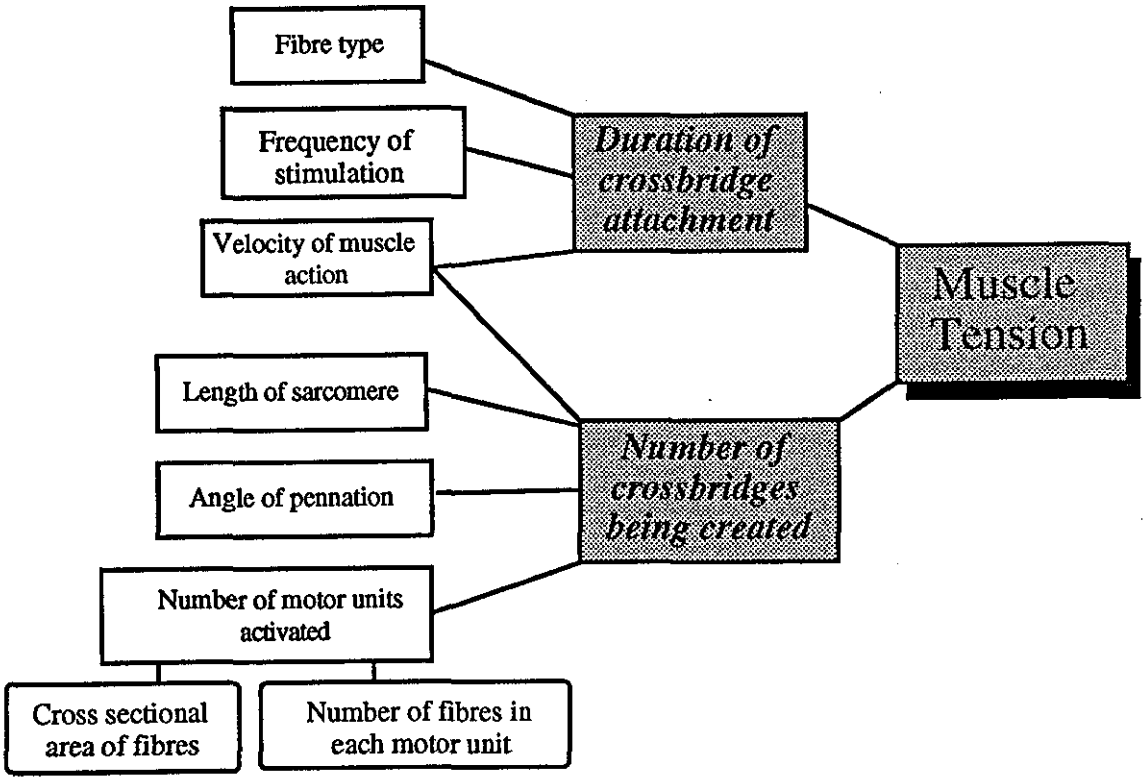



**FIGURE 2.10.** *Flow diagram of the determinants of force*

## 2.4 ENERGY SUPPLY DURING SHORT TERM HIGH INTENSITY EXERCISE

The immediate energy source for a muscle action is ATP (Sahlin, 1986), which is a mediator for energy transfer and is used in all cellular processes (McArdle et al., 1991). ATP is part of the adenine nucleotide family, adenine tri-, di-, and mono-phosphates (ATP, ADP and AMP respectively). Potential energy is stored in its chemical bonds and changed via free energy into kinetic energy (Astrand and Rodahl, 1986). The concentration of ATP in resting muscle is low, approximately 25.5 mmol.kg $dw^{-1}$ (Boobis et al., 1983; Cheetham et al., 1986; Jacobs et al., 1982; Nevill et al., 1989; Sahlin and Ren, 1989; Soderlund and Hultman, 1991; Spriet et al., 1987). Thus the muscle store of ATP is very limited which means it is essential that rapid resynthesis of ATP occurs.

Rapid ATP resynthesis is achieved via three major routes during high intensity exercise; phosphocreatine (PCr) breakdown, glycolysis which does not require oxygen and by oxidative metabolism involving the glycolytic pathway and the tricarboxylic acid (TCA) cycle (Sahlin and Ren, 1989).

### 2.4.1 Role of ATP

An ATP molecule is formed from a molecule of adenine and ribose (adenosine) linked to 3 phosphate molecules, as shown in Figure 2.11, with the two high energy bonds that link the two outermost phosphates.

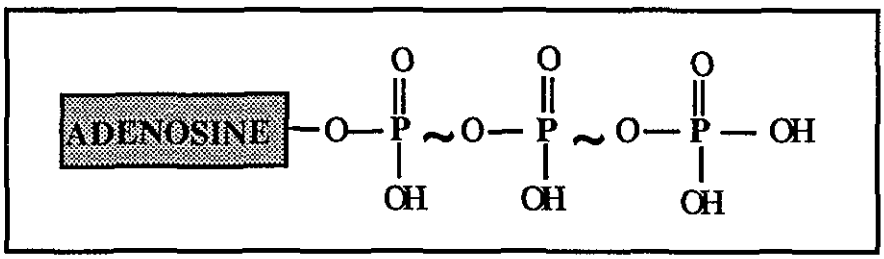



**FIGURE 2.11.** *Simplified structure of ATP.*
*The symbol ~ represents the high energy bonds. (From McArdle, W.D., Katch, F.I. and Katch, V.I., 1991)*

When ATP joins with water in a process called *hydrolysis*, catalysed by the enzyme ATPase the outmost phosphate bond is broken and a new compound is formed, adenosine diphosphate (ADP). ATP may be converted to and from ADP in this reversible reaction (1).

$$ATP + H_2O \xleftrightarrow{ATPase} ADP + Pi + Energy \quad (1)$$

ATP stores are limited and last only a few seconds if the muscles are acting maximally (Sahlin, 1978). However investigators have found ATP concentrations decrease only by a small amount during high intensity exercise (Hermansen, 1981), thus there must be resynthesis at an approximately equal rate to utilisation (Sahlin, 1978, Sahlin et al., 1978).

One mole of ATP can also be formed when 2 moles of ADP are used which produces AMP as shown in reaction (2). This acts as a buffer reaction to maintain ATP concentrations.

$$2\ ADP \xleftrightarrow{Adenylate\ Kinase} ATP + AMP \quad (2)$$

A further hydrolysis reaction breaks the final high energy terminal phosphate group from AMP, which is subsequently taken up by inosine to form inosine monophosphate (IMP), shown in reaction (3).

$$AMP + H_2O + H^+ \xleftrightarrow{AMP\ Deaminase} IMP + NH_4^+ \quad (3)$$

The relative concentrations of these three nucleotides (ATP, ADP and AMP) exert control upon cellular metabolism, i.e. ADP and AMP and Pi are powerful activators of the rate limiting enzyme in the glycolytic pathway, phosphofructokinase (PFK) (Sahlin, 1986). Activating PFK serves to increase the concentration of ATP by an increase in glycolysis and thereby restoring a high ATP/ADP ratio.

**2.4.2 ATP utilisation during high intensity exercise**

Several authors have investigated ATP utilisation during 30 s of high intensity sprint running and calculated the ATP turnover rate at being between 6.1-6.3 mmol.kg $dw^{-1}$ $s^{-1}$ (Cheetham et al., 1986; Nevill et al., 1989). Another factor influencing ATP turnover is the type of muscle fibre. A higher turnover in type II fibres has been found, 11.06 $\pm$ 1.53 mmol.kg $dw^{-1}$ $s^{-1}$ as

opposed to 7.32 ± 1.16 mmol.kg $dw^{-1}$ $s^{-1}$ in type I fibres (Greenhaff et al., 1992). It was suggested, based on these observations, that fatigue in maximal exercise is related to the inability to maintain the required rate of ATP resynthesis in type II fibres (Greenhaff et al., 1992).

In skeletal muscle the maximum depletion of ATP following exercise is about 40 % of the resting level (Gollnick and Hermansen, 1973). This percentage depletion is supported by McCartney et al., (1986) who found that muscle ATP was reduced by 40 % from resting concentrations after 30 s maximal isokinetic cycling. It is questionable whether or not the loss of ATP during high intensity exercise can be a main cause of fatigue as ATP stores are not completely depleted.

**2.4.3 PCr - ATP resynthesis**

Another high energy fuel used during high intensity exercise is phosphocreatine (PCr). PCr is utilised rapidly at the onset of exercise to resynthesise ATP via the *creatine kinase* reaction as shown in reaction (4). PCr binds with ADP and $H^+$ to produce ATP and creatine (Cr) and is thought to be the most powerful of all the processes which are used to regenerate ATP in the cell (Sahlin, 1986).

$$PCr + ADP + H + \xleftarrow{\text{Creatine Kinase}} \rightarrow ATP + Creatine \qquad (4)$$

The concentration of PCr in resting muscle is approximately 80 mmol.kg $dw^{-1}$ (Bogdanis et al., 1993; 1994a; 1994b; 1995a; Boobis, 1987; Sahlin and Ren, 1989; Soderlund and Hultman, 1991; Spriet et al., 1987). Following 40 s electrical stimulation in the quadriceps 93 % of the available PCr store was found to be utilised (Spriet et al., 1987).

Various studies have also found high rates of PCr degradation (Bogdanis et al., 1993; 1994a; 1994b; 1995a; Boobis et al., 1983; Boobis, 1987; Cheetham et al., 1986; Hultman and Sjoholm, 1983; Jacobs et al., 1982; Nevill et al., 1989). The rate of PCr utilisation is apparently linked to the intensity and duration of the exercise. ATP resynthesis from PCr breakdown has been reported to be 8-9 mmol.kg $dw^{-1}$ $s^{-1}$ during 3-4 s of electrical stimulation and 3.3 mmol.kg $dw^{-1}$ $s^{-1}$ during 10 s respectively (Hultman and Sjoholm, 1983). It would appear that the most rapid degradation of PCr occurs during the initial seconds of activity. Phosphocreatine concentration was found to fall to 40-66 % of

resting values following the first 10 s of a 30 s maximal isokinetic cycling bout whereas for the remaining 20 s ATP and PCr concentrations were maintained (Jones et al., 1985). A 57 % decrease in PCr concentration within the first 6 s of maximal ergometer cycling has been observed (Gaitanos et al., 1993). Thus it appears PCr is reduced markedly within the first few seconds of maximal exercise. ATP must be, therefore, resynthesised from other sources, as PCr could not exclusively maintain the supply during high intensity exercise as it is almost totally depleted within a few seconds.

**2.4.4 ATP - resynthesis from glycolysis**

ATP can be formed by the breakdown of glycogen or glucose in the absence of oxygen by substrate phosphorylation through a pathway known as *glycolysis.*

Glycogen stores at rest within skeletal muscle are between 250-400 mmol.kg $dw^{-1}$ (Bogdanis et al., 1994a; 1994b; 1995a; Boobis et al., 1983). Prior to exercise a difference in individual fibre types has been observed with higher concentration in type II than type I fibres (Greenhaff et al., 1992).

Glycolysis occurs in the cytoplasm of the cell and involves a chain of chemical reactions which convert either glycogen or glucose to *pyruvate* and which results in a net production of 2 moles of ATP. The breakdown of glycogen to glucose-6-phosphate is termed *glycogenolysis.* Degradation of glycogen is initiated by the action of the enzyme *phosphorylase* which forms *glucose-1-phosphate* (G-1-P). At the onset of exercise, adrenaline stimulates glycogen breakdown in the muscle via a rise in the concentration of cyclic AMP (cAMP). This rise leads to an increase in the activity of phosphorylase which is also controlled by other mechanisms such as $Ca^{2+}$ (Chasiotis et al., 1982).

The conversion from a glucose molecule into two molecules of pyruvate involves a sequence of ten enzymatic steps that create phosphate containing intermediates, as shown in Figure 2.12.

The ten steps can be divided into three parts. Part (A) involves steps 1 to 5 which converts a glucose molecule with six carbon atoms to two molecules of the aldehyde glyceraldehyde 3-phosphate that comprises of three carbon atoms. During steps 1 and 3 two molecules of ATP are used to add phosphate groups to glucose. Part (B) involves steps 6 and 7 which oxidise

the aldehyde group of each of the two glyceraldehyde 3-phosphate molecules into carboxylic acid and the energy from this reaction is coupled to the synthesis of two molecules of ATP. Part (C) involves the last three steps which forms 2 molecules of ATP, thereby repaying the original investment of ATP molecules hydrolysed in part A. However a total of four molecules of ATP are formed during part (B) and (C) providing a net gain of 2 or 3 ATP depending on whether glucose or glycogen was the substrate.

Key enzymes in this pathway are *phosphofructokinase* (PFK) and *pyruvate kinase*. Both enzymes catalyse essentially irreversible reactions (Newsholme and Leech, 1983). PFK is inhibited by an increased $H^+$ concentration, which prevents acidosis and possible death. PFK has been identified as the rate limiting enzyme in the glycolytic pathway (Newsholme and Leech, 1983). During high intensity exercise the substrate activity above the PFK reaction has been found to increase significantly in comparison to that beneath it (Boobis, 1987). Other investigators have found increased glucose 6-phosphate and fructose 6-phosphate late in exercise and a fall in fructose 1-6-phosphate between 10 s and 30 s (Cheetham et al., 1986; Jones et al., 1985; McCartney et al., 1986). According to Spriet et al. (1987) PFK activity is stimulated by AMP, ADP, Pi, cAMP, ammonium ($NH_4^+$), potassium, fructose-6-phosphate and fructose 1-6 biphosphate.

The redox (oxygenation) state of the tissue will determine if pyruvate will be converted to lactate or enter the TCA cycle (as described in Section 2.4.5) (Newsholme and Leech, 1983). During short term high intensity exercise pyruvate is predominantly reduced by the enzyme *lactate dehydrogenase* (LDH) as shown in reaction (5).

$$\text{Pyruvate} + \text{NADH} + H^+ \xleftrightarrow{\text{LDH}} \text{Lactate} + \text{NAD}+ \qquad (5)$$

The reoxidation of NADH in this reaction allows continuation of glycolysis or glycogenolysis by regenerating sufficient $NAD^+$ for step 5 in Figure 2.12 (Jones et al., 1985).

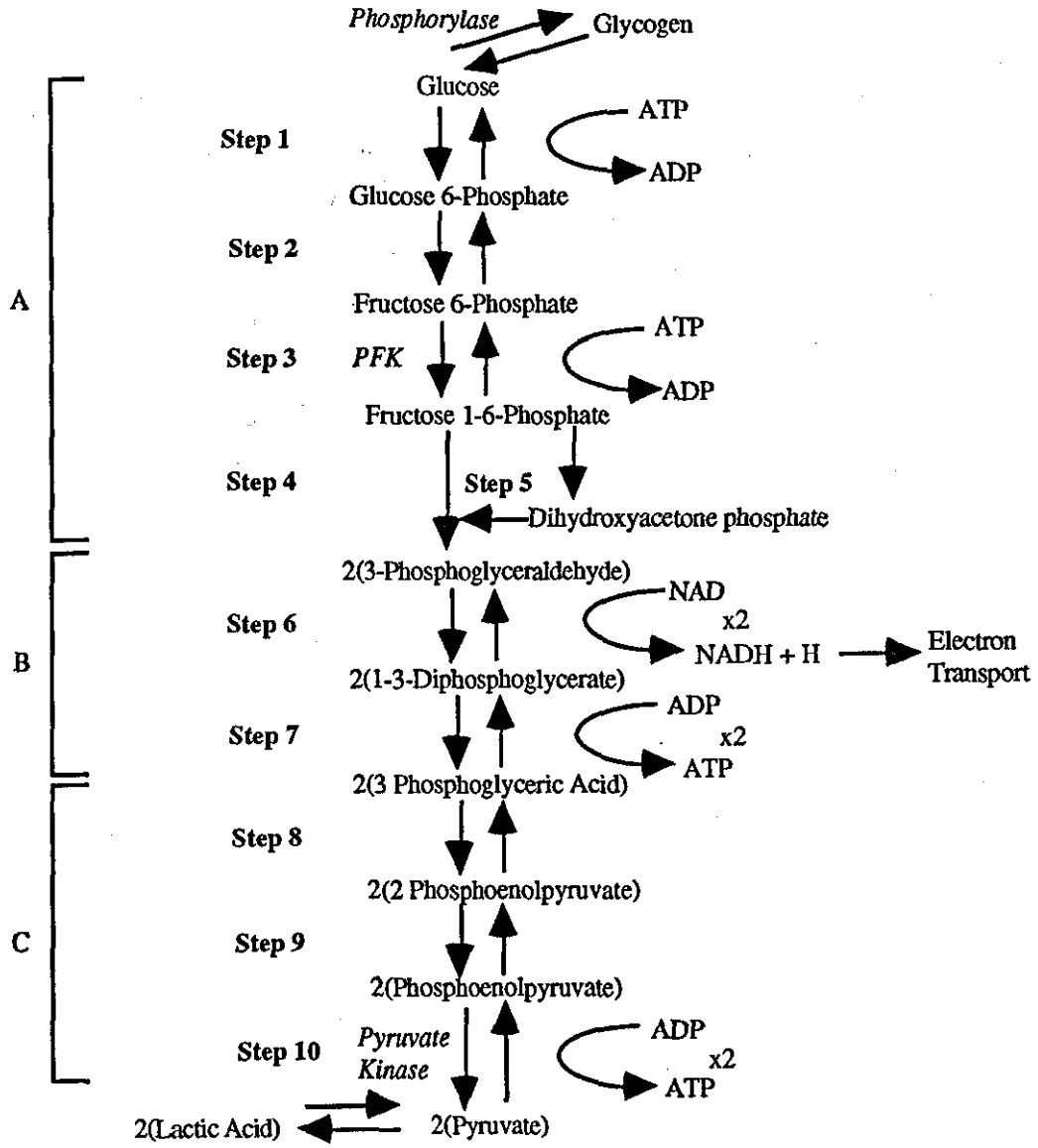



**FIGURE 2.12.** *Glycolytic pathway. (adapted from McArdle, W.D., Katch, F.I., and Katch, V.I., 1991)*

The net chemical change that occurs during glycolysis converts one molecule of glucose to two molecules of lactic acid and a net total of two molecules of ATP are formed (reaction 6).

$$\text{Glucose} + 2\text{ADP} + 2\text{Pi} \longrightarrow 2 \text{ lactic acid} + 2\text{ATP} + H_2O \quad (6)$$

Many investigators have reported that during high intensity exercise muscle glycogen is the predominant fuel for ATP production (Cheetham et al., 1986; Jacobs et al., 1982). As much as an 89 % contribution from glycolysis to total anaerobic ATP turnover has been found during 30 s of isokinetic cycling

(McCartney et al., 1986). It has been estimated that during 2.56 s and 1.28 s of electrical stimulation glycolysis contributed 50 % and 20 % respectively of the total anaerobic turnover of ATP (Hultman and Sjoholm, 1983). The energy required to sustain high mean power output generated during the first 6 s sprint of 10 was found to be provided by an equal contribution from anaerobic glycolysis and PCr degradation (Gaitanos et al., 1993). These observations are supported by the pronounced lactate accumulation occurring during maximal cycling sprinting of 10 s duration indicating that glycolysis is initiated from the onset of exercise (Bogdanis et al., 1993; 1994b; Boobis et al., 1983; Jacobs et al., 1982).

The rate of anaerobic glycolysis is maximal during the first few seconds of maximum intensity exercise and as the bout continues the rates of degradation of glycogen decreases (Bogdanis et al., 1994b; Hultman and Sjoholm, 1983). Rapid glycogenolysis has been found to occur in type II fibres but only a small rate in type I fibres (Greenhaff et al., 1992).

So although there is a large and rapid breakdown of glycogen only about half of the muscle glycogen stores are utilised during brief maximal exercise and fatigue occurs before muscle glycogen is depleted (Hermansen, 1981). Thus glycogen availability is not thought to limit performance during brief maximal exercise. However during repeated maximal exercise glycogen availability in type II fibres may limit the rate of ATP synthesis. A greater depletion of glycogen in type II fibres as a result of the rapid glycogenolysis has been reported using single fibre analysis (Greenhaff et al., 1991).

**2.4.5 ATP resynthesis from oxidative phosphorylation**

During high intensity exercise lasting approximately 30 s ATP resynthesis comes mainly from PCr and anaerobic glycolysis. However, ATP can also be derived by aerobic metabolism in a process called *oxidative phosphorylation*. This process appears to have a greater contribution either as the duration of the exercise increases and/or is repeated.

The oxidative process of ATP synthesis during high intensity exercise is initially the same metabolic pathway as glycolysis. The difference begins when pyruvate is transported across into the mitochondria where it is irreversibly converted to *acetyl Co-enzyme A* (acetyl Co-A). Acetyl Co-A then enters the pathway known as *tricarboxylic acid* (TCA) cycle. The TCA cycle

consists of seven reactions that give rise to three end products; ATP, carbon dioxide and hydrogen bound to co-enzyme molecules.

One molecule of ATP is formed during each cycle of the TCA cycle in addition to 11 ATP moles that can be formed by oxidative phosphorylation via electron transfer from the 4 pairs of hydrogen atoms that are derived in the cycle. Thus for every pyruvate entering the mitochondria, 12 moles of ATP are formed. Therefore from one glucose mole, 2 moles of ATP are produced by glycolysis, 24 moles by TCA cycle (two pyruvates) and a further 8 moles from electron transfer (not mentioned) of NADH from glycolysis (Figure 2.12, step 6) and conversion of pyruvate to acetyl Co-A. This results in a total of 36 moles of ATP (Astrand and Rodahl, 1986; McArdle et al., 1991). However this process proceeds at a much slower rate compared with glycolysis and will occur as long as oxygen is available in the mitochondria. Therefore it is important to determine the aerobic contribution during high intensity exercise.

It has been suggested that during repeated short term high intensity exercise an increase of ATP synthesis may be derived by oxidative metabolism (Gaitanos et al., 1993). The aerobic contribution to energy supply during high intensity exercise becomes more important with increasing duration. This contribution has been estimated at 40%, 50% and 65% during high intensity exercise for 30s, 1 and 2 mins respectively (Medbo and Tabata, 1989). An elevation of $H^+$ has been suggested to increase the activity of the enzyme, pyruvate dehydrogenase which is involved in the reaction from pyruvate to acetyl Co-A, thus enhancing aerobic metabolism (Newsholme and Leech, 1983). A significant drop in mean power output for subjects in a hypoxic condition group during a 30 s Wingate power test has been reported (Kavanagh et al., 1986). This was assumed to reflect the partial dependence on the aerobic metabolism. An inhibition has been reported of anaerobic glycogenolysis at the end of 10 x 6 s sprints and it was proposed ATP synthesis was mainly derived from PCr degradation and oxidative metabolism (Gaitanos et al., 1993). Hence, aerobic metabolism may provide the energy for maintaining high work output during repeated high intensity exercise.

## 2.5 AETIOLOGY OF FATIGUE DURING HIGH INTENSITY EXERCISE

The purpose of this section is to outline the current understanding of the mechanisms that may cause fatigue during high intensity concentric muscle actions.

Edwards (1981) defined fatigue as the inability to maintain a required or expected power output. Fatigue can be classified into two categories, central and peripheral. Central fatigue refers to the brain and spinal cord activation which are the first two echelons of the chain of command for a muscle action. Central fatigue relies upon motivation, thus it is difficult to physiologically measure the mental element. Therefore the following information will concentrate on the peripheral causes of fatigue.

Peripheral fatigue is a complex process and as yet the mechanisms are not fully understood. Edwards (1983) illustrated, as shown in Figure 2.13, the possible factors that could inhibit performance during the chain of command for muscular action.

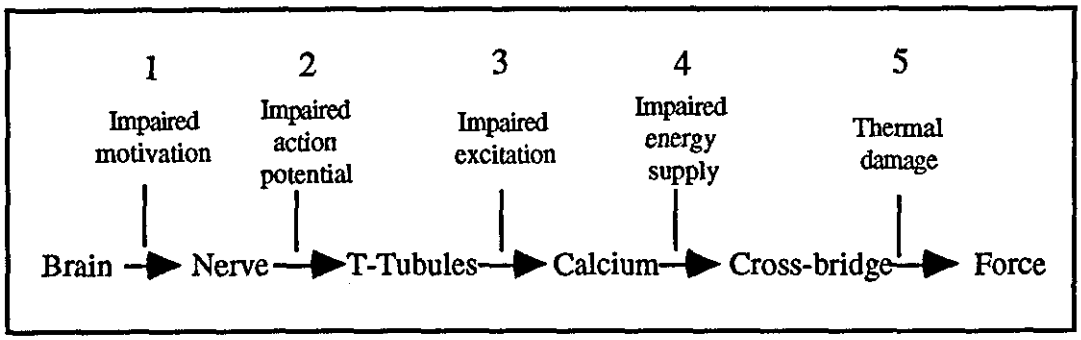



**FIGURE 2.13.** *Factors involved in fatigue during the chain of command for muscular command (adapted from Edwards, 1983)*

It has been suggested that fatigue results when ATP production is insufficient for muscular demand (Sahlin, 1986), causing a reduction in the number of simultaneously attached cross-bridges in an activated state. However most authors recognize that a multiplicity of factors will be involved in the fatigue process.

According to Rall (1985) there are 3 major ATP utilising systems active during an excitation-coupling cycle; myosin ATPase, $Ca^{2+}$ transport ATPase of the

sarcoplasmic recticulum and $Na^+$, $K^+$, ATPase of the sarcolemma. Thus availability of ATP might inhibit the excitation-coupling mechanism. Muscle ATP decreases following short term high intensity exercise by only 30-40% (Bogdanis et al., 1994a; 1995a). Thus it appears unlikely that the ATP concentration only can be the limiting factor unless there are areas or compartments of the cell where ATP are lower.

### 2.5.1 PCr and fatigue

PCr provides approximately 50% of the ATP utilised in a 6 s sprint and would be nearly fully depleted within 10 s (Gaitanos et al., 1993). The mean half-time of PCr resynthesis following a 30 s cycle ergometer sprint was found to be 56.6 $\pm$ 7.3 s. However, the restoration of PCr was incomplete (85% of control values) after 6 min rest (Bogdanis et al., 1995a).

Oral creatine supplementation has been shown to: increase creatine and PCr stores in the muscle and the speed of PCr resynthesis (Greenhaff, et al. 1993b; Greenhaff, 1994a; Harris et al., 1992); increase muscle power output (Balsom et al., 1993; Greenhaff et al., 1993a) and; increase recovery in intermittent high intensity exercise (Balsom et al., 1993). Creatine supplementation is discussed further in Section 2.7. Thus it appears from these findings that depletion of PCr affects the rate of ATP resynthesis which may cause the initial decrease in performance during high intensity exercise.

It has recently been observed the resynthesis of PCr and the restoration of peak power following 30 s sprint cycling proceeded in parallel throughout a 6 min recovery (Bogdanis et al., 1995a). Thus PCr resynthesis is important for recovery of peak performance during repeated bouts of sprint exercise and appears related to fatigue.

### 2.5.2 Glycolysis and fatigue

During short term high intensity exercise glycogen availability probably does not contribute to fatigue. After a 30 s cycle ergometer sprint mean muscle glycogen was found to decrease from pre-exercise resting values of 321 $\pm$ 18.2 to 211.6 $\pm$ 19.5 mmol.kg dry $muscle^{-1}$ (34%) (Bogdanis et al., 1995a). However Greenhaff et al. (1991) suggest that during repeated bouts of exercise glycogen availability may limit performance in type II fibres.

During high intensity exercise, glycolysis causes an increase in $H^+$ which reduces pH (acidosis). Acidosis will influence many physiological processes at many levels which can indirectly reduce force. Various studies have found a decrease of force with increased $H^+$ and reduced pH (Donaldson, 1983; Fabiato and Fabiato, 1978; Fretthold and Garg, 1978).

The relationship between acidosis and fatigue can be explained by an $H^+$ mediated inhibition of the ATP generating processes (Sahlin and Ren, 1989):

(i) $H^+$ competes with $Ca^{2+}$ for the binding sites on the troponin molecule (Bolitho and Donaldson et al., 1978- cited Edwards, 1983) and if the $H^+$ attaches onto the binding site it will prevent the conformational change in the troponin-tropomyosin complex, thus preventing cross-bridge attachment.

(ii) Nakamaru and Schwartz (1972-cited Edwards, 1983) suggest that $H^+$ reduces the amount of $Ca^{2+}$ released by the sarcoplasmic reticulum upon stimulation.

(iii) $H^+$ may affect the activity of the enzymes PFK and phosphorylase, thus inhibiting the glycogenolytic and glycolytic rates (Gaitanos et al., 1993).

Although acidosis is related to fatigue there is no conclusive evidence that $H^+$ directly inhibits a muscle action. A recent study found that the restoration of peak power output following a 30 s sprint occurred despite muscle pH remaining low throughout a 6 min recovery. The recovery of peak power output occurred in parallel with PCr resynthesis (Bogdanis et al., 1995a). Thus acidosis appears primarily to act indirectly in the fatigue process by decreasing the rate of ATP resynthesis (Sahlin, 1986).

### 2.5.3 Other causes of fatigue

The products of ATP hydrolysis, ADP, $H^+$ and Pi, in reaction (1), increase during high intensity exercise. Sahlin (1986) suggests the magnitude of the increase in ADP concentration in a fatigued muscle interferes with the enzyme, ATPase, involved in $Na^+$ and $K^+$ balance across the cell membrane. A decreased activity of $Na^+$, $K^+$, ATPase increases intracellular $Na^+$ and extracellular $K^+$ which is likely to cause a problem with excitation-coupling (Sahlin et al., 1978; Sjogaard et al., 1985).

In summary, the following mechanisms could be responsible for the decrease in force:

(i) Inability to regenerate ATP at the required rates.

(ii) Inhibition of any of the processes by products formed in the energy supply reactions.

(iii) Alterations of excitation-coupling cycle from the surface action potential to $Ca^{2+}$ release from the sarcoplasmic reticulum.

## 2.6 DIFFERENCES BETWEEN CONCENTRIC AND ECCENTRIC MUSCLE ACTIONS

There are considerable differences between concentric and eccentric muscle actions, not only in the production of forces but also in the metabolic cost, structural changes within the muscle and responses to voluntary and stimulated activation. This section will review the current understanding of the differences in force characteristics, neural activation and metabolic responses.

### 2.6.1 Electromechanical delay

Electromechanical delay is referred to by Cavanagh and Komi (1979) as the elapsed time from the biochemical response to the actual onset of muscular force. These authors showed that for elbow flexors and extensors the electromechanical delay was 4.4 ms shorter for eccentric than concentric muscle actions.

### 2.6.2 Stretch-shortening cycle

In most activities the muscle lengthens (eccentric) before it acts concentrically. This is commonly referred to as stretch-shortening cycle. A transfer of energy from the elastic properties and proprioceptive reflexes produces a greater magnitude of force than a concentric action alone (Asmussen and Bonde-Petersen, 1974).

The mechanical characteristics of a muscle have been modelled by Albert (1995) to comprise of three components; contractile, series elastic and parallel elastic (Figure 2.16).

The series and parallel elastic components can act as springs. For example, during an eccentric action the series elastic component is stretched and allowed to contribute to the force production. An eccentric action immediately preceding a concentric action will increase the force concentrically due to the stored energy from the eccentric action in the series elastic component. This added contribution is affected by time, magnitude of stretch and velocity of stretch (Enoka, 1988).

Another force producing mechanism related to the stretch-shortening cycle is the proprioceptive stretch reflex. Proprioceptors, called muscle spindles, are

located within the muscle and provide afferent information about the stretch of the muscle. Information is transmitted via the afferent axons to the central nervous system which controls the frequency of impulses back to the muscle. For example, when the muscle spindle is activated from stretching, a sensory afferent response is evoked and transmitted to the spinal cord, which sends impulses back to the muscle causing a motor response. This process is called the stretch or myotatic reflex. The greater rate of stretch corresponds to a greater muscle spindle response (Albert, 1995).

The improvement in force from a concentric action preceded by an eccentric action can be identified as the combined effects of both storage of elastic energy and the myotatic reflex activation of the muscle.

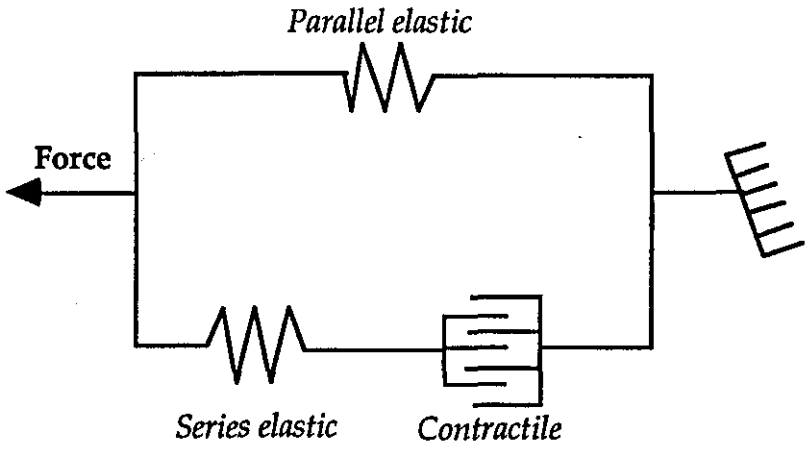



**FIGURE 2.14** *Three components model of muscle behaviour; parallel elastic component, series elastic component and contractile component (From Albert, M. 1995).*

### 2.6.3 Force characteristics

During eccentric muscle actions the maximum force developed is greater than for isometric or concentric actions. Various studies have found that maximally stimulated human and isolated animal skeletal muscle has the capacity to produce up to twice the force during an eccentric action than during an isometric action (Edman et al., 1978; Flitney and Hirst, 1978; Gravel et al., 1988; Katz, 1939; Levin and Wyman, 1927; Lombardi and Piazzesi, 1990; Westing et al., 1990). It was first reported by Singh and Karpovich (1966) that eccentric actions in maximally voluntary activated human muscles can also generate more force than concentric and isometric actions.

The existence of a relationship between force generation and velocity in contractile tissue during muscle shortening was first identified by Fenn and Marsh (1935). The relationship modelled mathematically by Hill (1938) was an hyperbolic curve between velocity and force in isolated animal muscles, i.e. as the velocity increases the force decreases. This curved inverse relationship has also been shown for intact human tissue (Dern et al., 1947; Wilkie, 1950). The force-velocity relationship for eccentric exercise is different from that found for concentric. During eccentric actions the force response at different velocities can be separated into three phases; the force increases, at a steeper course than the concentric curve, with an increase in velocity until the second phase where force production is relatively independent of any further increase (Flitney and Hirst, 1978; Gülch, 1994; Lakomy, 1994). The third phase occurs following the plateau when the tension reduces progressively as lengthening velocity increases further (Lombardi and Piazzesi, 1990). It has been postulated that at fast velocities there is insufficient time for re-attachment of the actin and myosin filaments (Flitney and Hirst, 1978). These curves are shown in Figure 2.15.

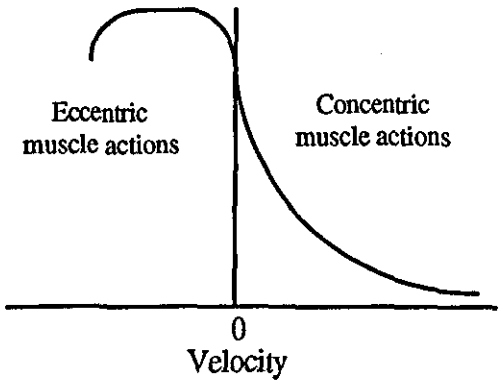



**FIGURE 2.15.** *The relationship between maximum muscle force and velocity for concentric and eccentric in vitro isolated animal muscle actions.*

The ratio between eccentric force and isometric force created in any given muscle varies with temperature and velocity of stretch, however investigators have found a ratio up to 1.8 at the critical velocity (Abbott et al., 1951; Flitney and Hirst, 1978). This difference in the maximum force and velocity curves appears to be related to the elastic properties within the muscle and differences in the binding and interaction of the actin and myosin filaments.

During eccentric actions the muscle is being stretched and additional force to that produced from the cross-bridge attachments, can be created by the series elastic component. It has been stated that during muscle lengthening a substantial fraction of the stretch is not taken up by the sarcomeres lengthening, but instead by series elastic components, which contributes to the force production (Flitney and Hirst, 1978). This additional force becomes greater as the velocity of lengthening increases. During concentric actions some of the force generated by the cross-bridge attachments is utilised to overcome the internal resistance or viscosity of the muscle. Therefore the force that can be transmitted by the tendons to the skeletal structure is reduced. The greater the velocity, the greater amount of force is required to overcome the internal viscosity, hence the reduced maximum force for concentric in comparison with eccentric actions.

Another possible explanation for the greater force developed in eccentric actions is due to the conformational changes that occur within a cross-bridge. During eccentric actions the myosin heads are forced to move apart in the opposite direction to that which occurs during concentric actions. The deformation in the myosin filament that must take place during this change might result in a greater production of maximum force during the attachment. Also it is possible that the change which occurs in the myosin filaments causes an increase in the duration of each cross-bridge attachment. Maximum force is a function of the magnitude and duration for each cross-bridge, hence by increasing duration, an increase of overall muscle tension occurs.

Stimulated skeletal muscle has been found to have the capacity to produce nearly twice as much force during an eccentric action than during a concentric or isometric action, but in a voluntary activated human muscle, eccentric force only marginally exceeds the isometric level (Smidt, 1973; Westing et al., 1988) and exceeds the concentric action by an amount dependent on the muscle groups examined (Komi and Viitasalo, 1977). During an electrical stimulation of an already voluntary activated muscle during an eccentric action the knee extensors, on average, were found to produce 21-24% more torque than the maximal voluntary level alone (Westing et al., 1990). Electrical stimulation reflects the actual uninhibited capacity of the muscle since it can be assumed that neural inhibition at the spinal level is bypassed. Therefore there appears to be a reluctance for

muscle to exert maximal force and/or there are difficulties in performing the co-ordination of maximal eccentric action.

### 2.6.4. Differences in Electromyography activity

Differences in electromyogram (EMG) activity between concentric and eccentric actions were first reported by Asmussen (1953) who found higher activity for concentric work. This has been supported by other studies which have measured EMG readings for the knee extensors during maximal voluntary tension and found less activity (10-30%) in eccentric loading compared with concentric (Eloranta and Komi, 1980; Seliger, et al., 1980; Tesch, et al., 1990; Westing, et al., 1991). EMG activity during concentric actions has been reported to exceed eccentric by a two to three fold factor (Bigland and Lippold, 1954). EMG activity measures the number of action potentials in a muscle, an indication of the level of recruitment and activation. Therefore assuming one hundred percentage effort was achieved during an eccentric action, full activation of the muscle groups appears not to be possible.

It appears that during eccentric actions some inhibitory mechanism prevents the development of full muscle activation in untrained individuals. This regulation may come from neural inhibitions limiting the amount of electrical energy reaching the muscles. This inhibition would maintain tension within 'safe' limits and also may help in protection against injury (Houk and Henneman, 1967; Stauber, 1989). Therefore the force-velocity relationship would probably not produce an exponential increase in force during an increase in velocity, but rather a plateau as described. As mentioned, at a certain point the plateau will be followed by a sudden loss of force. It has been reported that EMG decreased as velocity decreased during concentric actions (Westing et al., 1991) which suggests that non-maximal activation may also occur under slow concentric loading, possibly due to neural regulation. Increased inhibitory feedback could occur from joint receptors, free nerve endings in the muscle, cutaneous receptors, pain receptors and/or golgi tendon organs (Carew, 1981).

### 2.6.5. Differences in metabolic responses

During eccentric work the mechanical energy absorbed by the body tissues from the elastic properties may be stored or dissipated as heat (Pahud et al., 1980). This heat transfer in eccentric actions produces an early and extensive

sweat production compared to concentric actions generating the same external work. A threefold greater heat production for eccentric exercise in comparison with concentric exercise of approximately the same metabolic rate has been reported (Nielsen et al., 1972). Core temperature during eccentric exercise remains lower than during concentric actions at the same workload (Nielsen, 1966) probably due to lower muscle blood requirements as reflected in the metabolic responses, but skin and muscle temperatures were greater by 3 and 1.2 °C respectively during eccentric exercise (Nadel et al., 1972).

It may appear that activities involving eccentric actions are easier than concentric actions because they mostly work with gravity. However if both actions are performed at the same velocity, the same amount of force is generated during both lifting and lowering tasks. Studies which have examined equal sub-maximal work load for eccentric and concentric actions have found that EMG activity is less during the eccentric work (Bigland and Lippold, 1954). The lower EMG activity for eccentric actions implies less muscle activation to tolerate the same load than concentric actions which is supported by studies that have found a reduction of up to 25% in high energy phosphate hydrolysis during eccentric actions despite equal work compared with isometric and concentric actions (Abbott et al., 1951; Curtin and Davies, 1975; Fenn, 1924; Wilkie, 1968). Lower net energy output during eccentric than during isometric or concentric actions was first identified by Fenn (1924). Other studies have supported Fenn's original results by finding that during eccentric actions in comparison with concentric action of equal work, there is a lower oxygen uptake and/or metabolic energy expenditure (Abbott et al., 1951; Abbott et al., 1952; Asmussen, 1953; Bigland-Ritchie and Woods, 1976; Bonde-Petersen, et al., 1972; Davies and Barnes, 1972; Kaneko, et al., 1984; Knuttgen and Klausen, 1971; Knuttgen et al. 1982; Plante and Houston, 1984; Schwane et al., 1983), less phosphocreatine degradation (Bonde-Petersen et al., 1972), lower muscle and blood lactate concentrations (Bonde-Petersen et al., 1972; Plante and Houston, 1984) and lower muscle glycogen replenishment after 48 hours (Bonde-Petersen et al., 1972; Doyle et al., 1993). These studies seem to suggest that less metabolic activity is required to produce the same work for eccentric actions. Oxygen uptake per unit of muscle activity has been reported to be 3 times greater for concentric than for eccentric actions (Bigland-Ritchie and Woods, 1976). Similarly during

comparable work eccentric work requires 70-75% less oxygen than concentric, which is illustrated in Figure 2.16 (Knuttgen et al., 1982).

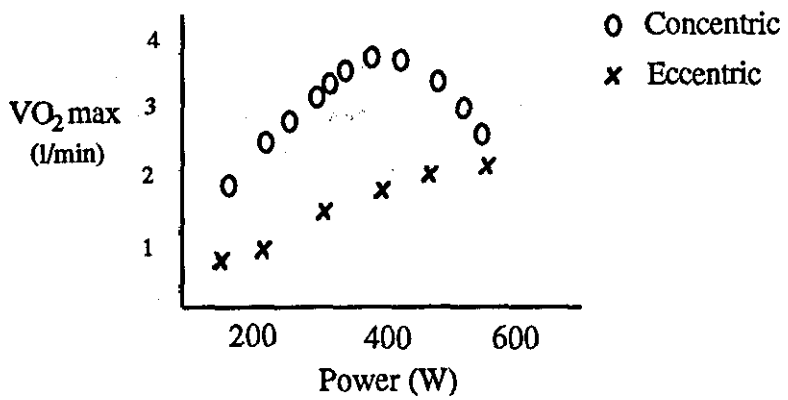



**FIGURE 2.16.** *Oxygen uptake at various force levels (From Knuttgen et al., 1982).*

These findings demonstrate that eccentric actions are mechanically more efficient compared to concentric actions. Mechanical efficiency is defined as the ratio of the output to the input energy (Kaneko et al., 1984). Menard et al. (1991) showed by measuring the metabolic cost of the muscle cells, that concentric energy expenditure was proportional to the rate of work production, whereas during eccentric actions energy expenditure was found to be independent of the rate of work. Thus at higher workloads the efficiency for eccentric exercise becomes higher. Additionally, Kaneko et al. (1984) found that mechanical efficiency increased for eccentric actions as the testing velocity increased, whereas efficiency during concentric actions remained constant.

It has also been found that during a tetanized eccentric action in an isolated muscle, less inorganic phosphate is consumed compared with concentric and isometric actions (Curtin and Davies, 1975). Since the entire muscle was assumed to be activated and all muscle fibres recruited the reduced energy consumption suggests differences in the contractile mechanism's responses.

### 2.6.6. Differences in the excitation-coupling mechanism

In section 2.3.3 the activation of the excitation-coupling mechanism was explained. The myosin heads bind to actin as the binding site is exposed. Once the attachment occurs, the potential energy stored on the myosin head is transformed and causes movement as the actin is pulled along the myosin filaments. This conformational change causes the muscle to shorten and tension is created. However during eccentric actions the external resistance

exceeds the force that can be created by the cross-bridge attachments and muscle lengthening occurs. The myosin head is forced in the opposite direction and once the displacement of the filaments in the axial direction exceeds 11-12 nm the attachment is forced apart, as shown in Figure 2.17 (Gülch, 1994; Lakomy, 1994). The length change is shared between backward rotation of the head and further extension of the elastic linkage (Flitney and Hirst, 1978).

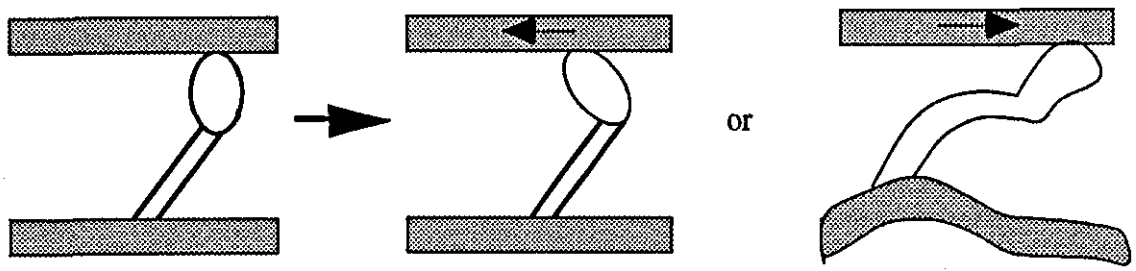



**FIGURE 2.17.** *Conformational change in myosin and actin filaments during either concentric or eccentric muscle actions (unpublished from Lakomy, H.K.A).*

An eccentric cross-bridge theory has been postulated that suggests the actin-myosin bond is broken before transduction of energy can occur. Therefore if the external force continues, the energised myosin is repeatedly reattached and pulled apart without the required transduction of energy (Curtin and Davies, 1975; Menard et al., 1991; Rall, 1985; Stauber, 1989).

### 2.6.7 Differences in fatigue

Studies have found differences in fatigue between repeated eccentric and concentric exercise bouts, however these differences vary depending on the velocity of movement. Komi and Viitasalo (1977) found eccentric actions to be highly fatiguing during slow velocities and suggested it was due to high degree of tension generated by the elastic properties of the muscles. A more recent study found eccentric actions to be more fatigue resistant than concentric for knee extensors during fast velocities (Gray and Chandler, 1989). Thus it appears from these studies that greater fatigue is found during eccentric actions at slow velocities and concentric actions at fast velocities. This suggestion is supported by Newham et al. (1983) who found dramatic decreases in force during low frequency electrical stimulation but not during high frequency stimulation. Slow velocity eccentric actions lack the force potential which can be generated from the stretch-shortening cycle (Komi and

Viitasalo, 1977). The findings in fatigue at different testing velocities suggest the contractile mechanism is 'stressed' less at intermediate and fast velocities for eccentric actions (Gray and Chandler, 1989).

## 2.7 ORAL CREATINE SUPPLEMENTATION

Creatine in it's phosphorylated form, PCr, functions in the maintenance of cellular ATP homeostasis. In addition PCr has been postulated to be involved in the buffering of $H^+$ (Harris et al., 1992). It has been reported that the mean content of total creatine in skeletal muscle (quadriceps femoris) is 124.4 $\pm$ 11.2 mmol.kg $dw^{-1}$ (Harris et al., 1974). Dietary sources of creatine are found mainly in red meat, for example, 1 kg of fresh, uncooked meat contains around 5 g of creatine. Creatine administered as a supplement was investigated by Harris et al. (1992) who found that ingestion of 5 g of creatine monohydrate 5 to 6 times a day for two or more days resulted in a significant increase in creatine content of the quadriceps femoris. It was also found that total creatine content was increased if the subjects undertook heavy exercise during the supplementation period.

The benefits to performance from oral creatine supplementation have only been investigated over the last four years. Studies have shown that supplementation can improve performance in; muscle peak and mean power output (Birch et al., 1994), improve running performance times (Harris et al., 1993; Stroud et al., 1994) and improve recovery in repeated bouts of high intensity activity (Balsom et al., 1993; Bogdanis et al., 1995b, Greenhaff et al., 1993a; Greenhaff et al., 1994a). These studies are described further in Table 2.3.

The creatine ingestion studies suggest that by increasing resting concentration of creatine and PCr and improving the rate of PCr and ATP resynthesis in skeletal muscle, oral creatine supplementation can improve performance in high intensity exercise.

At present no studies to the authors knowledge have examined the effect creatine supplementation on repeated eccentric muscle actions. The effect of creatine supplementation on repeated bouts of maximal voluntary concentric actions has been examined and peak isokinetic torque increased throughout four of the five sets (Greenhaff et al., 1993a). However as discussed there appears to be differences in the energy utilisation between concentric and eccentric actions, thus the different responses following creatine

**TABLE 2.2.***Previous literature measuring the effect oral creatine supplementation on high intensity exercise*

| Authors | Subjects | Performance measure | Dosage | Significant performance improvement following supplementation |
|---|---|---|---|---|
| Harris et al., (1993) | n=10, male middle distance runners | Sprint running<br>4 x 300 m<br>4 x 1000 m<br>at 90-95% best | 30 g.d-1 for 6 days | -Times for final repetition of both distances were reduced.<br>-Total time to run 4 x 1000 m was reduced |
| Balsom et al., (1993) | n=16, male, P.E. College students | Sprint cycling<br>10 x 6 s at 100% | 25 g.d-1 for 6 days | - Blood lactate accumulation decreased.<br>- A smaller decline in work output |
| Greenhaff et al., (1993b) | n=12, 2 females and 10 males | 20 x repeated electrically evoked isometric actions lasting 1.6 s (1:1 rest/stimulated ration) | 20 g.d-1 for 5 days | - increase in muscle PCr<br>- accelerated rate of PCr resynthesis following intense muscle action |
| Greenhaff et al., (1993a) | n=12, non-trained | Isokinetic actions<br>5 x 30 s at 180 deg.s-1 (1 min recovery between sets) | 20 g.d-1 for 5 days | - Peak torque increased in the final few extensions for the first set and throughout the last 4 sets<br>- plasma ammonia accumulation was lower |
| Greenhaff et al., (1994a) | n=6, male | Isokinetic actions<br>2 x 30 s<br>(4 min recovery between sets) | 20 g.d-1 for5 days | - increase total muscle creatine<br>- ATP degradation reduced by 50% on 2nd set, despite more work produced |
| Stroud et al., (1994) | n=10, middle distance runners | Sprint running<br>4 x 1000 m<br>4 x 300 m | 30 g.d-1 for 5 days | - best times for both distances increased<br>- improvement of total time for both set of distances |
| Birch et al., (1994) | n=14, male, untrained | Isokinetic cycling<br>3 x 30 s at 80 rpm<br>(4 min recovery) | 20 g.d-1 for 5 days | - increase peak power output in the 1st bout<br>- mean power output for bouts 1 and 2<br>- total work in bouts 1 and 2<br>- lower plasma ammonia despite more work |
| Bogdanis et al., (1995b) | n=16, male, active | Sprint running<br>6 x 10 s<br>1 x 30<br>(30 s recovery) | 300 mg.kg-1 for 5 days | - higher power for 6th sprint<br>- lower plasma ammonia |
| Cooke et al., (1995) | n=12, male, untrained | Sprint cycling<br>2 x 15 s | 20 g.d-1 for 5 days | no significant differences |

supplementation for both actions could help further understanding of the contractile mechanisms involved in eccentric actions.

## 2.8 SUMMARY

The review began by showing the joint movements and actions during a single leg cycle. All joint actions in the lower limb are involved during some part of the cycle, excluding concentric dorsiflexion. However ambiguity is seen to exist concerning which muscle action in which joint is best related to sprint performance. Further investigations are necessary to measure the relationships between muscle strength and sprint performance.

The review has also illustrated the mechanisms and factors involved in skeletal muscle actions from the activation procedures to the conformational changes that occur between the actin and myosin filaments. Many different responses have been found following concentric and eccentric actions. A greater amount of force can be generated during eccentric actions than both isometric and concentric actions. In addition eccentric actions utilise a lower amount of energy during equal work and electrically evoked isolated muscle in comparison with concentric actions. Thus it appears the conformational changes and energy utilisation during eccentric muscle actions are vastly different. One theory suggests that during muscle lengthening, the external resistance forces the mysoin head apart from the actin attachment before the breakdown of an ATP molecule can occur.

Another area which requires further understanding are the mechanisms involved in fatigue during repeated bouts of concentric and eccentric exercise. Currently it remains to be seen whether the mechanisms are the same for both types of muscle actions.

Therefore further studies are required to examine the force and metabolic differences following repeated bouts of either concentric or eccentric maximally activated actions. These findings will help to understand and explain further the actual mechanisms involved in eccentric actions.

# *CHAPTER 3. GENERAL METHODS*

## 3.1 INTRODUCTION

Three studies have been completed, the first examining the relationship between sprint times and strength and the second and third examining the differences in performance and metabolic responses between concentric and eccentric exercise bouts. Three types of measurements were used in the studies; anthropometric, sprint times and isokinetic measurements. All subjects in all studies had their anthropometric measurements recorded. Sprint times during two tests (Acceleration and Basic speed test) were recorded in the first study only. During all studies torque and total work were recorded during concentric and eccentric muscle actions using an isokinetic dynamometer. Two different protocols were used throughout the three studies, one measuring peak torque from the best of three repetitions and two involving repeated bouts of knee extension and flexion. Blood samples were taken during the second and third studies and were analysed for blood lactate, plasma ammonia, haemoglobin, haematocrit and changes in plasma volume.

## 3.2 ANTHROPOMETRIC MEASUREMENTS

Anthropometry is the means of quantifying variations in body size and shape for different subjects. Subjects in each study had standard measurements of height, body mass and in some studies, limb length taken, as described by Weiner and Lourie (1981). Skinfold thicknesses were also determined using protocols described by Durnin and Womersley (1974) to estimate percentage body fat by the same researcher for all subjects in every study.

### 3.2.1 Height

Each subject's height was recorded using a Holtain Stadiometer. All subjects were measured in bare feet with heels together. Each subject stretched upward to the fullest extent and was encouraged to *'stand tall, take a deep breath and relax'*. The counter-weighted board was brought down on to the subject's head. The subject's heels were watched to make sure they did not leave the ground. Measurements were taken to the nearest 0.1 cm.

### 3.2.2 Body mass

Each subject's body mass was recorded using a balance beam (Avery Ltd., Model 3306 ABV). All subjects were weighed in bare feet wearing only shorts. Measurements were to the nearest 0.05 kg.

### 3.2.3 Skinfold measurements

A Harpenden skinfold caliper (Holtain Ltd.) was used for the measurement of skinfold thicknesses from which percentage body fat and lean body mass values were estimated. Skinfold thicknesses were taken at four sites on the left side of the body (biceps, triceps, subscapula and suprailiac).

Each skinfold was taken by pinching skin from a wide sweep between the inverted thumb and forefinger and was lifted slightly away from the underlying tissues. The caliper jaws were applied to the skinfold at the point where the double fold was parallel. Measurements were recorded after the full pressure of the caliper jaws had been applied to the skinfold. Two measurements were taken at each site and an average value was calculated. If the two values differed by more than 0.4 mm the measurements were repeated. The readings were taken to the nearest 0.2 mm.

*(1) Biceps:*

The arm was bent at 90° at the elbow and the mid-point distance between the tip of the acromion process and olecranon process was marked. This mark was translated around to both the biceps and triceps. The biceps skinfold measurement was taken with the arm resting and slightly supinated.

*(2) Triceps:*

The triceps skinfold was measured on the triceps mark using the same procedure as for the biceps.

*(3) Subscapular:*

A mark was made 2 cm below the inferior angle of the scapular. A diagonal skinfold was measured opposite to the diagonal angle of the scapula.

*(4) Suprailiac:*

A vertical skinfold was taken 2 cm above the iliac crest in the mid-axillary line.

The sum of the four skinfolds was used in conjunction with regression equations derived by Durnin and Womersley (1974) for males between 20-29 years, illustrated below, to estimate percentage body fat from the calculated body density.

$$\textit{Body density} = 1.1631 - (0.0632 \times \textit{sum of skinfolds})$$
$$\textit{Body fat percentage} = ([4.950/\textit{Body Density}] - 4.5) \times 100$$

### 3.2.4 Limb lengths

A digital read-out Harpenden Anthropometer (Holtain Ltd.) was used to measure three lower limb lengths (buttocks-knee, tibial and foot lengths) all on the left side. Straight blades were used and the reading was taken to the nearest 1 mm.

*(1) Buttocks-knee length:*

The subject sat erect, with feet resting against a surface so the knees were bent at 90°. The horizontal distance from the rear-most point of the buttock to the front of the knee cap was measured using the anthropometer blades.

*(2) Tibial length:*

The subject sat erect facing the experimenter with the left ankle resting on the right knee so that the medial aspect of the tibia faced upwards. The anthropometer blades were applied to the marked proximal-medial border of the tibia and the marked distal border of the medial malleolus.

*(3) Foot length:*

The subject sat and rested one foot lightly along the horizontal blade of the anthropometer. The second blade was brought into contact with the end of the longest toe without pressure.

## 3.3 SPRINT PROTOCOL

Two sprint tests were performed in an indoor sprint corridor over distances of 15 (Acceleration test) and 35 (Basic speed test) metres. Photo-electric cells were used to record sprint times.

### 3.3.1 Equipment

A pacesetter training system (Split Second Ltd.) was used to measure split times. This system consists of a hand-held battery operated timing unit with 6-digit display and key pad controls, 4 infra-red sensors, 4 reflectors and 8 miniature tripods.

Each infra-red sensor was aligned with a reflector and mounted on a tripod. The sensor detected the subject as they ran through the infra-red beam. When a sensor was triggered it sent a coded radio signal back to the main hand-held unit. The main unit has a clock which runs continuously until all the sensors have been triggered. The first sensor to be triggered is used as a time reference for the other sensors and is allocated the time 00:00.00. The unit then displays the time for each sensor relative to that of the first sensor triggered. This equipment is illustrated in Figure 3.1.

### 3.3.2 Testing procedures

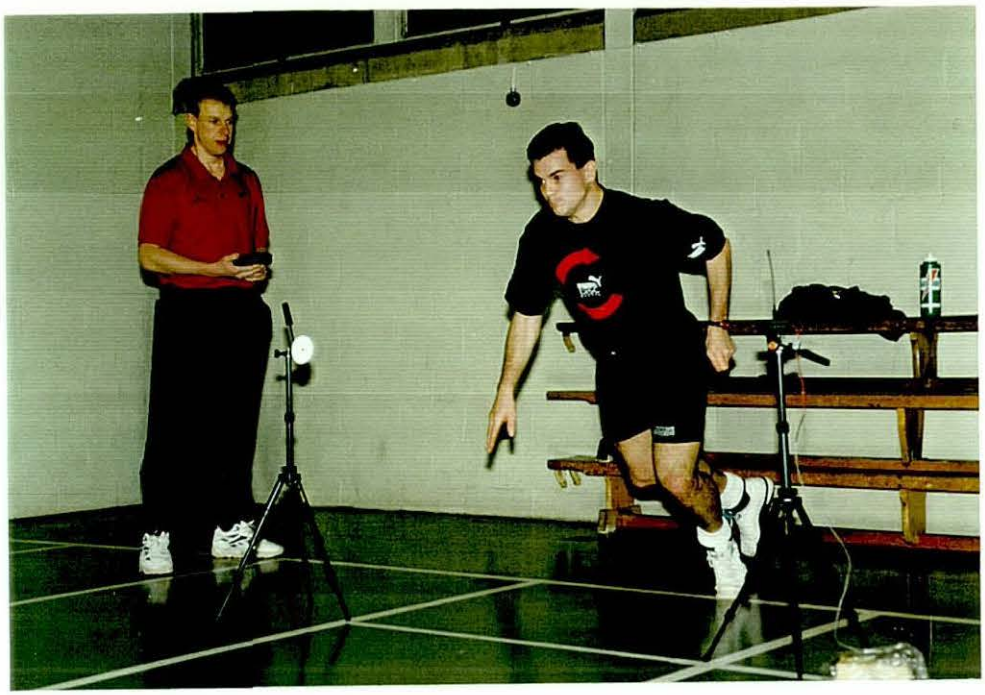
A standing static start was used for both tests.

*Acceleration Test:* The subject stood 1 m behind the first infra-red beam and sprinted 16 m in a straight line passing 4 sensors separated 5 m apart, as shown in Figure 3.1 (a). The trial with the fastest 15 m time was recorded as well as the corresponding times for each 5 m section.

*Basic Speed Test:* The subjects started sprinting 30 m away from the first infra-red beam and ran through the second beam set a further 5 m away, as illustrated in Figure 3.1 (b). The best trial was recorded.

Three trials over each distance were performed with three mins recovery time between each trial and five mins between tests. Prior to the tests a standardised 20 min warm up was performed (Appendix C). All measurements were electronically timed in seconds to two decimal places.

(a)



(b)



**FIGURE 3.1.** *Illustrations of the sprinting equipment and positioning for the Acceleration test from the starting position (a) and mid-sprint position (b).*

### 3.3.3 Calculations

The three recordings from the Acceleration Test were used to calculate 5 m split times by simple subtraction. These split times were then converted into velocity ($m.s^{-1}$) by dividing the distance, 5 m, by the calculated split time. Velocity was calculated from the Basic Speed Test (30-35 m) by the same method. The velocities for each 5 m split were compared with the Basic Speed Test and percentages of each subject's basic speed calculated.

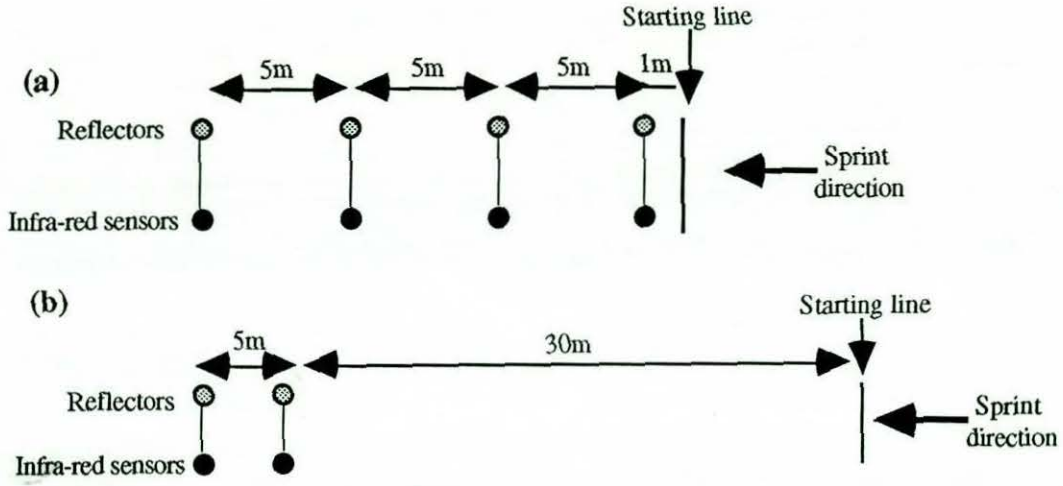



**FIGURE 3.2.** *Diagrams of the distances and equipment positioning for the Acceleration test (a) and Basic speed test (b).*

## 3.4 TORQUE AND WORK MEASUREMENTS USING THE ISOKINETIC DYNAMOMETER

### 3.4.1 Introduction - Isokinetic Dynamometer

The isokinetic measurement principle was introduced in the late 1960's as an isokinetic rehabilitative measure for investigation in man (Thistle et al., 1967). The commercially distributed isokinetic device (dynamometer) allows the subject to exert as much force during an angular movement as they can generate up to a predetermined velocity. The dynamometer produces a counterforce when the angular rate of movement of a limb equals or exceeds the pre-set velocity limit, which allows a constant movement rate. No matter how much force is exerted the velocity will not exceed the pre-selected speed.

Therefore isokinetic devices can measure muscle performance from a muscle group that is exercised to its maximum voluntary potential throughout the joints range of motion. Modern dynamometers are also advantageous because they have many safety features which minimise the risk of injury to the subject while measuring muscle performance. Also the corresponding computer software allows ease of calculation of force or torque, work and power. Furthermore since the early 1980's it has been possible to measure force/torque, work and power during eccentric muscle action.

These studies have used either the Cybex 6000 or KinKom AP II Isokinetic Dynamometer. Both dynamometers are very similar, but the slight differences which affect either set-up procedures or measurements will be described in detail in each relevant section.

Results from one dynamometer system cannot be compared with results on another device. Several studies have attempted to compare results and found significant differences between dynamometers (Francis and Hoobler, 1987; Timm, 1989). Each system employs its own algorithms, which are not published or standardised from manufacturer to manufacturer, thus these differences are probably related to the systems computer.

### 3.4.2 Computer Setting

#### 3.4.2.1 Ramping

An isokinetic dynamometer operates by a subject accelerating to a pre-set test velocity and then the dynamometer produces a counterforce to maintain a constant velocity. The overspeeding limb and lever arm will consequently decelerate once achieving this pre-set velocity which may produce a transient peak or spike in the resulting isokinetic torque curve. This is referred to as the *'overshoot'* phenomenon (Sapega et al., 1982). This artificial spike in the torque curve may be an inaccurate interpretation of a muscle's true capacity to produce maximum force (Perrin, 1993).

To avoid this misinterpretation, pre-programs have been incorporated into the instrumentation which controls the rate of acceleration and deceleration of the limb and lever arm. These pre-programs decrease overshoot and facilitate the identification of torque which is free from artefacts (Sinacore et al., 1983).

With both the Cybex 6000 and KinKom AP II this pre-program feature is named as 'ramping'. This ramping allows the subjects to meet the desired angular velocity more easily, by linearly accelerating the limb and lever arm at a pre-set speed until the pre-selected testing velocity is attained. Ramping is applied in both directions of movement for each repetition. The ramp mode will not be activated until the subject achieves a pre-determined force.

Ramping influences the torque curve because it allows the muscle more time to develop tension before getting to the most advantageous point in the range of motion, hence higher levels of torque will be recorded earlier in the range of motion (Perrin, 1993). This ramping will affect average torque and angle specific torque to a greater extent than peak torque (Jensen et al., 1991).

#### 3.4.2.2 Gravity Correction

Gravity correction accounts for the weight of the subject's limb being tested and the dynamometer's lever arm when it is moved through a gravity dependent position. For example, the acceleration of a limb and lever arm due to gravity erroneously adds to torque output, whereas additional force must be exerted to accelerate the limb and lever arm against gravity which would decrease torque output (Perrin, 1993). In the case of knee extensors and flexors gravity correction would add torque to the extensors by 4-43% and subtract torque from the flexors by 15-510% (Nelson and Duncan, 1983;

Winters et al., 1981). Gravity correction has a greater effect at fast velocities because as velocity increases, torque decreases, but gravity stays the same. Therefore the effect of gravity as a percentage of force output, increases.

A failure to gravity correct would affect the determination of reciprocal muscle group ratios. Without gravity correction the ratios are inflated. It has been established that gravity correction helps in obtaining valid strength measures (Perrin et al., 1991; Rothstein et al., 1987; Winters et al., 1981). If gravity correction is used in re-testing it is essential that reliability is achieved, i.e. the same gravity correction factor must be maintained throughout all re-tests.

### 3.4.3 Standardisation procedures

In the present study the following guidelines were adhered to, to ensure an accurate assessment and reliability of strength measures. Table 3.1. lists the factors associated with standardised protocols.

**TABLE 3.1.** *Factors involved in standardised testing procedures*

| |
|---|
| 1. Subject education |
| 2. Axis of rotation for each joint |
| 3. Body stabilisation |
| 4. Warm up |
| 5. Verbal encouragement |
| 6. Visual feedback |
| 7. Test velocity selection |
| 8. Number of repetitions |
| 9. Calibration |
| 10. Time of day |

#### 3.4.3.1. Subject education

Each subject was educated, informed and familiarised with the isokinetic dynamometer. Perrin (1993) suggested that to attain a reliable and valid assessment the patient or subject must be adequately educated and familiarised with isokinetics. This would probably make the subjects less apprehensive and more consistent with their test efforts. Each subject was educated before their first familiarisation session on isokinetic principles and was told:

*'The dynamometer will not resist you until you match the pre-selected speed. You must push and pull as hard and as fast as you can throughout the range of motion on every repetition. If you feel pain during the test, decrease the effort and the machine will decrease resistance. If you stop, the machine will stop'.*

Additional information was given to the subjects who were assessed during eccentric actions. They were informed *'The limb and you should resist the movement of the lever arm'*. Each subject had at least one familiarisation session. Fleck et al. (1984) and Kues et al. (1992) found 1-2 days familiarisation and training enhances reliability of measurement.

#### 3.4.3.2. Axis of rotation for each joint

In order to isolate the performance of a single muscle group isokinetic testing may be performed through any or all of the three crucial planes of the body, i.e. transverse, sagittal and coronal. In this thesis flexion and extension in different joints were examined in only the sagittal plane. The axis of rotation of each joint was aligned as closely as possible with the axis of rotation of the dynamometer head.

#### 3.4.3.3. Body stabilisation

To isolate the target muscle groups the subject was adequately stabilised in an appropriate manner by velcro straps. This eliminated contribution from the upper extremities. It has been found that stabilisation of the trunk and thigh significantly increase test retest reproducibility (Amundsen, 1990; Bohnannon et al., 1986; Lunner et al., 1981; Richard and Currier, 1977). Straps were required in all tests for the chest and waist. For all tests the subjects were instructed to fold arms during muscular exertion.

#### 3.4.3.4. Warm-up

A warm-up can increase performance by a subsequent increase in blood flow and muscular and core temperature (Franks, 1972). It has been reported that three submaximal and 3 maximal warm-up repetitions are sufficient to find stability of a peak torque measurement during knee extension (Johnson and Siegel, 1978). Good reliability of measurement after this warm-up protocol in the knee joint for peak torque, work and power has been supported by Perrin (1986). Amundsen (1990) suggests a warm-up should precede each isokinetic velocity so the subject can 'get used' to each new speed of movement. Warm

ups did precede each test for each testing velocity for each study and these are described in the methods section of each study.

#### 3.4.3.5. Verbal encouragement

The presence or absence of verbal encouragement has a dramatic influence on the ability to produce maximum effort (Perrin, 1993). Standardised verbal encouragement was used to achieve maximal effort from the subject. The verbal commands were presented in a strong and aggressive manner and consisted of '*push up*', '*pull down*' and for protocols consisting of 10 repetitions, '*five to go*' and '*three to go*'.

#### 3.4.3.6. Visual feedback

Visual feedback involves the subject observing the computer monitor and its visual display during the test. This knowledge of results during an assessment may improve maximal voluntary exertion. Greater peak torque has been reported following knee extension and flexion when visual feedback was allowed during slow and intermediate testing velocities (Baltzopoulos et al., 1991; Hald and Bottjen, 1987; Figoni and Morris, 1984). However at fast testing velocities, Baltzopoulos et al. (1991) and Figoni and Morris (1984) found visual feedback had no effect. It has been suggested that the central nervous system has insignificant time to process the visual information at fast velocities (Perrin, 1993). During all tests and for each testing velocity visual feedback was allowed.

#### 3.4.3.7. Test velocity selection

A good indicator of muscular capabilities is testing at velocities which simulate actual tasks performed in an activity. The functional angular velocity of the knee during walking is 233 $deg.s^{-1}$ (Hart et al., 1984) and during running 1200 $deg.s^{-1}$ (Dillman, 1970- cited Elliot and Blanksby, 1979; Parker, 1981). However modern isokinetic dynamometers maximum testing velocities are only 500 (Cybex 6000) and 300 $deg.s^{-1}$ (KinKom AP II). During isokinetic testing, recruitment of both slow and fast twitch muscle fibres may be accomplished exclusively at slower velocities (Perrin, 1993). However, motor unit recruitment patterns vary with velocity and therefore it appears it is important to test through a wide spectrum of velocities. During eccentric actions, according to Stauber (1989) healthy subjects have motor control difficulty which accentuates as the testing velocity increases. It has been reported there is a higher risk of subjects sustaining an injury during eccentric

actions at fast testing velocities (Perrin, 1993). The testing velocities in each study were chosen based on these findings and from the suggested speeds for slow, intermediate and fast velocities in the Cybex manual (Table 3.2.).

**TABLE 3.2.** *Test velocity catagories (From Cybex Manual)*

| Slow | Intermediate | Fast |
|---|---|---|
| 0-60 deg.s$^{-1}$ | 90-180 deg.s$^{-1}$ | 180-300 deg.s$^{-1}$ |

#### 3.4.3.8. Number of repetitions

To obtain true maximal value of torque, regardless of velocity, multiple actions are necessary (Perrin, 1993). Maximum torque is typically found between the first 2-6 repetitions (Baltzopoulos and Brochie, 1989). Perrin (1993) states 3-4 repetitions are sufficient. Based on these findings, three maximal repetitions were performed when assessing maximal muscle performance.

#### 3.4.3.9. Calibration

Calibration is important to ensure validity and reliability (Mayhew and Rothstein, 1985). The dynamometer was stabilised on a level floor to prevent artefacts, overshoot and oscillation (Amundsen, 1990). Equipment was calibrated, using each system's calibration protocol, at the start of each testing day (Appendix C).

#### 3.4.3.10. Time of day

Peak torque may vary during different times within a day (Wyse et al., 1994). Thus, all re-tests were performed at the same time of day.

#### 3.4.3.11. Summary

Therefore by following these findings and guidelines the reliability for testing was enhanced. The reliability of each subject's effort however was less controllable, i.e. the willingness of the subject to provide maximum effort and tolerance to discomfort at each testing occasion. However verbal encouragement and visual feedback did reduce this error. Additionally, Perrin (1993) suggests it is impossible to produce maximal force if pain is experienced. Therefore all subjects were instructed and monitored to stop the test if pain was evident during testing. Finally the mechanical reliability for

Cybex and KinKom have been previously reported which indicate that these dynamometers provide a valid and reliable test measure (Farrell and Richards, 1986; Moffroid et al, 1969; Johnson and Siegel, 1978; Molnar et al., 1979).

### 3.4.4 Seat Positioning

The major muscle groups of the lower body were tested: hip extension and flexion, knee extension and flexion and, ankle plantarflexion and dorsiflexion. The Cybex 6000 and KinKon AP II dynamometers had incorporated into their software a program which took the experimenter through a series of seat positioning procedures, which were followed closely. First, the dynamometer was correctly positioned according to the selected joint movement. Appropriate accessories were added and adjustments were made to the chair position and/or Utility Bench Exercise Table (U.B.X.T.) seats and back positions. Adjustments were required for individual limb length variations. Appropriate stabilisation was attached and secured to each subject as described in the instruction manual supplied by the manufacturer. By eye and palpation the axis of rotation of the lever arm was aligned with the anatomical axis of the joint being tested, as described in the test manuals. To ensure the alignment of rotational axes and accessory arm length was correct, the subject moved through the active range of motion. Each subjects anatomical zero position was set before commencing the test.

Finally the range of motion stops were located at both extreme ranges as an added safety feature. Each joint tested required specific considerations:

*Knee:* The adjustable arm was installed in the dynamometer input tube. The back rest was adjusted to 10° to the vertical. The axis of the knee was taken at the line passing transversely through the femoral condyles. The shin pad was positioned 2 cm proximal to the medial malleolus. A thigh velcro strap for the Cybex 6000 and a V-shaped pad for the KinKom AP II was placed securely across the top of the subject's thigh. Anatomical zero was set at full




**FIGURE 3.3.** *Subject and equipment placement for testing the knee joint on the Cybex 6000.*

at full knee extension. The positioning of the knee joint on the Cybex 6000 is shown in Fig. 3.3.

*Hip:* The short adjustable arm was installed in the dynamometer input tube. The back rest was adjusted to the horizontal and the U.B.X.T. was placed perpendicular to the seat. The dynamometer's height was adjusted and aligned with the subject's hip. The axis of rotation was taken approximately 1 cm superior and anterior to the greater trochanter. The shin pad was positioned superior and proximal to the knee joint line. Several straps were placed around the hip and waist. Anatomical zero was set at full leg extension. The positioning of the hip joint on the Cybex 6000 is shown in Fig. 3.4.

*Ankle:* The dynamometer was set forward and the ankle adapter and footplate were installed into the input tube. The position of the U.B.X.T. was set with the backrest to the lowest position and seat to highest position. A thigh stabiliser was installed into the U.B.X.T. and positioned proximal to the knee joint line. The subject's knee was in 90 degrees flexion to emphasise the

solelus. The test with the knee at full extension was perceived, during pilot testing, to exhibit inaccuracies due to insignificant strapping and the instability of the U.B.X.T. under maximal muscle exertion. The axis of rotation passed obliquely through the tip of the fibula and the troclea of the talus and exited just distal to the tip of the tibia. This was accomplished by appropriately positioning the foot on the footplate. All subjects wore tennis type shoes with a flat heel and were strapped into the footplate over the ankle and across the foot. Two additional velcro straps were placed around the waist and upper chest. Anatomical zero was set with the ankle at $90^{o}$. The positioning of the ankle joint on the Cybex 6000 is shown in Fig. 3.5.

### 3.4.5 Testing Procedures

Two protocols were designed, one to measure maximal muscle performance (strength test) and one an exercise protocol.

#### 3.4.5.1 Strength test protocol

All joint movements were tested concentrically in the extensors and flexors and eccentrically in the extensors only. The dominant leg, based on kicking preference, was tested. All movements were tested at slow, intermediate and fast speeds ranging from 60-240 $deg.s^{-1}$. These speeds and joint actions were determined by pilot testing the subjects to find the slowest and fastest velocities during both concentric and eccentric actions that they could comfortably be tested without a perceived risk of injury. Several subjects perceived the risk of injury during eccentric flexion in all the joints to be high, thus this test was excluded. All subjects were tested in the evening. Readings were taken during knee concentric extension and flexion at 60, 150, and 240 $deg.s^{-1}$ and for eccentric extension at 60 and 240 $deg.s^{-1}$. The readings were taken during hip and ankle concentric extension and flexion at 60, 120 and 180 $deg.s^{-1}$ and for eccentric extension at 60 and 180 $deg.s^{-1}$.

#### 3.4.5.2 Repeated bouts of knee extension and flexion protocol

The exercise test protocol consisted of 10 sets of 10 maximal repetitions for knee extension and flexion at a velocity of 120 $deg.s^{-1}$. The dominant leg, based on kicking preference, was tested. The repeated bouts of knee extension and flexion protocol involved either concentric or eccentric actions. Three min before the start of the test, three sets of the corresponding muscle actions were performed, which served both as a warm-up and to re-accustom the subjects to the task. Sixty seconds separated each set.

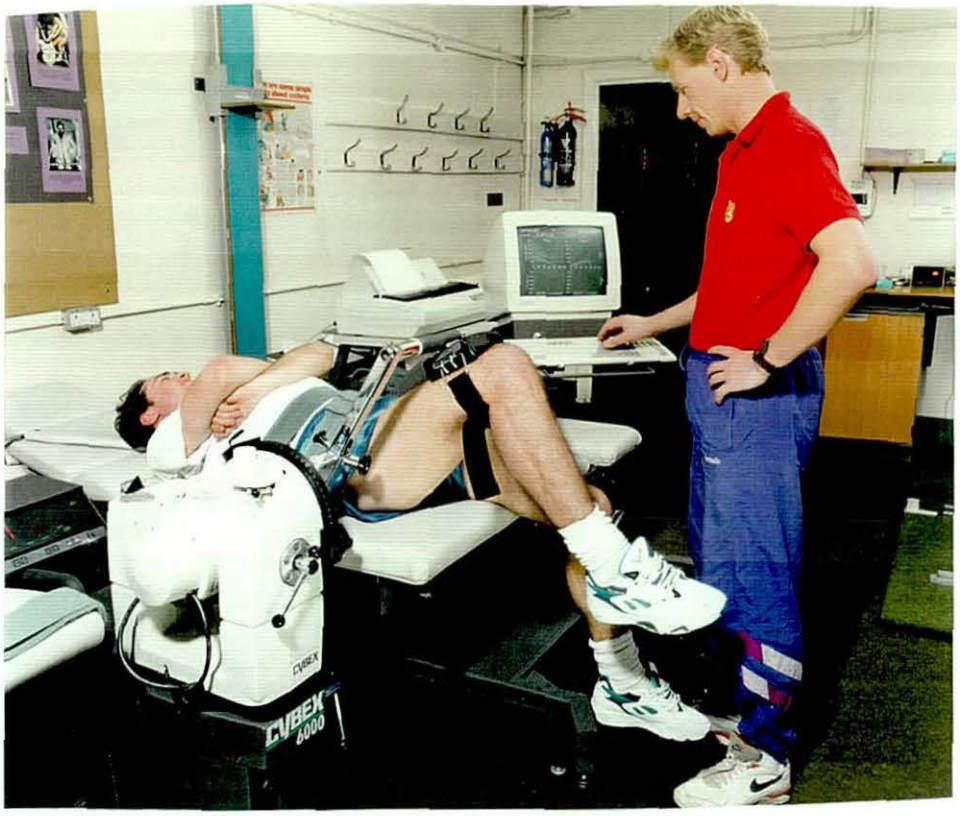



**FIGURE 3.4.** *Subject and equipment placement for testing the hip joint on the Cybex 6000.*

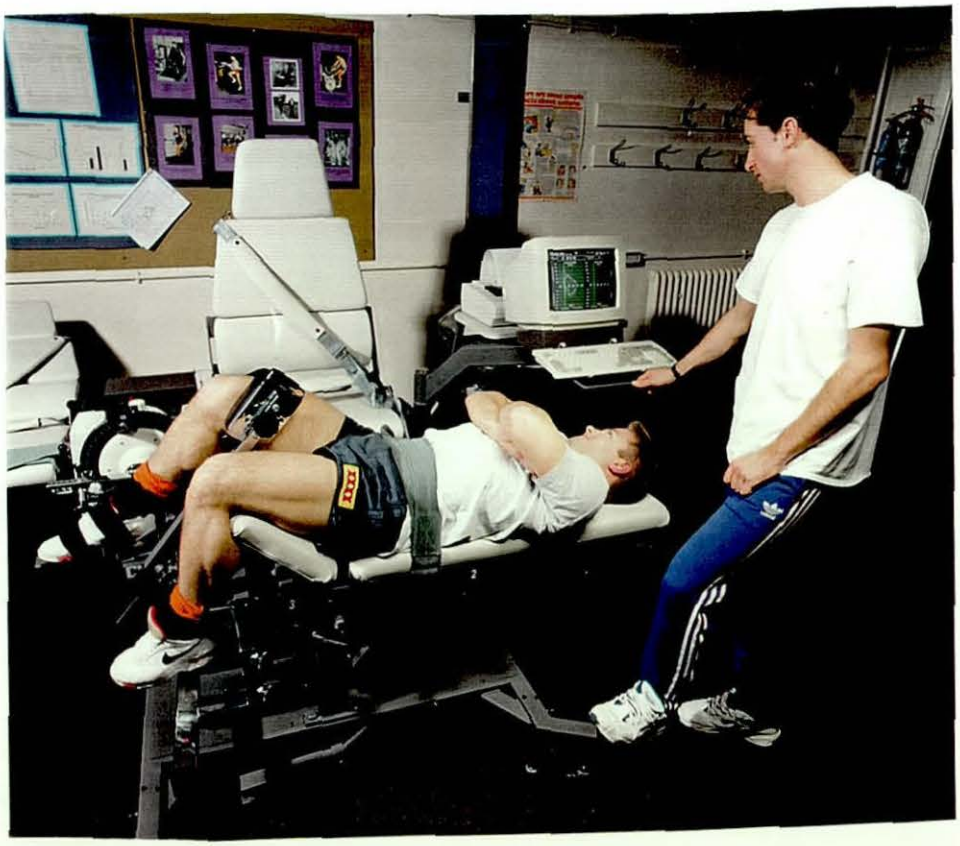


**FIGURE 3.5.** *Subject and equipment placement for testing the ankle joint on the Cybex 6000.*

### 3.4.6 Measurements

#### 3.4.6.1 Torque

The reliability of peak torque and work have all been estabilished for all the muscle groups tested in these studies (Perrin, 1986). For each repetition in all tests, torque was measured throughout the range of motion. Torque is a measurement of force about the axis of rotation for a joint. The Cybex 6000 measures the tension produced by a muscle from the axis of rotation of the dynamometer, thus this device is capable of measuring only torque. In contrast the KinKom AP II assesses the capacity of muscle to produce tension against the resistance pad of the dynamometer, which is appropriately fixed to a lever arm, and thus force can be measured. However, for standardisation force measurements were converted to torques by a pre-programmed package on the software, by determining the distance of the resistance pad from the joint's axis of rotation. The Systeme International d'unites (S.I units) for torque is the Newton meter (N.m). Combining the overall effectiveness of a muscle action and the length-tension relationship, the greatest torque is shown through the mid-range of the available range of motion for a joint and is less at the extreme range of motion. Torque can be reported as either peak or average torque. Peak torque is the highest point on the torque curve, whereas average torque is calculated from the torque production throughout the range of motion. Both peak torque and average peak torque have been reported to produce a valid assessment of a muscle's ability to generate tension (Perrin, 1993). Therefore peak torque only was used as the assessment of the ability of muscle to generate tension. The repetition for each test which exhibited the greatest peak torque value was taken as the measure of maximal strength.

In addition peak torque was expressed, in some studies, in relation to body mass. This individualises the interpretation of results because of the variations in body size and somatotypes within subjects, thus allowing comparison between subject groups in spite of marked differences in body size. Peak torque was divided by each subject's body mass yielding a Newton metre per kilogram ($N.m.kg^{-1}$) value.

Peak torque angle can also be calculated from each repetition performed isokinetically. This is the angle where the peak torque measurement was recorded. The unit of measurement is degree ($^{o}$).

Ratios between the reciprocal muscle groups were calculated for each joint. It has been long recognized that it is important to train both muscle groups which produce opposite actions about a joint. It has been suggested that excessive imbalances in reciprocal muscle group ratios predispose the muscle groups to injury (Perrin, 1993).

#### 3.4.6.2 Work

Work is the amount of tension produced by the muscle over a given distance which can be determined if the torque and range of movement of a muscle action is known. The S.I. units for work is the joule (J).

*Work Formula:* Torque x Angular Displacement = Work

In the present study total work refers to the sum of work performed throughout the 10 repetitions from the repeated knee extension and flexion exercise bout.

#### 3.4.6.3 Fatigue Index

During the repeated knee extension and flexion exercise bout, which consists of 10 sets of 10 repetitions, a fatigue index was calculated. The difference between the highest and last total work was taken from the ten sets and calculated using the formula below. The unit of measurement is percentage (%).

*Fatigue Index Formula:* $$\frac{\text{Highest value - Last value}}{\text{Highest value}} \times 100$$

## 3.5 COLLECTION AND ANALYSIS OF BLOOD SAMPLES

### 3.5.1 Blood sampling collection

During the exercise test, venous blood samples were taken via an indwelling cannula (Venflon 2 Model 1453-0I-V cannula, Viggo-Spectramed) that remained in place throughout the duration of each test (approximately 60 min). The cannula was placed in a large forearm vein such as the cubital or anticubital vein and a three way tap (Connecta, 3 Way Stopcock) was attached to the cannula via a length of flexible transparent tubing secured to the forearm using adhesive tape. The tube and cannula were flushed with a 0.9 % sodium chloride saline solution after each sample to maintain patency.

Prior to taking a sample the saline solution remaining in the cannula and tubing was drawn off and disposed of. Then a 6 ml aliquot of blood was drawn off using a 10 ml syringe (Becton Dickinson). This sample was dispensed into two calcium heparinised tubes. Using blood samples from one tube haematocrit was determined using 30 µl microhaematocrit tubes which were centrifuged (Hawksley Micro-haematocrit centrifuge) and read (Hawksley Micro-haematocrit centrifuge reader), and haemoglobin was assayed spectrophotometrically (Cecil series 2, CE393 Digital grating spectrophotometer) using a commercially available kit (Boehringer Mannheim). Additionally two 20 µl samples of whole blood were dispensed into 200 µl aliquots of perchloric acid in an eppendorf tube. These samples were then stored at -20 $^{o}$C, and assayed at a later date fluorimetrically (Locarte Mk 8-9 Mercury lamp Fluorimeter) for lactate. The second heparinised tube was immediately centifuged (Eppindorph Model 5414) for 3 min and the supernatant was taken and stored at -70 $^{o}$C and assayed spectrophotometrically (Cecil series 2, CE 393 Digital grating spectrophotometer) for ammonia using a commercially available kit (Boehringer Mannheim).

### 3.5.2 Analysis of blood samples

#### 3.5.2.1 Blood lactate

Blood samples were assayed for lactate according to the method described by Maughan (1982), reaction (7), and as outlined below.

In alkaline conditions (pH 9.1) the forward reaction is favoured and continues to completion, thus an equivalent molar amount of both pyruvic acid and

NADH (reduced form of nicotinamide adenine dinucleotiude) are produced from lactate. NADH is a flourescent compound so the flourescence can be quantitatively determined and will be directly proportional to the concentration of lactate in the blood sample.

Reaction mixture:

i) 2 mg NAD per ml hydrazine buffer.

ii) 10 µl lactate dehydrogenase (LDH) per ml hydrazine buffer.

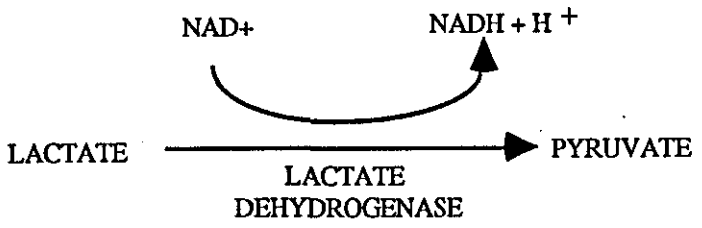
Hydrazine buffer:

i) 1.3 g hydrazine sulphate.

ii) 5.0 g hydrazine hydrate.

iii) 3.0 g EDTA (complexing agent).

per 100 ml distilled water.




(7)

Lactate standards of known concentrations (0, 0.5, 2, 5, 10 and 15 $mmol^{l-1}$) were used to provide a calibration curve ($r \geq 0.999$). Samples taken from the -20°C freezer and allowed to thaw at ambient room temperature (40 min). Each sample was then homogenised (whirlmixer) and centrifuged (Eppendorf model 5414). A 200 µl aliquot of reaction mixture was added to a 20 µl aliquot of supernatent. The tube contents were then mixed. Samples were incubated for 30 min at room temperature after which 1 ml of lactate diluent (0.07 HCL) was added and the solution was remixed before reading the fluorescence (Locarte Mk 8-9 Mercury lamp Fluorimeter). Blood lactate was determined using the regression equation derived from the lactate standards.

### 3.5.2.2 Plasma ammonia

The method for the Boehringer Mannheim assay kit used was based upon the reaction below;

$$\text{a- OXOGLUTARATE} + NH_4^+ + \text{NADPH} \xrightarrow{\text{GLDH}} \text{L-GLUTAMATE} + \text{NADP}^+ + H_2O \quad (8)$$

Samples were stored in a freezer at -70 C until analysis. All analysis was performed within 48 hours of the sample being drawn. After the samples were thawed at ambient room temperature (40 min), they were centrifuged (Eppendorf model 5414) and 100 µl of the supernatant was taken and placed in a cuvette. Next, 500 µl of the kit reaction mixture was added, and the solution was gently agitated. Blank samples (600 µl reaction mixture) and standards of known concentration were also prepared. An incubation period of 10 min at ambient room temperature followed. The absorbance of the samples and the blanks were then measured (Cecil series 2, CE393 Digital grating spectrophotometer: 340 nm) and 4 µl of the enzyme GLDH were added and the solution gently agitated. Absorbance was read following a 10 min incubation period. The final step was repeated and from the three sequential readings and the blank absorbance values. Ammonia concentration was determined using an equation provided by the Boehringer Mannheim assay kit.

### 3.5.2.3 Haemoglobin

The cyanmethaemoglobin method was used. This is a colourimetric technique based on the following principle;

$$\text{HAEMOGLOBIN} + \text{CYANIDE} + \text{FERRICYANIDE} \longrightarrow \text{CYANMETHAEMOGLOBIN} \quad (9)$$

(Vam Kampen and Zilstra, 1961)

A single reagent from the Boehringer Mannheim assay kit is used to produce the reaction mixture. The reagent is known as 'Drabkins' and contains phosphate buffer, potassium cyanide, potassium ferricyanide and detergent. The prepared Drabkins is diluted in distilled water (1000 ml) and stored in a brown glass bottle at +15 $^{o}$C to +25 $^{o}$C. A 20 µl aliquot of venous blood was added to 5.0 ml of Drabkins reagent in a glass tube with screw top lid and mixed well. The solution was then incubated at ambient room temperature for 15 mins. Absorbance was measured against a reagent blank (Drabkins reagent) using the Cecil series 2, CE 393 Digital grating spectrophotometer from which the haemoglobin concentration was calculated.

#### 3.5.2.4 Haematocrit

Haematocrit was determined immediately after sampling by taking duplicate aliquots of venous blood using 30 µl microhaematocrit tubes, which were then centrifuged (Hawksley Micro-haematocrit centrifuge) and read (Hawksley Micro-haematocrit centrifuge reader).

#### 3.5.2.5 Changes in plasma volume

Changes in plasma volume were calculated, on a Macintosh computer using Microsoft excel package, from the haemoglobin and haematocrit values (Dill and Costill, 1974) and were expressed as a percentage change from the resting plasma volume.

## 3.6 STATISTICAL ANALYSIS

The results were analysed statistically by analysis of variance to determine significant differences. Where significant F ratios were found, the means were compared using a Scheffe post-hoc test.

Relationships between variables were examined by calculating the Pearson product-moment correlation coefficient (r).

To predict a variable further analysis was conducted using Stepwise regression analysis ($R^2$).

Values are presented as the mean and standard deviation (mean $\pm$ S.D) of the results.

# *CHAPTER 4. THE RELATIONSHIP BETWEEN CONCENTRIC AND ECCENTRIC MUSCLE STRENGTH AND SPRINT RUNNING PERFORMANCE*

## 4.1 INTRODUCTION

A primary factor in athletic success is the capacity of a muscle to generate a high explosive force. Muscles in the lower limb need to develop great amounts of tension rapidly for high speed movements (Manning et al., 1988). Sprinters have been aware of this and have for many years, used strength training techniques to enhance their performance.

The view that strength training may enhance sprint performance is also common amongst participants in other sports, such as hockey, soccer and rugby. However unlike track sprinting these sports involve many players competing within confined spaces. This makes the ability to accelerate into positions a very important quality as well as good maximal velocity. It is also widely accepted that in sprint events on the track a powerful start is essential to reach a high level of performance (Mero, 1988). Therefore there are two aspects to consider with relation to muscle strength in sprinters and other sports: acceleration and maximal velocity.

Authors have stated, as described in Section 2.2.1., that strength training enhances motor performance in short sprints of distances from 5-60 yards (4.6-54.9 metres) (Adams et al., 1991; Chui, 1950; Dintiman, 1964; Peterson, 1975; Schultz, 1967; Smith & Melton, 1981; Taiana et al, 1992). However, many of these studies have been poorly designed or controlled and conflicting results have been reported. Ambiguity can therefore, be seen to exist within the literature, on whether or not strength training across particular joints and or during different muscle actions or at fast or slow velocities may be beneficial for the enhancement of sprint performance.

By analysing the muscle strength of track sprinters and relating strength to sprint performance it may be possible to suggest which muscle groups and which muscle action at which testing velocity may be most beneficially incorporated into strength training programmes to enhance sprint performance. Figure 2.2 (see review) summarises the joint movements and concentric and eccentric muscle actions involved in each phase in a single leg

cycle during sprinting. Each muscle action, in the three lower limb joints are all involved in a sprint cycle excluding concentric ankle dorsiflexion. However the importance and influence of each muscle action on sprint perfrormance is currently not completely known.

One approach is to examine the literature regarding significant relationships between muscle strength in any of the lower limb movements at various velocities with sprint performance. However the literature is extremely limited and most studies have documented only poor relationships, which are described in Section 2.2.2 (Anderson et al., 1991; Berg et al., 1986; Berger and Blaschke, 1967; Costill et al., 1968; Farrar and Thorland, 1987; Liba, 1967; Manning et al., 1988; Osinski, 1988). These poor relationships may be partly due to use of inappropriate strength and speed tests, the examination of only one joint action or to an incomplete investigation of the relationship between strength and sprint performance measures. Alexander (1989) used elite sprinters and significant correlations were found between 100 m sprint times and peak torque of the concentric knee extensors and eccentric dorsiflexors. This study used a stepwise multiple regression and found two variables as predictors for 100 m sprint times, concentric knee extension and eccentric hip flexion at 230 and 180 deg.$s^{-1}$ respectively ($R^2$=0.67). Guskiewicz et al. (1993) also used athletes and examined hip extension and flexion at various velocities and found significant relationships with 40 yard sprint times at 60 deg.$s^{-1}$ (r=-0.57 and r=-0.56 respectively). However this study and others, have only examined the relationship between strength and sprint times, whereas theoretically one would expect body mass and dimensions to also contribute to sprint performance.

Astrand and Rodahl (1986) stated that units such as acceleration, force and power can be expressed as derivatives from basic units such as length, mass and time. Gunther (1975) expressed these body dimensions into theoretical models. Two of these are presented in equation 1 and 2.

| Equation 1 and 2. Descriptive Analysis Model | |
|---|---|
| *Equation 1*: $a=Lt^{-2}$ | *Equation 2*: $F=LMt^{-2}$ |
| Key: Acceleration (a), Limb length (L), Time (t), Force (F), Body mass (M). | |

Despite the existence of these theoretical models, no study to the authors knowledge, has attempted to examine the relationship between strength and

sprint performance using both time and acceleration as a performance measure and by taking body dimensions, in addition to body mass, into account.

The modern isokinetic dynamometers now allows the examination of eccentric torque. There has been limited research on identifying the differences in the relationships between concentric and eccentric muscle force and motor performance.

Previous studies have examined the relationship between strength and sprint performance from standing start sprints over a set distance or by using personal best times over 100 m. The best distance suited for assessment of maximal running velocity has been suggested to be 30 m (Fleck and Kraemer, 1987). However, Kollath and Quade (1992) illustrated that identical times over a set distance may have different split times. This study shows that a greater analysis of acceleration and running speed in detail can be made if split times are included.

The purpose of this study was to examine the relationship in elite sprint related performers between concentric and eccentric isokinetic muscle strength at three lower limb joints and sprint performance including the use of Gunther's (1975) theoretical models. This information may facilitate recognition of the muscle strength characteristics which are beneficial for sprint performance.

In addition, another purpose of this study was to compare the differences in torque production and ratio calculations at different testing velocities between concentric and eccentric actions. This provides an opportunity to validate the torque results with established relationships and examine other differences in the two types of muscle actions such as; flexion/extension ratios and peak torque angles.

## 4.2 METHODS

### 4.2.1 Subjects

Eight rugby players (one scrum-half, one fly-half, one centre, three wing, three quarters and two fullbacks), eight track sprinters (two 100m, one 200m and five 400m runners) and eight 'active' competitive sportsmen (two from soccer, one squash player, one rugby forward, one tennis player, one swimmer, one endurance athlete and one gymnast) participated as subjects in this experiment which had University Ethical Committee approval. Test procedures were explained to the subjects, who were all male. Each subject completed a medical questionnaire to ensure no-one possessed a musculo-skeletal injury or health problem. Several members from each group were among the best in the country in their respective sports, while the others were at elite regional level.

### 4.2.2 Experimental procedures and protocol

#### 4.2.2.1 Anthropometric Measurement

Standard measurements of height, body mass and lower limb lengths were taken for each subject (Weiner and Lourie, 1981). Skinfold thicknesses were taken at four sites on the body and used in conjunction with regression equations derived by Durnin and Womersley (1974) to estimate percentage body fat. All measurements are described in Chapter 3.

#### 4.2.2.2 Sprint Protocol

Timing for the sprint tests was measured using photo-electric cells. All sprints were performed in an indoor sprint corridor over distances of 15 m (acceleration test) and 35 m (basic speed test). The order of the two sprint distances was randomised for each subject. All subjects were tested in the evening. All measurements were electronically timed in seconds to two decimal places.

#### 4.2.2.3 Strength Protocol

A Cybex 6000 Isokinetic Dynamometer was used for the strength tests. Each subject had a standardized familiarisation session which included all tests at least one week before their experiment. The major muscle groups of the lower body were tested: hip extension and flexion, knee extension and flexion and, ankle plantarflexors and dorsiflexors. All tests were completed in a

single session. All joint movements were tested concentrically in the extensors and flexors and eccentrically in the extensors only, as described in Chapter 3.

Each subject was measured on an adjustable chair and the Upper Body Exercise Table (U.B.X.T.) and stabilised with velcro straps. The axis of rotation of the Cybex lever arm was aligned with the anatomical axis of the joint being tested, as described in Chapter 3. Both the Ramping and Gravity Correction features were used in all tests to avoid previously found problems such as torque overshoot and gravity effects respectively (Winters et al. 1981, Sapega et al., 1982, Sapega, 1990).

The repetition for each test which exhibited the greatest peak torque value was taken as the measure of maximal strength. In addition peak torque was expressed in relation to body mass.

Readings were taken for concentric knee extension and flexion at 60, 150, and 240 deg.$s^{-1}$ and for eccentric knee extension at 60 and 240 deg.$s^{-1}$. The readings for concentric hip extension and flexion, and ankle plantarflexors and dorsiflexors were taken at 60, 120 and 180 deg.$s^{-1}$ and for eccentric hip extension and ankle plantarflexion at 60 and 180 deg.$s^{-1}$. The joint movements and velocity orders were randomised for all subjects which reduces the chance of fatigue or the learning effect influencing the torque averages (Morris et al., 1983).

### 4.2.3 Statistical anaylsis

#### 4.2.3.1 Analysis of results

The results were analysed statistically using a one way analysis of variance and a Scheffe test, except where modelling was used (as described in Section 4.2.3.2):

Anthropometry
Sprint times
Isokinetic peak torque
Peak torque ratios
Peak torque angles.

A Pearson Product Moment Correlation was used to examine the relationship between variables. Results are presented as mean $\pm$ S.D.

#### 4.2.3.2 Modelling

Using multiple log-linear regression*, all the peak torque values (F) were predicted, based on Gunther's force performance model, equation 2, using the three variables: body mass (M), the limb length of knees to buttock (L) and the time to sprint 0-15 m (t), i.e., by defining $\log_e$ (F) as the dependent variable and allowing $\log_e$ (L), $\log_e$ (M) and $\log_e$ (t) to be the independent or predictor variables, i.e.,

$$\log_e (F) = \log_e (L). \log_e (M). \log_e (t)^{-2}$$

---

**Footnote:**

* Log-linear regressions is the term used to describe a linear regression analysis having previously adjusted logarithmically both the dependent and independent variables.

## 4.3 RESULTS

### 4.3.1 Track, rugby and active group differences

#### 4.3.1.1 Sprint performances for the sprint, rugby and active groups

The physical characteristics and sprint times of the subjects in each group are presented in Table 4.1. The Rugby group had significantly higher percentage body fat than the Sprint group ($p<0.05$). No other significant differences were found in the anthropometric results. The Sprint group were faster over 0-15 m, 5-10 m, 10-15 m and 30-35 m than the Active group ($p<0.05$). The Rugby group were also faster than the active group for 10-15 m ($p<0.05$). The split times over 10-15 m measured as a percentage of the 30-35 m split shows a significantly higher percentage for Rugby and Active groups compared with the Sprint group.

#### 4.3.1.2 Muscle strength

Peak torque relative to body mass for the knee extensors and flexors during concentric and eccentric actions are shown in Table 4.2. The sprinters had higher torque values ($N.m.kg^{-1}$) than both the rugby and active groups for the concentric extensors at all velocities and at the fast velocity during eccentric actions ($p<0.05$). However there was no significance between groups for absolute peak torque values. There were no significant differences between the groups for concentric or eccentric hip extensors, hip flexion, ankle dorsiflexors and ankle plantarflexors when expressed as absolute or relative torque values.

Peak torque ratios for the flexors/extensors and dorsiflexors/plantarflexors were calculated for each group at each velocity and there was no significant difference between the groups.

### 4.3.2 Isokinetic differences between concentric and eccentric (n=24)

The isokinetic muscle strength results for all subjects are reported and illustrated in Table 4.3 and Figure 4.1a and 4.1b. Eccentric peak torques were significantly higher ($p<0.01$) compared with corresponding concentric torques for each joint and testing velocity. The greatest mean peak torque was $324.7 \pm 64.4$ N.m, produced by eccentric knee extensors at 60 $deg.s^{-1}$. Peak torque during concentric actions decreased as the velocity increased ($p<0.01$ and 0.05). There were no significant differences in peak torques between the fast and slow velocities during eccentric actions.

**TABLE 4.1.** *Anthropometric results and sprint times for each subject group (mean ± S.D.)*

| | | Rugby Group (n=8) | Sprinters Group (n=8) | Active Group (n=8) | All Data (n=24) |
|---|---|---|---|---|---|
| Age (yr) | | 23.6 ± 1.9 | 22.5 ± 3.7 | 28.5 ± 8.4 | 24.9 ± 6.1 |
| Height (cm) | | 177.3 ± 5.3 | 180.9 ± 5.4 | 180.1 ± 7.5 | 179.4 ± 6.1 |
| Body mass (kg) | | 82.1 ± 8.1 | 77.3 ± 8.2 | 75.5 ± 6.5 | 78.3 ± 7.8 |
| Body Fat (%) | | 16.8[a] ± 3.7 | 12.1[a] ± 1.9 | 14.7 ± 4.1 | 14.6 ± 3.8 |
| Lean Body Mass (kg) | | 68.1 ± 5.2 | 68.0 ± 7.8 | 64.4 ± 5.5 | 66.8 ± 6.2 |
| Tibial Length (cm) | | 41.9 ± 1.5 | 41.6 ± 1.6 | 42.7 ± 2.7 | 42.0 ± 2.0 |
| Foot Length (cm) | | 24.6 ± 1.0 | 25.9 ± 0.9 | 25.8 ± 1.7 | 25.5 ± 1.4 |
| Buttocks-Knee Length (cm) | | 62.5 ± 2.5 | 62.8 ± 2.6 | 62.4 ± 4.8 | 62.6 ± 3.6 |
| Sprint Times | | | | | |
| 0-5 metres | Time (s) | 1.00 ± 0.06 | 0.97 ± 0.09 | 1.03 ± 0.06 | 1.00 ± 0.07 |
| | Percentage of 30-35 m | 57.1 ± 4.3 | 55.2 ± 3.7 | 58.9 ± 7.5 | 57.1 ± 4.6 |
| 5-10 metres | Time (s) | 0.71 ± 0.04 | 0.68[b] ± 0.04 | 0.74[b] ± 0.04 | 0.71 ± 0.05 |
| | Percentage of 30-35 m | 80.0 ± 3.8 | 78.9 ± 3.9 | 81.4 ± 0.66 | 80.1 ± 3.8 |
| 10-15 metres | Time (s) | 0.63 ± 0.03 | 0.63 ± 0.03 | 0.66 ± 0.05 | 0.64 ± 0.05 |
| | Percentage of 30-35 m | 90.3[a] ± 4.2 | 84.8[ab] ± 3.8 | 91.2[b] ± 4.4 | 88.7 ± 4.9 |
| Time to reach 15 metres (s) | | 2.34 ± 0.10 | 2.28[b] ± 0.11 | 2.43[b] ± 0.11 | 2.35 ± 0.12 |
| Time to sprint 30-35 m (s) | | 0.57 ± 0.04 | 0.53[b] ± 0.02 | 0.60[b] ± 0.05 | 0.57 ± 0.05 |

*a* Significant difference between the Rugby and Sprinters groups $p<0.05$

*b* Significant difference between the Sprinters and Active groups $p<0.05$

**TABLE 4.2.** *Peak torque, relative to body mass, N.m.kg$^{-1}$ (mean ± S.D.) measured from the knee joint on an Isokinetic Dynamometer for three subject groups of rugby, sprinters and active sportsman.*

| | | | Rugby Group (n=8) | Sprinters Group (n=8) | Active Group (n=8) |
|---|---|---|---|---|---|
| Concentric | Extensors | 60 deg.s$^{-1}$ | 3.05$^{a}$ ± 0.36 | 3.58$^{ab}$ ± 0.26 | 3.07$^{b}$ ± 0.47 |
| | | 150 deg.s$^{-1}$ | 2.37$^{a}$ ± 0.19 | 2.71$^{ab}$ ± 0.25 | 2.33$^{b}$ ± 0.23 |
| | | 240 deg.s$^{-1}$ | 1.97$^{a}$ ± 0.12 | 2.27$^{ab}$ ± 0.25 | 1.94$^{b}$ ± 0.27 |
| | Flexors | 60 deg.s$^{-1}$ | 1.95 ± 0.21 | 2.16 ± 0.19 | 1.83 ± 0.44 |
| | | 150 deg.s$^{-1}$ | 1.58 ± 0.19 | 1.79 ± 0.19 | 1.52 ± 0.33 |
| | | 240 deg.s$^{-1}$ | 1.39 ± 0.16 | 1.60 ± 0.20 | 1.25 ± 0.38 |
| Eccentric | Extensors | 60 deg.s$^{-1}$ | 4.03 ± 0.69 | 4.45 ± 0.21 | 3.86 ± 0.67 |
| | | 240 deg.s$^{-1}$ | 3.73$^{a}$ ± 0.69 | 4.20$^{ab}$ ± 0.22 | 3.57$^{b}$ ± 0.44 |

*a* Significant difference between the Rugby and Sprinters groups $p<0.05$

*b* Significant difference between the Sprinters and Active groups $p<0.05$

**TABLE 4.3.** *Isokinetic results (mean ± S.D.) of the knee, hip and ankle joints at different velocities.*

| | | | Peak Torque (N.m) | Peak Torque ($N.m.kg^{-1}$) |
|---|---|---|---|---|
| Knee | Concentric Extensors | 60 $deg.s^{-1}$ | 254.7 ± 43.5 | 3.23 ± 0.43 |
| | | 150 $deg.s^{-1}$ | 194.5 ± 30.4 | 2.47 ± 0.28 |
| | | 240 $deg.s^{-1}$ | 162.8 ± 28.4 | 2.06 ± 0.26 |
| | Concentric Flexors | 60 $deg.s^{-1}$ | 156.8 ± 32.1 | 1.98 ± 0.32 |
| | | 150 $deg.s^{-1}$ | 129.3 ± 27.4 | 1.63 ± 0.26 |
| | | 240 $deg.s^{-1}$ | 112.3 ± 27.9 | 1.41 ± 0.29 |
| | Eccentric Extensors | 60 $deg.s^{-1}$ | 324.7* ± 64.4 | 4.13* ± 0.67 |
| | | 240 $deg.s^{-1}$ | 306.4* ± 66.9 | 3.85* ± 0.66 |
| Hip | Concentric Extensors | 60 $deg.s^{-1}$ | 228.3 ± 35.6 | 2.92* ± 0.45 |
| | | 120 $deg.s^{-1}$ | 227.4 ± 42.3 | 2.90* ± 0.47 |
| | | 180 $deg.s^{-1}$ | 221.6 ± 46.5 | 2.82* ± 0.49 |
| | Concentric Flexors | 60 $deg.s^{-1}$ | 188.3 ± 33.3 | 2.39 ± 0.30 |
| | | 120 $deg.s^{-1}$ | 169.7 ± 23.7 | 2.16 ± 0.19 |
| | | 180 $deg.s^{-1}$ | 162.9 ± 23.0 | 2.07* ± 0.20 |
| | Eccentric Extensors | 60 $deg.s^{-1}$ | 305.9* ± 67.4 | 3.98* ± 0.99 |
| | | 180 $deg.s^{-1}$ | 295.6* ± 82.9 | 3.84* ± 1.09 |
| Ankle | Concentric Plantarflexors | 60 $deg.s^{-1}$ | 94.0 ± 13.4 | 1.20 ± 0.17 |
| | | 120 $deg.s^{-1}$ | 71.1 ± 13.0 | 0.91 ± 0.17 |
| | | 180 $deg.s^{-1}$ | 58.4 ± 10.7 | 0.74 ± 0.13 |
| | Concentric Dorsiflexors | 60 $deg.s^{-1}$ | 24.7 ± 5.7 | 0.31 ± 0.07 |
| | | 120 $deg.s^{-1}$ | 18.0 ± 4.0 | 0.23 ± 0.06 |
| | | 180 $deg.s^{-1}$ | 16.5 ± 3.3 | 0.21 ± 0.04 |
| | Eccentric Plantarflexors | 60 $deg.s^{-1}$ | 155.1* ± 33.9 | 1.99* ± 0.46 |
| | | 180 $deg.s^{-1}$ | 148.0* ± 37.6 | 1.90* ± 0.50 |

* No significant difference between the nearest testing velocity

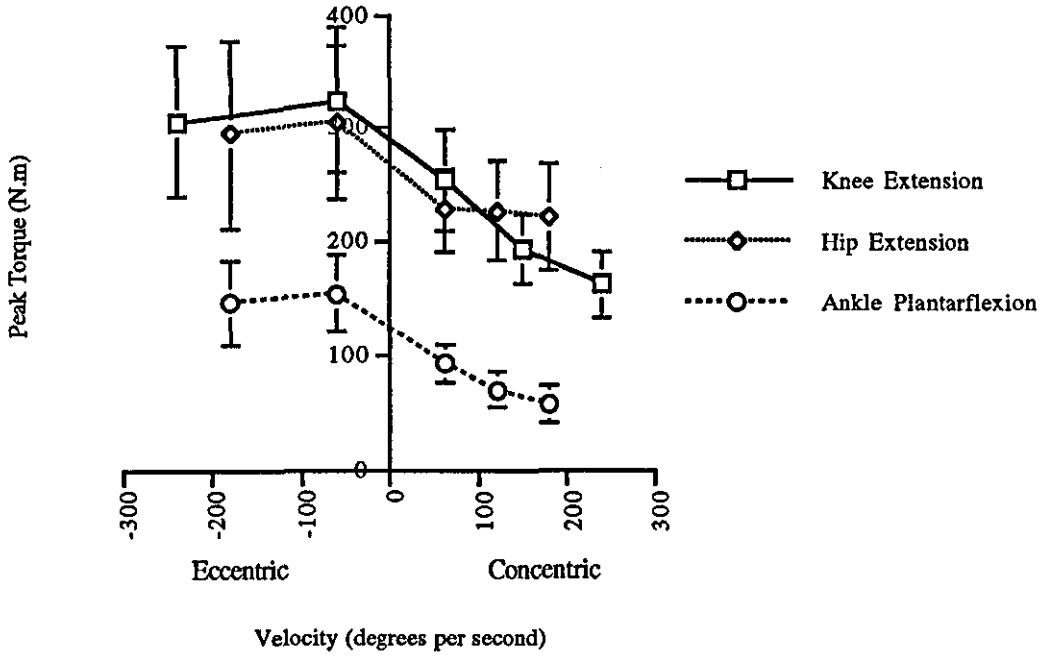



**FIGURE 4.1a** *The relationship between peak torque and testing velocity for both concentric and eccentric muscle actions (mean ± S.D., n=24) in the extensors for the three joints.*

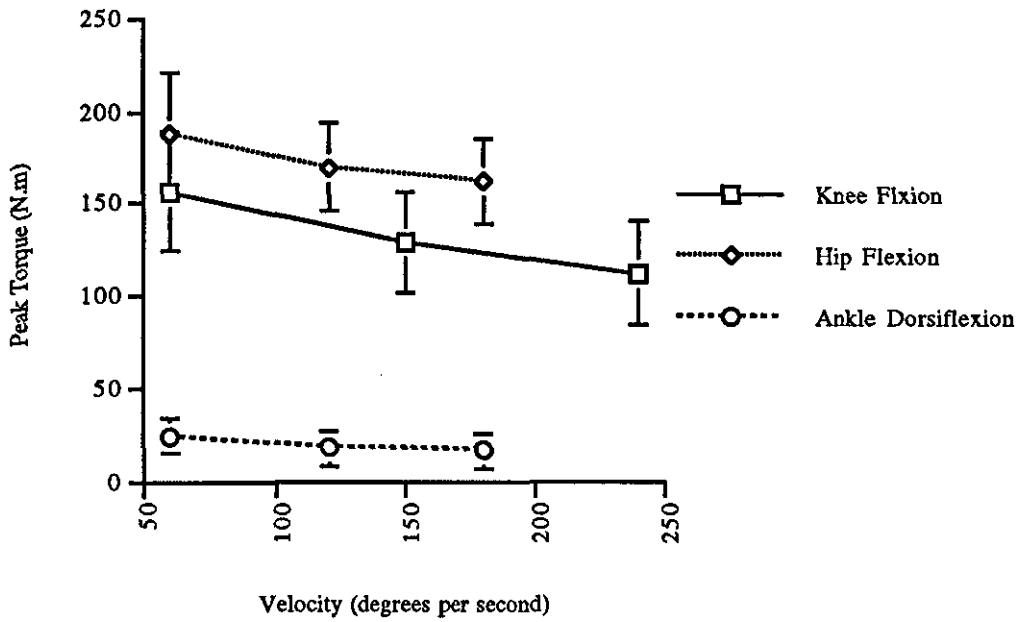



**FIGURE 4.1b** *The relationship between peak torque and testing velocity for concentric muscle actions (mean ± S.D., n=24) in the flexors for the three joints.*

Peak torque ratios between the each joint's flexors and extensors were calculated for the concentric actions and are illustrated in Table 4.4. The knee ratio significantly increased between the slow and fast velocities ($p<0.05$). There was no significant change in the ratios for the different velocities in the hip and ankle joints.

**TABLE 4.4.** *Peak torque ratios (flexors:extensors) for the knee, hip and ankle joints (mean ± S.D.)*

| Velocity ($deg.s^{-1}$) | Knee (%) | Hip (%) | Ankle (%) |
|---|---|---|---|
| 60 | 61.4* ± 6.3 | 83.3 ± 14.1 | 26.5 ± 6.1 |
| 120 | | 75.7 ± 10.7 | 25.6 ± 4.9 |
| 150 | 66.1 ± 8.1 | | |
| 180 | | 75.1 ± 12.0 | 28.6 ± 4.7 |
| 240 | 68.3* ± 10.3 | | |

* Significant differences between testing velocities $p<0.05$

The joint angles at which peak torque was reached (peak torque angles) were calculated for each joint measurement. The average peak torque angle across all test velocities for concentric knee extension and flexion were 60.8 ± 7.7 deg and 35.0 ± 8.8 deg respectively. Peak torque angles decreased during knee extension from 64.1 ± 6.1 deg to 57.5 ± 6.7 deg at 60 $deg.s^{-1}$ and 150 $deg.s^{-1}$ respectively ($p<0.05$). No significant change occurred in either hip extensors or ankle plantarflexion. Peak torque angles significantly increased with increasing velocities during hip flexion and ankle dorsiflexion ($p<0.01$). The average peak torque angles across all test velocities for concentric hip extension and flexion, and ankle plantarflexion and dorsiflexion were 86.9 ± 12.2, 16.0 ± 6.3, 1.8 ± 6.6 and 33.4 ± 9.0 deg respectively. There was no significant difference between peak torque angles for the two eccentric velocities in each joint.

### 4.3.3 Concentric and eccentric relationship with sprint performance

#### 4.3.3.1 Strength and sprint time relationships using linear regression

A number of significant relationships were found between the sprint times and muscle strength, for all subjects (Table 4.5). The strongest relationships

were between peak torque concentric knee extension and flexion and 30-35 m time. These correlations became stronger as the testing velocity increased. Correlations were higher between relative, rather than absolute, peak torque and 0-15 m times, whereas the correlation between 30-35 m time and peak torque was higher for absolute values.

**TABLE 4.5.** *Peak torque and peak torque in relation to body mass relationships with 0-15 m sprint and top speed, for the knee tests (n=24).*

| | | 0-15 metre time | | 30-35 metre time | |
|---|---|---|---|---|---|
| | | Peak Torque (N.m) | Peak Torque (N.m. $kg^{-1}$) | Peak Torque (N.m) | Peak Torque (N.m. $kg^{-1}$) |
| Knee | Concentric, extension, 60 $deg.s^{-1}$ | | | -0.490* | |
| | Concentric, extension, 150 $deg.s^{-1}$ | -0.421* | -0.444* | -0.644** | -0.579** |
| | Concentric, extension, 240 $deg.s^{-1}$ | -0.518** | -0.581* | -0.688** | -0.659** |
| | Concentric, flexion, 60 $deg.s^{-1}$ | | | -0.526** | -0.464* |
| | Concentric, flexion, 150 $deg.s^{-1}$ | -0.471* | -0.526** | -0.638** | -0.625** |
| | Concentric, flexion, 240 $deg.s^{-1}$ | -0.514* | -0.562** | -0.666** | -0.644** |
| | Eccentric, extension, 60 $deg.s^{-1}$ | -0.428* | -0.408* | -0.561** | |
| | Eccentric, extension, 240 $deg.s^{-1}$ | | | -0.473* | |
| Hip | Concentric, extension, 60 $deg.s^{-1}$ | | | | |
| | Concentric, extension, 120 $deg.s^{-1}$ | | | -0.426* | |
| | Concentric, extension, 180 $deg.s^{-1}$ | | | -0.456* | |
| | Concentric, flexion, 60 $deg.s^{-1}$ | | | -0.602* | -0.569** |
| | Concentric, flexion, 120 $deg.s^{-1}$ | | | -0.488* | |
| | Concentric, flexion, 180 $deg.s^{-1}$ | | | | |
| Ankle | Concentric, Plantarflexion, 60 $deg.s^{-1}$ | | | | |
| | Concentric, Plantarflexion, 120 $deg.s^{-1}$ | -0.513* | | -0.460* | |
| | Concentric, Plantarflexion, 180 $deg.s^{-1}$ | -0.474* | | -0.450* | |

* $p<0.05$

** $p<0.01$

### 4.3.3.2. Strength and acceleration relationships using correlations

It is well known that for an individual subject, the <u>average</u> acceleration for the 0-15 metre sprint performance is given by:

$$a(m.s^{-2})=15\ m/(\text{sprint time})^2 \qquad (3)$$

When the relationship between the average acceleration and the various torque measurement were investigated, the correlations were slightly higher than those found by using sprint times. Knee extension concentric (240 deg.s$^{-1}$) was found to have the best correlation with acceleration over 0-15 m ($r=0.520$; $P<0.01$). Correlations were also calculated between acceleration and peak torques relative to body mass. These correlations were similar to those obtained using sprint times, but were slightly higher. Once again relative knee extension concentric (240 deg.s$^{-1}$) torque was found to have the highest correlation with acceleration over 0-15 m ($r=0.590$; $P<0.01$).

**4.3.3.3 Modelling the relationship between sprint time and strength using a curvilinear power function model**

Gunther (1975) proposed that force or torque should be proportional to the components of a linear dimension of body size (L), body mass (M) and time (t) as shown in equation (2). Therefore, choosing knee-to-buttocks as the linear dimension of body size, body mass and the subjects' 0-15 m sprint times as the appropriate components, the validity of Gunther's model can be assessed for the present results. Using the multiple log-linear regression model described in the methods section, all the peak torque values were examined. The five best multiple regressions equations from different joint actions in the knee and hip are shown in Table 4.6. Knee extension concentric torque (240 deg.s$^{-1}$) was found to be predicted best by limb length, body mass and sprint time ($R^2=0.771$). This regression equation estimates the body mass exponent as (0.79), knees to buttock exponent as (1.19) and the time exponent as (-1.35) for the group, similar to the theoretical equation for force, equation (2), proposed by Gunther (1975).

Taking average acceleration for each subject as proportional to $t^{-2}$ as described in equation (3), an alternative expression for acceleration can be deduced, based on Gunther's model (Equation 2)

$$a=F/M.L \qquad (4)$$

Using knee-to-buttocks, body mass and knee extension concentric (240 deg.s$^{-1}$) as the most appropriate linear dimension of body size, mass and force respectively, the ratio, F/M.L, was calculated and correlated with the average acceleration, given by Equation 3. The resulting correlation was found to be ($r=0.619$; $P<0.001$).

**TABLE 4.6.** *Regression equations to predict peak torque using Gunther's force performance models (n=24).*

| *Equation* | $R^2$ |
|---|---|
| Knee extension concentric 240 deg.s$^{-1}$ = -2.13 . Knee to buttocks $^{1.19}$ . Body mass $^{0.79}$ . Time to sprint 0-15m $^{-1.35}$. | 0.771 |
| Knee extension eccentric 240 deg.s$^{-1}$ = -1.32 . Knee to buttocks $^{0.576}$ . Body mass $^{1.19}$ . Time to sprint 0-15m $^{-0.63}$. | 0.456 |
| Knee flexion concentric 150 deg.s$^{-1}$ = -4.21 . Knee to buttocks $^{1.12}$ . Body mass $^{1.23}$ . Time to sprint 0-15m $^{-1.54}$. | 0.715 |
| Hip flexion conentric 60 deg.s$^{-1}$ = -1.20 . Knee to buttocks $^{0.70}$ . Body mass $^{1.01}$ . Time to sprint 0-15m $^{-1.00}$. | 0.653 |
| Hip extension concentic 180 deg.s$^{-1}$ = -1.95 . Knee to buttocks $^{0.10}$ . Body mass $^{0.92}$ . Time to sprint 0-15m $^{-1.13}$ | 0.359 |

## 4.4 DISCUSSION

The main finding was that peak torque in the knee extensors measured concentrically at 240 deg.s$^{-1}$ gave the best indication, amongst those limb actions and velocities tested, of sprint running performance expressed as either time or acceleration. The relationship between strength and acceleration was found to be slightly greater than that between strength and time, but the relationship between strength and sprint performance was markedly enhanced by taking body dimensions, in addition to body mass into account.

### 4.4.1 Group differences

The Sprint group was found to have significantly lower body fat than the rugby players. This appears appropriate for the type of sport i.e. for sprinters low lean body mass improves power output in relation to body mass and for the rugby players, larger body masses aid in breaking tackles and provide greater protection from physical contact. The sprinters were significantly faster than the Active group but not the rugby players. This is not surprising considering rugby is an intermittent sport based on explosive short periods of play (Maclean, 1992) and all of the rugby players in the present study were backs who need good sprinting ability over these tested distances to achieve success (Hazeldine and McNab, 1991). The results of the *Top Speed Percentages* showed the rugby and active groups accelerate quicker to their 30-35 m velocity than the sprinters but they do not have as high a top speed. This would suggest Rugby and Active groups have a better acceleration capability in relation to top speed in comparison to sprinters. The findings from the 1987 World Championships showed 100m sprinters achieved their top velocity at 60.5 $\pm$ 11.5 metres (International Athletic Foundation, 1987). This may reflect that 30-35 m distance is too short for the sprinters group to achieve their maximal velocities.

The sprinters have significantly greater torque, adjusted to body mass, than the rugby and active groups in concentric and eccentric knee extensors. The knee extensor torques appear to be the most significant difference between sprinters and the other athletes which adds weight to earlier statements that the ability to generate high levels of peak torque during leg extension is among the factors that distingish those who excel in sprints activities from

those in other events (Thorstensson et al., 1977). Furthermore, in other studies where sprinters and endurance athletes have been compared, the sprinters produced greater torques in the knee joint actions than endurance athletes at fast velocities (Campbell, 1979; Kuhn et al., 1991; Taylor et al., 1991; Thorland et al., 1987) and when corrected for body mass (Thorstensson et al., 1977; Thorland et al., 1990). The production of greater peak torques at high velocities is influenced by the proportion of fast twitch fibres. Numerous studies have found a significant correlation between isokinetic torque in the knee joint at fast velocities and the proportion of fast twitch fibres in the muscle (Ivy et al., 1981; Tesch et al., 1978; Tesch and Karlsson, 1978; Thorstensson et al., 1976; Thorstensson et al, 1977). In addition, athletes who require high muscle strength and fast powerful explosive actions do have a greater proportion of fast twitch fibres in the leg muscles, in contrast to endurance athletes (Bergh et al., 1978; Costill et al., 1976; Thorstensson et al., 1976; Thorstensson et al., 1977; Tihanyi et al., 1982). Therefore subject's who possess predominantly fast twitch fibres are able to achieve higher force and power output at relatively higher angular velocities than those who have a predominance of slow twitch fibres. These findings suggest that the sprint group may be able to produce greater peak torques in relation to their body mass due to a greater proportion of fast twitch fibres.

There were no significance differences between the three groups for absolute peak torque, which suggests superior sprinting among sprint related athletes is more closely related to strength per unit of body mass. This suggestion is supported by the findings of Thorland et al. (1990) who observed differences between young sprinters and middle distance runners only when torques were adjusted to fat free mass. No significant differences in peak torque were found between the groups for knee flexion and all the hip and ankle actions.

### 4.4.2. Correlations between concentric and eccentric muscle strength and sprint performance

A statistically significant relationship was found, using all subjects, between concentric and eccentric knee torques and 0-15 m and 30-35 m sprint times in the present study. The relationships became greater for sprint times and concentric torques as the testing velocity increased. Adult athletes reach stride cadences exceeding 5 strides per second or leg velocities up to 500 $deg.s^{-1}$ (Berg et al., 1986; Mann and Herman, 1985). However isokinetic testing provides the closest match to angular limb velocities. These higher

correlations are expected as the velocities are closer to the actual limb angular velocities achieved in sprinting.

All the concentric and eccentric knee tests showed significant correlations with 30-35 m and 0-15 m times. Alexander (1989) also found a strong correlation between sprint performance, 100 m personal best sprint time, and concentric knee extension torque, at 230 deg.s$^{-1}$, among elite sprinters ($r=-0.710$ $p<0.01$). This is close to the relationship found in the present study with 30-35 m time and concentric extension at 240 deg.s$^{-1}$ ($r=-0.688$ $p<0.01$). Thus it appears both concentric and eccentric knee extension are important for sprinting. It has previously been shown that eccentric and concentric muscle actions for the knee extensors are important in sprinting, especially during heel-strike when vertical ground reaction forces in excess of three times body mass are exerted (eccentric) and later on in the ground phase by generating positive vertical forces (concentric) (Mann, 1981).

Correlations were also found for the concentric knee flexors and ankle plantarflexion with 30-35 m and 0-15 m times. This study is the first to report significant relationships between knee flexion and ankle plantarflexion and sprint times. However Adams et al. (1991) discovered that after strength training in different joint actions, only the toe flexors training group improved 5-20 and 20-40 yards sprint times. The relationships in the present study between knee and ankle flexors and sprint times suggest that concentric strength in these actions are important components for superior sprint performances. Knee flexors act primarily during the ground phase of sprinting, generating slight knee flexion at heel strike and ankle plantarflexion generates positive vertical and horizontal velocity during the ground phase immediately prior to the heel lifting off to toe-off phase (Elliott and Blanksby, 1979; Mann 1981; Williams, 1985).

Concentric hip extension and flexion were only significantly related to 30-35 m sprint times. Only one study has previously reported a significant relationship between sprint times (over distances of 36.6 m) and isokinetic concentric hip flexion and extension at 60 deg.s$^{-1}$ ($r=-0.57$) and at 60 deg.s$^{-1}$ ($r=-0.56$) and 240 deg.s$^{-1}$ ($r=-0.41$) respectively (Guskiewicz et al., 1993). However hip measurements were performed in a functionally standing position whereas in the present study the measurements were performed in a supine position which was perceived to be easier to standardise.

Investigators have noted the importance of hip flexion during the recovery phase, which is an indication of knee lifting, and the hip extensors during the extension of the leg into the ground phase (Mann and Sprague, 1983; Simonsen et al., 1985). No correlations for the hip extensors or flexors were found with 0-15 m sprint times. This may indicate that the hip muscle actions assist performances more as the sprinting distance is increased. Mann et al. (1986) concluded from biomechanical analysis that the main muscle group that appears to increase the speed of gait is the hip flexors.

This study did not measure eccentric knee flexion or ankle dorsiflexion, but Wood (1988) states the ability to transverse the ground as quickly as possible is predomiantly due to eccentric muscle activity in the knee flexors and extensors, with the knee flexors acting powerfully to control leg momentum and prepare the foot-strike. Further studies are required to measure the relationship between eccentric joint flexion and sprint times.

**4.4.3. Relationships between sprint performance and muscle strength using curvilinear power function models**

Using linear regression to examine the relationship between torque or torque relative to body mass, and sprint performance expressed as time, may not be the best way of examining the relationship between strength and sprint running. Theoretically one would expect a better relationship if performance was expressed as acceleration which is directly proportional to force. Thus, performance was recalculated as average acceleration. This was only possible for the 0-15 m trials. The correlation between average acceleration and peak torque during concentric knee extension at 240 $deg.s^{-1}$ was r=0.520 compared with r=-0.518 for time. A similar very small improvement in the correlation was observed when torques were expressed relative to body mass (r=-0.581 for time and r=0.590 for acceleration). Thus, expressing performance as acceleration rather than time had only a small effect on the resulting relationship between performance and strength.

Of more influence was the addition of a body dimension to the analysis of the relationship between strength and performance. Gunther's (1975) force performance model, illustrated in equations (1) and (2), proposes that performance is better related when body dimensions are taken into account. To validate this theoretical approach the log-linear regression model was used to predict the peak torque results using, knee to buttocks, body mass

and 0-15 m time as limb length, body mass and time predictors respectively. The peak torque for knee extension concentric at 240 deg.s$^{-1}$ was found to be the best predictor for equation (2) ($R^2$=0.771). The exponents for each variable for this regression equation, as shown in Table 4.6., are similar to those in the descriptive analysis model proposed by Gunther (1975), equation (2). The model is theoretical and situated in the perfect world and therefore, slight variations are expected. However these findings support Gunther's model and Astrand and Rodahl's theory that acceleration, force and power can be expressed as derivatives from length, mass and time and highlight the importance of taking body dimensions into account when examining the relationship between strength and sprint performance.

Equation (2) can be rearranged as shown in equation (4) to predict the group's acceleration. The relationship between acceleration predicted in this manner from knee to buttocks length, body mass and knee extension torque at 240 deg.s$^{-1}$ and the measured acceleration was r=0.619, in comparison with r=0.581 for the relationship between measured acceleration and torque alone. These findings lend further support to the suggestion that the relationship between strength and sprint performance over 0-15 m is best examined by taking body dimensions into account. As it was not possible to use the 30-35 m times in association with Gunther's model because of the lack of timing from zero velocity, it remains to be seen whether or not the relationship between top speed and strength, as opposed to acceleration over the first few metres, is enhanced by taking body dimensions into account.

**4.4.4 Isokinetic differences between concentric and eccentric muscle actions**

The results from one isokinetic dynamometer system cannot be directly compared to results on another model. This is related to the system's computer, which employs its own algorithmic calculations which are not published nor standardised from manufacturer to manufacturer (Amundsen, 1990). Also comparisons are difficult with other studies because of the lack of consistency in testing velocities, subject testing protocols and use of testing modes, such as, gravity correction. Therefore there has been a limited attempt to compare the isokinetic results in this study with others. However the trends and relationships can be contrasted with previous research.

#### 4.4.4.1 The relationship between peak torque and testing velocity

Concentric isokinetic torques decreased as the testing velocity increased for all subjects. Eccentric torques, however, were unaffected by testing velocity. These relationships between torque or force and concentric and eccentric actions in muscle fibres are universally accepted (Gülch 1994). Hill (1938) found an inverse hyperbolic relationship between velocity and force in an isolated animal muscle, i.e. as the velocity increases the force decreases for concentric muscle shortening. Whereas for eccentric actions there is an initial increase in tension at slow velocities until a plateau is reached (Flitney and Hirst, 1978; Gülch, 1994; Lakomy, 1994). These differences in maximum force and in velocity curves appear to be related to the elastic properties within the muscle and differences in the binding and interaction of the actin and myosin filaments (Section 2.6.1.).

#### 4.4.4.2. Peak torque angles

Peak torque angles showed a significant decrease for concentric knee extension as the testing velocity increased. This supports previous studies which have also found a negative function between concentric knee extension's peak torque angle and angular velocity (Kannus, 1994; Thorstensson et al., 1976; Taylor et al., 1991). Thus by increasing testing velocity the peak torque will occur later in the range of movement for knee extension. Eccentric knee extension illustrated a similar trend to concentric extension between slow and fast velocities. The peak torque angles at 60 $deg.s^{-1}$ for concentric and eccentric knee extension were found to be very similar, 64.1 $\pm$ 8.3 and 64.3 $\pm$ 6.4 degrees respectively. There were no significant differences found between concentric and eccentric actions for hip extension or ankle plantarflexion. Therefore, it appears that the torque curves may have different magnitudes between concentric and eccentric maximal actions but are similar in shape.

#### 4.4.4.3. Unilateral muscle ratios

The unilateral peak torque muscle ratio between flexors and extensors could only be calculated from concentric actions due to the subjects not performing eccentric flexion. The knee ratio showed a positive function with angular velocity and this supports numerous investigations (Imwold et al., 1983; Morris et. al, 1983; Kannus, 1994; Read and Bellamy, 1990; Amundsen, 1990; Alexander, 1990). No significant changes were found for the hip and ankle actions as testing velocity changed. Previous studies have reported that the

hip ratios decrease and ankle ratios increase as velocity increases (Alexander, 1990; Fugl-Myer, 1981).

Alexander (1989) examined both eccentric extensors and flexors for the knee, hip and ankle joints and found the flexor/extensor ratios for both concentric and eccentric actions to be similar. The knee ratios for concentric and eccentric actions at 60 deg.s$^{-1}$ were calculated at 0.64 and 0.63 respectively. Therefore based on these findings it appears that the ratio between the extensors and flexors in the lower limb joints are similar for both concentric and eccentric actions.

**4.4.5 Summary**

The relationships found between strength of the leg muscle groups and sprinting times and acceleration suggest that strength is a major contributor to sprint performance. The strength of the knee extensors at fast velocities appears to have the most influential role as 77 % of the variation in Gunther's theoretical model could be explained by the peak torque generated during concentric knee extension at 240 deg.s$^{-1}$. The closeness of the fit of the empirical data to the theoretical model and the small improvements in the relationship when acceleration rather than time was derived suggest that the relationship between sprinting performance over short distances and strength is improved (i) slightly, by expressing performance as acceleration rather than time and (ii) for 0-15 m, to a greater extent by taking an appropriate linear body dimension, as well as mass, into account.

# *CHAPTER 5. TOTAL WORK AND BLOOD LACTATE RESPONSES TO MAXIMAL CONCENTRIC AND ECCENTRIC BOUTS OF EXERCISE*

## 5.1 INTRODUCTION

There are considerable differences between concentric and eccentric muscle actions, not only in the production of force but also in the metabolic cost, actin-myosin conformational changes and metabolic responses to voluntary and stimulated activation. These differences have been reviewed in Section 2.6.

It is universally accepted that eccentric actions produce greater force or torque in intact human muscles during maximal voluntary conditions and electrically evoked stimulation. There are distinct differences in the metabolic responses to concentric and eccentric actions when the external work is the same. The energy expenditure during concentric actions, such as raising a weight, is greater than the amount during eccentric actions, such as lowering a weight, when both tasks are producing equal submaximal work output (Abbott et al., 1952; Asmussen, 1953). It has been found that during eccentric muscle actions less activation and recruitment is required to generate equal work in comparison with concentric actions (Bigland and Lippold, 1954). This knowledge helps to explain why less metabolic activity is necessary to perform the same tasks (Abbott et al., 1951; Abbott et al., 1952; Asmussen, 1953; Bigland-Ritchie and Woods, 1976; Bonde-Petersen et al., 1972; Kaneko et al., 1984; Knuttgen and Klausen, 1971; Knuttgen et al., 1982; Plante and Houston, 1984). Therefore raising and lowering a resistance during weight training has a different metabolic efficiency and requires different neural strategies for each movement. The findings of these investigators are described in Section 2.6.4. During maximal voluntary muscle activation there also appears to be differences between concentric and eccentric actions. The energy expenditure following 7 s of maximal voluntary concentric actions has been found to be significantly higher compared with eccentric actions (Seliger et al., 1980). However there is little information concerning the metabolic responses to repeated isokinetic eccentric muscle actions during maximal voluntary effort in intact human muscle. Therefore, the metabolic responses to repeated eccentric and concentric maximal voluntary muscle actions have

not been compared thoroughly. Furthermore, the current understanding of the conformational changes and energy utilisation during eccentric actions are still not fully understood.

The mechanisms that may cause fatigue during high intensity concentric actions are described in Section 2.5, but it cannot be assumed that the magnitude of fatigue and the mechanisms involved are the same in concentric and eccentric actions. In addition the extent of fatigue may be dependent on the testing velocity (Gray and Chandler, 1989; Komi and Viitasalo, 1977).

The purpose of this study was to investigate the metabolic responses and fatigue profile during concentric and eccentric actions by measuring the responses to maximal isokinetic exercise bouts in intact human muscles. These findings may help in explaining or confirming some of the theories associated with eccentric muscle actions.

## 5.2 METHODS

### 5.2.1 Subjects

Eight competitive sportsmen (two from soccer, one squash player, one rugby forward, one tennis player, one swimmer, one endurance athlete and one gymnast) participated as subjects in this experiment which had University Ethical Committee approval. Test procedures were explained to the subjects prior to them giving their informed consent to participate. Each subject completed a medical questionnaire to ensure they were free from musculo-skeletal injuries and that none were on medications or hormonal therapy. All subjects were male.

### 5.2.2 Experimental procedures and protocol

#### 5.2.2.1 Anthropometric Measurement

Standard measurements of height and body mass were taken for each subject (Weiner and Lourie, 1981). Skinfold thicknesses were taken at four sites on the body and used in conjunction with regression equations derived by Durnin and Womersley (1974) to estimate percentage body fat. All measurements are described in Chapter 3.

#### 5.2.2.2 Repeated knee extension and flexion protocol

The repeated knee extension and flexion test was performed on a Cybex 6000 Isokinetic Dynamometer. Each subject had two familiarisation sessions which included five sets of 10 repetitions either performing concentric or eccentric extension and flexion actions at 120 deg.$s^{-1}$. Each familiarisation session was separated by 2 to 3 days and at least a week before their first testing session.

All subjects performed the repeated knee extension and flexion test on two occasions, one performing eccentric actions for both movements and another performing concentric actions. The protocol design of the repeated knee extension and flexion tests are illustrated in Fig 5.1 and described further in Chapter 3. The two tests were performed at the same time of day and approximately a week apart. The order of tests was randomised between subjects.

Each subject was measured on an adjustable chair and stabilised with velcro straps. The axis of rotation of the Cybex lever arm was aligned with the

anatomical axis of the knee joint, as described in Chapter 3. Both the Ramping and Gravity Correction features were used in all tests to avoid previously found problems such as torque overshoot and gravity affects respectively (Sapega et al., 1982; Sapega, 1990; Winters et al., 1981).

The repetition for each set which exhibited the greatest peak torque and the total work from the ten sets were measured. Each subject was measured throughout their full range of movement which remained constant for both tests.

#### 5.2.2.3 Fatigue index

The difference between the highest and the last total work were taken from the ten sets and used to calculate fatigue as described in the formula in Section 3.4.6.3.

#### 5.2.2.4 Muscle soreness questionnaire

Each subject completed a questionnaire 48 hours after the completion of each test. The questionnaire asked for the perceived muscle soreness by indicating a scale between 1 (no soreness) to 10 (very very sore) in the following muscle groups, hamstring, quadriceps, inside thigh, calf and back, for the exercised and non-exercised leg (Appendix A).

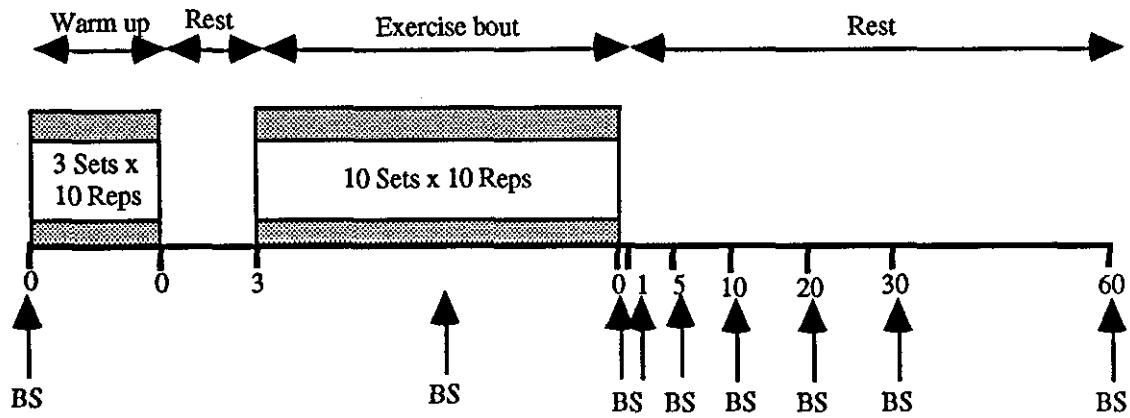
### 5.2.3 Blood sampling and analysis

Blood samples were taken, as described in Chapter 3., before the exercise test with the subjects in a seated position. Further samples were taken after the fifth set during the test and at 1, 5, 10, 20, 30, 45 and 60 mins after the completion of the test. Following collection of blood the analysis of blood lactate, haematocrit, haemoglobin and percentage change in plasma volume was made as described in Chapter 3.

### 5.2.4 Statistical analysis

A two way analysis of variance (ANOVA) with repeated measured on both factors was used to compare total work, fatigue, the blood lactate response, the changes in plasma volume and the rating of muscle soreness during the exercise tests involving eccentric and concentric muscle actions. Where significant F ratios were found, the means were compared using a Scheffe F-post-hoc test.

Results are presented as mean ± standard deviation.




BS - Venous blood sample

**FIGURE 5.1** *Schematic representation of the repeated knee extension and flexion protocol and blood sampling*

## 5.3 RESULTS

### 5.3.1 Total work output

Total work for the ten sets for the knee extensors and flexors are presented in Table 5.1. Total work produced for the extensors and flexors were significantly greater for each set in eccentric exercise as opposed to concentric ($p<0.01$). For each muscle action the highest mean total work was produced in the first set. The highest total work measured, 2369.0 $\pm$ 416.0 J was produced by eccentric extensors.

**TABLE 5.1** *Mean total work (J) for each set from the concentric and eccentric exercise tests (mean $\pm$ S.D., n=8)*

| | Flexors | | Extensors | |
|---|---|---|---|---|
| *Set Number* | **Concentric** | **Eccentric** | **Concentric** | **Eccentric** |
| *1* | 1381.6** $\pm$ 313.6 | 1682.6** $\pm$ 259.6 | 1771.5* $\pm$ 291.8 | 2369.0* $\pm$ 416.0 |
| *2* | 1354.9** $\pm$ 284.2 | 1567.6** $\pm$ 305.3 | 1744.4* $\pm$ 287.8 | 2313.6* $\pm$ 349.9 |
| *3* | 1377.6** $\pm$ 285.8 | 1605.5** $\pm$ 335.9 | 1748.6* $\pm$ 304.9 | 2333.8* $\pm$ 468.7 |
| *4* | 1355.5** $\pm$ 267.4 | 1584.0** $\pm$ 355.9 | 1726.6* $\pm$ 268.0 | 2355.0* $\pm$ 437.1 |
| *5* | 1353.6** $\pm$ 258.3 | 1473.6** $\pm$ 302.8 | 1690.5* $\pm$ 259.0 | 2303.3* $\pm$ 402.6 |
| *6* | 1316.9** $\pm$ 261.9 | 1414.4** $\pm$ 318.8 | 1648.9* $\pm$ 308.8 | 2192.5* $\pm$ 512.9 |
| *7* | 1299.0** $\pm$ 272.8 | 1440.9** $\pm$ 307.3 | 1623.9* $\pm$ 352.1 | 2213.9* $\pm$ 517.8 |
| *8* | 1295.4** $\pm$ 273.6 | 1470.0** $\pm$ 388.6 | 1584.6* $\pm$ 323.5 | 2191.9* $\pm$ 524.5 |
| *9* | 1282.8** $\pm$ 257.0 | 1395.0** $\pm$ 298.7 | 1558.4* $\pm$ 286.3 | 2183.8* $\pm$ 505.0 |
| *10* | 1291.6* $\pm$ 275.7 | 1438.8* $\pm$ 269.3 | 1567.3* $\pm$ 316.6 | 2297.4* $\pm$ 431.9 |

* Significant difference between concentric and eccentric exercise tests $p<0.05$

* * Significant difference between concentric and eccentric exercise tests $p<0.01$

The fatigue indexes for total work are reported in Table 5.2. For the knee flexors the fatigue index was higher during eccentric exercise, 17.2 $\pm$ 9.4 %, compared to concentric exercise, 9.8 $\pm$ 7.0 % ($p<0.05$). No significant difference was found between eccentric and concentric fatigue in the knee extensors.

**TABLE 5.2.** *Fatigue Index (%) measured from the difference between the mean total work from the highest and last sets for knee extension and flexion (mean ± S.D.)*

| | Flexors | Extensors |
|---|---|---|
| Concentric | 9.8* ± 7.0 | 13.5 ± 10.6 |
| Eccentric | 17.2*# ± 9.4 | 7.4 #± 6.7 |

* Significant difference between concentric and eccentric exercise tests $p<0.05$

\# Significant difference between knee extensors and flexors $p<0.05$

### 5.3.2 Blood lactate

Blood lactate concentrations are illustrated in Figure 5.2. Blood lactate concentration was higher after concentric than eccentric exercise at 1, 5, 10, 20 and 30 min post-exercise ($p<0.01$ and $p<0.05$). Mean peak values were found for both conditions 5 min post-exercise, measured at 5.23 ± 2.17 $mmol^{l-1}$ and 3.04 ± 2.45 $mmol^{l-1}$ respectively.

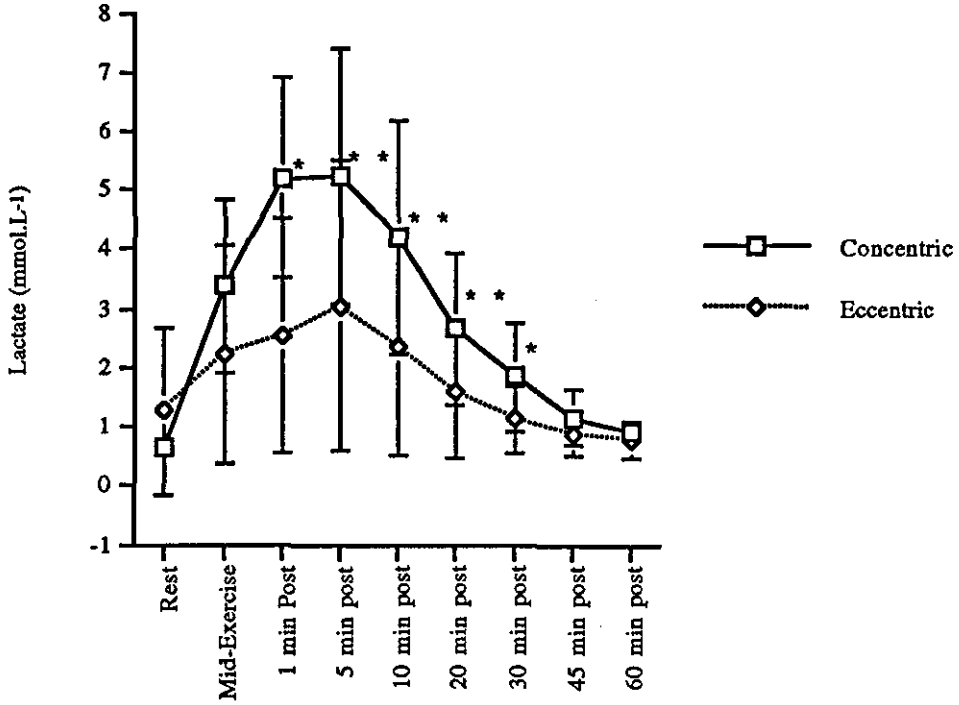



**FIGURE 5.2** *Blood lactate concentrations (mean ± S.D., n=8) for the concentric and eccentric tests. (main effect condition, P<0.01; main effect time, P<0.01; condition-time interaction, P<0.05). * P<0.05 difference between concentric and eccentric tests, ** P<0.01 difference between concentric and eccentric tests.*

### 5.3.3 Changes in plasma volume

The estimated changes in plasma volume are presented in Table 5.3. The decrease in plasma volume during and immediately post-exercise was corrected more quickly following eccentric than concentric exercise as reflected by differences between the two conditions at 5, 10 and 20 min after the repeated knee extension and flexion tests.

**TABLE 5.3.** *Estimated changes in plasma volume (%) for the concentric and eccentric tests (mean + S.D., n=8)*

| | Concentric | Eccentric |
|---|---|---|
| *Mid-exercise* | -6.03 ± 3.49 | -6.06 ± 4.69 |
| *1 min post-exercise* | -8.94 ± 4.82 | -4.91 ± 5.48 |
| *5 min post-exercise* | -5.65** ± 3.31 | -0.77** ± 4.52 |
| *10 min post-exercise* | -2.03** ± 3.65 | 0.95** ± 5.58 |
| *20 min post-exercise* | 0.01** ± 4.22 | 2.81** ± 5.65 |
| *30 min post-exercise* | 2.11 ± 3.42 | 2.80 ± 5.34 |
| *45 min post-exercise* | 3.22 ± 2.82 | 2.93 ± 5.14 |
| *60 min post-exercise* | 2.67 ± 2.68 | 3.05 ± 5.60 |

** Significant difference between the two tests $p<0.01$

### 5.3.4 Muscle soreness

Eight subjects completed the muscle soreness questionnaire. The results are shown in Table 5.4. The perceived muscle soreness ratings 48 hours post exercise for the exercised leg were higher for eccentric in comparison with concentric exercise for the hamstring, quadriceps and inside thigh ($p<0.01$). Muscle soreness in the hamstring and inside thigh of the exercised leg was greater than that for the quadriceps ($p<0.01$ and $p<0.05$ respectively). There was no difference in muscle soreness between the two conditions in the non-exercised leg muscle groups or in the exercised calf or back.

**TABLE 5.4** *Muscle Soreness Questionnaire (mean + S.D., n=8), 48 hour post exercise. Scale between 1-10, 1 being no soreness to 10 being very very sore).*

| | Concentric | Eccentric |
|---|---|---|
| Exercised Hamstring | 1.6* ± 0.7 | 7.4* ± 2.1 |
| Non-Exercised Hamstring | 1.3 ± 0.7 | 1.4 ± 1.1 |
| Exercised Quadriceps | 1.5* ± 0.5 | 4.0* ± 2.0 |
| Non-Exercised Quadriceps | 1.5 ± 1.4 | 1.5 ± 0.9 |
| Exercised Inside Thigh | 1.4* ± 0.5 | 6.6* ± 1.6 |
| Non-Exercised Inside Thigh | 1.1 ± 0.4 | 1.0 ± 0.0 |
| Exercised Calf | 2.1 ± 1.7 | 1.6 ± 1.1 |
| Non-Exercised Calf | 1.0 ± 0.0 | 1.0 ± 0.0 |
| Back | 1.9 ± 1.4 | 2.1 ± 1.6 |

* Significant difference $p<0.01$ between ratings for concentric and eccentric actions

## 5.4 DISCUSSION

The main finding of the present study was that blood lactate concentration was lower for the eccentric exercise bout compared with the concentric exercise bout, even though average work was greater for eccentric exercise. These findings suggest that less metabolic activity is required for an activated muscle to lengthen than to shorten during maximal voluntary actions.

### 5.4.1 Total work and blood lactate concentrations

The mean total work production for each set was 13% and 37% greater for the flexors and extensors respectively during eccentric in comparison with concentric actions. Under maximal conditions it is accepted that eccentric actions produce significantly greater force and work than concentric actions (Gülch, 1994). An activated isolated muscle following lengthening has been found to be 'stiffer' by 10-20% (higher change in tension per unit change in length) than when it shortens (Bressler et al., 1988; Lombardi and Piazzesi, 1990). Therefore, when a stretched muscle is activated the external force causes stiffness (cross-bridge elasticity) which is able to support higher external loads. This force or load increases until a point where the force remains at steady state throughout the faster velocities until it becomes too fast when failure to maintain tension occurs (Gülch, 1994; Lakomy, 1994). Therefore, force produced during eccentric actions in intact human muscle is independent of velocity and is greater than force recorded during isometric or concentric actions until the velocity exceeds the binding rates of the cross-bridges.

Peak blood lactate concentrations occurred at 5 min post-exercise for both concentric and eccentric muscle actions. The average peak concentration for concentric exercise was $5.23 \pm 2.17$ $mmol \cdot l^{-1}$ compared with $3.04 \pm 2.45$ $mmol \cdot l^{-1}$ for eccentric exercise. These findings suggest that during maximal voluntary repeated knee extension and flexion exercise, the energy expenditure from glycolysis is greater for concentric exercise than eccentric exercise. The differences in energy consumption can be partly explained by the differences in muscle activation that take place during the two muscle actions. Various authors have found that a maximal voluntary effort during an eccentric action activates the muscle 10-30% less than a concentric action (Eloranta and Komi, 1980; Seliger et al., 1980; Tesch et al., 1990; Westing et al.,

1991). During eccentric muscle actions motor control patterns are poorly understood. By electrically stimulating knee extensor muscles that were already undergoing voluntary activation it has been reported a 21-24 % increase in eccentric torque output was produced on average over maximal voluntary levels (Westing et al., 1990). Thus, during eccentric actions some inhibitory mechanism prevents the development of full muscle activation, probably preventing excessive forces which might tear the muscle apart. The inhibitory feedback could occur from joint receptors, free nerve endings in the muscle, cutaneous receptors, pain receptors and/or golgi tendon organs (GTO) (Carew, 1981). A unique control mechanism has been suggested for eccentric actions caused by the increased mechanoreceptor input which is associated with muscle tension levels three- to five fold greater than during concentric actions (Knuttgen and Klausen, 1971). The mechanoreceptor activity was postulated to be from GTO. The main function of the GTO's are to respond to tension within the tendon, where they are located. The sensory neuron of a GTO travels to the spinal cord where it synapses with the alpha motor neurons of that muscle and the antagonistic muscles. If the tension increases to a point where damage to the muscle or tendon is possible, this inhibitory mechanism operates by decreasing activation and/or altering the muscular recruitment patterns in the agonist muscle and increases the activation of the antagonist muscle. This protective mechanism prevents an exponential increase in force during eccentric actions as velocity increases and a plateau in force output occurs, thus preventing damage of the muscles, connective tissue and/or joints.

Therefore, lower blood lactate concentrations can be partly explained by the reduced amount of muscle activation during eccentric actions by neural inhibition. However previous studies have examined energy expenditure differences in tetanized muscle lengthening (eccentric) and shortening (concentric) for isolated muscle and found less energy is still consumed during muscle lengthening (Curtin and Davies, 1975). Since a muscle is fully activated during tetanized conditions it can be assumed that for both shortening and lengthening actions there is equal activation.

A further explanation is required to fully understand why energy consumption is different for concentric and eccentric muscle actions. To do this it is necessary to examine the energy utilising systems and the 'cross-bridge theory' for concentric and eccentric actions. Energy is consumed for

each cross-bridge cycle. According to Rall (1985) there are 3 major ATP utilising systems active during an actin-myosin coupling cycle; actomyosin ATPase, $Ca^{2+}$ transport ATPase of the sarcoplasmic recticulum and $Na^{+}$, $K^{+}$, ATPase of the sarcolemma. The latter system is insignificant in terms of contributing to energy cost during muscle activity (Smith, 1972). During a maximal muscle action about 30 % of the energy utilised is for the continual cycling of $Ca^{2+}$ from and eventually back to the sarcoplasmic reticulum at the expense of a phosphate molecule. Therefore about 70% of the energy utilised derives from actomyosin ATPase (Rall, 1985). The hydrolysis of ATP from actomyosin ATPase can proceed through the reaction sequence only if the filaments are allowed to slide relative to each other (Homsher, 1987). According to the current cross-bridge theory postulated by recent authors, eccentric actions cause the cross-bridge to be forcibly pulled back. For a cross-bridge to remain intact, when an activated muscle fibre is stretched, it extends to the utmost and breaks possibly before the completion of the cycle, thus before ATP hydrolysis can occur (Curtin and Davies, 1975; Menard et al., 1991; Rall, 1985; Stauber, 1989). This may prevent the actomyosin ATPase from proceeding through the cycle. The myosin would then remain in a high energy form and could reattach to the next available binding site of the actin, only to be forced away again if the external force was maintained. Each of these attachment-separations would generate tension by the muscle, but without the consumption of energy.

This theory explains why eccentric exercise utilises less energy than concentric, but it assumes negligible energy is consumed for eccentric actions. However, the blood lactate response in the present study indicates that energy is still consumed during eccentric exercise. The suggestion that eccentric actions do require hydrolysis of ATP is supported by experiments on both isolated and intact muscles which have shown that inorganic phosphate-ATP exchange reactions do occur during isolated lengthening muscles (Gillis and Marechal, 1971; Marechal et al., 1971). Thirty percent of energy in isometric actions is utilised by the sarcoplasmic reticulum which pumps $Ca^{2+}$ (Rall, 1985), which suggests that energy is still consumed even if eccentric actions cause a complete suppression of the actomyosin ATPase. In muscles where no overlap of the filaments has occurred a 27 % change in ATP breakdown was found in comparison to a tetanized muscle at resting length (Sandberg and Carlson, 1966). Similarly, Curtin and Davies (1975) found the

net chemical change during eccentric actions at slow velocities to be 25 % of the change in unstimulated isometric resting control.

Therefore, the energy cost of an eccentric action to the muscle cell may appear to be only the amount necessary to maintain the cell in an activated state. The energy required in lengthening the muscle derives from the external source. This would explain the blood lactate concentration differences for the eccentric exercise bout found in the present study. So although the results obtained were anticipated based on EMG literature and on the findings of Curtin and Davies (1975), no study has previously examined and compared the blood lactate responses to repeated intact human maximal isokinetic eccentric and concentric muscle actions.

### 5.4.2 Fatigue index

The fatigue index showed that the knee flexors had a significantly greater fatigue for eccentric exercise compared with concentric exercise. No difference in the two conditions was found for the knee extensors. Extensors have also shown to be more fatigue resiliant by Emery et al. (1994) who found a greater number of repetitions were performed for eccentric extensors than flexors until the subject performed under 50 % of their highest torque. This may suggest that the knee extensors are more accustomed to cope with repetitive eccentric actions compared to the flexors. During running and moreso in sprinting the eccentric knee extension, as discussed in Chapter 4, has a significant role during the ground phase as a stabiliser and acting as a braking force. Eccentric knee flexors act mainly during the air phase as a limb decelerator. Therefore the external forces which the knee extensors experience eccentrically are considerable. Landing forces have been reported to be 3-3.5 times the sprinters body weight (Mann, 1981; Williams, 1985). Thus knee extensors are more accustomed to large external forces which could possibly explain why the extensors are more fatigue resistant.

It appears from previous research that greater fatigue is only found for eccentric actions in comparison to concentric actions at slow testing velocities (Komi and Viitasalo, 1977). During fast testing velocities eccentric actions have been found to be more fatigue resistent than concentric actions (Gray and Chandler, 1989). Fatigue was calculated during eccentric exercise at an intermediate velocity (150 deg.$s^{-1}$) and a greater resistance to fatigue was found compared with concentric exercise (Emery et al., 1994), whereas no

difference was found in fatigue after 3 bouts of 30 repetitions for knee extension at 120 deg.$s^{-1}$ between concentric and eccentric actions (Bilcheck et al., 1993). Therefore it appears at different testing velocities the relationship between eccentric and concentric actions and fatigue are different. Subjects in the present study performed each muscle action at 120 deg.$s^{-1}$ (intermediate velocity).

Fatigue has been associated with the inability to regenerate ATP at the required rates (Sahlin and Ren, 1989; Soderlund et al., 1992). During repeated maximal short duration exercise the high rate of ATP generation is provided mainly by PCr breakdown and glycolysis (Gaitanos et al., 1993). In the present study ten knee extensions and flexions were performed, which lasted for 13-15 s duration depending on the subject's range of movement, on ten occasions with one min rest between sets. A recent study found that following 10 s sprint cycling, muscle PCr had decreased by 55.3 % from resting values in comparison to only an 11.5 % decrease in muscle glycogen and a 21.1 % in muscle ATP (Bogdanis et al., 1994b). The duration of maximal effort in the present study was similar and thus it can be assumed a high degradation of PCr also occurred. Phosphocreatine needs to be resynthesised to prevent a fatigue (Bogdanis et al., 1995a). The resynthesis of PCr and the restoration of peak performance has been observed to proceed in parallel during 1.5 to 6 min recovery following a 30 s sprint (Bogdanis et al., 1995a). The mean half-time of PCr resynthesis has been found to be 56.6 $\pm$ 7.3 s following passive recovery (Bogdanis et al., 1995a). Subjects in the present study had 1 min passive recovery which suggests total PCr resynthesis did not occur before the start of each set. Therefore it appears the ability to resynthesise PCr limits the maintenance of total work performed in the first set for the other nine sets. Another factor involved in fatigue may include the increase in $H^+$ caused from glycolysis. Following 10 s sprint cycling muscle lactate increased from resting levels of 4.5 $\pm$ 0.4 $mmol \cdot l^{-1}$ to 51.0 $\pm$ 4.6 $mmol \cdot l^{-1}$ (Bogdanis et al., 1994b). Also, following ten 6 s sprints with 30 s recovery muscle lactate increased from resting values to 112.3 $\pm$ 30.6 $mmol \cdot l^{-1}$ (Gaitanos et al., 1993). The increase in lactate observed in these studies, which had a similar exercise time to that in the present study, confirms the strong contribution of anaerobic gylcolysis during repeated short term high intensity exercise. The increase in $H^+$ has been shown to inhibit the release of $Ca^{2+}$ from the sarcoplasmic reticulum (Nakamaru and Schwartz, 1972- cited Edwards, 1983), inhibit the activity of the enzymes PFK and phosphorylase

(Gaitanos et al., 1993) and to compete with $Ca^{2+}$ for the binding sites on the troponin molecule (Bolitho and Donaldson et al., 1978- cited Edwards 1983). However studies examining the muscle metabolic responses following short term high intensity exercise have limited their exercise to concentric actions only. No studies, to this authors knowledge, have examined the muscle ATP, PCr and lactate following repeated high intensity eccentric actions.

It has been postulated that the energy cost for eccentric actions at intermediate velocities is only the amount to mantain the cell in an activated state. If this theory is correct it would appear fatigue caused by the lack of available energy and increased $H^+$ would be lower than during concentric exercise. However even though eccentric exercise required lower energy utilisation than concentric exercise, fatigue was greater during eccentric knee flexion compared with concentric knee flexion. In agreement to these findings another study examined eccentric actions performed on a cycle ergometer to exhaustion and found greater fatigue for eccentric actions even though elevations in blood lactate concentration and heart rate were not as high as compared with concentric actions (Klausen and Knuttgen, 1971). Greater fatigue has been reported in maximal voluntary force one hour after eccentric actions compared with one hour after concentric actions and it was postulated that fatigue was related to the mechanical damage to the sarcoplasmic reticulum which resulted in altered $Ca^{2+}$ release and corresponding nerve electrical activity (Newham et al., 1983). Following intense exercise a reduction in the surface EMG signal has been found which is reflected in a decrease in performance (Kroon and Naeije, 1991). The recovery of the full EMG signal for eccentric actions is delayed (up to 7 days) compared with concentric actions (1-2 days) following exercise to exhaustion (Kroon and Naeije, 1991). The delay in EMG signals restoring to reference activity levels were attributed to muscle damage affecting the electrical excitability of the membrane.

Therefore, if the energy cost theory is correct for eccentric actions it appears fatigue could be affected by muscle damage. However, it still remains unclear the aetiology of fatigue during eccentric exercise and more research is required, including an examination of fatigue during actions at different angular velocities.

### 5.4.3 Muscle soreness

The results from the questionnaire demonstrated a significant difference in muscle soreness during the eccentric exercise bout for the exercised hamstrings, quadriceps and inside thigh. No significant soreness was felt after the concentric exercise and no differences were found in the back, exercised calf and non-exercised muscle groups for both exercise bouts. These findings are consistent with the results from previous studies. For some time it has been recognised that delayed onset of muscle soreness occurs 1-2 days after the completion of exercise (Talag, 1973). This sensation is very common and has been found to follow exercise involving deceleration or eccentric muscle actions (Abraham 1977; Schwane et al., 1983). Later studies have found that after unaccustomed eccentric exercise, delayed onset muscle soreness, muscle damage and inflammation are all experienced (Clarkson et al., 1986; Costill et al., 1990; Friden et al., 1983; O'Reilly et al., 1987). Remmers and Kaljot (1963) first linked exercise and muscle damage together after detecting cellular damage by measuring intracellular proteins leaking out due to exercise. Delayed damage in the muscle fibre structure or function is the best established injury, related to eccentric exercise (Jones et al. 1986). Damage can be seen from up to 3 days to two weeks post-exercise, indicating that the contractile unit is degraded for a considerable amount of time (Stauber, 1989). Evidence also suggests there is damage to the connective tissue, endomysium, following eccentric actions (Abraham, 1977). The muscle damage and soreness that occurs is also accompanied by an inflammation response (Armstrong et al., 1983; Evans and Cannon, 1991).

### 5.4.4. Summary

In summary, eccentric actions produce high force and work with low energy expenditure. Thus eccentric actions have a higher mechanical efficiency than concentric actions. The greater amount of force produced maximally during eccentric actions can be attributed to the effects of internal resistance of the muscle, which causes stiffness that is capable of supporting higher external loads. Lower energy expenditure for eccentric exercise has been explained by two theories: (i) lower muscle activation takes place during maximal eccentric actions than concentric, caused by a neural inhibition mechanism, probably GTO, which would account for less cross-bridge cycles per unit of time; (ii) the current eccentric cross-bridge theory regarding the breakage of the cross-bridge before the completion of the cycle which prevents energy transduction. Therefore it has been postulated and supported by the present

study finding's that the energy cost during an eccentric action/exercise, at intermediate angular velocities, appears to be only the amount necessary to maintain the cell in an activated state. Fatigue has been observed to be greater for knee flexors following eccentric actions compared with concentric actions. A greater fatigue in eccentric actions questions the validity of the energy cost theory for eccentric actions unless the processes involved in fatigue are different to those occurring during concentric actions. Muscle damage has been suggested as one unique factor involved in eccentric fatigue. Further research is required to understand the fatigue processes involved in eccentric actions at different testing velocities.

# *CHAPTER 6. THE EFFECT OF ORAL CREATINE SUPPLEMENTATION ON THE METABOLIC AND MUSCLE WORK RESPONSES FROM VOLUNTARY ECCENTRIC AND CONCENTRIC EXERCISE.*

## 6.1 INTRODUCTION

During repeated muscle actions the skeletal store of adenosine triphosphate (ATP) is rapidily utilised. Therefore for action to continue, ATP must be resynthesised. This is achieved from the breakdown of muscle phosphocreatine (PCr) and glycogen and through the adenylate kinase reaction in which 2 adenosine diphosphates (ADP) combine to form ATP and adenosine monophosphate (AMP) (Hultman et al., 1990). Degradation of PCr in type II fibres is very rapid during the initial seconds of maximal muscle action (Soderlund et al., 1992; Hultman et al., 1991). The depletion of PCr is one of the limiting factors for maintaining the high rates of energy turnover and is commonly associated with the onset of fatigue by reducing the rate of ADP rephosphorylation to ATP (Hultman et al., 1967). Therefore by increasing intramuscular creatine stores it might prolong PCr depletion and therefore slow the rate of fatigue. Recent studies have shown that dietary creatine intake influences total body creatine (Crim et al., 1976). Oral creatine supplementation has been shown to increase creatine stores in the muscle and speed of PCr resynthesis (Harris et al., 1992; Greenhaff et al., 1993b; Greenhaff et al., 1994b), increase muscle power output (Birch et al., 1994; Greenhaff et al., 1993a; Greenhaff et al., 1994a) and increase recovery in intermittent high intensity exercise (Balsom et al., 1993; Bogdanis et al., 1995b; Greenhaff et al., 1993a; Greenhaff et al., 1993b).

Peak isokinetic torque production has been found to be sustained at a higher level during repeated bouts of maximal voluntary concentric actions after creatine supplementation (Greenhaff et al., 1993a). The effect supplementation has on improving fatigue suggests that an improvement in performance can be achieved for concentric exercise by increasing resting levels of creatine and PCr and improving the rate of PCr and ATP resynthesis in skeletal muscle. No studies have yet investigated the effect of oral creatine supplementation on eccentric exercise.

It has been found that during muscle lengthening in isolated frog muscle a lower ATP hydrolysis occurs compared with shortening and active static actions (Curtin and Davies, 1975). Since the entire muscle was assumed to be activated and all muscle fibres recruited, the reduced energy consumption suggests differences in the muscle contractile mechanism. According to the cross-bridge theory proposed by recent workers, during eccentric muscle actions at intermediate velocities the energy cost may only appear to be the amount necessary to maintain the cell in an activated state. (Curtin and Davies, 1975; Menard et al., 1991; Rall, 1985; Stauber, 1989). However there appears no difference between concentric and eccentric fatigue from repeated maximal muscle actions. Instead, results from the last study found eccentric knee flexors fatigued more than concentric knee flexors. A decline in the rate of resynthesis of ATP due to the depletion of PCr has been recognised as a possible cause of fatigue during maximal high intensity exercise (Bogdanis et al., 1995a). Therefore if the theory regarding energy cost for eccentric actions is accurate fatigue caused from PCr depletion would be lower than concentric actions.

The aim of this study was to examine the effect of creatine supplementation on performance and metabolic responses during concentric and eccentric muscle actions. The findings of this study may increase understanding of the differences in energy utilisation between concentric and eccentric muscle actions.

## 6.2 METHODS

### 6.2.1 Subjects

Twenty eight competitive sportsmen (nineteen from rugby, [fourteen backs and five forwards], seven from soccer and two from hockey) participated as subjects for this experiment which had University Ethical Committee approval. Test procedures were explained to the subjects prior to them giving their informed consent to participate. Each subject completed a medical questionnaire to ensure no-one possessed a musculo-skeletal injury or were on medications or hormonal therapy. All subjects were male. The subjects were randomly assigned to two groups (n=14 in each) which performed either concentric or eccentric muscle actions only.

### 6.2.2 Experimental procedures and protocol

#### 6.2.2.1 Anthropometric measurement

Standard measurements of height and body mass were taken for each subject (Weiner and Lourie, 1981). Skinfold thicknesses were taken at four sites on the body and used in conjunction with regression equations derived by Durnin and Womersley (1974) to estimate percentage body fat. All measurements are described in Chapter 3. The body mass and skinfold measurements were taken prior to both tests.

#### 6.2.2.2 Repeated knee extension and flexion protocol

The subjects in this investigation were required to perform either two concentric or two eccentric repeated bouts of maximal voluntary exercise. The repeated bouts of exercise were administered by use of an isokinetic dynamometer (Kin-Com AP), which was connected on-line to a microcomputer. Each subject had two or three familiarisation sessions for the concentric and eccentric groups respectively to establish reproducible torque measurements (Appendix C). Each familiarisation session included five sets of ten repetitions for the chosen muscle action. One week separated the final familiarisation and the first test. During the second familiarsation session all subjects in both groups performed 3 maximal concentric repetitions at 120 $deg.s^{-1}$ for knee extensors and flexors and the repetition which produced the highest peak torque was recorded.

Each group performed ten sets involving ten repetitions of knee extension and flexion. This protocol was conducted on two occasions, one trial before and one trial immediately after 5 days of either a placebo or oral creatine supplementation period. One group performed only concentric actions whereas the other group performed only eccentric actions. The re-tests were performed at the same time of day and two weeks apart, on the day following the completion of their supplementation. Each subject standardised their dietary intake by eating the same foods prior to both tests (See Appendix A). No strenuous activity was performed 24 hours before each test.

Each subject was measured on an adjustable chair and stabilised with velcro straps. The axis of rotation of the Kin-Kom AP II lever arm was aligned with the anatomical axis of the knee joint, as described in Chapter 3. The Ramping feature was used in all tests to avoid previously found problems such as torque overshoot. Gravity Correction was not selected because of the difficulty found in pilot testing, in standardising the correction during re-tests.

Total work for knee extension and flexion for each repetition was measured. Each subject was measured throughout a standardised range of movement, 5-85$^{o}$, for both tests.

#### 6.2.2.3 Oral supplementation

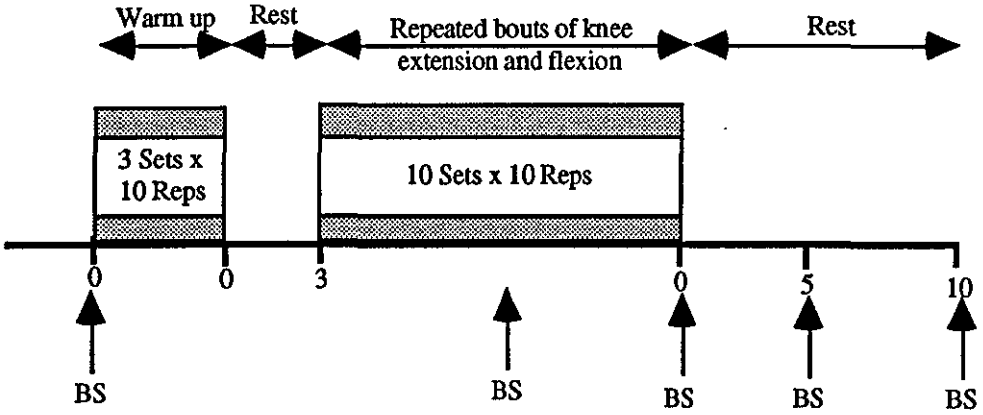
Subjects were provided with either 250 mg.kg body mass$^{-1}$ of creatine plus glucose or a placebo consisting of glucose only per day for 5 days. The supplements were provided in 15 pre-measured packets of powder of either 83.3 mg.kg body mass$^{-1}$ of creatine plus 6 g glucose or 83.3 mg.kg body mass$^{-1}$ of glucose plus 6 g glucose and were given instructions to mix the powder in a hot drink for consumption at 9.00, 13.00 and 17.00 hours during the 5 day supplementation period. All subjects followed their normal training in the days before trial one and trial two, but rested on the day before the tests. The two treatments were administered in a randomised double blind design. All creatine used in the study was purchased by the Department of P.E., S.S., and Rec. Man. at Loughborough University.

### 6.2.2.4. Fatigue index

The difference between highest and the last peak torque and total work were taken from the ten sets and used to calculate fatigue as described in the formula in Section 3.4.6.3.

### 6.2.3. Blood sampling and analysis

Blood samples were taken, as described in Chapter 3., before the exercise test with the subjects in a seated position. Further samples were taken after the fifth set during the test and at 0, 5, and 10 min after the completion of the test. Samples were analysed for blood lactate and plasma ammonia, haematocrit, haemoglobin and percentage change in plasma volume as described in Chapter 3.




BS - Venous blood sample

**FIGURE 6.1** *Schematic representation of the repeated bouts of knee extension and flexion protocol and blood sampling*

### 6.2.4 Statistical anaylsis

A three way analysis of variance (Statistica: MANOVA) was used to examine differences between the control and creatine group (main effect group), between all subjects before and after supplementation (main effect treatment) and to examine the response of all subjects over time (main effect time). As the main effect time was always statistically significant at the $p<0.01$ level, this main effect is not referred to in the tables or figures. Differing responses between the groups as a result of supplementation were identified by group-treatment and group-treatment-time interactions. interactions:

Total work

Plasma ammonia
Blood Lactate
Plasma volume change

A two way analysis of variance (Statistica: MANOVA) was used to examine the differences between the control and creatine group (main effect group) and between all subjects before and after supplementation (main effect treatment) for:

Antropometry
Maximum isokinetic peak torque
Fatigue index

Where a significant F value was found, a Tukey post-hoc test was used to examine where the differences lay.

Results are presented as mean $\pm$ standard deviation.

## 6.3 RESULTS

### 6.3.1 Peak torque and total work output

Anthropometric results and maximum concentric torque at 120 deg.s$^{-1}$ are shown in Table 6.1. Body mass, body fat percentage and lean body mass are shown pre- and post-placebo and creatine ingestion. In both the concentric and eccentric groups (n=14) body mass and lean body mass increased significantly ($p<0.05$) following creatine ingestion by 1.0 ± 0.7 and 0.9 ± 0.6 kg respectively.

**TABLE 6.1** *Anthropometric results and maximum concentric isokinetic torque at 120 deg.s$^{-1}$ for each subject group including body mass, body fat and lean body mass pre- and post-supplementation period (mean ± S.D.)*

| | Concentric placebo group (n=7) | Concentric creatine group (n=7) | Eccentric placebo group (n=7) | Eccentric creatine group (n=7) |
|---|---|---|---|---|
| Age (yrs) | 21.6 ± 1.3 | 21.9 ± 1.6 | 22.9 ± 2.2 | 22.7 ± 2.2 |
| Height (cm) | 180.7 ± 3.3 | 180.6 ± 4.8 | 184.8 ± 6.6 | 180.9 ± 3.9 |
| Weight (kg) pre- | 81.5 ± 10.4 | 86.1 ± 9.6 | 83.7 ± 16.1 | 77.7 ± 5.2 |
| Weight (kg) post- | 81.0 ± 9.4 | 86.6 ± 9.2 | 84.3 ± 16.0 | 79.0 ± 5.2 |
| Body fat (%) pre- | 14.0 ± 2.8 | 16.0 ± 4.1 | 16.1 ± 6.3 | 14.9 ± 2.4 |
| Body fat (%) post- | 13.7 ± 2.6 | 15.7 ± 4.2 | 16.3 ± 5.9 | 15.0 ± 2.4 |
| Lean body mass (kg) pre- | 70.0 ± 7.7 | 72.0 ± 4.0 | 69.5 ± 8.4 | 66.0 ± 4.1 |
| Lean body mass (kg) post- | 69.8 ± 7.2 | 72.7 ± 3.7 | 69.8 ± 8.2 | 67.1 ± 4.2 |
| Maximum isokinetic knee extension torque at 120 deg.s$^{-1}$ (N.m) | 208.1 ± 21.3 | 205.8 ± 19.1 | 208.5 ± 37.8 | 202.7 ± 39.4 |
| Maximum isokinetic knee flexion torque at 120 deg.s$^{-1}$ (N.m) | 154.6 ± 30.4 | 156.3 ± 22.9 | 155.6 ± 31.6 | 162.8 ± 29.9 |

The total work produced during the ten repetitions in each set were added together for the knee extensors during sets 1 to 10 before ($T_1$) and after ($T_2$) the supplementation period (Fig. 6.2a-b). The oral creatine supplementation

had no significant ($p>0.05$) effect on the total work performed by concentric knee extensors (Fig 6.2a). However after supplementation there was a tendency for an increased total work. During eccentric muscle actions creatine supplementation significantly ($p<0.05$) increased total work (Fig 6.2b). The improvement in fatigue for total work in each set appears to increase as the number of sets increase.

No differences ($p>0.05$) were found in total work production for the knee flexors during the 10 sets for both concentric and eccentric groups following creatine ingestion.

The total work in all subjects performing eccentric exercise (n=14) was higher ($p<0.01$) during T1 and T2 than all subjects performing concentric exercise. The total work during $T_1$ in set 1 for eccentric exercise was 66.2 $\pm$ 49.2 % greater than for concentric exercise. The exercise pattern over 10 sets was similar in both groups with the highest mean total work recorded during set 1 followed by a decline to a plateau level.

### 6.3.2 Fatigue index

The fatigue index for the placebo and creatine groups performing concentric and eccentric knee extension are shown in Fig. 6.3. For the concentric group the fatigue index was 31.3 $\pm$ 10.0 % and 30.2 $\pm$ 10.0 % for the placebo group and 14.8 $\pm$ 8.0 % and 19.7 $\pm$ 11.2 % for the creatine group before and after supplementation respectively. The eccentric group fatigue index was 14.9 $\pm$ 9.9 % and 17.3 $\pm$ 7.3 % for the placebo group and 25.7 $\pm$ 14.9 % and 16.0 $\pm$ 12.8 % for the creatine group before and after supplementation respectively. After 5 days of creatine ingestion the fatigue index for the eccentric group significantly ($p<0.05$) decreased by 9.8 $\pm$ 8.2 % whereas supplementation had no significant effect on fatigue index for the concentric group. No difference was found for the fatigue index following creatine ingestion for both concentric and eccentric knee flexion.

No significant difference was observed when comparing fatigue index for the two types of muscle actions, concentric and eccentric.

### 6.3.3 Plasma ammonia

No significance influence was found following oral creatine supplementation in plasma ammonia concentrations for both concentric and eccentric groups

($p>0.05$) (Figure 6.4a-b). However, a decrease in plasma ammonia was observed in the second trial for both concentric and eccentric creatine groups in comparison with the respective placebo groups.

For both concentric and eccentric groups peak plasma ammonia occurred 5 min post exercise with values during $T_1$ of 65.8 $\pm$ 13.8 $\mu mol \cdot l^{-1}$ and 35.9 $\pm$ 12.9 $\mu mol \cdot l^{-1}$ respectively. Comparing concentric with eccentric groups the changes in plasma ammonia concentration from rest, during exercise and 3 samples in 10 mins of recovery during T1 and T2 were significantly ($p<0.01$) lower for the eccentric exercise even though greater work was performed. However it must be noted that the eccentric and concentric groups comprised of different subjects which limits the value of comparisons.

### 6.3.4 Blood lactate

Blood lactate concentrations at rest, during exercise and recovery measured before ($T_1$) and after ($T_2$) placebo and creatine ingestion are presented in Fig. 6.5a-b. For the eccentric group blood lactate was lower after creatine ingestion ($p<0.05$) (Fig 6.5b). For the concentric group creatine supplementation did not affect the blood lactate response ($p>0.05$) (Fig 6.5a).

Mean peak blood lactate concentration was observed during the concentric group at 5 min post exercise whereas the peak values in the eccentric group was observed immediately upon completion of exercise. Mean peak values for the two subject groups during $T_1$ in concentric and eccentric exercise were 5.10 $\pm$ 1.10 $mmol \cdot l^{-1}$ and 3.17 $\pm$ 1.23 $mmol \cdot l^{-1}$ respectively. Again despite the fact less work was performed during concentric exercise a significantly ($p<0.05$) higher blood lactate accumulation was found in both $T_1$ and $T_2$ than that observed for the group performing eccentric exercise.

### 6.3.5 Changes in plasma volume

On all occasions the peak change was observed immediately following the completion of exercise. Following creatine supplementation the decrease in plasma volume was greater than that observed prior to supplementation for the concentric group -4.2 $\pm$ 4.6 % to -8.3 $\pm$ 4.0 % ($p<0.05$). No significance ($p>0.05$) differences were found for the eccentric groups after creatine ingestion.

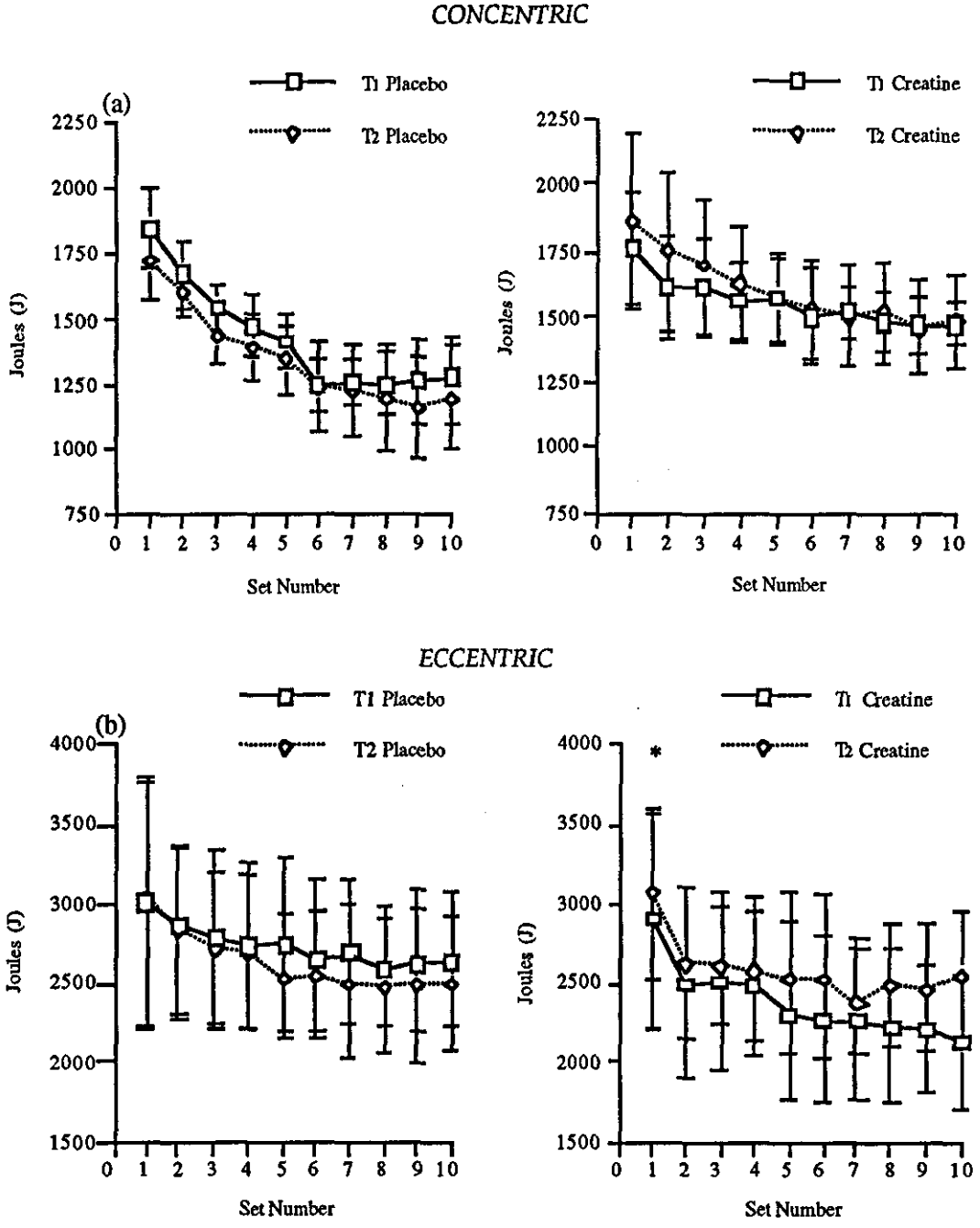



**FIGURE 6.2.** *Total work production of the knee extensor muscle groups for each set during 10 sets of 10 repetitions before ($T_1$) and after ($T_2$) 5 days placebo (250 mg.kg body weight$^{-1}$ + 6g of glucose.day$^{-1}$) or creatine (250 mg.kg body weight$^{-1}$ of creatine + 6g of glucose.day$^{-1}$) ingestion. Each bout of 10 repetitions was interspersed with 60s recovery. Graphs are arranged with concentric muscle actions as (a) and eccentric muscle actions as (b). Values are means ± S.D. Treatment-group interaction for concentric, n.s., treatment-group interaction for eccentric, P<0.05 represented as *. Treatment-group-set number interaction for concentric, n.s., treatment-group-set number interaction for eccentric, n.s.*

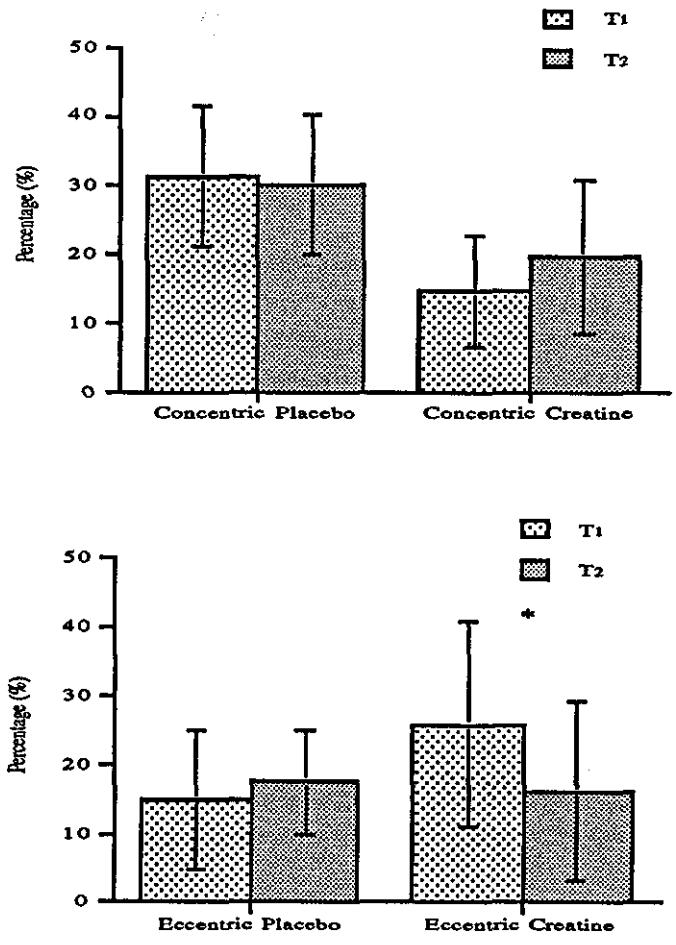



**FIGURE 6.3.** *Fatigue index for the knee extensor muscle groups from concentric and eccentric muscle actions before ($T_1$) and after ($T_2$) 5 days of placebo (250 mg.kg body weight$^{-1}$ + 6g of glucose.day$^{-1}$) or creatine (250 mg.kg body weight$^{-1}$ of creatine + 6g of glucose.day$^{-1}$) ingestion. Treatment-group interaction for concentric, n.s., treatment-group interaction for eccentric, $P<0.05$ represented as *. Values are means $\pm$ S.D.*

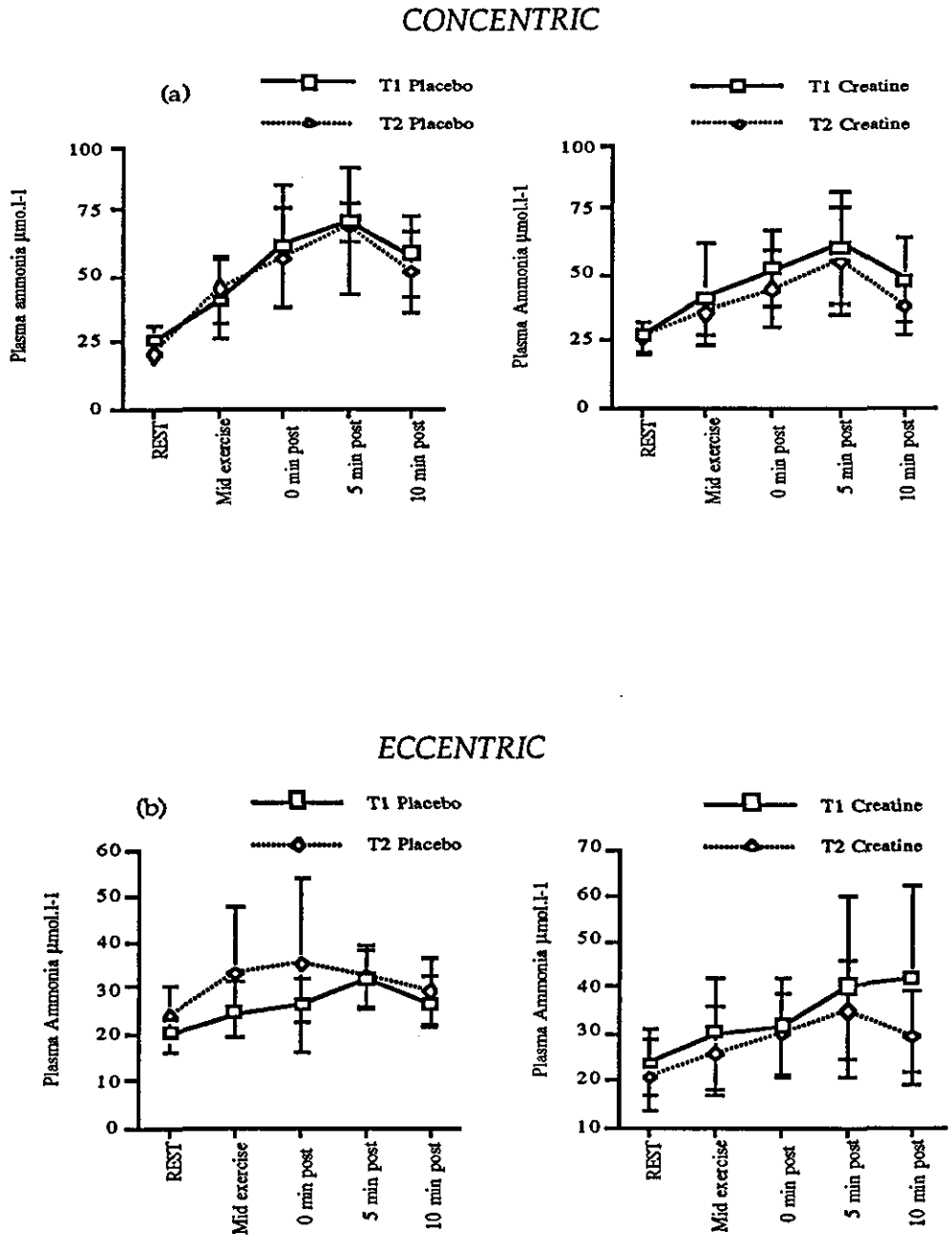



**FIGURE 6.4.** *Plasma ammonia concentration ($\mu mol^{.l-1}$) measured at rest (1), immediately after 5 sets, 10 sets and 5 and 10 min recovery, before ($T_1$) and after ($T_2$) 5 days placebo (250 mg.kg body weight$^{-1}$ + 6g of glucose.day$^{-1}$) or creatine (250 mg.kg body weight$^{-1}$ of creatine + 6g of glucose.day$^{-1}$) ingestion. Each bout of 10 repetitions was interspersed with 60s recovery. Graphs are arranged with concentric muscle actions as (a) and eccentric muscle actions as (b). Values are means ± S.D. Treatment-group interaction for concentric, n.s., treatment-group interaction for eccentric, n.s. Treatment-group-time interaction for concentric, n.s., treatment-group-time interaction for eccentric, n.s.*

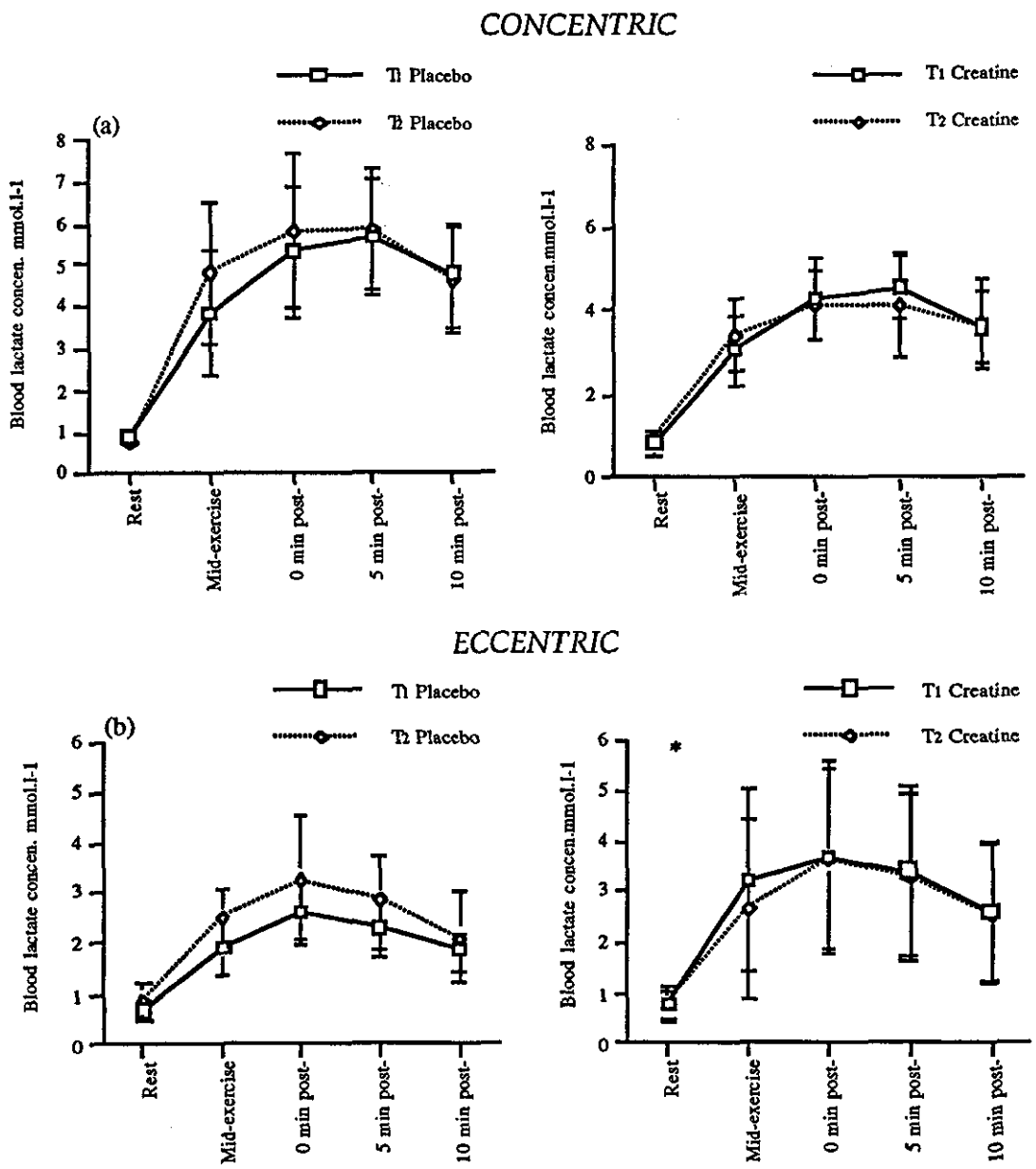



**FIGURE 6.5.** Blood lactate concentration ($mmol \cdot l^{-1}$) measured at rest, immediately after 5 sets, 10 sets and 5 and 10 min recovery, before ($T_1$) and after ($T_2$) 5 days placebo (250 mg.kg body $weight^{-1}$ + 6g of $glucose.day^{-1}$) or creatine (250 mg.kg body $weight^{-1}$ of creatine + 6g of $glucose.day^{-1}$) ingestion. Each bout of 10 repetitions was interspersed with 60s recovery. Graphs are arranged with concentric muscle actions as (a) and eccentric muscle actions as (b). Values are means ± S.D. Treatment-group interaction for concentric, n.s., treatment-group interaction for eccentric, $P<0.05$ represented as *. Treatment-group-time interaction for concentric, n.s., treatment-group-time interaction for eccentric, n.s.

## 6.4 DISCUSSION

The main finding of this study was that total work increased and the fatigue index decreased following 5 days of creatine ingestion, at a rate of 250 mg.kg $bw^{-1}$ per day, during maximal unilateral isokinetic eccentric knee extension muscle actions.

**6.4.1. The effect of creatine supplementation on repeated eccentric actions**

Blood analysis for the eccentric group showed lower blood lactate accumulation following oral creatine ingestion. Mid-exercise blood lactate concentrations were 3.23 $\pm$ 1.79 $mmol.l^{-1}$ pre-supplementation compared with 2.68 $\pm$ 1.79 $mmol.l^{-1}$ post-supplementation. Some studies have found no difference in blood lactate concentration following creatine ingestion and concluded that supplementation has no direct effect on glycolysis during exercise and the increase in work which was found arose from other sources (Greenhaff et al., 1993a; Greenhaff et al., 1994a; Birch et al., 1994). Oral creatine supplementation has been postulated to reduce ATP loss during high intensity muscle actions by maintaining better the rate of ATP resynthesis from ADP (Greenhaff et al., 1994a). However the results of Balsom et al. (1993) support the present study by showing lower accumulation of blood lactate following 10 x 6 s maximal cycling bouts. Assuming that the rate of lactate efflux is unaltered, the lower blood accumulation and greater work performed suggests that the increase in creatine stores has influenced the rate of anaerobic glycolysis during the present study's protocol. This is possibly caused by a feedback mechanism regulated by the reduced accumulation of ADP and AMP (Ren and Hultman, 1990). The nucleotides, ADP and AMP have been found to be powerful activators of the rate limiting enzyme, PFK, in the glycolytic pathway (Sahlin, 1986). Thus a change in activation of PFK will cause a change in the rate of gylcolysis.

No significant ($p<0.05$) differences were observed in the plasma ammonia concentration in eccentric exercise as a result of creatine ingestion, but a small decrease was observed (ns). Plasma ammonia is an accepted marker of muscle adenine nucleotide loss and the decline in muscle ATP (Lowenstein, 1972). A reduction in the rate of ADP rephosphorylation to ATP causes an increase in plasma ammonia as a consequence of a decrease in the availability of PCr and/or the rate of glycogenolysis (Sahlin and Katz, 1988). Although

no significance was found, a reduction would be expected for plasma ammonia concentrations, indicating lower ADP rephosphorylation, an improvement of performance following oral creatine ingestion, based on similar findings from recent investigators (Birch et al., 1994; Bogdanis et al., 1995b).

Fatigue during eccentric actions was 25.7 $\pm$ 14.9 % and 16.0 $\pm$ 12.8 % before and after creatine ingestion period respectively ($p<0.05$). The onset of muscle fatigue is commonly associated with the depletion of the muscle PCr store (Hultman et al., 1967). Depletion will limit the rate of ADP rephosphorlation to ATP. Following a similar supplementation regimen to that used in the present study, total creatine concentration of human skeletal muscle has been reported to increase by about 22 mmol.kg $dm^{-1}$ and PCr content to increase by about 6 mmol.kg $dm^{-1}$, both with large individual variations (Harris et al., 1992). The importance of the PCr system to muscle action is in maintaining a high intracellular ATP/ADP ratio (Kammermeier, 1987). This function is achieved by using PCr as an immediate buffer to ATP and also as a facilitator of energy transport from the mitochondria to sites in the cytosol requiring ATP utilisation (Bessman and Geiger, 1981; Bessman and Savabi, 1988). PCr also contributes to the buffering of $H^+$. Therefore, an increase in PCr stores in skeletal muscle will aid performance as a result of a higher pre-exercise concentration, a higher rate of resynthesis during recovery periods and a smaller decrease in muscle pH (Balsom et al., 1994). Fatigue is also associated with $H^+$ which affects the activity of glycolytic enzymes, the binding sites for actin and myosin and PCr resynthesis (Hermansen, 1981; Sahlin, 1986). However blood lactate concentration during eccentric exercise peaked at only 3.17 $\pm$ 1.23 $mmol^{l-1}$, which suggests that fatigue is unlikely to be caused by an increase of $H^+$ from anaerobic glycolysis. Therefore, PCr appears to be the limitation for this eccentric exercise protocol in maintaining total work.

According to the cross-bridge theory, during eccentric actions the cross-bridge breaks before the completion of the excitation coupling cycle, thus before a breakdown of an ATP molecule can occur. The energy utilisation measured during eccentric exercise has been proposed to be consumed predominantly by the transduction of $Ca^{2+}$ across the sarcoplasmic reticulum and the additional energy required for muscle action is derived from the external resistance causing the muscle to lengthen. It would appear unlikely

that the energy demand of $Ca^{2+}$ transduction would deplete PCr stores. Therefore the results from the present study conflict this theory. If eccentric actions relied predominantly upon the external source for energy then it would be expected that creatine supplementation would have no effect. However despite total work significantly improving in the creatine group, blood lactate significantly decreased in comparison with the placebo group and plasma ammonia was observed to decrease (ns). These findings suggest that the probable increase in total stores of creatine and PCr, as a result of supplementation dosage, allowed faster PCr resynthesis during the exercise. This indicates eccentric actions rely upon PCr to sustain work and prolong the onset of fatigue.

Psychological factors must also be taken into consideration when examining differences between maximal concentric and eccentric exercise. During isokinetic measurements, valid results rely on the subject's compliance for maximal effort. Subjects regularly commented during familiarisation that they were unaccustomed to eccentric exercise. Some investigators have found that during fast eccentric exercise subjects perceive the activity to be unnatural, uncontrolled and therefore inhibitory (Johnson et al., 1976). It has been reported during unaccustomed eccentric activity that subjects have a reluctance to exert maximally or have difficulty in performing the co-ordination of the task (Sale, 1988). EMG has been reported to increase progressively during continuous eccentric exercise whereas during concentric exercise the EMG reported no increase (Bigland and Lippold, 1954). The increase in muscle activation during a bout of eccentric exercise suggests greater amount of effort is exerted as more repetitions or time progresses. During pilot testing in the first study subjects were uncomfortable and felt uncontrolled for eccentric knee flexion, hence it was excluded from the tests. During the present study the testing velocity selected was 120 $deg.s^{-1}$ which was considered the most comfortable eccentric velocity by the subjects. Nevertheless for all studies the subjects were unaccustomed to eccentric exercise, thus it must be considered that subjects may not have put maximal effort during part or whole of the exercise bout. This creates an uncertainty when attempting to compare equal amount of efforts for the two different muscle action types. However each subject was given considerable verbal assurance and provided with at least two familiarisation sessions in the attempt for maximal effort. Nevertheless, the psychological influence on effort was the same for both the placebo and creatine ingestion groups. Thus

the psychological factor has not affected statistical significance shown for the eccentric group.

**6.4.2. The effect of creatine supplementation on repeated concentric actions**

The lack of change in performance during concentric actions is in contrast to previous studies (Greenhaff et al., 1993a; Greenhaff et al., 1994a; Birch et al., 1994). For example, Greenhaff and co-workers (1993a) demonstrated that performance improved during 5 x 30 s isokinetic concentric knee extensions during the final 10 repetitions for each set with 20 g of creatine per day. Also, muscle ATP degradation has been shown to reduce by about 50 % on the second bout of 2 x 30 s isokinetic concentric exercise despite more work produced following creatine ingestion (Greenhaff et al.,1994a). In addition, an increase in total work has been found during the first 2 bouts of 3 on an isokinetic cycle ergometer for 30 s (Birch et al., 1994). It appears from these studies that oral creatine supplementation does have a positive effect on fatigue during repeated concentric muscle actions, thus it would be expected that the results of the present study would have been consistent with earlier reports. It has been claimed that performance in maximal high intensity exrecise is limited by PCr affecting the rate of ATP resynthesis (Bogdanis et al., 1995a). Therefore if creatine ingestion did not affect performance during this protocol, the exercise protocol in the present study may not fully deplete PCr. However the design was identical for both exercise groups and eccentric creatine group did show a significant change following creatine ingestion.

It is possible that the sample size was too small to detect statistical differences in the metabolic and performance variables following creatine supplementation. An upper limit of total creatine stores appears to range between 150-160 mmol.kg $dm^{-1}$ (Harris, et al., 1974) and if subjects are near this upper limit before supplementation then the effect of creatine ingestion would be minimal. As all subjects compete at a high level in soccer, rugby and hockey it could be possible some of these subjects have high creatine stores due to natural selection. It has been shown that type II fibres have higher levels of PCr than type I fibres (Tesch et al., 1989; Soderlund et al., 1992) and that athletes who compete in these sprint related sports have a greater proportion of type II fibres compared with endurance athletes and untrained people (Bergh et al., 1978; Costill et al., 1976; Thorstensson et al., 1976; Thorstensson et al., 1977; Tihanyi et al., 1982). Therefore even though a significant change was found for the eccentric creatine group the sample size

for the concentric group might need to have been larger to show a significant performance change. Such a suggestion is supplied by the tendency for a higher total work, lower ammonia and a significant ($p<0.05$) reduction in the change in plasma volume after creatine supplementation.

Various studies have shown that a 5 day supplementation period significantly increases body mass (Harris et al., 1992; Balsom et al., 1993). Creatine supplementation has also increased body mass in the present study with an average increase of $1.0 \pm 0.7$ kg ($p<0.05$). Lean body mass was also calculated and a similar increase of $0.9 \pm 0.6$ kg was shown ($p<0.05$). The increase in mass could be due to a structural change at the myofibril level in the muscle however the most likely explanation of this mass gain is water retention.

### 6.4.3. Summary

The findings of the present study question the current theories regarding energy utilisation during repeated eccentric exercise in that as creatine supplementation was effective in reducing fatigue during eccentric muscle actions, it suggests that energy supply (and probably myosin ATPase activity) may be a limitation in eccentric as well as concentric actions.

# *CHAPTER 7. GENERAL DISCUSSION.*

The studies described in this thesis were designed to examine the functional and mechanical differences between concentric and eccentric muscle actions by measuring isokinetic torque and work, and by the analysis of blood samples following repeated exercise bouts. New methods for analysing the relationship between sprint performance and strength have been examined and the established theories concerning the energy utilising mechanisms for eccentric exercise examined.

## 7.1 RELATIONSHIP BETWEEN STRENGTH AND SPRINT RUNNING PERFORMANCE

The first study examined the relationship between concentric and eccentric muscle actions and sprint running times. Sports such as rugby, soccer and hockey require both acceleration and maximal velocity; therefore these two aspects were considered in relation to sprint performance. The involvement of both concentric and eccentric actions during sprinting were analysed (Figure 2.2) and it was discovered that all types of movements and actions occur for each lower limb joint, excluding concentric ankle dorsiflexion. This analysis indicated that acceleration and deceleration of the lower limbs take place throughout a sprinting cycle.

It is acknowledged that isokinetic movements are unnatural and measure an isolated joint movement and therefore cannot completely predict the functional capacity of the subject in actual human sprinting. However, in the first study of the thesis significant relationships were found between muscle strength of the lower leg joint actions and sprint times which implies the importance of concentric and, to a smaller degree, eccentric strength as major components in superior sprint performances.

The findings supported some of the work of earlier researchers and highlighted new areas of significance in the analysis of the relationship between strength and sprint performance. Elite sprinters were found to produce greater torques than non-elite sprinters at fast angular velocities, which supports previous findings. It was suggested that this greater torque at fast angular velocities may be accounted for by elite sprinters possessing a greater percentage of fast twitch muscle fibres. The concentric strength of the

knee extensors at fast velocities appears to have the stongest relationship with sprint times, especially strength in relation to body mass. Absolute eccentric knee extension torque also showed a significant relationship with both 0-15 m and 30-35 m sprint times. Eccentric knee extension has been described as important for terminating negative vertical velocity at heel strike and assisting the concentric action during the push off. The correlations between 0-15 m and 30-35 m sprint performances and concentric knee flexion, concentric hip extension and flexion and concentric plantarflexion, indicate that each action has a significant degree of importance in sprinting. Previous studies have found a significant relationship between sprint times and hip extension and flexion. However, the relationship between concentric knee flexion and ankle plantarflexion reported in Chapter 4., has not been previously observed. All the correlations found between sprint times and each muscle action were similar for both 0-15 m and 30-35 m times except that the relationship between sprint times and concentric hip extension and flexion was statistically significant only for 30-35 m times. With concentric hip extension and flexion having a significant relationship with 30-35m sprint time rather than 0-15 m, it may indicate that these muscle actions are more influential once a sprinter has achieved near maximal running velocity.

Another purpose of the first study was to examine the relationship between strength and sprint performance using both time and acceleration as the performance measure also taking body mass and dimensions into account. Previous literature has only examined the relationship using sprint times and predominantly using only the knee actions. Thus using Gunther's theoretical models and measuring hip and ankle joint actions provided an opportunity for original work. A finding of this study was that a stronger relationship appears to exist between force and acceleration rather than between force and time. This suggests that force is a better predictor of acceleration rather than sprint times. When body dimension was taken into account, using Gunther's proposed models, the relationship became even stronger. In simple and practical terms, if two sprinters can produce equal force or torque and have the same body mass, the shorter subject should have the greatest acceleration. This may already be known, but this study was the first to confirm this finding using theoretical models.

The relationship found between peak torque and angular velocity for both concentric and eccentric actions supports the previous literature. The torque-

velocity relationship has been extensively studied using both whole animal muscle, single fibre preparations using electrical stimulation and intact human muscles. The overall shape of the concentric torque-velocity curve, or so-called force-velocity relationship, for intact human muscles is similar to that of animal preparations (Thorstensson et al., 1976; Tihanyi, et al., 1982; Wilkie, 1950). However, since the development of isokinetic dynamometry, the eccentric force-velocity relationship has been examined extensively and in vivo human muscle the relationship appears to differ in comparison with the isolated animal muscle findings. Differences include the amount of increase in force or torque above isometric production (Smidt, 1973; Westing et al., 1988) and the lack of force or torque increase as testing velocity increases (Westing et al., 1988). The explanation for these differences lead the research described in Chapters 5 and 6 into another direction, to discover more about the responses to and the mechanisms of action of eccentric exercise compared with the established understanding of both the metabolic responses to and mechanism of muscle action during concentric exercise.

## 7.2 METABOLIC DIFFERENCES FOR ECCENTRIC MUSCLE ACTIONS COMPARED WITH CONCENTRIC

It has been well documented in the literature that for equal work, eccentric actions have a reduced energy expenditure of up to 25 % compared with concentric actions. During equal submaximal work eccentric actions have been reported to display lower EMG activity compared to concentric (Bigland and Lippold, 1954). An EMG measures the number of action potentials in a muscle, an indication of the level of recruitment and activation. This knowledge helps to explain why less metabolic activity is necessary to perform the same tasks.

In the second study of the thesis, lower blood lactate concentrations were found for eccentric exercise in comparison with concentric, during maximal voluntary actions. Blood lactate concentration is mainly influenced by the type and time of muscular work (Scharf et al., 1994). Environmental conditions, range of movement, exercise time and source of blood samples which may also affect the results, were standardised for both tests. Therefore the changes in blood lactate reflected the differences in muscle actions. Reports in the literature have revealed, through EMG recordings, that under maximal voluntary effort, eccentric actions do not appear to represent the maximal torque producing capacity, which suggests that some inhibitory mechanism prevents the development of full muscle activation. This inhibitory mechanism would explain the energy expenditure differences between muscle actions. However, Curtin and Davies (1975) found less energy was utilised by an isolated tetanized muscle during lengthening compared to shortening. The lower energy cost from fully activated eccentric actions has been explained by the theory, that the cross-bridge is forced apart as the muscle lengthens, before the completion of the excitation coupling cycle, which prevents the breakdown of an ATP molecule. When the muscle fibre remains activated the detached cross-bridge is able to re-attach to the next available binding site. Thus, the energy required during eccentric actions has been postulated to derive from the external source and the amount of energy that is utilised is required to keep the muscle in an activated state only. This theory has been accepted and used to explain other differences in metabolic responses following repeated bouts of eccentric exercise.

The results of the third study lead to a closer examination of this theory. Following 5 days of creatine ingestion, work output during repeated bouts of eccentric exercise was increased. If eccentric actions relied upon energy only to sustain an activated state, creatine ingestion would have had no effect. The results of this study require further explanation compared with the findings of lower energy consumption during tetanized lengthening muscle (Curtin and Davies, 1975). During tetanized conditions the energy consumed during lengthening and shortening with all other factors remaining constant, should be equal if both actions rely on the same amount of energy to act. However the current understanding of the mechanism of action suggests that eccentric actions produce a greater amount of work with less energy expenditure. The findings of the third study however indicate that the energy cost for repeated eccentric exercise is sufficient for PCr to be the limiting factor in maintaining work output. The following theories attempt to explain why creatine supplementation had an effect on repeated eccentric muscle action.

One possible reason could be the temperatures associated with eccentric actions. A three-fold greater production of heat has been found during eccentric exercise compared to the same metabolic rate as concentric (Nielsen et al., 1972). Eccentric exercise has been reported to produce 3 and 1.2 °C greater temperatures in the skin and muscle respectively following 40 min of work in comparison to work performed at the same metabolic load as in concentric exercise (Nadel et al., 1972). The muscle temperature in the quadriceps femoris has been reported to be as high as 41.8 °C (Nadel et al., 1972). Chemical reactions increase their rate with an increase in temperature until a certain point where reactions slow down and/or stop. As the temperature rises enzymes are denatured by heat so that the activity will tend to decrease. A decreased enzyme activity has been reported after short exposures to temperatures above 60 °C (Newsholme and Leech, 1983). However the research is limited on the effect of muscle temperature on chemical reaction rates following eccentric actions. Therefore, the increase in muscle temperature to 41.8° may not inhibit or slow down chemical reactions such as the hydrolysis and formation of ATP and PCr. However the protocol in the third study required maximal actions and produced a higher workload than the Nadel et al. (1972) protocol which may indicate that muscle temperature could be higher in these conditions. It is therefore, just possible that high muscle temperatures may inhibit chemical reactions, but it is much

more likely that such reactions will be increased with muscle temperatures of around 40 $^{o}$C and just over. Thus, the increase in muscle temperature cannot explain the lower energy cost of eccentric action.

Another factor which may influence the energy cost of eccentric muscle actions is direct damage to the structure or function of muscle fibres. The muscle soreness questionnaire (illustrated in Table 5.4.) indicated that subjects experienced significant soreness following eccentric actions for the hamstrings, quadriceps and inside thigh. Muscle soreness is associated with inflammation, damage to the connective tissue, endomyosium and myofibril damage. Severe cellular disruption within the muscle fibre structure can be observed immediately after eccentric exercise (Lieber et al., 1994). This may include sarcomere derangement, swollen mitochondria, fragmented or swollen sarcoplasmic reticulum elements, dilated T-tubules and lesions in the plasma membrane (Stauber, 1989). Disturbances have been reported of the striated pattern with Z bands streaming and broadening, mostly in the type II muscle fibres and affecting nearly 50% of the fibres examined, following intense cycling involving eccentric actions (Friden et al., 1983). This may imply that the sarcoplasmic reticulum and T-tubules are also damaged, which would interfere with normal $Ca^{2+}$ metabolism, thus affecting the energetics involved in the contractile mechanism. It has also been postulated that the physiological alterations could manifest themselves by faulty actin- myosin coupling, impaired $Ca^{2+}$ transport, inability to produce tension and abnormal energetics (Stauber, 1989). Therefore, cell damage during repeated eccentric exercise bouts may affect the energetics involved. These findings increase the likelihood of PCr in some way becoming a limiting factor for the protocol used in the third study. Further investigations with subjects immediately evaluated during or after eccentric exercise bouts for signs of muscle fibre damage are required.

A final explanation which might affect the energy cost of performing 10 sets of 10 repetitions of eccentric knee extension and flexion, is the co-activation of the antagonists. Antagonist muscles take an active part by their braking effect for accurate termination of the limb when there is excessively strong impulsation of the agonist at high velocities (Osternig et al., 1986). When subjects move through a small distance quickly, antagonist activity has been found to be large, whereas movements performed slowly and over a large distance, the antagonist activity was relatively small (Marsden et al., 1983).

An isokinetic dynamometer was used in all studies and the movements for the repeated exercise protocol were performed at 120 deg.s$^{-1}$ (intermediate velocity) and over a short range of movement (80 °). EMG recordings have been measured from the quadriceps and hamstrings for track athletes performing maximal isokinetic concentric knee extensions and flexions at 100 deg.s$^{-1}$ (Osternig et al., 1986). During knee extensions, the hamstring activity was 33 % of its agonist activity throughout the range of movement. Hamstring co-activation during knee extensions also generated 58 % of its agonist activity during the last 25 % of the subject's range of movement. The authors concluded that during isokinetic measuring the activity of antagonists should be considered, although no ramping was used for this study. Osternig et al. (1986) however postulated that co-activation was also induced to assist in knee stabilization. This suggestion is supported by Freund and Budingen (1978 - cited Osternig et al., 1986) who found co-activation with the onset of rapid and vigorous isometric force adjustments. Therefore co-activation of the antagonists appears to serve as a decelerator during knee extension prior to terminal extension and act as a joint stabiliser throughout the range of movement. Osternig and co-workers (1986) only examined concentric actions, however the same stabilization of the joint would be necessary for eccentric actions. Therefore it is proposed that during an eccentric exercise bout, activation of the antagonists existed for both eccentric knee extension and flexion which would contribute to the energy expenditure of the exercise. Based on these findings and explanations, the eccentric cross-bridge theory could still be valid and the effect of creatine on the eccentric group in the third study may have been for the antagonist activation as well as maintaining activation of the agonist muscle while performing eccentric actions. Further research is required to examine EMG activity for the agonist and antagonist muscles during isokinetic eccentric exercise to validate these theories.

The fatigue index was found to be greater during eccentric actions following repeated knee flexion compared with concentric actions. It has been suggested that greater fatigue is found for eccentric actions at slow testing velocities compared to concentric, however this trend is reversed for fast velocities (see Section 2.6.7.). In the case of eccentric actions performed at slow velocities, Stauber (1989) postulated that some of the cross-bridges may have time to complete their cycles and consume a greater amount of energy. This is supported by Kaneko et al. (1984) who reported a greater mechanical

efficiency for eccentric exercise as the angular velocity in the knee increased. Efficiency was calculated from work generated and energy expenditure. Therefore at slow velocities the amount of fatigue could be influenced by the depletion of PCr. At fast velocities a greater contribution of force could be generated during eccentric actions by the elastic properties (passive component) of the muscle which could account for the reduced fatigue in relation to concentric exercise. The difference in fatigue with a change in velocity implies the excitation-coupling cycle contribution to force and energy expenditure for eccentric actions changes at various velocities, however the explanation remains unclear and requires further investigation. The results in Chapter 5. revealed that at an intermediate velocity, 120 deg.$s^{-1}$, fatigue from repeated knee flexion was significantly greater during eccentric actions than during concentric actions. This finding may also question the eccentric energy cost theory. Currently it is unknown what causes fatigue during repeated eccentric actions, however the literature regarding concentric actions have described the importance of PCr resynthesis and the effect of $H^+$ on the maintainance of peak power and total work. However if these factors were equally responsible for fatigue during eccentric actions it would appear likely that the energy cost is greater than the proposed theory suggests. If the energy cost is the amount only to maintain the cell in an activated state during eccentric actions then the processess involved in fatigue appear to differ from that for concentric actions. One theory postulated by Newham and co-workers (1983) related fatigue during eccentric exercise to mechanical damage to the sarcoplasmic reticulum, resulting in altered $Ca^{2+}$ release and neural electrical activity. Various investigators have supported the influence of muscle fibre damage following eccentric exercise on the energetics involved in muscle action, however these have involved measuring the recovery time for EMG and metabolites between 1 hour to 7 days (Doyle et al., 1993; Kroon and Naeije, 1991; Newham et al., 1983).

In overview then, the results in this thesis do question exsisting theories relating to the mechanism of muscle action during eccentric exercise. As creatine supplementation was effective in enhancing performance it is possible at least some ATP hydrolysis is required at intermediate velocities during the excitation-coupling cycle for during eccentric actions. Further investigations are necessary to measure the effect of muscle damage on the energy expenditure systems during repeated high intensity eccentric actions.

## 7.3. SUMMARY

The thesis has provided new methods for analysing the relationship between sprint performance and strength and has identified the different responses following repeated concentric and eccentric actions. The proposed theory regarding the actin-myosin cross-bridge being forced apart without a breakdown of an ATP molecule at intermediate to fast velocities has been questioned. Following oral creatine supplementation fatigue was reduced and a lower concentration of blood lactate was found for repeated eccentric actions. This finding indicates that PCr is relied upon during eccentric actions to maintain total work and delay the onset of fatigue. In addition there appears to be no major difference in fatigue at intermediate velocities between concentric and eccentric actions. It is widely understood that fatigue is influenced by the rate of PCr resynthesis and by an increase of $H^+$ from anaerobic glycolysis during high intensity short term exercise. Therefore the fatigue processes during eccentric actions are either different compared with concentric actions or the eccentric cross-bridge theory in relation to energy cost must be questioned.

In conclusion the findings in this thesis suggest further study is required on eccentric actions to understand and confirm the contractile mechanisms involved and differences that occur with a change of velocity. The following list cites recommended areas for further investigation based on the findings of the present studies:

* Measurement of muscle ATP, PCr and lactate at rest and post-exercise following both maximal concentric and eccentric exercise in an attempt to understand the fatigue processes for eccentric exercise.
* Fatigue appears different at different testing velocities for eccentric actions. Muscle metabolites need to be examined following exercise performed at different velocities.
* Examination of the effect of cell damage on the energetics and fatigue during repeated bouts of eccentric exercise.
* Examination of the increase in muscle temperature following repeated bouts of maximal eccentric exercise and its effect on the rate of chemical reactions.

* Measurement of EMG recordings during maximal isokinetic testing to detect the extent of co-activation for the antagonist muscle for eccentric knee extension and flexion.
* Examination of the relationship between sprint running performance and eccentric flexion for each lower limb joint.

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# APPENDICES

**APPENDIX A** ..... **Subject information**

**I. First study:** Subject information prior to participating in the study

**II. Second study:** Subject information prior to participating in the study

**III. Third study:** Subject information prior to participating in the study

**IV. Study readiness form**

**V. Food record diary**

**VI. Muscle soreness questionnaire**

**APPENDIX B** ..... **Blood Metabolite Assays**

**I. Fluorimetric assay for the determination of blood lactate**

**II. Spectrophotometric determination of plasma ammonia**

**APPENDIX C** ..... **Calibrations, Reliability and Calculations**

**I. Isokinetic Calibration**

**II. Subject test re-test reliability using electronic sprint timing equipment**

**III. Calculations**

I. FIRST STUDY

SUBJECT INFORMATION PRIOR TO PARTICIPATING IN THE STUDY

LOUGHBOROUGH UNIVERSITY OF TECHNOLOGY

DEPARTMENT OF PHYSICAL EDUCATION, SPORTS SCIENCE AND RECREATION MANAGEMENT

| | |
|---|---|
| Supervisors: | Dr. Mary E. Nevill, Dr. Henryk K.A. Lakomy and Mr Rex Hazeldine. |
| Investigators: | Mr Martin Dowson |
| Telephone: | 223496 (Martin) |


---

**Study Title:** The relationship between muscle strength and sprinting performance.

**Aims and Outline of the Study**

The aim is to investigate the relationship between muscle strength and sprinting speed. You will be required to perform a series of strength tests and sprint over two distances to record your sprint times. All strength measurements will be determined by use of an isokinetic dynamometer (Cybex 6000). The Cybex 6000 can measure strength in both a concentric (shortening) and eccentric (lengthening) mode, which is useful in that both these modes are used in sprinting. It is possible to test torque output of a muscle group at a range of different speeds from 0 degrees/second to 500 degrees/second. The sprint times will be measured using electronic timing (photo-electric beams). A relationship will be found between the different muscle groups tested, the different speeds of the strength test, concentric and eccentric, and limb lengths with the your sprint times. This will provide information on the questions of how important is concentric and eccentric muscle strength at different speeds to sprinting speed and which muscle groups make the greatest contribution?

**Time Commitment**

Familiarisation of the sprint and strength tests will take place over a period of one week. This requires only one session. The strength and sprint tests will take place over three weeks. You will be required on two separate occasions. The strength test will take approximately one hour. The sprint tests will take approximately half an hour. All sessions will occur during the evening.

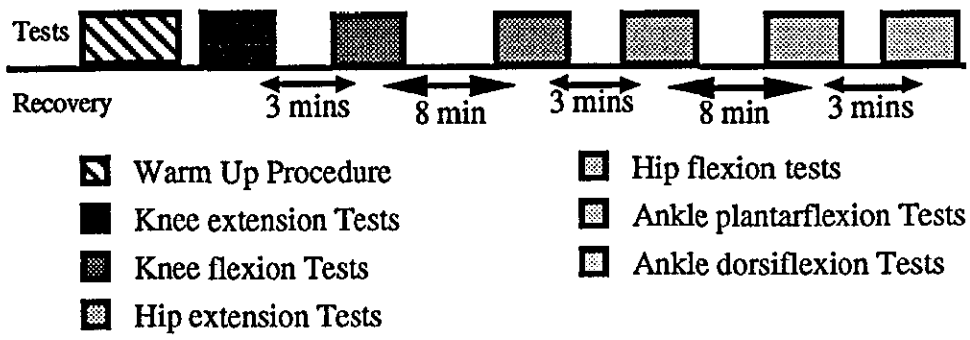
**Testing Procedures:**

Strength Testing

Prior to testing at each velocity 3 warm up trials will be performed. The major muscle groups of the lower body which produce force in sprinting will be tested, which will include: hip extensors, hip flexors, knee extensors, knee flexors, ankle plantarflexors and dorsiflexors. All joint movements will be tested for both concentric and eccentric contraction types. All movements

will be tested at a slow, Intermediate and fast ranging from 30-300 degrees/speed. This totals up to eighteen separate tests. Sufficient rest will be allowed between each test. Following the warm up trials, three maximal trials will be performed for each test. The repetition for each test which exhibits the greatest torque value will be taken as the measure of maximal strength.

**Schematic representation of the main test protocol**




Sprint Testing

All sprint tests will be performed in an indoor sprint corridor at distances of 15 and 35 metres. A standing static take-off will be used. Three trials of each distance will be performed with approximately five minutes elapsing between each trial. Prior to testing a standardised warm up will be performed.

**Measurements:**

Standard measurements of height and weight will be taken. Skinfold thicknesses will be taken at four sites on the body (biceps, triceps, subscapula and suprailiac). Limb lengths will be measured using flexible rules.

Strength Test

The dominant leg, based on kicking preference, will be measured. The variables for each test will include: peak torque over the range of motion, total work, and total power.

Sprint Tests

The mean of the best two trials will be recorded. The 15 metre test will measure 5 metre split times over 15 metres. The 35 metre test will measure basic speed over 10 metres after a 25 metre flying start. The 5 metre split times can be compared to the basic speed measurement (converted to metres per second) and percentage of their basic speed for each 5 metres can be determined.

**Possible risks, discomforts and/or distress :**

Although the chance of injury during strength testing of top athletes is remote, the chance does exist and you should be aware of this. During the familiarisation session you will be gradually introduced to each speed, to determine if you can comfortably be tested without a perceived risk of injury.

## STATEMENT OF INFORMED CONSENT

I have read the above outline of the procedures which are involved in this investigation and **I understand** what will be required of me. I have had the opportunity to ask for further information and for clarification of the demands of each of the procedures. I am aware that I have the **right to withdraw** from the study at any time, with **no obligation to give reasons** for my decision.

I agree to take part in the 'The relationship between strength and sprinting speed study'.

SIGNED _____ DATE: _____

WITNESSED BY _____

## II. SECOND STUDY

SUBJECT INFORMATION PRIOR TO PARTICIPATING IN THE STUDY

LOUGHBOROUGH UNIVERSITY OF TECHNOLOGY

DEPARTMENT OF PHYSICAL EDUCATION, SPORTS SCIENCE AND RECREATION MANAGEMENT


| | |
|---|---|
| Supervisors: | Dr. Mary E. Nevill, Dr. Henryk K.A. Lakomy and Mr Rex Hazeldine. |
| Investigators: | Mr Martin Dowson |
| Telephone: | 223496 (Martin) |


---

**Study Title:** Hormonal and metabolic responses to concentric and eccentric single bouts of high intensity work-outs.

**Aims and Outline of the Study**

The aim is to investigate differences in growth hormone and lactate responses following an eccentric and a concentric high intensity exercise test. No previous research has investigated growth hormone following exercise on an Isokinetic Dynamometer. You will be required to perform an exercise test which will last 30 minutes. Following the test venous blood samples will be taken to measure growth hormone, insulin, glucose and lactate. This will provide information on responses following concentric and eccentric high intensity work-outs. Concentric contractions involves the muscle actively shortening and thickening with the two end attachments of the muscle moving closer together. An example of this would be having your thigh fully flexed seated in a leg extension machine and then extending the knee to raise your leg. The knee extensor muscles would be contracting concentrically and shortening as the movement took place. Eccentric contractions involves the muscle lengthening and becoming thinner with the two attachments of the muscle being drawn apart under control. An example for the knee extensors muscles would be lowering the resistance in a controlled fashion back to the starting position.

**Time Commitment**

Familiarisation of the high intensity exercise work-out will take place over a period of one week. This requires only one session. The test will take place over three weeks. You are required in the laboratory on two occasions for a period of approximately two hours.

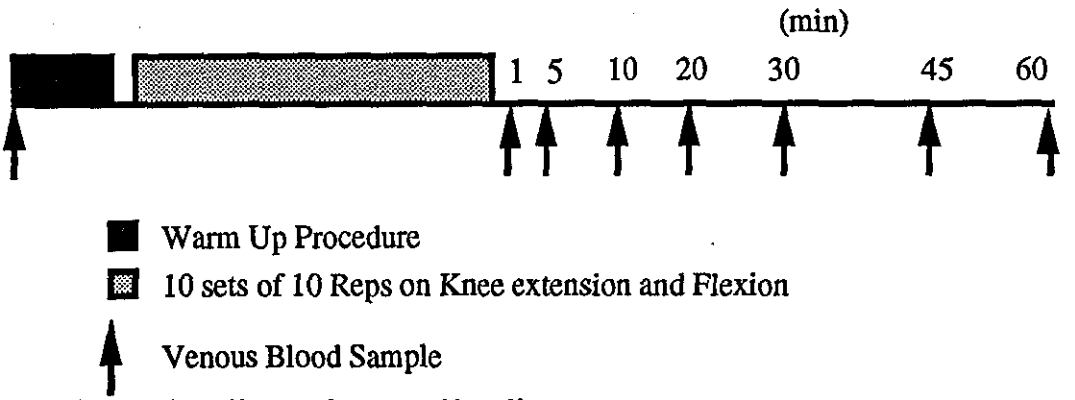
**Testing Procedures:**

Two bouts of exercise will be performed on separate occasions. The exercise test will consist of ten sets of ten repetitions for knee extension and flexion. There will be ninety seconds recovery between sets. You will be requested to avoid ingestion of alcohol or caffeine for 24 hours prior to testing and to avoid strenuous exercise for 48 hours .

**Measurements:**
Standard measurements of height and weight will be taken for each subject. Skinfold thicknesses will be taken at four sites on the body (biceps, triceps, subscapula and suprailiac).

**Exercise Test**
Venous blood samples will be withdrawn from the antecubital vein via an indwelling catheter placed by Dr.Nevill at rest at 1, 5, 10, 20, 30, 45 60 minutes after completion of exercise. The catheter will be inserted 30 minutes prior to exercise commencement. Samples will be analysed for pH, lactate, glucose, growth hormone and insulin.

**Schematic representation of the main test protocol**




**Possible risks, discomforts and/or distress :**
Although the chance of injury during the exercise test is remote, the chance does exist and you should be aware of this. During the familiarisation session you will be gradually introduced to each speed, to determine if you can comfortably be tested without a perceived risk of injury. The caheter will be placed under local anesthetic therefore discomfort will be minimal. Intravenous cannulation carries a small risk of plastic or air embolism but good practice minimises this risk.

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STATEMENT OF INFORMED CONSENT

I have read the above outline of the procedures which are involved in this investigation and I **understand** what will be required of me. I have had the opportunity to ask for further information and for clarification of the demands of each of the procedures. I am aware that I have the **right to withdraw** from the study at any time, with **no obligation to give reasons** for my decision.

I agree to take part in the 'Hormonal and metabolic responses to concentric and eccentric single bouts of high intensity work-outs study'.

SIGNED _____ DATE: _____

WITNESSED BY _____

## III. THIRD STUDY

### SUBJECT INFORMATION PRIOR TO PARTICIPATING IN THE STUDY


**LOUGHBOROUGH UNIVERSITY OF TECHNOLOGY**

DEPARTMENT OF PHYSICAL EDUCATION, SPORTS SCIENCE AND RECREATION MANAGEMENT

| | |
|---|---|
| Supervisors: | Dr. Mary E. Nevill, Dr. Henryk K.A. Lakomy and Mr Rex Hazeldine. |
| Investigators: | Mr Martin Dowson<br>Mr Andi Knight |
| Telephone: | 223474 (Martin)<br>(Andi) |


---

**Study Title:** *The effect of oral creatine supplementation on the metabolic and muscle torque responses from voluntary eccentric and concentric exercise*

**Introduction**

You have probably heard about the new dietary supplement for athletes 'creatine'. It is now very popular in the sports circles, and a number of successful athletes are supplementing their diets with it. Creatine is used by the body in order to synthesise phosphocreatine which is the rapid source of energy for the muscles. Creatine supplementation has been likened to carbohydrate supplementation, with the difference that it provides substrate for quick energy release. Creatine is NOT a doping substance, and many studies have shown that creatine supplementation has NO side affects.

An increased total creatine level in the muscle can:

* Improve *sprint* performance
* Prolong *fatigue* during repeated sprints

**Aims and Outline of the Study**

The aim is to investigate the differences in the responses between eccentric and concentric exercise following creatine supplementation. No previous research has investigated the effect oral creatine supplementation has on eccentric actions. You will be required to perform an exercise test which will last 20 minutes. Peak torque for each repetition will be recorded for the knee flexors and extensors. Following the test, venous blood samples will be taken to measure blood lactate and ammonia. This will provide information on responses following concentric and eccentric high intensity work-outs. Concentric actions involves the muscle actively shortening and thickening with the two end attachments of the muscle moving closer together. An example of this would be having your thigh fully flexed seated in a leg

extension machine and then extending the knee to raise your leg. The knee extensor muscles would be working concentrically and shortening as the movement took place. Eccentric actions involves the muscle lengthening and becoming thinner with the two attachments of the muscle being drawn apart under control. An example for the knee extensors muscles would be lowering the resistance in a controlled fashion back to the starting position.

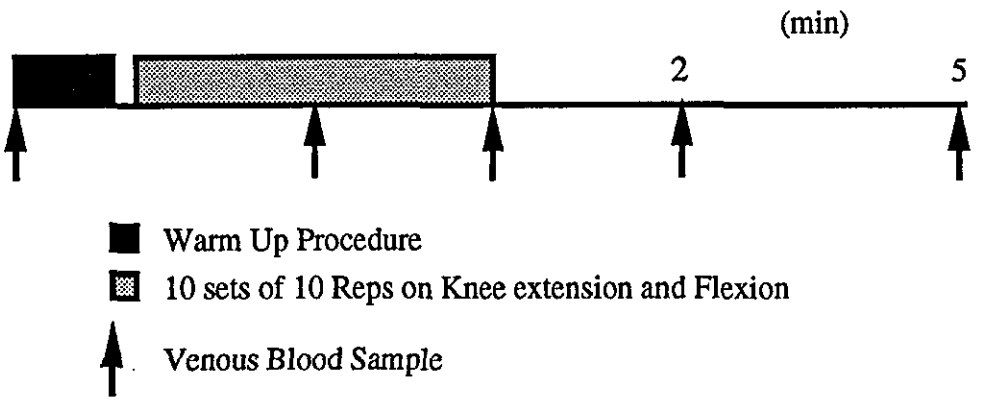
**Time Commitment**
Familiarisation of the high intensity exercise work-out will take place over a period of one week. This requires at least two sessions lasting approximately 20 minutes each. The test will take place over three weeks. You are required in the laboratory on two occasions for a period of approximately one hour each.

**Testing Procedures:**
Two bouts of exercise will be performed on separate occasions. The exercise test will consist of ten sets of ten repetitions for knee extension and flexion. There will be sixty seconds recovery between sets. You will be requested to avoid ingestion of alcohol or caffeine for 24 hours prior to testing and to avoid strenuous exercise for 48 hours .

**Measurements:**
Standard measurements of height and weight will be taken for each subject. Skinfold thicknesses will be taken at four sites on the body (biceps, triceps, subscapula and suprailiac).

**Exercise Test**
Venous blood samples will be withdrawn from the antecubital vein via an indwelling catheter placed by Dr.Nevill at rest, mid-exercise, 0, 2, and 5 minutes after completion of exercise. Samples will be analysed for lactate, ammonia and plasma volume concentration.

Schematic representation of the main test protocol



**Possible risks, discomforts and/or distress :**
Although the chance of injury during the exercise test is remote, the chance does exist and you should be aware of this. During the familiarisation session you will be gradually introduced to each speed, to determine if you can comfortably be tested without a perceived risk of injury. Following the eccentric exercise muscle soreness is commonly experienced for 2-3 days. The catheter will be placed under local anaesthetic therefore discomfort will be minimal. Intravenous cannulation carries a small risk of plastic or air embolism but good practice minimises this risk.

---

**STATEMENT OF INFORMED CONSENT**
I have read the above outline of the procedures which are involved in this investigation and **I understand** what will be required of me. I have had the opportunity to ask for further information and for clarification of the demands of each of the procedures. I am aware that I have the **right to withdraw** from the study at any time, with **no obligation to give reasons** for my decision.

I agree to take part in the 'The effect of oral creatine supplementation on muscle torque and metabolic responses from voluntary eccentric and concentric exercise'.

SIGNED _____ DATE: _____

WITNESSED BY _____

IV.

## STUDY READINESS QUESTIONNAIRE

Name: _____ Age: _____

Address: _____ Telephone (Home):_____
_____ (Office): _____
_____ Date of Birth: _____

Doctor: _____ Address: _____

Please tick either the Yes or No box

**PAST HISTORY**
Have you ever had?

| | YES | NO | | YES | NO |
|---|---|---|---|---|---|
| Rheumatic fever/heart murmur | ___ | ___ | Asthma | ___ | ___ |
| High blood pressure | ___ | ___ | Kidney disease | ___ | ___ |
| Any heart trouble | ___ | ___ | Gout | ___ | ___ |
| Disease of arteries | ___ | ___ | Diabetes | ___ | ___ |
| Varicose veins | ___ | ___ | Epilepsy | ___ | ___ |
| Lung Disease | ___ | ___ | Thyroid disease | ___ | ___ |

**HOSPITALIZATIONS**

Year _____ Reason _____

Year _____ Reason _____

**PRESENT SYMPTOMS REVIEW**
Have you recently had?

| | YES | NO | | YES | NO |
|---|---|---|---|---|---|
| Chest pain/discomfort | ___ | ___ | Cough on exertion | ___ | ___ |
| Shortness of breath | ___ | ___ | Coughing of blood | ___ | ___ |
| Heart palpitations | ___ | ___ | Frequent headaches | ___ | ___ |
| Skipped heart beats | ___ | ___ | Frequent colds | ___ | ___ |
| Orthopeadic problems | ___ | ___ | Recurrent sore throat | ___ | ___ |
| Back pain | ___ | ___ | Dizzy spells | ___ | ___ |

Arthritis/swollen, stiff, and painful joints ___ ___
Unexplained weight loss (> 5 Lbs) ___ ___
Are you preently taking any medications ___ ___

Are there any other medical problems not already indicated? ___ ___

Please state _____

Please state any current musculo-skeletal injuries: _____

List all current prescription and non-prescription medications

| MEDICATION | REASON FOR TAKING | FOR HOW LONG |
|---|---|---|

Signed: _____ Date: _____

V.

## FOOD RECORD DIARY

Confidential

**Subject Name:** _____

**DATE OF TEST 1:** _____

**DATE OF START OF DIET:** _____

**DATE OF TEST 2:** _____

**DATE OF START OF DIET:** _____

Please record everything You eat and drink during the two days prior to each experimental trial. It is important that you eat the same type and quantity of food bfore the second trial as the first. The timing of your last meal/snack before each trial should be the same.
Instructions and an example are given inside.

NB DO NOT EAT BREAKFAST ON THE MORNING OF THE TRIALS.
A 10-12 hr fast is required. Therefore, if your test starts at 8am, you should not eat after 10pm the night before. If the test is at 11am, the last meal or snack should be no later than 1am.

Information about your diet will be treated in confidence and results will be returned to you as soon as possible.

Please use a separate line for each item eaten: write in weight of plate; leave a line between different 'plate entries.

| | | | | Day Month Year | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | Date: | | | | |
| A | B | | C | D | E | F | Office use only | |
| Time | food eaten | | Brand name of | Full description of each item including: | weight | weight | Actual | food |
| am/ pm | home | away | each item (except fresh food) | -how cooked - whether fresh, frozen, dried, canned | served gms | leftovers gms | weight gms | code |
| | | | | | | | | |
| | | | | | | | | |
| | | | | | | | |

VI.

## MUSCLE SORENESS QUESTIONNAIRE

Please fill in 48 hours after the completion of *each* exercise test. Circle the number which best fits the *'soreness'* that is felt 48 hours after the test for each muscle group.

**NAME:** _____ **EXERCISE TEST:** _____
(Concentric or Eccentric)

**Date:** _____

**1. Do you have any muscle soreness in your Hamstring from the exercised leg.**

| No Soreness | | Uncomfortable | | Sore | | Very Sore | | Very Very Sore | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

**2. Do you have any muscle soreness in your Hamstring from the non-exercised leg.**

| No Soreness | | Uncomfortable | | Sore | | Very Sore | | Very Very Sore | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

**3. Do you have any muscle soreness in your Quadriceps from the exercised leg.**

| No Soreness | | Uncomfortable | | Sore | | Very Sore | | Very Very Sore | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

**4. Do you have any muscle soreness in your Quadriceps from the non-exercised leg.**

| No Soreness | | Uncomfortable | | Sore | | Very Sore | | Very Very Sore | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

**5. Do you have any muscle soreness in your Inside Thigh from the exercised leg.**

| No Soreness | | Uncomfortable | | Sore | | Very Sore | | Very Very Sore | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

**6. Do you have any muscle soreness in your Inside Thigh from the exercised leg.**

| No Soreness | | Uncomfortable | | Sore | | Very Sore | | Very Very Sore | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

**7. Do you have any muscle soreness in your Calf from the exercised leg.**

| No Soreness | | Uncomfortable | | Sore | | Very Sore | | Very Very Sore | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

**8. Do you have any muscle soreness in your Calf from the non-exercised leg.**

| No Soreness | | Uncomfortable | | Sore | | Very Sore | | Very Very Sore | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

**9. Do you have any muscle soreness in your Back.**

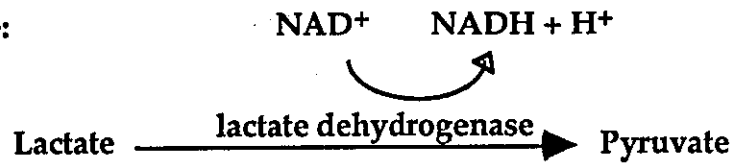
| No Soreness | | Uncomfortable | | Sore | | Very Sore | | Very Very Sore | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

## APPENDIX B

## BLOOD METABOLITE ASSAYS

**I. Fluorimetric assay for the determination of blood lactate (modified from Maughan, 1982)**

**Principle:**




**Reagents:**

| | |
|---|---|
| Buffer: | Hydrazine 1.1 mol·l$^{-1}$, pH 9.0 with 1 mmol·l$^{-1}$ EDTA·$Na_2$ |
| Cofactor: | NAD |
| Enzyme: | lactate dehydrogenase (LDH) 5500 U·ml$^{-1}$ (undiluted) |
| Standard: | L-Lactate 1 mol·l$^{-1}$ (stock solution) |
| Diluent: | 0.07 mol·l$^{-1}$ HCl |

Stock standards were prepared before each study and stored at -20 °C:

| | | | | | | |
|---|---|---|---|---|---|---|
| L-Lactate 1 mol.l$^{-1}$ (µl) | 0 | 5 | 20 | 50 | 100 | 150 |
| 0.4 mol.l$^{-1}$ perchloric acid (ml) | 10 | 9.995 | 9.98 | 9.95 | 9.90 | 9.85 |
| Lactate concentration (mmol.l$^{-1}$) | 0 | 0.5 | 2 | 5 | 10 | 15 |

Working standards were prepared by diluting 20 µl of each of the above standards into 200 µl of 0.4 mol·l$^{-1}$ (~2.5%) perchloric acid.

**Reaction mixture (final concentration):**

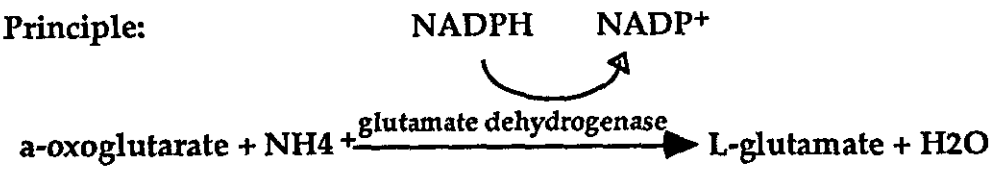
Buffer 1 ml
NAD 2 mg (2.98 mmol·l$^{-1}$)
LDH 10µl (54.46 U·ml$^{-1}$)

**Procedure:**

1. 200 µl of reaction mixture was added to 20 µl aliquots of duplicate samples, perchloric acid blanks and standards (mix well).
2. After 30 min incubation at room temperature, 1 ml of diluent (0.07 mol.l-1 HCl) was added, the contents were mixed and fluorescence was read. Lactate concentration was calculated from the standard curve.

## II. Spectrophotometric determination of plasma ammonia (340 nm)

**Principle:**




**Reagents:** The Boehringer Mannheim MPR 1 Ammonia kit was used (Cat. No. 125 857)

**Initial concentrations of solutions:**

Reagent solution: NADPH 0.10 mmol.l-1
triethanolamine buffer 0.15 mol.l-1, pH 8.6 a-oxoglutarate 15 mmol.l-1
ADP 1.5 mmol.l-1

Enzyme: Glutamate dehydrogenase (GLDH) ≥ 755 U.ml-1

**Procedure:**

1. 500µl of the reagent solution was added to 100 µl aliquots of samples or standards* in a 1 ml disposable cuvette (light path: 1 cm). 2-4 reagent blanks were used for every assay (600 µl of the reagent solution in each cuvette).
2. The contents of each cuvette were mixed well upon addition of the reagent solution and were incubated at room temperature for 10 min.
3. After the 10 min of incubation the initial absorbance ($A_1$) of samples, blanks and standards was read at 340 nm (N.B. absorbance decrease).
4. After the absorbance was read, 4 µl of enzyme (GLDH) was added to each cuvette with a positive displacement pipette, the contents of each cuvette were mixed well and incubated for 10 min.
5. After the 10 min of incubation the absorbance of samples, blanks and standards was read again ($A_2$).
6. Steps 4 and 5 were repeated, and a final absorbance of samples, blanks and standards was read ($A_3$).

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* To control accuracy and precision of ammonia determinations, a set of 3 standards (58.8 µmol·$^{-1}$, 117.6 µmol·$^{-1}$ and 176.5 µmol·$^{-1}$) was used (Preciset® Ammonia, Cat. No. 166 570)

Cuvettes were sealed during incubations with a plastic cap.
If the ammonia concentration exceeded 412 $\mu mol \cdot^{-1}$, plasma was diluted (1:2) with double distilled water and assay was repeated.
The concentration of ammonia in the sample was calculated using the extinction coefficient for NADPH, as follows:
$(A_1-A_2) - (A_2-A_3) = \Delta A_{BLANK}$ or $\Delta A_{SAMPLE/STANDARD}-\Delta A_{BLANK}$)
Standards were used only to check the assay.

I.

# APPENDIX C
# ISOKINETIC CALIBRATION

Both the Cybex 6000 and Kin-Com AP isokinetic dynamometers had Diagnostics and Calibration programs in their software.

The Diagnostic portion checks out the major functions of the unit, reports any faults and provide suggestions where the problem could be arising from.

The Calibration programs takes the user through a step by step procedure. The programs were designed to be user friendly. The prompts displayed for the Kin-Com AP are as follows for the force and velocity calibration.

## FORCE CALIBRATION

1. Rotate the actuator arm to a true horizontal position pointing away from the unit. Rotate the mechanical stop to a position under the actuator arm that will support the arm when a weight is placed on the load cell.
2. Hang known weight (we used 20 kg) on the load cell.
3. Adjust potentiometer for a displayed force value equal to the weight you have on the load cell (in our case, 196 Newtons). An error of 2% is acceptable.
4. Remove the weight from the Loadcell Assembly.
5. Remove the Loadcell Assembly from the Motor Shaft. Press (ESC).

## VELOCITY CALIBRATION

1. Display Velocity: should be indicating a velocity of 0+1. If incorrect, adjust potentiometer R27 for a correct indication.
2. Turn both Motor (F1) and Servo (F2) to ON, then press (F3) and enter a speed of 60. The motor shaft will start rotating in order to find the maximum range of motion. This range of motion will depend on where the mechanical stops are placed. For maximum range of motion, locate the two tops close together.
3. Adjust potentiometer (R18, R49, R52) for a displayed velocity of 60 degrees per second.
4. Press (F1) then (F2). The system should stop. Press (F3) and enter a speed of 250.
5. Verify that the displayed velocity reads 250 + 4 deg/s. If incorrect, repeat procedure starting from 2. If correct press (ESC).

## II. SUBJECT TEST RE-TEST RELIABILITY USING THE ELCTRONIC TIMING EQUIPMENT

### *STANDARDISED SPRINT TESTING WARM UP*

1. Jog 4 lengths of the hall [4 x 40m]
2. Stretch calf and quadriceps muscle groups
3. For 3 length of the hall 'stride out'
4. Stretch hamstring and hip flexor muscle groups
5. For 2 lengths of the hall 'stride out'
6. Stretch lower back muscle groups
7. Two reaction starts
8. Stretch of subjects choice

**0-15 m Sprint Times**

n=13

| | Mean | Std. dev. |
|---|---|---|
| Test 1 | 2.34 | 0.11 |
| Test 2 | 2.34 | 0.12 |
| Test 3 | 2.35 | 0.10 |

One factor ANOVA - repeated measures : treatments p value 0.467

Reliability estimates for: all treatments: 0.987
single treatment: 0.962

**30-35 m Sprint Times**

n=13

| | Mean | Std. dev. |
|---|---|---|
| Test 1 | 0.568 | 0.033 |
| Test 2 | 0.571 | 0.031 |
| Test 3 | 0.572 | 0.035 |

One factor ANOVA - repeated measures : treatments p value 0.4214

Reliability estimates for: all treatments: 0.974
single treatment: 0.927

## III. CALCULATIONS

*** To calulate the Ammonia values ($\mu mol \cdot l^{-1}$)**

$$(A_1 - A_2) - (A_2 - A_3) = \Delta AR_B \text{ or } \Delta A_{SAMPLE}$$

$$\text{Concentration} = 863 \times (\Delta A_{SAMPLE} - \Delta AR_B)\ \mu mol \cdot l^{-1}$$

*Key:*

$A_1$: First reading on the spectrophotometer
A2: Second reading
A3: Third reading
RB: Reaction of the blank

*** To calculate haemoglobin concentration ($g \cdot 100ml^{-1}$)**

$$\text{Concentration} = (37.2 \times A) + 0.06$$

*Key:*

A: Absorbance

*** To calculate body fat percentage**

$$X = \text{sum of the four skinfolds (mm)}$$

$$\text{Body density} = 1.1631 - (0.0632 \times \log X)$$

$$\textit{Body fat percentage} = ((4.950/\text{body density}) - 4.5) \times 100$$

*** To calulate lean body mass**

$$\text{Absolute body fat} = (\text{Body mass} - \text{Body fat percentage}) / 100$$

$$\textit{Lean body mass} = \text{Body mass} - \text{Absolute body fat}$$

*** To calculate plasma volume change**

$$A = \text{Hb-pre} \times (100 - \text{Hct-post})$$
$$B = \text{Hb-post} \times (199 - \text{Hct-pre})$$
$$PVC = 100 \times (A/B) - 100$$

Hb-pre - resting sample (haemoglobin)
Hb-post - mid exercise or post test sample (haemoglobin)
Hct-pre - Resting sample (haematocrit)
Hct-post-mid exercise or post test sample (haematocrit)