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Neuromuscular functioning in older women and their adaptations to resistance power training

by

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CONTENTS **Page**

ABSTRACT

The purpose of this thesis was to investigate some of the neural mechanisms underlying the decline in older women's muscle power, and to determine the neural adaptations to a resistance training program that aimed to produce optimal gains in their muscle power.

The initial study demonstrated that motion analysis can be utilised as a suitable alternative method to measure the change in flywheel velocity, using cycle ergometry, for determining short-term maximal peak power and its determinants in older women.

To the author's knowledge, for the first time during fast cyclic movements linear array surface electromyography showed that the decline in older women's short-term maximal peak power, in comparison to younger women, was associated with an overall lower neural activity, from all loads tested. The lower signal amplitude, which indicates a decreased neural drive, can be regarded as a contributory factor in the decline of muscle power with advancing age. Conversely, neural activation patterns remained the same with age.

In the third study, an overall lower neural activity accompanied the decline in instantaneous peak power during single leg extensions, which reflect the functional gesture of rising out a chair, but only against loads requiring a high generation of torque in older women. Whereas, the detrimental changes in muscle twitch characteristics suggest that more peripheral mechanisms within the muscle are likely to explain the decline in explosive power requiring high movement velocities.

Finally, two modalities of cycle resistance training, with both uniquely allowing maximal power output but with significantly different velocities, showed that adopting a high training load (low movement velocity) produced significantly greater gains in explosive power than adopting a low training load (high movement velocity). Therefore, resistance training at maximal power can not be regarded as the stimulus for optimal gains in older women's muscle power. Improvements in muscle power were accompanied by significant increases in the ability to generate torque, thus adopting high resistance loads may be more beneficial for improving muscle power in older women.

LIST OF FIGURES

LIST OF TABLES

CHAPTER 1

GENERAL INTRODUCTION

The ageing process inevitably leads to individuals over 60 years of age, especially women, experiencing a significant decline in muscular functioning to the extent that it can fall below the 'critical' level required to perform everyday functional tasks unaided (Skelton *et al.*, 1994; Lindle *et al.*, 1997; Daily and Spinks 2000; Foldvari *et al.*, 2000; Ploutz-Snyder *et al.*, 2002; Tanaka and Seals 2003; Dean *et al.*, 2004; Kuh *et al.*, 2005). The functional tasks in question include walking, rising from a seated position and climbing stairs (Foldvari *et al.*, 2000; Kuh *et al.*, 2005).

The age-related decline in the maximal force (strength) generated from a concentric muscle contraction has been well documented for over 100 years, and is considered to be the main contributing factor for the reduction in functional ability (Larsson *et al.*, 1979; Narici *et al.*, 1991; Brooks and Faulkner 1994; Lindle *et al.*, 1997; Roos *et al.*, 1999; Signorile *et al.*, 2002; Docherty 2003; Macaluso and De Vito 2004). However, muscle power, which is the product of muscle force generated and movement velocity, has been found to decline at a greater rate than muscle strength per se with age (Skelton *et al.*, 1994; Bassey *et al.*, 1992; Izquierdo *et al.*, 1999; Pearson *et al.*, 2002; Lauretani *et al.*, 2003). Furthermore, as muscle power has a greater association with the performance of dynamic movements, which occur more often in daily life, the decline in muscle power will thus have a greater detrimental impact on functional ability than the loss of maximal strength per se (Bassey *et al.*, 1992; Evans 2000; Foldvari *et al.*, 2000; Bean *et al.*, 2002; Lauretani *et al.*, 2003; Cuoco *et al.*, 2004; Dean *et al.*, 2004; Herman *et al.*, 2005). Therefore, muscle power may be regarded as a better predictor of functional disability than muscle strength per se (Bassey *et al.*, 1992; Evans 2000; Foldvari *et al.*, 2000; Cuoco *et al.*, 2004; Macaluso and De Vito 2004; Herman *et al.*, 2005; McNeil *et al.*, 2007).

The reduction in muscular strength and power with advancing age is significantly correlated with an increased likelihood of disability, risk of falls and incidence of hip fractures due to postural instability, and ultimately the loss of functional independence and institutionalisation (Narici *et al.*, 1991; Skelton *et al.*, 1994; Foldvari *et al.*, 2000; Ploutz-Snyder *et al.*, 2002; Signorile *et al.*, 2002; Vandervoort 2002; Brooks 2003; Hortobagyi *et al.*, 2003; Dean *et al.*, 2004). All of which have severe detrimental consequences on the older individual's quality of life

(ACSM 1998; Daily and Spinks 2000; Vandervoort 2002; Narici *et al*., 2003). Nonetheless, exercise interventions based on resistance training methods have been shown to successfully reverse and attenuate the effects of ageing on muscle strength, power and functional ability in older subjects (Frontera *et al*., 1988; Fiatarone *et al*., 1990; Miszko *et al*., 2003; Macaluso *et al*., 2003; Latham *et al*., 2004; de Vos *et al*., 2005; Hazell *et al*., 2007; Henwood *et al*., 2008). Crucially, research shows that ageing muscle still maintains its plasticity, i.e. its ability to adapt structurally and functionally to 'resistance type' exercise (Frontera *et al*., 1988; Fiatarone *et al*., 1990; Brown *et al*., 1990; Trappe *et al*., 2000; Ferri *et al*., 2003; Macaluso and De Vito 2004; Narici *et al*., 2004; 2005; Onambélé *et al*., 2008).

More recently, exercise research has focused on improving muscle power due to traditional heavy resistance training programs producing larger gains in maximal strength than peak power, and functional ability having a significantly greater correlation with muscle power than strength (Evans 2000; Fielding *et al*., 2002; Macaluso *et al*., 2003; Macaluso and De Vito 2004; Hazell *et al*., 2007). However, 'power based' resistance training studies have been proved inconclusive as to the best method and mode of training to improve muscle power and functional ability optimally in older people, and thus highlights that further investigations are required to develop effective exercise regimes (Evans 2000; Foldvari *et al*., 2000; Signorile *et al*., 2002; Vandervoort 2002; Docherty 2003; Macaluso *et al*., 2003; Caserotti *et al*., 2005; de Vos *et al*., 2005; Kuh *et al*., 2005; Hazell *et al*., 2007). In order to develop effective exercise regimes the physiological mechanisms responsible for the decline in functional ability, but more specifically muscle power, with ageing require further investigation (ACSM 1998; Foldvari *et al*., 2000; Höök *et al*., 2001; Vandervoort 2002; Doherty 2003; Macaluso *et al*., 2003; Macaluso and De Vito 2004).

The physiological mechanisms responsible for the decline in muscle strength and power with advancing age include muscular, neural and tendinous factors (Frontera *et al*., 2000a; 2008; Doherty 2003; D'Antona *et al*., 2003; Narici *et al*., 2004; 2005; Maganaris *et al*., 2004; Reeves *et al*., 2004; Thom *et al*., 2005), all of which will be extensively investigated and discussed in more detail in the next chapter of this thesis. Most notability is the decline in muscle mass with advancing age termed sarcopenia, which is regarded as the main contributing factor for a

decline in muscle strength (Narici *et al.*, 1991; 2005; Lexell 1993; Janssen *et al.*, 2000; Roubenoff and Hughes 2000; Trappe *et al.*, 2001; Frontera *et al.*, 1988; 2008). Furthermore, it has been demonstrated that muscle quality is affected, i.e. a muscle produces less force than expected for its size (Lynch *et al.*, 1999; Narici *et al.*, 2003; Reeves *et al.*, 2004; Thom *et al.*, 2005; Narici and Maganaris 2006). Several physiological factors have been identified as contributing to a decline in muscle quality including a selective atrophy and reduced number of type II muscle fibres, which produce a higher degree of force and contract at a faster rate than type I fibres, leading to muscles appearing to posses a greater proportion of type I fibres (Lexel 1993; D'Antona *et al.*, 2003). In addition, muscle architectural changes such as a change in fibre pennation angle and fibre length ultimately causes muscle force and contraction speed to decline (Klein *et al.*, 2001; Narici *et al.*, 2003; 2005; Morse *et al.*, 2005). Hence, affecting not only muscle strength per CSA but also the two determinants of muscle power (force and velocity).

Although the detrimental impact of ageing on neural drive, which refers to motor unit behaviour, has been established in relation to muscle strength (Roos *et al.*, 1997; Connelly *et al.*, 1999; Vandervoort 2002; Doherty 2003; Klass *et al.*, 2008), the effects of ageing on motor unit behaviour during the generation of muscle power are however less well understood. This can be attributed to limitations of surface electromyography (sEMG) ability to detect motor unit activity during dynamic movements that are crucial for the development of muscle power (Farina *et al.*, 2004; 2004a; McNeil *et al.*, 2005; 2007; Klass *et al.*, 2008). Advancements in linear array sEMG, devised by Farina *et al.* (2004), now provide a method of studying MU behaviour during dynamic explosive movements that represent activities of daily living through estimating the conduction velocity of a MU action potential (or muscle fibre conduction velocity [MFCV]) (Pozzo *et al.*, 2004). As MFCV is regarded as a size principle parameter, which increases in accordance with recruitment of higher threshold MUs, thus changes in central neural control strategies governing muscular contractions causing movement can be inferred during the generation of muscle power (Farina *et al.*, 2004; 2004b; Pozzo *et al.*, 2004). Therefore, linear array sEMG provides a measurement technique to improve our understanding of the central neural mechanisms responsible for not only a decline in

muscle power but also for improvements in muscular power after resistance training in the older population (Farina *et al.*, 2004; 2004b).

The thesis now continues with a review of the literature, initially examining the techniques utilised in the measurement of muscle strength and power. This is followed by a review on the effects of ageing on muscle strength and power and the physiological mechanisms leading to the decline in muscle strength and muscle power. Finally, the thesis will evaluate the effects of resistance training on strength and power, leading to a novel hypothesis for improving muscle power in older women.

CHAPTER 2

REVIEW OF LITERATURE

TECHNIQUES FOR MEASURING MUSCLE STRENGTH AND POWER

Muscle Strength

In order to discuss a review of the literature on the effects of ageing on muscle strength and power the different measurement techniques have to be elucidated.

The techniques used to measure muscle strength involve either performing a concentric or isometric muscular contraction. These types of muscular contractions relate to the amount of force a muscle can generate, as described by the muscle's force-velocity (F-V) relationship (Hill 1938; Wickiewicz *et al*., 1984; Jones and Round 1990). The muscle's F-V relationship (Figure 1.1) describes how the amount of force generated by an isolated muscle is dependent upon the velocity of muscle shortening, with the amount of muscle force produced declining in a hyperbolic path along with the increase in muscle shortening velocity (Hill 1938; Wickiewicz *et al*., 1984; Jones and Round 1990). The force generated eventually approaching zero as the muscle reaches its maximum shortening velocity (V_{max}). Typically, in relation to functional ability, the maximum force (torque) generated by a muscle is measured when the muscular torque is equal to the opposing resistance or load during a maximal voluntary effort, causing the movement around the joint to be static with no change in muscle length (isometric contraction [P_o]) (Jones and Round 1990; McArdle *et al*., 1996; Doherty 2003). Hence, maximal muscle strength is assessed during performance of a maximal isometric voluntary contraction (MVC). Furthermore, the amount of torque a muscle can generate is also influenced by its length-tension relationship, with maximum torque generated occurring at an optimal length (Jones and Round 1990; Lieber and Friden 2000; Maganaris 2001; Lanza *et al*., 2003). With regards to the literature review, since the majority of the studies measure quadriceps' MVC using a similar knee angle of approximately 90^o the influence of the length-tension relationship on strength in older people may be discounted.

Numerous studies measured muscle strength as the maximum force produced during dynamic contractions, i.e. the heaviest weight that can be lifted through a

predetermined range of motion (1 repetition maximum [1RM]). The measurement of an individual's muscle force from performing a 1RM is typically lower than that recorded from their MVC due to the different measurement techniques involving different portions of the muscle's F-V relationship (Bassey 1997; Izquierdo *et al.*, 1999; Lanza *et al*., 2003; Doherty 2003; Macaluso and De Vito 2004).

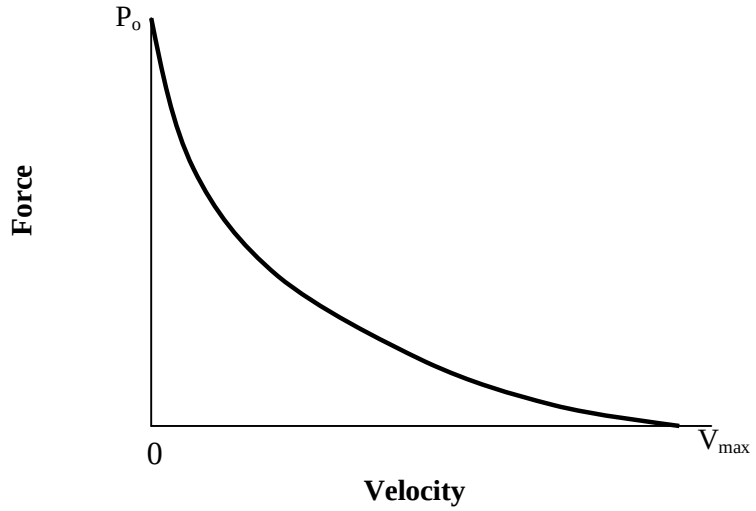

Figure 1.1 The Force-Velocity relationship (isolated muscle): P_o represents maximal isometric voluntary contraction force and V_{max} represents maximal shortening velocity of muscle (AV Hill (1938) cited from Jones and Round (1990) p26.

Another method of measuring strength (and power) refers to isokinetic torque, which utilises a dynamometer that measures the amount of torque (rotational force) produced from a specific limb action at a constant predetermined velocity of muscle shortening (Bassey 1997; Lieber and Friden 2000; Lanza *et al*., 2003; Macaluso and De Vito 2004; Frontera *et al*., 2008). The measurement of isokinetic muscle torque from the lower limbs is commonly assessed using a knee extension [KE] (Bassey 1997; Lanza *et al*., 2003; Trappe *et al*., 2003; Macaluso and De Vito 2004).

Muscle Power

Muscle power can be defined as the rate of performing mechanical work, which is the product of muscle force and shortening velocity [Power = Force x Velocity] (Meriam 1978 cited from Maud and Foster 1995; Bassey 1997; Signorile *et al*., 2002; Kawamori and Haff 2004). For this thesis, muscle power will refer to both instantaneous muscle power, produced from a single explosive contraction lasting a fraction of a second, and short-term muscle power, produced from repeated explosive muscular contractions lasting approximately 6 seconds or less performed using cycle ergometry, or where otherwise stated (Sargeant *et al*., 1981; Bassey *et al*., 1992; Martin *et al*., 1997; Macaluso and De Vito 2003; Pearson *et al*., 2006).

As discussed previously, the amount of force a muscle generates is dependent upon the velocity of shortening (Figure 1.1) and as mechanical power output is proportional to force and velocity therefore the muscle's F-V relationship can be used to determine the mechanical power output of a muscle. Mechanical power output is found to form its own distinct parabolic relationship against velocity or force (Jones and Round 1990; Newton and Kraemer 1994; Macaluso and De Vito 2004). However, for this review muscle power will be shown against velocity, Power-Velocity (P-V) relationship, as shown in Figure 1.2.

The muscle power calculated from the F-V relationship results in maximum power output occurring at an optimal velocity and force, V_{opt} and F_{opt} respectively. These values are estimated to be approximately 30% of P_o and V_{max} from a single isolated muscle fibre. However, during *in vivo* measurements of V_{opt} and F_{opt} at maximal power may differ from *in vitro* measurements due to involvement of numerous muscle-tendon units, which move around one or more articulated joints (Wickiewicz *et al*., 1984; Jones and Round 1990; Newton and Kraemer 1994; Izquierdo *et al*., 1999; Macaluso and De Vito 2004). For example, Izquierdo *et al*. (1999) study showed that the optimal loads for the instantaneous maximal power of the upper limbs is 30-45% 1RM and the lower limbs 60-70% 1RM, using an instrumented traditional weight training barbell apparatus that only allowed the barbell to move in a vertical direction.

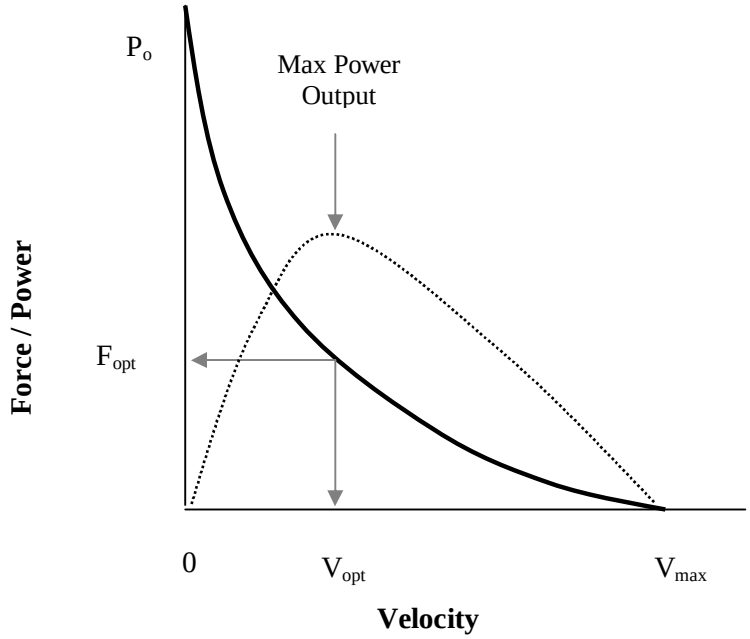

Figure 1.2 The Force / Power –Velocity relationships using an isolated muscle. The solid line (—) represents the Force-Velocity relationship and the dotted line (····) represents the Power-Velocity relationship, calculated from the Force-Velocity curve (Power = Force x Velocity). The arrows represent the optimal force (F_{opt}) and velocity (V_{opt}) on the F-V curve where maximum power output occurs. AV Hill (1938) cited from Jones and Round (1990).

The findings that the optimal load for maximal peak power from the lower limbs is approximately 60-70% of the individuals strength has also been confirmed using more accurate methods of measuring peak power, i.e. Thomas *et al.* (1996) using a pneumatic bilateral LP system and Macaluso and De Vito (2003) using isotonic chair dynamometry, methods that will be discusses later. The different optimal loads for maximal peak power between the lower and upper limbs are possibly attributed to the fact that the F-V relationship during performance of dynamic movements is also influenced by the differences in muscle mass (volume), muscle architecture and predominant fibre types between the muscle groups (Wickiewicz *et al.*, 1984; Martin *et al.*, 1997; Lieber and Friden 2000; Driss *et al.*, 2002; Cronin and Sleivert 2005).

There are fewer studies assessing the effects of aging on muscle power than strength due to the technical difficulties and costing (Hruda *et al.*, 2003; Laurentani

et al., 2003; Macaluso and De Vito 2004; Herman *et al*., 2005). The measurement of muscle power has evolved from a timed step test ascent, developed by Margaria *et al*. (1966), to studies measuring instantaneous muscle power utilising a force platform during performance of a vertical squat jump (Bosco and Komi 1980; De Vito *et al.* 1998). In these studies the vertical ground reaction force produced from the explosive muscular actions are measured over a short instance of time, which are used to calculate muscle power by direct dynamics (refer to De Vito *et al.*, 1998). The measurement of muscle power on a force platform is described as instantaneous or peak explosive power but not as maximal power (De Vito *et al.*, 1998; Macaluso and De Vito 2003; 2004). This is due to the involvement of body weight, regarded as a 'fixed inertia', ultimately affecting performance by not enabling the muscles to reach their V_{opt} required for maximal power, as described by the F-V and P-V relationships. Hence, the power output generated is regarded as a peak (Macaluso and De Vito 2003; 2004). The disadvantage of this method when comparing older and younger populations is that older people tend to be weaker and thus have to work at a higher portion of their muscle's F-V relationship in order to overcome the same fixed inertia (Harridge and Young 1998 cited in Ferri *et al.*, 2003; Hortobagyi *et al*., 2003; Macaluso and De Vito 2003; Narici *et al*., 2003). The consequence of older people's muscles working at a different portion of the F-V relationship will only exacerbate the differences in the generation of muscle power in comparison to younger people (Ferri *et al*., 2003; Hortobagyi *et al*., 2003; Macaluso and De Vito 2003; 2004). These findings are applicable to any assessment of instantaneous muscular power against a single 'fixed inertia', such as the Nottingham Power Rig (NPR: devised by Bassey and Short 1990) that involves performing a single explosive leg thrust action from a seated position (Sargeant *et al.*, 1981; Macaluso and De Vito 2003).

To overcome the limitations of the NPR, Pearson *et al*. (2004) modified the NPR test through using a series of different inertial loads to allow the identification of the load that produces V_{opt}, and thus determine maximum power from a single explosive muscular action. Other methods that can be adopted to measure unilateral or bilateral instantaneous maximal power include instrumented weightlifting apparatus (Izquierdo *et al*., 1999), discussed previously, chair dynamometry in

isokinetic (see muscle strength) or isotonic mode (Macaluso and De Vito 2003; Signorile *et al*., 2002; Petrella *et al*., 2005) or pneumatic resistance machines (Thomas *et al*., 1996; Fielding *et al*., 2002; de Vos *et al*., 2005). With regards to isotonic dynamometry, Macaluso and De Vito (2003) devised an optimisation procedure, which involves performing a series of unilateral explosive limb movements against a number of predetermined loads varying in resistance relative to individual's MVC, in order to determine maximum peak power. This methodology also enables the determinants of maximum peak power to be measured, the optimal torque (F_{opt}) and corresponding V_{opt}, which are crucial parameters when investigating the effects of ageing on muscle power and functional ability (Macaluso and De Vito 2003; 2004; Petrella *et al*., 2005; Pearson *et al*., 2006). An optimisation procedure can also be conducted, using the individual's 1RM, on a pneumatic resistance machine to produce a valid measure of instantaneous unilateral and bilateral LP and KE maximal peak power (Thomas *et al*., 1996; Fielding *et al*., 2002). One advantage of this system, like chair dynamometry, is that there is no 'fixed' inertia to overcome at the start of the contraction, as the resistance is a pneumatic piston that can be adjusted in accordance with the individuals strength. Muscle power is calculated through computer software sampling the system pressure at 400Hz over a predetermined range of movement (Fielding *et al*., 2002).

Short-term muscle power can be determined using isokinetic cycle ergometry, which measures the force production from the pedals during repeated explosive pedalling at a pre-determined constant velocity (Sargeant *et al*., 1981). More commonly, friction-loaded cycle ergometry has been adapted to measure short-term maximum muscle power (Arsac *et al*., 1996; Martin *et al*., 1997; MacIntosh *et al*., 2000; Morin and Belli, 2004), which can be attributed to both the financial cost of the isokinetic equipment and isokinetic muscular actions being regarded as 'unnatural' with respect to activities of daily living (Martin *et al*., 1997; Izquierdo *et al*., 1999; Macaluso and De Vito 2004). In order to accurately measure power output from friction-loaded cycle ergometry not only does the flywheel velocity, due to pedaling, and the load placed on the flywheel need to be measured, but also the additional torque required to overcome the flywheel's inertia during its continual acceleration (Arsac *et al*., 1996; Lakomy 1986; Martin *et al*., 1997). The

measurement of the 'additional torque' required to overcome the flywheel inertia has been established using recommendations devised by Lakomy (1986), which have been found to produce a valid and reliable method of assessing muscle power using friction-loaded cycle ergometry (Arsac *et al*., 1996; Martin *et al*., 1997; Watson *et al*., 2007). It has also been demonstrated that short-term supra-maximal cycling results in a higher mechanical power output than that produced from single explosive muscular actions (Sargeant *et al*., 1981; Arsac *et al*., 1996; MacIntosh *et al*., 2000; Pearson *et al*., 2004; 2006). This is attributed to the torque generated from the repeated explosive muscular limb actions during each pedal stroke having a cumulative effect on the flywheel's acceleration, which is used to measure mechanical power output (Martin *et al*., 1997; MacIntosh *et al*., 2000; Pearson *et al*., 2004).

In recent times, Pearson *et al*. (2004) have demonstrated that short-term maximum peak powers generated against different inertial loads were not significantly different between each other, which has been highlighted previously by Martin *et al*. (1997) using friction loaded cycle ergometry. Pearson and co-workers explain their results through the muscles' ability to travel along their F-V relationship, in accordance with repeated explosive muscular actions accelerating the flywheel, until an optimal velocity is reached for peak power. However, the authors add that maximum peak power is only achieved if individuals can generate the required torque and are provided with enough time in order to accelerate the flywheel until the muscles reach an optimal velocity. Therefore, short-term efforts on a friction-loaded cycle ergometer, involving the muscles performing along their F-V relationship, appear to replicate the physical conditions the muscles would experience during the performance of numerous activities of daily living, such as climbing stairs. Hence, short-term cycle ergometry may be regarded as a suitable assessment of muscle power in older people but also in relation to their functional ability (Farina *et al*., 2004; Sargeant *et al*., 1981; Pearson *et al*., 2006).

EFFECTS OF AGEING ON MUSCLE STRENGTH AND POWER

Muscle Strength

Maximal muscle strength in humans is reported to decline slowly by approximately 10% from the 3rd decade until the 6th decade in life. Thereafter, the rate of decline significantly accelerates to 1.5% per year. This may even increase to 3% per year or greater after the 9th decade (Larsson *et al.*, 1979; Vandervoort and McComas 1986; Narici *et al.*, 1991; Skelton *et al.*, 1994; Lynch *et al.*, 1999; Docherty 2003; Lauretani *et al.*, 2003; Short *et al.*, 2005). However, the rate of decline in maximal muscle strength with ageing is still regarded as ambiguous possibly due to studies comparing muscle strength using a cross sectional or longitudinal research design (Frontera *et al.*, 1991; 2000; 2008; Macaluso *et al.*, 2002; Doherty 2003; Lanza *et al.*, 2003; Lauretani *et al.*, 2003).

Cross sectional designed studies, assessing older versus younger populations at a given time, have observed that older adults in their 7th and 8th decade possess a lower limb MVC, from the quadriceps, approximately 20-40% of that of younger adults in their 3rd decade (Larsson *et al.*, 1979; Skelton *et al.*, 1994; Macaluso *et al.*, 2002; Lanza *et al.*, 2003; Doherty 2003; Lauretani *et al.*, 2003; Dean *et al.*, 2004; Morse *et al.*, 2005; Short *et al.*, 2005). Conversely, longitudinal designed studies reassess the same participant after a specific period of time has elapsed to determine the effects of ageing with studies being conducted over 4-25 years (Doherty 2003). The results from longitudinal studies indicate that cross sectional studies often underestimate the decline in quadriceps' strength with ageing (Frontera *et al.*, 2000; 2008). For instance, Frontera e*t al.* (2000) observed a 30% reduction in isokinetic KE torque after a 12-year time period using male subjects with an average initial age of approximately 65 years. Whereas, their previous cross sectional study, Frontera *et al.* (1991), showed KE torque was lower by 20-22%, using similar isokinetic velocities, when comparing men aged between 45 and 78 years.

The major limitation of cross sectional design is that it makes the assumption that the strength of the older population was similar to that of the younger population when they where the same age. Whereas, older adults would have experienced

different levels of physical activity, nutritional habits, and quality of health care in comparison to today's younger population (Macaluso and De Vito 2004; Frontera *et al*., 2008). Nonetheless, not all longitudinal studies indicate that muscle strength declines with ageing. Kallman *et al*. (1990) showed that 15% of their older subjects, average initial age 60 years, experienced no change in grip strength over a 9 years period. Similar results where also highlighted by Greig *et al*. (1993) utilising subjects with an average age of 81 years over an 8 year period analysing strength from the quadriceps. These results may be explained through participants maintaining their level of daily activity or even due to assessing strength at different times of year which can influence physical activity levels (Greig *et al*., 1993; Doherty 2003; Macaluso and De Vito 2004; Frontera *et al*., 2008). Doherty (2003) emphasizes that many studies show a significant 'inter subject variability', which makes the rate of decline in strength within the ageing population difficult to determine. The lack of homogeneous findings within the older population in relation to the decline in muscle strength can be attributed to several factors. These include the interacting factors that influence the individual's rate of ageing, e.g. genetics, prevalence of disease, calorific intake, and physical activity levels, thus explaining how older subjects of the same age experience various degrees of decline in muscular and functional capacity (ACSM 1998; Macaluso and De Vito 2004). Furthermore, the conflicting results can also be attributed to studies adopting different methods of measuring strength, muscle groups assessed, sexes recruited, and age of populations recruited (Doherty 2003; Macaluso and De Vito 2004). Importantly, Short and Nair (1999) add that the rate of ageing within the whole population may be even greater than that highlighted, as only healthy older participants are recruited in these studies.

Further evidence that suggests the loss of strength is an inevitable part of the ageing process may be provided by comparative studies using master athletes or trained older people, as the influence of physical inactivity may be excluded (Chamari *et al*., 1995; Brooks and Faulkner 1994; Pearson *et al*., 2002; Hawkins *et al*., 2003; Tanaka and Seals 2003). Pearson *et al*. (2002) analysed the strength of trained male weightlifters from different age categories, age ranging from 40 to 87 years, during a competition phase against healthy non-trained men of a similar age range. Their results show that the winner of the oldest age category (aged 87 years)

lifted a weight 36% of that of the youngest winner in the 40-49-age category. Pearson and co-workers commentated that this is extraordinary, as sedentary older people in their 9th decade often cannot raise themselves out of a chair unaided. These results highlight that trained older men experience a similar relative decline in strength in comparison to healthy non-trained older men. Nonetheless, the absolute strength of resistance trained older population is significantly greater than that of the healthy sedentary older population. To the extent that trained older men in their 7th decade can possess a similar MVC to young sedentary men in their 3rd decade, and strength trained older male athletes in their 9th decade can have strength comparable to sedentary older people in their 7th decade or younger (Pearson *et al*., 2002; Doherty 2003). For this reason, resistance trained older people in their 7th decade and older are more likely to prolong their functional independence into later life where sedentary individuals of a similar age are vulnerable to functional disability and institutionalisation [refer to Figure 1.3] (Brooks and Faulkner 1994; Pearson *et al*., 2002; Hawkins *et al*., 2003). Crucially, these findings clearly demonstrate the benefits of regular resistance training in attenuating the effects of ageing on muscular strength and functioning (Doherty 2003).

Ageing has been found to result in the muscle groups of the lower extremities experiencing a greater decline in strength than that of the upper extremities (Larsson *et al*., 1979), which is supported by a number of other studies cited in a review paper by Macaluso and De Vito (2004). These findings are contradicted by Doherty's (2003) review paper highlighting the 'In CHIANTI' population study conducted by Laurentani *et al*. (2003) comparing knee extensor and handgrip strength on 469 men and 561 women. However, Hughes *et al*. (2001), a study cited by Doherty (2003), clearly demonstrated a greater decline in isokinetic strength of the lower limbs than upper limbs in older women between the 5th and 8th decades, 16% versus 2% respectively. In addition, Frontera *et al*. (2000) found that the isokinetic strength of the knee extensor muscles declined at a significantly greater rate than the elbow flexors, 23.7% versus 19.4 % respectively, using the same male and female participants over a 12 year period with an initial average age of 65.4 years.

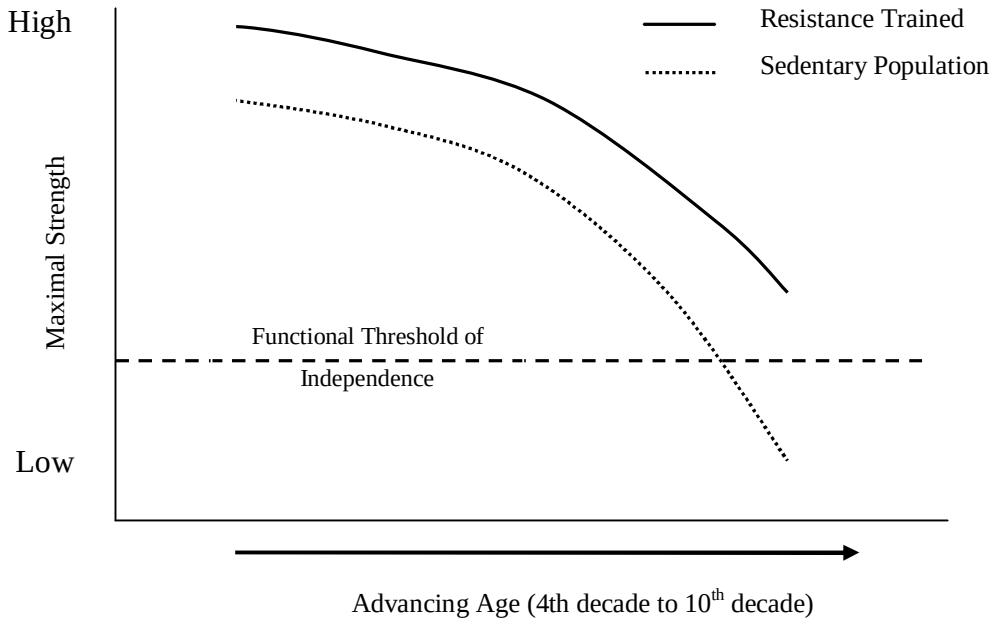

Figure 1.3 Theoretical model of ageing between resistance trained athletes and non-resistance trained sedentary individuals. The X axis refers to increasing age and the Y axis refers to maximal strength. The model indicates that for older people of the same age the resistance trained athletes possess a greater absolute maximal strength, but also experience the same relative decline with age. The higher absolute strength enables ageing resistance trained athletes to delay the age that it takes for maximal strength to decline towards the threshold that leads to a loss of functional independence, in some cases as much as 20-30 years in relation to the sedentary ageing population (Pearson *et al.*, 2002).

Further support is provided by Izquierdo *et al.* (1999), showing that the rate of decline in dynamic strength (bilateral 1RM) of the upper body (using bench press) appears to be less than the lower body (using squat) between middle-aged and older men. One possible factor that could account for the differences in regional strength between the studies is the complexity of the movements performed, i.e. how often encountered in everyday living (Izquierdo *et al*., 1999). More recently, results from Candow and Chilibeck (2005) comparative study, analysing the muscle groups of the lower and upper limbs of 50 men aged between the 3rd and 8th decade, found that the peak torque produced at slow and high velocities was significantly lower in the upper limbs than the lower limbs. For instance, peak torque at 3.14 rad·s^{-1} declined by 39%

in the knee extensors compared to 16% in the elbow extensors. Hence, providing support that ageing affects the muscles of the lower limbs greater than the upper limbs. Therefore, as regional differences in strength may be likely to occur with ageing, the rate of decline in strength of one muscle group should not be generalised to another (Frontera *et al.*, 1991).

Moreover, studies have demonstrated that ageing has a differing effect even between the individual muscles from the same limb. Klein *et al*. (2001) found that strength declined at significantly different rates between the elbow extensors and flexors with the elbow extensors declining greater than flexors, 26% compared to 17% respectively. This is supported by the findings of Vandervoort and McComas (1986), which showed that MVC of the plantar flexor muscles declined significantly greater than dorsi flexors, 63% versus 48%, between women aged in their 3rd to their 9th and 10th decades. Nevertheless, conclude that the disparate effects of aging on regional strength may reflect the daily activities performed by older people. Support for this conclusion is emphasized by studies that found small to no difference in strength between lower and upper limbs with ageing, as there is a possibility that the individual's level of physical activity was sufficient to prevent the effects of ageing affecting muscle strength (Frontera *et al.*, 2000; Lanza *et al.*, 2003; Tanka and Seals 2003; Bazzucchi *et al.*, 2004).

Cross sectional study show that men tend to possess and maintain a significantly greater absolute value of maximal strength than women throughout all ages of the life span, and that both sexes experience the same relative decline in muscle strength (Vandervoort and McComas 1986; Skelton *et al*., 1994; Lanza *et al*., 2003; Doherty 2003; Lauretani *et al*., 2003; Short *et al*., 2005). Frontera *et al.* (1991) found women's knee extensor isokinetic strength ranged from 42.2-62.8% of that of males between the ages of 65-70 years. These findings agree with Short *et al*. (2005) using 38 men and 39 women between the ages of 21 to 87 years. However, men tend to experience a greater magnitude of decline in strength (Frontera *et al.*, 1991; Doherty 2003; Lauretani *et al*., 2003), with Short an co-workers finding that men experienced a 13% decline per decade compared to a 8% per decade decline in women from the 3rd to 9th decade (Short *et al*., 2005). Conversely, Hughes *et al*. (2001) found that women aged in their 5th and 8th decades experienced similar

percentage declines in isokinetic KE strength over a 10-year period in comparison to males of a similar age. Nevertheless, the most important finding is the confirmation that the muscle strength of older women is found to reach significantly lower levels than men of a similar age, and that women experience a significantly greater loss in ability to perform functional tasks (Frontera *et al*., 1991; Skelton *et al*., 1994; Doherty 2003; Tanaka and Seals 2003; Lauretani *et al*., 2003; Macaluso and De Vito 2004).

Muscle Power

Muscle power has been found to decline at a significantly greater rate than muscle strength with ageing (Bosco and Komi 1980; Bassey and Short 1990; Skelton *et al*., 1994; Kostka *et al*., 1997; Izquierdo *et al*., 1999; Foldvari *et al*., 2000; Lauretani *et al*., 2003; Petrella *et al*., 2005; Yanagiya *et al*., 2004; Candow and Chilibeck 2005; Kostka 2005; McNeil *et al*., 2007). Skelton *et al*. (1994) observed a decline in explosive peak muscle power of 3-4% per year, using the modified NPR, whereas isometric knee extension strength declined 1-2% per year between the ages of 7th and 9th decades. Nonetheless, the greater decline in muscle power was only significant in older men, results that are supported by Lauertani *et al*. (2003) population study using the modified NPR. In contrast, Kostka *et al*. (1997), utilising short-term supra-maximal cycling, found that maximal power declined at an even higher rate of 4.5% per year using women aged between the 7th and 9th decades.

Studies utilising isokinetic dynamometry knee extensions support the exacerbated decline in instantaneous muscle power in comparison to the decline in MVC strength in both men and women between the 3rd and 8th decades (Lanza *et al*., 2003; Trappe *et al*., 2003; Ferri *et al*., 2003; Candow and Chilibeck 2005). For instance, Lanza *et al*. (2003) found that the average instantaneous muscle power loss was 33%, from all isokinetic velocities tested, compared to a 21% decline in MVC between the 3rd and 8th decades in older people, whereas Trappe *et al*. (2003) found that maximal peak power declined on average by 45% using a similar methodology. Furthermore, Macaluso and De Vito (2003) comparative study utilising 'isotonic' explosive knee extension contractions, found that maximal explosive knee extension

power of older women in their 7th and 8th decades was approximately 60% less than younger women in their 3rd decade compared to a reduction of a 50% in MVC. In addition, the ratio between MVC and maximal muscle power was 21% lower in older women, supporting the phenomenon that muscle power output is more susceptible to the ageing process than maximal strength. They also reported an interesting and important fact that older women could not move the load at which younger women where achieving F_{opt} for maximal power. This observed 'age trend' provides an indication of the degree to which older women's muscles F-V relationship experiences a leftwards shift with age. More crucially, McNeil *et al*. (2007) highlight the importance of testing muscle power over strength, as they found that even though older men's MVC was unchanged significant declines in peak power were observed.

De Vito *et al*. (1998) study found that the decline in V_{opt} was the most significant factor contributing to the loss in 'instantaneous peak power' (using a vertical jump on a force platform) with ageing in healthy older women aged 50-75 years. They also observed that the data from Bosco and Komi (1980) support their findings. Results from Kostka *et al*. (1997; 2005) also found a significant decline in older women's V_{opt} (1.7% per year) with short-term maximal power between the 7th to the 9th decades. Whereas, Martin *et al*. (2000), utilising maximal cycling to investigate the short-term power output across the life span on trained men cyclists, found that the significant 30% decline in maximal power correlated with a 9% decline in V_{opt} from the 5th to the 7th decade. However, unreported data calculated from their results indicates that F_{opt} at maximal power declined to a greater extent than V_{opt}, 24% versus 9%, respectively. Conversely to De Vito *et al*. (1998) findings, ageing studies that utilised single explosive isokinetic knee extensions predominantly found that the reduction in F_{opt} is the most significant factor contributing to the decline in maximal power (Ferri *et al*., 2003; Lanza *et al*., 2003; Macaluso and De Vito 2004). Possibly, more relevant are the findings of Macaluso and De Vito (2003) comparative study of older and younger women, who where the first to establish the effects of ageing on both determinants of maximal peak power using isotonic dynamometry. Their results show that the decline in maximal peak power (61%) was attributed to a significantly greater reduction in F_{opt} (52%) than V_{opt} (21%) [Refer to Figure 1.4]. These findings concur with Chamari *et al*. (1995) comparing master

endurance trained athletes in their 7th and 8th decade to endurance trained athletes in their 4th decade, utilising a 'supra-maximal' cycle test to measure short-term power. Overall, the conflicting results produced between these studies suggest that the different methodologies adopted in assessing muscle power are likely to explain the differences in the effects of ageing on the determinants of muscle power (Macaluso and De Vito 2004).

The regional differences found in relation to the decline in muscle strength with ageing are also relevant to explosive muscle power, with the quadriceps muscle power being affected greater than distal muscles of the lower and upper limbs (Izquierdo *et al*., 1999; Candow and Chilibeck 2005), e.g. average instantaneous peak power declined by 26% in dorsi flexors and 33% in knee extensors from the 3rd to 8th decade (Lanza *et al*., 2003). The functional consequences of the greater loss of peak power output in the quadriceps muscles are observed in older people's walking speed, which declines significantly by 12% and 16% per decade after the 7th decade in females and males, respectively, and other activities of daily living important for maintaining functional independence (Himann *et al*., 1988; Skelton *et al*., 1994; Vandervoort 2002; Cuoco *et al*., 2004).

Research shows that the absolute peak muscle power output produced by women is significantly lower in comparison to men of similar ages throughout the life span (Bosco and Komi 1980; Skelton *et al*., 1994; Lanza *et al*., 2003; Lauertani *et al*., 2003; Trappe *et al*., 2003; Petrella *et al*., 2005). The initial difference in peak muscle power between men and women in their 3rd decade is approximately 16%, however the difference between the sexes increases to 30-50% and greater by the 8th decade (Bosco and Komi 1980; Skelton *et al*., 1994; Lauertani *et al*., 2003). Moreover, results from Trappe *et al*. (2003) indicate that the peak power of younger women in their 3rd decade is similar to that produced by older men in their 8th decade.

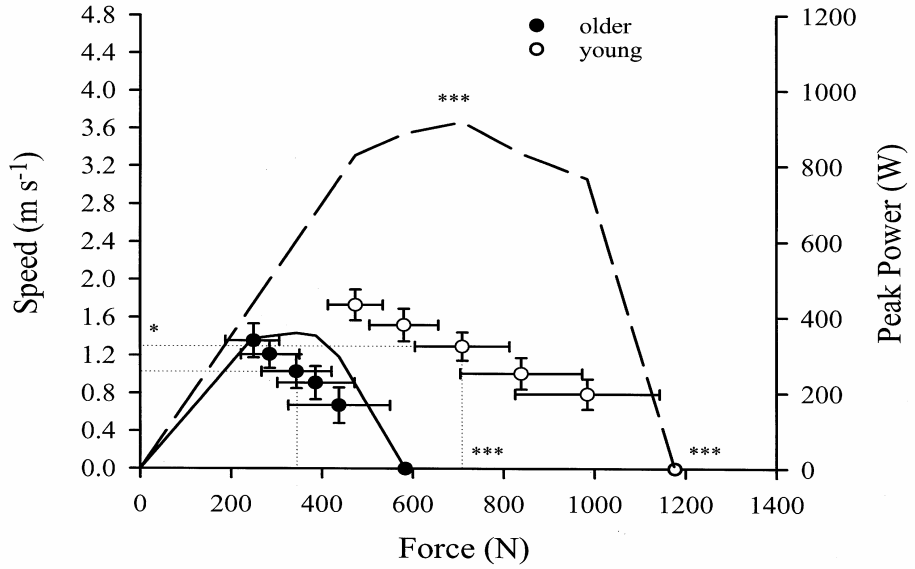

Figure 1.4 Force-Power and Force-Velocity relationships between older and younger women in their 8th and 3rd decades taken during single leg extensions. The F-V curve (represented by circles ● older ○ younger) is significantly shifted to the left leading to significant reductions in optimal speed, force and maximal power (represented by solid line for older and broken line for younger). The lighter broken lines represent corresponding optimal velocity and force for maximal power. Cited from Macaluso and De Vito (2003).

Crucially, this demonstrates the vulnerability of older women's muscle power in reaching or falling below the threshold required for functional independence earlier than older men of a similar age, and thus women can be regarded as being more prone to functional disability and institutionalisation (Skelton *et al.*, 1994; Lanza *et al.*, 2003; Macaluso and De Vito 2004; Kuh *et al.*, 2005). Therefore, women should be identified as the first group considered for interventions that aim at reversing or attenuating the effects of ageing on muscle power (Skelton *et al.*, 1994; Kuh *et al.*, 2005).

THE PHYSIOLOGICAL MECHANISMS RESPONSIBLE FOR THE DECLINE IN MUSCLE STRENGTH AND POWER

Muscle Strength

Given that the maximal amount of force generated by a muscle is proportional to its cross sectional area (CSA) any changes in muscle CSA will affect maximal muscle strength (Narici *et al*., 1991; 1992; 2005; Bruce *et al*., 1997; Lieber and Friden 2000; Frontera *et al*., 2000; Reeves *et al*., 2004; Morse *et al*., 2005; Jones *et al*., 2008). The CSA of muscle can be measured using anthropometrical measurement or with several types of imaging techniques (Doherty 2003; Macaluso and De Vito 2004). The imaging techniques commonly utilised include ultrasonography, computerized tomography (CT) and magnetic resonance imaging (MRI), the later being regarded as the gold standard method of measurement (Greig *et al*., 1993; Doherty 2003; Janssen *et al*., 2000; Reeves *et al*., 2004; Candow and Chilibeck 2005; Short *et al*., 2005; Morse *et al*., 2005; McNeil *et al*., 2007).

Muscle CSA has been found to decline with age by approximately 30-50% in the lower limbs (quadriceps femoris) from the 3rd to 9th decade of life in healthy older adults (Young *et al*., 1984; 1985; Frontera *et al*., 2000; 2008; Janssen *et al*., 2000; Trappe *et al*., 2001; Macaluso *et al*., 2002; Vandervoort 2002; Doherty 2003; Trappe *et al*., 2003; Short *et al*., 2005). A phenomenon that is even observed in resistance trained master athletes (Grassi *et al*., 1991; Pearson *et al*., 2002), who maintain physical activity levels above that recommended by the American College of Sports Medicine (ACSM 1998) in their Position Stand on 'exercise and physical activity for maintaining muscle mass in older people'. Hence, the loss of muscle mass can be regarded as an inevitable and a major part of the ageing process contributing to a decline in muscle strength (Narici *et al*., 1991; 2005; Brookes and Faulkner 1994; Janssen *et al*., 2000; Roubenoff and Hughes 2000; Trappe *et al*., 2001; 2003; Hakkinen *et al*., 2002; Doherty 2003; Frontera *et al*., 2008). The decline in muscle mass accompanying the loss in strength with advancing age has been termed 'sarcopenia' by Rosenberg (1989) cited in Roubenoff and Hughes (2000).

Sarcopenia in older women has greater detrimental connotations for remaining functional independent than older men. This is attributed to the absolute values of skeletal muscle mass tending to be significantly lower in women than men from the lower and upper limbs by an average of 33% and 40%, respectively, from the 2nd to 9th decades (Janssen *et al*. (2000). These results concur with the decline in muscle strength observed in older men and women, and with the decline in strength being greater in the lower limbs compared to the upper limbs (Izquierdo *et al*., 1999; Frontera *et al*., 2000; Hughes *et al*., 2001).

Studies measuring whole muscle and single muscle fibre CSA, between older and younger populations using cross-sectional or longitudinal methodologies, attribute sarcopenia to both a reduction in muscle fibre CSA (atrophy) and more notably to a loss in the number of muscle fibres [hypolasia] (Larsson *et al*., 1979; Grimby and Saltin 1983; Lexell *et al*., 1988; Klitgaard *et al*., 1989; Lexell 1993; Welle 2002; Doherty 2003; Frontera *et al*., 2008). Several of these studies have been able to identify specific changes in muscle fibre types with ageing utilising ATPase histochemistry. The classification of muscle fibres types is performed by analysing the enzyme 'ATPase' that relates to the muscle fibres functional characteristics, i.e. type I fibres refer to a muscle that that exhibits a slow contraction velocity, and are regarded as non-fatiguing due to utilising aerobic metabolic pathways for energy re-synthesis. Whereas, type II fibres predominately refer to fibres that possess a faster contraction speed and utilise fatiguing anaerobic pathways, in addition to having a larger CSA and developing muscular tension 3-5 times greater than type I fibres (Larsson *et al*., 1978; Narici *et al*., 1991; McArdle *et al*., 1996; D'Antona *et al*., 2003; Close *et al*., 2005; Short *et al*., 2006). Type II fibres are commonly termed fast twitch (FT) and type I fibres slow twitch (ST), with type II fibres being further subdivided into type IIa and IIb. Type IIa fibres are intermediate in terms of speed of contraction and the amount of force generated (Lexel *et al*. 1988; Lexell 1993; McArdle *et al*., 1996; Close *et al*., 2005).

Studies adopting ATPase histochemical analysis demonstrate that there is a greater decline in the CSA of type II fibres (predominantly type IIa) than type I from the vastus lateralis, 25-50% reduction observed from type II versus a 1-25% from type I fibres by the 9th decade in men and women (Lexell *et al*., 1988; Lexell 1993).

Therefore, suggesting that the ageing process causes a 'selective atrophy' of type II fibres (Larson *et al*., 1979; Aniansson *et al*., 1986; Lexell *et al*., 1988; Klitgaard *et al*., 1989; Lexell 1993; Andersen 2003). More significantly, the ageing process causes a reduction in the number of muscle fibres, with approximately a 50% reduction in the number of fibres from the vastus lateralis observed between the 3rd and 9th decades in older adults (Lexell *et al*., 1988; Lexell 1993). The loss in the number of muscle fibres is not pertinent to a specific fibre type, but can be regarded as the principal cause sarcopenia in the vastus lateralis or quadriceps (Lexell *et al*., 1988; Lexell 1993; 1997). However, Larsson *et al*. (1979) indicates that the area occupied by type I fibres increases within the quadriceps due to the proportion of type II fibres declining, caused by hypoplasia and selective atrophy of type II fibres. These debatable findings have been disputed by Lexel (1993) finding that both types of fibres are similarly affected, as supported by the recent studies of Frontera *et al*. (2000a; 2008) using ATPase hitochemisty. In contrast to the findings of Lexell *et al*. (1988) and Larsson *et al*. (1997), Frontera *et al*. (2008), analysing muscle fibre type and size changes from older men and women over a 9 year period with an initial age of approximately 64 years, found a significant increase in CSA from type IIa fibres. This led Frontera and co-workers to conclude that the decline in muscle strength can be explained through the significant loss in whole muscle CSA, with the remaining muscle fibres appearing to compensate for the changes in whole muscle CSA.

The electropheric technique SDS-PAGE (refer to Harridge *et al*., 1996) has been adopted by more recent studies to investigate the effects of ageing on muscle fibre types due to its greater ability to differentiate between single fibre types than histochemical analysis, and thus less likely to misinterpret fibre type changes with age (Klitgaard *et al*., 1989; Andersen *et al*., 1999; Frontera *et al*., 2000a; Ross and Leveritt 2001; Macaluso and De Vito 2004; Short *et al*., 2005; Pearson *et al*., 2006). The technique of SDS-PAGE specifically identifies the Myosin Heavy Chain (MHC) isoforms (related to the ATPase enzyme) within a muscle fibre, which are classified in relation to contractile characteristics, i.e. MHC-I, IIA and IIX that are similar to the classification of type I, IIa and IIb fibres (Andersen *et al*., 1999; Bottinelli and Reggiani 2000; Höök *et al*., 2001; Ross and Leveritt 2001; D'Antona *et al*. 2003; Trappe *et al*., 2003). This approach was adopted by D'Antona *et al*. (2003) to

investigate the effects of ageing and ageing combined with immobilisation on single fibre MHC isoforms from the vastus lateralis through comparing 7 sedentary young males (average age 30.2 years) against 9 sedentary older males (average age 72.7 years: 2 of which where bedridden for 3.5 months due to knee surgery). Their results demonstrated that the numbers of fibres containing MHC-IIA isoforms (type IIa fibres) were significantly reduced and fibres containing MHC-IIX isoforms (type IIb fibres) were significantly increased between older and younger sedentary men. Whereas, the elderly immobilised men experienced a significantly greater increase in MHC-IIX (24%), indicating a 'default' towards a fast phenotype with muscular disuse. Conversely, Trappe *et al*. (2003) only found a significant reduction in MHC-IIA isoform between younger and older women in their 3rd and 8th/9th decades, and that there appears to be no difference in fibre proportions containing MHC-I and IIX isoforms even with 2-3 months immobilisation in both males and females at an advanced age.

Several studies have shown that the amount of fibres containing MHC-I isoform (type I fibres) remains constant between young and old sedentary men and women in their 3rd and 8th decades (D'Antona *et al*., 2003; Trappe *et al*., 2003; Canepari *et al*., 2005), which supports Lexell *et al*. (1988) findings utilising histochemical analysis. However, Klitgaard *et al*. (1989) found an increase in amount of MHC-I content, from the vastus lateralis, between older and younger trained men in their 3rd and 7th decades, which supports Larsson *et al*. (1979). This observation may be explained by ageing muscle experiencing both a selective atrophy and decline in the number of type II fibres. In addition, ageing muscle has been shown to contain a significant greater number of fibres containing a co-expression of MHC-I-IIA isoforms, which are dominated by MHC-I in both older sedentary men and women (Andersen *et al*., 1999; D'Antona *et al*., 2003), and thus may explain the increased proportion in fibres containing MHC-I with age. Conversely, immobilisation compounded by advancing age has been found to cause a significant reduction in MHC-I content in older men (D'Antona *et al*., 2003). A more recent study by Short *et al*. (2005), showing a significant increase in the proportion of fibres expressing MHC-I and decline in MHC-IIA expression from the 3rd to 9th decade in both men and women, may provide significant evidence of the contentious issue

relating to the proportion of type I fibres increasing with age, as they utilised a larger number of participants (n=77). Hence, it appears that ageing causes muscle fibres to change towards a slower phenotype that results in the whole muscle experiencing an increase in the proportion of fibres dominated by MHC-I isoform.

The conflicting results may, however, be attributed to not only the increase in fibres containing a co-expression of MHC isoforms (Andersen *et al*., 1999; D'Antona *et al*., 2003), but due to the possibility that the MHC isoform within a muscle fibre may not be uniform along the whole length of the fibre (Andersen 2003). Therefore, inferring changes in fibre phenotype may be difficult from a small muscle biopsy. Other factors that influence muscle phenotype differences between older adults can be attributed to their individual experiences of physical inactivity, hormonal changes, and neurological processes (Doherty 2003; D'Antona *et al*., 2003; Macaluso and De Vito 2004; Canepari *et al*., 2005).

Additionally, D'Antona *et al*. (2003) results show that the CSA of fibres containing MHC-I and MHC-IIA isoforms of older and older immobilised men were significantly reduced in comparison to younger men. Both the older sedentary and immobilised men experienced a greater decline in CSA of fibres containing MHC-I than MHC-IIA isoform, 22% versus 12% respectively. However, the decline in CSA of these fibres was 50% greater in older immobilised compared to the older sedentary men. Whereas, Trappe *et al*. (2003) found that only sedentary older women experienced a significant decline in CSA of MHC-IIA fibres. The results from D'Antona and co-workers contradict Lexell *et al*. (1988) 'selective atrophy of type II fibres', however Lexell did comment on the fact that there was a large variability in the reduction in size and number of type II fibres between older individuals of the same age. The methodological differences between these studies, analyzing small *in vivo* muscle biopsies (D'Antona *et al*. 2003; Trappe *et al*. 2003) against direct measures of large *in vitro* muscle samples from cadavers (Lexel *et al*., 1988; Lexell 1993), may account for the discrepancies in results.

The effect of a greater reduction in the number of fibres expressing MHC-IIA (D'Antona *et al*., 2003; Trappe *et al*., 2003; Short *et al*., 2005), an increased co-expression of MHC I-IIA isoforms (D'Antona *et al*., 2003; Andersen *et al*., 1999; 2003), and a selective atrophy of type II fibres (Lexel 1993), is that ageing muscle,

specifically from the quadriceps, will lose its maximal force generating capacity due to both a decline in muscle CSA but also to a loss of higher tension / force generating fibres per CSA of muscle (Larsson *et al*., 1979; Aniansson *et al*., 1988; Lexell *et al*. 1988; Lexell 1993; Vandervoort 2002; Doherty 2003; D'Antona *et al*., 2003; Trappe *et al*., 2003; Macaluso and De Vito 2004; Narici *et al*., 2005; Thom *et al*. 2005). Nonetheless, the loss in muscle mass is regarded by numerous studies to be the most significant cause for the decline in strength with ageing (Frontera *et al*., 1991; 2000; 2008; Roos *et al*., 1997; Roubenoff and Hughes 2000; Vandervoort 2002; Doherty 2003), as the decline in muscle mass can totally account for the decline in strength (Frontera *et al*., 2000; 2008). Whereas, other studies have tried to demonstrate that the magnitude of decline in muscle mass appears to parallel the decline in muscle strength with advancing age (Grimby and Saltin 1984; Doherty 2003). For instance, the observed slow reduction in muscle mass from the quadriceps of approximately 10% from the 3rd to the 6th decade in life is followed by the rate of decline accelerating to 1% or greater per year, which is similar to the observed rate of decline in muscle strength (Lexell *et al*., 1988; Narici *et al*., 1991; Pearson *et al*., 2002; Doherty 2003). Conversely, Bruce *et al*'s (1997) review paper indicates that the time course of the decline in maximal strength in older participants occurs earlier than the decline in muscle mass, thus suggesting that other factors also contribute to the decline in maximal strength. Numerous cross-sectional studies support this observation, as they have demonstrated that the decline in muscle strength is greater in relation to reduction in whole muscle CSA (Young *et al*., 1985; Izquierdo *et al*., 1999; Klein *et al*., 2001; Macaluso *et al*., 2002; Narici *et al*., 2003; Short *et al*., 2005) and in regards to the strength and CSA of single muscle fibres (Frontera *et al*., 2000a; D'Antona *et al*., 2003). In order to determine if other factors contribute to the total loss of strength the ratio between the force generating capacity of a muscle to its unit of CSA (termed specific strength) has to be investigated (Brooks and Faulkner 1994; Narici *et al*., 1991; Bruce *et al*., 1997; Lynch *et al*., 1999).

Specific strength or 'muscle quality' is regarded as a more accurate indicator of the loss of muscle strength with age (Brooks and Faulkner 1994; Bruce *et al*., 1997; Lynch *et al*., 1999; Doherty 2003; 2003; Trappe *et al*. 2003; Macaluso and De Vito 2004; Narici *et al*., 2005). A reduction in specific strength refers to a 'weaker

muscle' that produces a force lower than expected in relation to its CSA, as a smaller muscle may still produce an expected force relating to its CSA (Brooks and Faulkner 1994; Klein *et al*., 2001; Doherty 2003; Reeves *et al*., 2004; Narici *et al*., 2005). For instance, Young *et al.* (1984), utilising ultrasonography, found no change in specific strength between older women in their 8th decade compared to younger women in their 3rd decade, thus concluding that the total loss of strength can be attributed to the decline in muscle mass. In contrast, Young *et al.* (1985), again utilising ultrasonography, concluded that older men in their 8th decade quadriceps specific strength was lower by 19%, in comparison to younger men in their 3rd/4th decades. The findings that older women's quadriceps specific strength was not affected by age is disputed by the findings of Lynch *et al*. (1999), comparing 364 men and 339 women subjects over an age range of 19 to 93 years. Specifically, Lynch and co-workers were the first to establish that women's quadriceps specific strength is significantly affected with advancing age by approximately 20%. In addition, highlight the validity of their measures of strength from isokinetic dynamometer and muscle CSA using whole body dual-energy x-ray absorptiometry, which is crucial for accurately measuring specific strength (Klein *et al*., 2001; Narici *et al*., 2005). Hence, the conflicting results produced by studies may possibly be attributed to the different methods adopted to calculate muscle strength and estimate muscle mass (Frontera *et al*., 2000a; Welle 2002; Doherty 2003; Macaluso and De Vito 2004; Narici *et al*., 2005). Specifically, ultrasonography is unable to differentiate between contractile and non-contractile tissue, and thus possibly overestimated CSA and specific strength (Klein *et al*., 2001; Macaluso *et al*., 2002; Narici *et al*., 2003). This is due to non-contractile tissue having been shown to increase within the muscle belly from older adults, which is caused by an increase in intra-muscular fat deposits and connective tissue (Overend *et al*., 1992; Kent-Braun *et al*., 2000; Macaluso *et al*., 2002; McNeil *et al*., 2007). Specifically, Overend *et al*. (1992) found that non-contractile material significantly increased by approximately 59% in the quadriceps and 127% in the hamstrings from the age of 20-80 years in men. These findings suggest that the measurement of specific strength may be inaccurate due to the overestimation of muscle CSA, as the non-contractile material will have been measured as muscle contractile material (Macaluso *et al*., 2002; Reeves *et al*., 2004).

A more accurate method of differentiating between contractile and non-contractile material is Magnetic Resonance Imaging (MRI) (Macaluso *et al.*, 2002; Doherty 2003). Despite the accuracy of MRI the results on specific strength have also been proved inconclusive, as Kent-Braun and Ng (1999), Frontera *et al*. (2000a; 2008) and McNeil *et al*. (2007) found no changes in specific strength between the 4th to 7th/8th decades in men or women. Whereas, Macaluso *et al*. (2002) study provides support for significant detrimental changes in specific strength of older women in their 7th decade, which was reduced by 17% and 24% from the knee extensor and flexor muscles, respectively, in comparison to younger women in their 3rd decade. In addition, McNeil *et al*. (2007) observed that dorsiflexion specific strength was only significantly affected in older men by the 9th and 10th decades in comparison to young men in their 3rd decade. Similar finding are reported by Klein *et al*. (2001) from the elbow flexors, but they utilised the physiological cross sectional area (PCSA) and conclude that sarcopenia is not only associated with a reduction in muscle quantity but also 'quality' i.e. less force per cross sectional area.

Discrepancies between studies are more likely to explained through measuring the anatomical CSA (ACSA) instead of the PCSA, which takes the muscle fibres' pennation angle (the angle of the muscle fibre to the direction of the force generating axis), muscle volume and fibre length into account (Lieber and Friden 2000; Klien *et al.*, 2001; Macaluso and De Vito 2004; Reeves *et al*., 2004; Narici *et al*., 2005). The PCSA is an important measurement in estimating specific strength as it has a significantly greater association with muscle force capacity than ACSA, and thus will produce a more accurate measure of specific strength (Lieber and Friden 2000; Klien *et al.*, 2001; Reeves *et al*., 2004; Narici *et al*., 2005). Taking the PCSA and muscle co-activation level (neural factor that restricts the amount of force from the agonist muscle, discussed in more detail later) into account, Morse *et al*. (2005) found that older men in their 8th decade still had a significantly lower specific strength by approximately 30% from the planter flexors in comparison to younger men in their 3rd decade. This led to the conclusion that possibly intrinsic factors within the muscle are more likely responsible for the lower specific strength with ageing.

Studies analysing single fibre specific strength have reported significant declines with age between young and older adults in their 4th and 8th decades (Larsson *et al*., 1997; Frontera *et al*., 2000a; D'Antona *et al*., 2003; Yu *et al*., 2007), but disputed by Frontera *et al*'s (2008) longitudinal study across a 9 year period in older people with an initial age of 64 years. Nonetheless, Yu *et al*. (2007) cross-sectional study found different effects of ageing between the sexes on their single fibre specific strength, as the MHC-I fibres specific force declined by 20-28% in both older sedentary men and women, but only MHC-IIA fibres in older men experienced a significant 29% decline in specific strength in comparison to young adults in their 3rd decade. D'Antona *et al*. (2003) found similar reductions in single fibre specific strength, using older sedentary men, but more notably discovered a significant correlation between a reduction in MHC concentration of single fibres and the loss of muscle force production. Therefore, the reduction in single fibre specific strength with ageing may be attributed to a lower number of acto-myosin cross bridges caused by a loss of MHC concentration or a 'reduced binding strength'. Hunter *et al*. (2004) add that a lower availability of adenosine triphosphate (ATP) at the acto-myosin cross bridge may also be a contributing factors in the decline of muscle fibre specific strength, as reduced availability of high energy phosphates correlated with the decline in MVC.

Neural Factors affecting muscle strength

The decline in muscle quality may also be attributable to a reduction in the quantity of functionally active muscle mass stimulated by the central nervous system during a voluntary contraction (Narici 1999 cited in Macaluso and De Vito 2004; Klass *et al*., 2005). The ability of older population to voluntary activate their remaining muscle mass for force and power production is dependent upon their neural drive, which refers to the 'integrity of motor units (MUs)' including their number, type, firing rate and ability to summate (Lexell 1997; Roos *et al*., 1997; 1999; Martin *et al*., 2000; Vandervoort 2002; Pozzo *et al*., 2004; Macaluso and De Vito 2004; Klass *et al*., 2008). Hence, studying MU behaviour with ageing may provide further insight into the physiological mechanisms of ageing that contribute to

the decline in muscle strength, power and functional ability with age (Roos *et al.*, 1997; 1999; Connelly *et al.*, 1999; Kamen *et al.*, 1995; Vandervoort 2002; Doherty 2003; Merletti *et al.*, 2002; Bazzucchi *et al.*, 2004).

A motor Unit (MU) can be described as the '*functional unit*' of a muscular contraction that refers to a nerve cell (motoneuron) and all the muscle fibres it innervates, via axons from the ventral root in the spinal cord where the motor neuron is located [refer to Figure 1.5] (Sherrington 1929 cited in Macaluso and De Vito 2004).

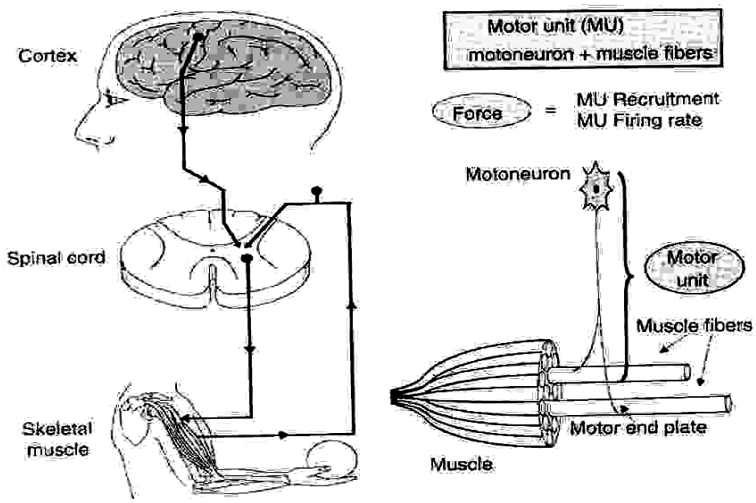

Figure 1.5 Schematic diagram of descending drive pathway and a motor unit. From Merletti and Parker (2004. p.3).

Motor units are categorised in accordance with the type of muscle fibres they innervate (McArdle *et al.*, 1996; Merletti and Parker 2004), with fast twitch (FT) MUs being regarded as large motoneurons that produce high conduction velocities of the action potential along their axons and innervate a large number of type II fibres (300-500 fibres). Whereas, slow twitch (ST) MUs innervate a smaller amount of type I muscle fibres and produce slower action potential conduction velocities than FT MUs (Jones and Round 1990; McArdle *et al.*, 1996; Merletti and Parker 2004). Therefore, in order to investigate the effects of ageing on muscular functioning in older people the muscular and nervous systems have to be regarded as one, termed

the neuromuscular system (Vandervoort 2002; Scaglioni *et al.*, 2003; Macaluso and De Vito 2004).

The number of functionally active MUs within a muscle can be estimated using intramuscular electrophysical techniques but only during static muscular contractions (Doherty *et al.*, 1993; Roos *et al.*, 1997; Vandervoort 2002; McNeil *et al.*, 2005). Cross sectional studies show the number of 'functional motor units' from the upper and lower limbs can decrease by up to 70% between older people in their 8^{th} to 9^{th} decade in comparison to younger population in their 3^{rd} and 4^{th} decades (Vandervoort and McComas 1986; Doherty *et al.*, 1993; Kamen *et al.*, 1995; Roos *et al.*, 1997; Connelly *et al.*, 1999; Vandervoort 2002; McNeil *et al.*, 2005). McNeil *et al.* (2005) add that the decline in MU number may not significantly affect muscle functioning until losses reach 40 to 60% of that of younger people. However, the extent of the loss in the number of functionally active MUs may totally account for the decline in strength after the 7th decade (Doherty *et al.*, 1993; Kamen *et al.*, 1995; Connelly *et al.*, 1999; Vandervoort 2002; Doherty 2003; McNeil *et al.*, 2007).

The reduction in the number of functionally active MUs is explained by a loss in the number of large alpha (α) motoneurons of the lumbospinal segments in the spinal cord, which is estimated to be approximately 25% from the 2^{nd} to the 10^{th} decade and may even reach 50% in some individuals (Tomlinson and Irving 1977 cited in Vandervoort 2002; Roos *et al.*, 1997). The loss of α motoneurons with ageing is regarded as the principal factor in the loss of the number of muscle fibres with ageing (sarcopenia) and muscle excitability (Larsson *et al.*, 1979; Doherty *et al.*, 1993; Lexell 1997; Roos *et al.*, 1997; Roubenoff and Hughes 2000; Vandervoort 2002; Doherty 2003).

Brooks and Faulkner (1994) add that animal studies analysing the effects of age found that the selective atrophy of type II fibres and decline in fibre number is attributable to a gradual loss of fast twitch motor neurons through denervation, which led to a theory of motor unit (MU) remodelling. MU remodelling theorises that after denervation of FT motoneurons there is an 'incomplete' reinnervation of these muscle fibres by a sprouting of ST motoneuron axon branches. The process of reinnervation of denervated muscle fibres with ST motoneuron branches is regarded as a 'mechanism' that attempts to prevent the loss of muscle fibres, which ultimately

leads to an increase in the size of ST MUs and number ST fibres with ageing (Larsson *et al.*, 1979; Lexell 1997; Vandervoort 2002; Doherty 2003; Enoka *et al.*, 2003; McNeil *et al.*, 2005). Apoptosis of a spinal motor neuron, which is highly likely to occur in human muscle due to the large change in muscle fibre type composition experienced with advancing age, is thought to be caused by free radicals within the cell (Brooks and Faulkner 1994; Weinert and Timiras 2003). This refers to the Free Radical Theory (Harman 1957), which states that leakage of free radical-containing reactive oxygen species, produced in living cells where energy is derived from oxidative processes within the mitochondria, leads to the production of superoxide anions (free radials) that are highly toxic and cause cellular oxidative damage and modification of the DNA. Hence, cell senescence is considered to occur due to the build up of oxidative damage over time (Weinert and Timiras 2003).

Moreover, MU remodelling provides an explanation of how ageing muscle experiences a decline in the number of fibres (possibly a preferential loss of fibres expressing MHC-IIA isoform) and an increase in the number of hybrid fibres with a co-expression MHC isoforms dominated by MHC-I, e.g. why FT fibres are experiencing a change towards a slower phenotype (Andersen 2003; Lexell 1997; D'Antona *et al.*, 2003). Furthermore, the conflicting results relating to the change in proportion of fibres expressing MHC-I isoform with advancing age between studies may also be explained by the age of participants recruited. As Andersen (2003) states that the MU remodeling process may initially affect FT MUs from the 7^{th} to 8^{th} decades and thereafter in the 9^{th} decade and onwards ST MUs may be predominately affected. Thus, he is suggesting that the ageing process involves muscles experiencing an initial increase in the proportion of ST fibres during the 7^{th} to 8^{th} decades followed by 'a regaining of equal status' between FT and ST fibre type proportions from the 9^{th} decade, as ST MUs become mainly affected by MU remodeling (Andersen 2003; McNeil *et al.*, 2005).

Further evidence that suggest MU remodelling is occurring in humans is found in the 'grouping' or an increase in the number of 'enclosed' fibres with advancing age especially after the 7^{th} decade (Lexell 1993; 1997; Andersen 2003). This refers to a fibre that is completely surrounded by the same type of fibres, which indicates that fibre phenotype types are changing with age. In addition, McNeil *et al*.

(2005) found that the significant augmented rate of decline of muscle mass and strength in the very old (above the 9th decade) coincides with an accelerated loss of functional MUs. Hence, MU remodelling processes can be regarded as a significant factor for muscle morphological and functional changes with advancing age (Doherty *et al*., 1993; Roos *et al*., 1997; 1999; Vandervoort 2002). However, it is still debatable if denervation is responsible for the initial losses in muscle mass and strength from the 3rd to the 6th decade (Vandervoort and McComas 1986; McNeil *et al*., 2005)

Motor unit behaviour changes have been observed with ageing using intramuscular electromyography during steady-state isometric voluntary contractions ranging from 10-100% MVC (Connelly *et al*., 1999; Vandervoort 2002; Doherty 2003; McNeil *et al*., 2005; Rubinstein and Kamen 2005). Connelly *et al*. (1999) found that MU firing rate decreases by about 30-35% with age from the tibialis anterior at all relative percentages of MVC in men aged from the 3rd to the 8th decade. Nonetheless, a study by Roos *et al*. (1997) concluded that the reduction in MU firing rates is only significant during contractions performed at 75% MVC or greater. The effect of a reduction in MU firing rate is that ultimately maximal muscular force is produced at lower tetanising frequencies in older people, and therefore reduces the muscle force and specific strength with age when fully activated (Roos *et al*., 1997; Connelly *et al*., 1999; Vandervoort 2002; Macaluso and De Vito 2004; Klass *et al*., 2008). These findings are contradicted by Roos *et al*. (1999), who observed no differences in the mean firing rates of MU from the vastus medialis (quadriceps) between men in their 9th and 3rd decades, therefore suggesting that the effects of ageing on MU firing rate may not have a detrimental impact on force development from the quadriceps due to relying more on the size principle (recruiting larger MUs to generate force) to modulate force than other muscle groups (Roos *et al*., 1999; Merletti and Parker 2004).

Several studies have shown that the 'innervation ratio' of slow twitch MUs increase with ageing, thus providing evidence of reinnervation (MU remodeling) occurring in humans (Doherty and Brown 1997; Roos *et al*., 1997; Connelly *et al*., 1999; McNeil *et al*., 2005). Roos *et al*. (1997) explains that the higher innervation ratios may possibly lead to an increased 'load' placed on the slow twitch MUs, as

they have to stimulate a greater number of fibres, bringing about a decline in MU firing rates and a reduced 'excitable' muscle mass. Nonetheless, the reduced ability to excite the remaining muscle mass in older people is contradicted by Roos *et al*. (1999). Their study was the first to utilise older men over 80 years of age to study MU changes in vastus medialis, which can be regarded as a crucial muscle in relation to performing functional tasks. The results from Roos and co-workers show that even though there was a 50% reduction in force production (voluntary and electrically stimulated) older men where still able to excite their muscle mass to a similar extent as young men in their 3rd decade. Furthermore, the lack of change in MU firing rate with age suggests that ageing does not affect the neuromuscular systems and that peripheral changes are possibly more accountable for the reduction in force generating capacity in the quadriceps (Roos *et al*., 1999).

Studies adopting surface electromyography (sEMG) provide some additional evidence of MU remodeling and more importantly changes in neural activation, which may contribute to a reduction in muscle contraction force and specific strength in older humans (Moritani and de Vries 1980; De Luca *et al*.,1997; Roos *et al*., 1997; Hakkinen *et al*., 1998; Izquierdo *et al*., 1999; Stashuk 2001; Macaluso *et al*. 2002; Merletti *et al*., 2002; Pearson *et al*., 2002; Macaluso and De Vito 2004; Bazzucchi *et al*., 2005). Surface EMG is a non-invasive technique that reflects changes in neural activation (both peripheral and central neural properties) from the muscle being voluntary activated during a maximal or sub-maximal isometric contraction (De Luca *et al*., 1997; Merletti *et al*., 2002; Farina *et al*., 2002; 2002a; 2004a; Macaluso and De Vito 2004; Arabadzhiev *et al*., 2009). The signals recorded from sEMG are regarded as a 'global' measure of MU activity that provides an insight into the level of neural activation or neural drive from the spinal cord to the motoneurons governing muscle force production (Stegeman *et al*., 2000; Farina *et al*., 2004a). The amount of force generated by the muscle is determined by the size, number and firing rate of the active MU's, which produce MU action potentials (MUAP) that in turn are detected and shown by the sEMG signal recordings (Basmajian and De Luca, 1985; Stegeman *et al*., 2000; Arabadzhiev *et al*., 2009).

The detection of neural activation from sEMG requires using surface electrodes, which can be either a pair (bipolar) or a linear electrode array that utilises

4 or more electrodes in series (Macaluso *et al.*, 2002; Pozzo *et al.*, 2004). Electrodes are attached to the skin above the muscle being voluntary activated using standardised procedures during the experimental protocol, as a method of minimising the negative effects from the non-physiological factors that can confound the EMG recording measurement (De Luca *et al*., 1997; Solomonow *et al.*, 1990; Merletti *et al*., 2001; Farina *et al*., 2004a; Merletti and Parker 2004). These confounding factors include the surface electrode's size, shape and material, interelectrode distance, the presence of gel, cleansing of the skin with alcohol, abrasion of the skin, shaving of hair, electrode location over the muscle with respect to tendons, MU innervation zone and fibre direction (De Luca *et al*., 1997; Merletti *et al*., 2001; Farina *et al*., 2004a). All of which have been taken into account when conducting the experimental work relating to this thesis, using standard linear array electrodes and procedures according to those utilised by Farina *et al*. (2004).

The global myoelectrical activity measured using sEMG during a muscular contraction can be differentiated into two main parameters (Merletti *et al*., 2001; Macaluso *et al*., 2000; Farina *et al*., 2002; 2002a; 2004a; Merletti and Parker 2004). Firstly, the amplitude of the sEMG signal can be determined using the root mean square (RMS), which reflects the overall number, firing rate and synchronisation of activated MUs and 'quantifies' the level of muscle activation termed neural drive (Basmajian and De Luca, 1985; Esposito *et al*., 1996; Macaluso *et al*., 2002; Farina *et al*., 2002a; 2004a; Rau *et al*., 2004). The behaviour of single MUs cannot be differentiated between using sEMG due to the likelihood of several MUAP, recorded within the detection area of the electrode, firing stochastically producing the termed 'interference sEMG' (Stegeman *et al*., 2000; Enoka *et al*., 2003; Farina *et al*., 2004a). Hence, the behaviour of individual MU can not be observed, but the sEMG signal characteristics do reflect the sum of the behaviour of the contributing MUs i.e. size and firing rate (Basmajian and De Luca, 1985; Stegeman *et al*., 2000; Arabadzhiev *et al*., 2009). Secondly, the frequency or myoelectrical power spectrum of the signal is more commonly reflected by the median frequency (MDF), which is determined to reflect the conduction velocity of the action potential travelling along the muscle fibre's sarcolemma, i.e. muscle fibre conduction velocity [MFCV], and can be used to infer changes in MU recruitment strategies during muscular fatigue

(Bigland-Ritchie *et al*., 1981; Stulen and De Luca, 1982; Solomonow *et al.*, 1990; Macaluso *et al*., 2002; Farina *et al*., 2002a; 2004a; Rau *et al*., 2004). The MDF is estimated to correlate with the muscle's fibre type composition, as an increase in MDF infers a greater number of fast twitch fibres activated, and thus could be utilised as a non-invasive method of fibre typing (Merletti *et al*., 2001; Pearson *et al*., 2002; Macaluso and De Vito 2004; Merletti and Parker 2004). However, some doubt has been placed on the correlation between MDF and MFCV (a size principle parameter) during isometric contractions, as it has been demonstrated that the an increase in MU synchronization occurring during sustained isometric fatiguing contractions influences the rate of decline MDF and MFCV differently (Farina *et al*., 2002; Farina *et al*., 2004a). Furthermore, MU discharge frequency has a small limited effect on MDF and thus may be regarded as a poor indicator of determining changes in MU recruitment strategies (Solomonow *et al*., 1990; Farina *et al*., 2004a). Recently, the use of MDF as a form of determining predominant fibre typing in a muscle has been highlighted as highly debatable, as changes in MDF do not always coincide with changes in MFCV due to the influence of muscle fibre length and depth of active MU having differing effects on MDF (Farina *et al*., 2004a; Farina 2008). A more accurate method of estimating MU recruitment strategies is through measuring MFCV using linear array sEMG with a double differential technique and an algorithm devised by Farina *et al*. (2004), which is discussed later in this thesis.

The interpretation of results from sEMG has to be conducted with some degree of caution due to the influence of the non-physiological confounding factors, as previously described, and the physiological factors of skin conductivity, the amount of subcutaneous fat that acts like a low pass spatial filter termed volume conductor, and depth of MU activated (De Luca *et al*., 1997; Farina *et al*., 2004a; Merletti and Parker 2004; Rau *et al*., 2004). In order to determine if differences in sEMG amplitude between individuals can be attributed to changes in neural drive then all the confounding factors, both physiological and non-physiological, have to be taken into account during the interpretation of the sEMG data (De Luca *et al*., 1997; Farina *et al*., 2004a). Several review studies indicate that in order to speculate that the central neural factors (neural drive) are primarily responsible for the differences in sEMG amplitude the sEMG data should be normalised to an

estimation of maximal activation (Merletti and Parker 2004; Farina *et al*., 2004a; Rouffet and Hautier 2008; Arabadzhiev *et al*., 2009). However, there are several contentious issues relating to the best method of normalising EMG amplitude data, which include normalising the EMG activation to that produced during an MVC, or to the corresponding M-wave or to a sub-maximal load relative to the individual's body mass when comparing sEMG during dynamic movements (Merletti and Parker 2004; Rouffet and Hautier 2008; Arabadzhiev *et al*., 2009). Nonetheless, in relation to comparing EMG amplitude during isometric contractions, older participant's ability to achieve full neural activation may be underestimated due to difficulties in their ability to fully activate their MU pool, which may lead to a scenario where normalisation of the sEMG data from older participants may falsely show no differences in neural drive when there are (Staudenmann *et al*., 2009).

Cross-sectional studies utilising sEMG during a MVC or sub-maximal isometric contraction have found that the absolute values from older adult's RMS are significantly reduced with age (Merletti *et al*., 2002; Macaluso *et al*., 2000a; Pearson *et al*., 2002; Bazzucchi *et al*., 2004). For instance, Macaluso *et al*. (2000a) found that older women in their 7th and 8th decade had a RMS approximately 50% lower, whereas MDF reduced to a lesser degree (24%), in comparison to younger women in their 3rd decade during a knee extension MVC. The reduction in MDF possibly infers a slower MFCV indicating a selective atrophy of fast twitch fibres or a decline in the ability to recruit FT MUs, which provides speculative evidence of neural denervation of type II fibres. While the reduction in RMS may be regarded as an indicator of a loss in neural drive with age, due to either a decline in number of functional motor units, lower firing rate of motor units or reduction in ability to synchronize motor units, as sEMG is unable to differentiate between these factors (Macaluso *et al*., 2002). The speculative conclusion that RMS is a reflection of neural drive is supported by both Merletti *et al*. (2002) and Macaluso *et al*. (2002), who state that differences in skin conductivity and subcutaneous fat would not have accounted for the large declines observed in their signal amplitude data with age, and thus differences in RMS are most likely due to a lower neural drive in older participants. These results support the studies highlighted previously using intramuscular EMG (Doherty *et al*., 1993; Roos *et al*., 1997; Connelly *et al*., 1999; McNeil *et al*., 2005)

The decline in RMS (neural drive) will ultimately reduce the force generating capacity of muscle either through a lower frequency of tetanic fusion (Narici *et al*., 1991; Roos *et al*., 1997; 1999; Merletti *et al*., 2002), a reduced ability to activate all MUs or a loss in the number of MUs (Roos *et al*., 1997; Hakkinen *et al*., 1998; 2001; Macaluso *et al*., 2000a; Vandervoort 2002; McNeil *et al*., 2005). Moreover, Pearson *et al*. (2002), investigating the effects of ageing between strength-trained and non-trained males between the 5th and 9th decades, provides further evidence for changes in overall neural activation, as resistance trained athletes had a significantly greater RMS and MDF compared to non-trained males of similar ages. These authors indicate the possibility of a reduction in the number of active MUs providing indirect support for MU remodelling theory occurring with ageing in humans, as demonstrated by McNeil *et al*. (2005). In addition, as MU remodelling appears to predominately affect the FT fibres the effects of ageing will thus be more pronounced in muscles that contain a higher percentage of fast twitch fibre (Narici *et al*., 1991; Lexell 1997). Hence, as the lower limbs contain a greater amount of fast twitch fibres than the upper limbs this provides a possible explanation for regional differences in strength with ageing. Importantly, as the quadriceps muscle is crucial for performing numerous everyday tasks a reduction of type II fibres in size and number from this muscle group would have a significant detrimental impact on an older person's functional ability (Evans 2000; Kubo *et al*., 2003b; Narici *et al*., 2003; Macaluso and De Vito 2004; Pearson *et al*., 2006).

Further evidence to support MU remodelling theory is shown by several studies reporting a 'fatigue paradox' with ageing, as older subjects appear to be able to sustain sub-maximal isometric contractions longer than younger subjects (Narici *et al*., 1991; Merletti *et al*., 2002; Hunter *et al*., 2004; Lanza *et al*., 2004; Bazzucchi *et al*., 2005; Rubinstein and Kamen 2005). This is attributed to MU remodelling process possibly leading to an increased proportion of slow twitch fibres or hybrid fibres that are more fatigue resistant within ageing muscle. Whereas, Rubinstein and Kamen (2005) found a slower decline in MU firing rate in older women compared to younger women during fatiguing isometric contractions, using intramuscular EMG, that suggest neural factors also influence the 'fatigue paradox' in older adults. Merletti *et al*. (2002), using linear array sEMG, confirms the phenomenon of a

fatigue paradox with ageing thorough greater myoelectrical manifestations observed in the younger men in their 3rd decade compared to older men in their 8th decade. The younger men demonstrated a significantly greater rate of decline in MFCV and MDF in comparison to older subjects in their 8th decade, which is associated with changes in intramuscular pH due to anaerobic glycolysis and possibly a lower MU firing rate (Merletti *et al*., 2002). Merletti and co-workers conclude that older people's muscles possibly contain a lower proportion of fast twitch fibres and a higher proportion of slow twitch fibres. However, highlight that their results may have been affected by the degree of blood flow occlusion. Recently Farina *et al*. (2005) have demonstrated that the degree of ischemia significantly influences the decline in MFCV, thus if the younger participants in Merletti *et al*. (2002) study experienced a greater degree of ischemia than the older participants, using the same relative loading, this would explain the greater decline in MFCV in the younger subjects and thus disputing the fatigue paradox with ageing. This suggestion has been contradicted by the findings of Hunter *et al*. (2005), which showed that strength-matched young and older men, in their 3rd and 8th decades, still resulted in older men being able to sustain a sub-maximal isometric contraction 42% longer than the younger men. Thus, highlighting that the absolute maximal strength and relative sub-maximal torque does not appear to influence the time to fatigue, and that possibly a higher proportion of type I fibres, slower recruitment of FT MUs and lower MU firing rate may explain the 'fatigue paradox' in older people (Narici *et al*., 1991; Merletti *et al*., 2002; Lanza *et al*., 2004; Hunter *et al*., 2005; Rubinstein and Kamen 2005).

As highlighted previously, co-activation between muscle groups in both older women and men has been found to increase in comparison to younger individuals in several studies but disputed by others (Hakkinen *et al*., 1998; Izquerdo *et al*., 1999; Klein *et al*., 2001; Macaluso *et al*., 2002; Doherty 2003; Hortobagyi *et al*., 2003; McNeil *et al*., 2007). Muscle co-activation restricts the amount of force generated from agonist muscles due to an increased activation of the opposing antagonistic muscles during a concentric contraction (static and dynamic), contributing to the decline in MVC and dynamic strength (Hakkinen *et al*., 1998; Klein *et al*., 2001; Macaluso *et al*., 2000a). Several studies have shown significant greater co-activation occurring during a knee extension between older people in their 7th-8th decade than in

younger people in their 3rd and 4th decades (Hakkinen *et al*., 1998; 2001b; Klein *et al*., 2001; Macaluso *et al*. 2002). More notably, Hortobagyi *et al*. (2003) found that the co-activation between the biceps femoris and the vastus medialis was significantly increased in older women in their 8th decade compared to women in their 3rd decade during the performance of functional tasks, which included climbing stairs and rising from a seated position. Importantly, these findings support Hakkinen *et al*. (1998) stating that older people experience an increased co-activation during performance of 'dynamic multi-joint actions'.

Another possible neural mechanism responsible for the decline in MVC is that older people may experience a decline in 'descending neural drive' from the 'supraspinal centres' to the motoneurons (Roos *et al*., 1997; Vandervoort 2002). These studies predominantly use a twitch interpolation technique to study 'central drive' by stimulating the motor neuron while the subject is performing a MVC. Roos *et al*. (1997) review paper indicates that studies predominantly indicate no significant difference in the ability to activate the muscle voluntarily with age, with small reductions of approximately 3-5% that are regarded as being unlikely to be 'functionally significant', which is supported by Klass *et al*. (2005). Conflicting results may however be due to the older subjects being unfamiliar with performing a MVC, as differences in 'descending drive' can be eliminated after a familiarisation period of the task (Macaluso and De Vito 2004). Macaluso and De Vito (2004) add that electrical stimulation techniques only results in an increase in MU synchronization to increase force production, and thus can be regarded as irrelevant as MU synchronization refers to situations that are unlikely to occur in everyday activities in older population.

Muscle architectural and tendinous factors affecting muscle strength

Other potential physiological mechanisms that affect whole muscle specific strength with age can be related to changes in muscle architecture, i.e. fibre pennation angle and fascicle length (Lieber and Friden 2000; Klein *et al*., 2001; Kubo *et al*., 2003b; Narici *et al*., 2003; 2005; Morse *et al*., 2005; Narici and Maganaris 2006). This is due to changes in muscle architecture affecting the

contractile characteristics of a muscle (Lieber and Friden 200; Narici *et al*., 2005). Narici *et al*. (2003) found a 13.2% reduction in pennation angle and a 10.2% reduction in fibre length from the gastrocnemius muscle leading to a 15.2% reduction PCSA, between males in their 4^{th} and 8^{th} decade of life. The findings of a significant reduction in pennation angle, due to a loss in muscle mass, are supported by Kubo *et al*. (2003b) from the vastus lateralis and medial gastrocnemius, but they found no significant effect of age on fascicle length. Whereas, Morse *et al*. (2005) reported that changes in muscle architecture may also contribute to the decline in whole muscle specific strength. Furthermore, Klein *et al*. (2001) conclude that the changes in muscle architecture with ageing would cause a decline in specific strength through a leftwards shift in the F-V relationships, which is supported by Narici *et al*. (2003; 2005). As a reduction in muscle pennation angle would lead to a decline in P_o and a shorter muscle fascicle length would results in a slower contraction velocity (Lieber and Friden 2000; Kubo *et al*., 2003b; Narici *et al*., 2003; 2005). This would ultimately detrimentally change the muscle groups F-V relationship, as highlighted by changes in isokinetic muscle strength, discussed previously. Kubo *et al*. (2003b) add that the declines in muscle mass and pennation angle from the medial gastrocnemius and vastus lateralis are significantly greater than those reported from the tricep brachii of the upper limbs with ageing, which supports the previous findings that the functional characteristics of the lower limbs are affected more with age than the muscles from the upper limbs. Therefore, exercise interventions should be predominately targeting the quadriceps as this muscle group is more vulnerable to the effects of ageing and is fundamental for performing numerous activities of daily living and preserving functional independence.

Tendinous factors influence force production due to the mechanical force produced by a contracting muscle having to be transmitted via tendons to the bones in order to bring about limb movement, and thus are important for performing functional tasks (Revees *et al*., 2003; Maganaris *et al*., 2004; Narici and Maganaris 2006). The ability of a tendon to transmit mechanical forces from a muscular contraction that produces human movement is determined by its *viscoelastic* properties (Kubo *et al*., 2003a; Revees *et al*., 2003; Maganaris *et al*., 2004; Narici *et al*., 2005; Narici and Maganaris 2006). Narici *et al*. (2005) states that there is some

debate over whether or not ageing has a detrimental influence on the transmitting of force via tendons. This is attributed to numerous *in vitro* animal and human studies showing that tendon stiffness can either increase or decreases with age, however the problem may stem from the different methodologies used by these studies. More recently investigations have turned towards *in vivo* human measurements using a methodology devised by Maganaris (2002), where ultrasound can be utilised to assess the elongation of the tendon under different degrees of muscular tension (Reeves *et al*., 2003; Maganaris *et al*., 2004; Narici *et al*., 2005; Narici and Maganaris 2006). These studies have shown that the patella tendon of ageing adults is approximately 15% more compliant than that of younger adults in their 3rd decade. This ultimately results in the tendons of older people stretching more when placed under tension, which leads to an increase in the time to transmit the force from the contracting muscle to produce movement. The consequences of which are observed in the slower rate of force development or time to peak torque (Davies *et al*., 1983; Reeves *et al*., 2003; Thom *et al*. 2005; Klass *et al*., 2008), and the ability to use the elastic energy during short-stretch cycle (required for squat jump peak power assessment) and electromechanical delay (time between initiating a muscular contraction and limb movement). The observed increase in the stretch of tendons means that older people are less able to transmit muscle force, which ultimately makes them vulnerable to falls (Magnaisis *et al*., 2001; 2004; Narici *et al*., 2005).

Muscle Power

Given that muscle power is the product of force and velocity, the physiological mechanisms discussed previously that bring about the decline in muscle strength (force) are thus applicable to the decline in muscle power (Macaluso and De Vito 2004; Thom *et al*., 2005). However, muscle power declines to a greater extent than maximal strength with advancing age (Bassey and Short 1990; Skelton *et al*., 1994; Izquierdo *et al*., 1999; Lanza *et al*., 2003; Macaluso and De Vito 2003; Thom *et al*., 2005). Hence, indicating that the mechanisms responsible for the augmented decline in muscle power relate more to a reduction in the muscles velocity of shortening. Figure 1.6 below illustrates how the decline in maximal

strength (P_o) and maximal shortening velocity of a muscle (V_{max}) affects the muscle's F-V and P-V relationships in the isolated muscle.

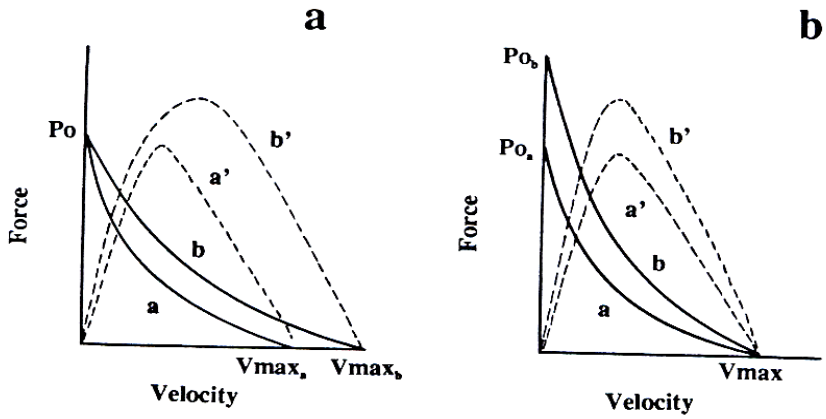

Figure 1.6 The effects of a decline in maximal shortening velocity (V_{max}) and strength (Po) on the Force-Velocity (—) and Power-Velocity (-----) relationships. **a.** The effects of a decline in V_{max} ($Vmax_b$ to $Vmax_a$) on the F-V and P-V relationship. The graph illustrates a theoretical leftwards shift in the F-V curve causing a reduction in V_{opt} and maximal power (b' to a') **b.** The effects of a decline in Po (Po_b to Po_a) that also causes a theoretical leftwards shift on the F-V curve causing a decline in F_{opt} and maximal power. Cited from Jones and Round (1990, p.100).

Numerous studies indicate that the decline in quadriceps (knee extensor) maximal muscle power is due to both a reduction in V_{opt} and F_{opt}, in addition to the speed at which the muscle contracts for a given amount of torque generated with age (Larsson *et al.*, 1979; 1997; Lanza *et al.*, 2003; Macaluso and De Vito 2003; Trappe *et al.*, 2003; Narici *et al.*, 2005; Pearson *et al.*, 2006; McNeil *et al.*, 2007). This suggests a slowing of the muscle's maximum shortening velocity with age, as demonstrated by Lanza *et al.* (2003). Lanza and co-workers found that the majority of older men and women in their 8th decade could not generate torque with a muscle shortening velocity greater than 270^o s^{-1}, during performance of isokinetic knee extensions, whereas younger men and women in their 3rd decade were all able to generate torque with an isovelocity of 300^o s^{-1} and the majority with 400^o s^{-1}. In

addition, the deficit in explosive power with age magnified with increasing 'isovelocities', which is also supported by McNeil *et al*. (2007) using isotonic contractions comparing three age groups of men in their 3rd, 7th and 9th decades. Trappe *et al*. (2003) describes this as a loss of *in vivo* maximal shortening velocity. Further evidence that muscle contraction velocity is reduced in the older population is provided by studies carrying out single twitch electrical stimulation (Davies *et al*., 1986; Vandervoort and McComas 1986; Duchateau and Hainaut 1987; Pääsuke *et al*., 2000; Thom *et al*., 2005; Klass *et al*., 2005; 2008). These investigations show that single twitch muscle contractile properties of time to peak tension (TPT) and half relaxation times (½RT) are significantly longer (by approximately 23% and 30%, respectively) between older adults in their 8th decade compared to younger adults in their 3rd decade from the planter flexors (Davies *et al*., 1986; Vandervoort and McComas 1986; Thom *et al*., 2006). However, there appears to be conflicting evidence on the effects of age on ½RT, a measure of the relaxation of a muscle contraction, as Pääsuke *et al*. (2000) and Thom *et al*. (2005) have shown that both older women and men's ½RT was unaffected using the same muscle groups. This shows that the efficiency of the sarcoplasmic reticulum and cross-bridge mechanics in the relaxation phase of the contraction may be unaffected by age in women (Klass *et al*., 2005). Nonetheless, the longer TPT is widely reported with age in both sexes, indicating a slower muscle contraction velocity that may be due to factors affecting the excitation-contraction coupling, changes in muscle architecture and/or an increase in tendon compliance (Vandervoort and McComas 1986; Pääsuke *et al*., 2000; Reeves *et al*., 2004; Thom *et al*., 2005; Klass *et al*., 2005; Narici and Maganaris 2006). Therefore, several physiological factors could account for the decline in contraction velocity that includes muscular and neural changes, which will now be discussed.

Muscle architecture changes regarding the muscle fibre (fascicle) length, which refers to the number of sarcomeres in series, has a significant influence on the velocity of muscle shortening (Wickiewicz *et al*., 1984; Lieber and Friden 2000; Kubo *et al*., 2003b; Narici *et al*., 2003). Thus, the observed reduction in fibre length with advancing age, as discussed previously, will contribute to a slowing of 'whole muscle' V_{max} (Kubo *et al*., 2003b; Narici *et al*., 2003; 2005; Morse *et al*., 2005).

Furthermore, the degree of influence the fibre length has on the muscle's functional characteristics is the acknowledgement that the variation in V_{max} between different muscle groups may relate more to differences in the fibre length than biochemical differences relating to the MHC isoform (Wickiewicz *et al*., 1984; Kubo *et al*., 2003b). Although a reduction in V_{max} does not affect P_o it does however result in a lower force production at all contraction velocities [Figure 1.6] (Lieber and Friden 2000; Macaluso and De Vito 2003; Narici *et al*., 2003; Morse *et al*., 2005). Combined with the decline in maximal muscle strength (P_o) this ultimately causes a significant leftward shift in the muscles F-V relationship, as demonstrated by older people during the production of both instantaneous peak power (Figure 1.4) and maximum short-term (De Vito *et al*., 1998; Martin *et al*., 2000; Macaluso and De Vito 2003; Lanza *et al*., 2003; Kostka 2005; Thom *et al*., 2005; Pearson *et al*., 2006).

Since, muscle PCSA and fascicle length are proportional to muscle strength and velocity of shortening, respectively, thus muscle volume (muscle volume = muscle PCSA x muscle length) is in turn proportional to muscle power (Sargeant *et al*., 1981; Martin *et al*., 2000; Trappe *et al*., 2003; Thom *et al*., 2005; Pearson *et al*., 2006). Therefore, by measuring the ratio between the muscle's power output and volume (specific power) it can be determined if factors other than muscle mass and architectural changes account for the decline in muscle power. Previously, Martin *et al*. (2000) found that the upper thigh limb muscle volume from trained younger and older men, across an age range from the 3rd to 8th decade, was reduced by approximately 16% and that specific power (that also included normalising for differences in V_{opt} at maximum peak power) accounted for 82% of the variability in short-term maximal power with age. In contrast, Thom *et al*. (2005), utilising MRI to measure muscle volume, found a 29% lower muscle volume that only accounted for approximately 50% of the decline in explosive maximal power of plantar flexors between the 3rd and 8th decade in sedentary men. They state that their measures of muscle volume and specific power are more accurate than those reported by Martin *et al*. (2000), as Martin and co-workers overestimated muscle volume due to including both the leg extensor and flexor muscles. Hence, the changes in muscle volume would not have account for the majority of the decline in muscle power. McNeil *et al*. (2007) also found similar results to Thom *et al*. using dorsiflexion

explosive power in young, old and very old men in their 3rd, 7th and 9th decades, respectively.

The literature on the effect of ageing on the *in vivo* V_{max} is found to be inconsistent (Thom *et al*., 2006). This may be due to differences in the body segments analysed, where the muscle groups studied would have employed different PCSA's, tendon arrangements, mechanical leverage and neural activation strategies (Bruce *et al*., 1997; Larsson *et al*., 1997; Thom *et al*., 2005). However, investigations have focused on single fibre maximum unloaded shorting velocity (V_o) from each fibre type in order to infer the effects of ageing on whole muscle contraction velocity. The V_o of a muscle fibre is dependent upon its MHC isoform expression, as fibres containing MHC-IIA or IIX isoforms produce a shortening velocity 4 -5 times faster and generate power 6 times greater than that of MHC-I type fibres (Larsson and Moss 1993; Larsson *et al*., 1997; Bottinelli and Reggiani 2000; Höök *et al*., 2001; Trappe *et al*., 2003; 2004; D'Antona *et al*., 2003; Canepari *et al*., 2005; Pearson *et al*., 2006). Analysis of single muscle fibres, using SDS-PAGE to identify fibre types and their V_o measured from slack-test manoeuvres, demonstrate that the V_o of fibres containing MHC-I or MHC-IIA isoforms declines within ageing muscle by approximately 46% and 30%, respectively, from the vastus lateralis (Larsson *et al*., 1997; Höök *et al*., 2001; Krivickas *et al*., 2001; D'Antona *et al*., 2003). Whereas, the effect of age on the V_o of MHC IIX is difficult to assess due to the small amount of these fibres found within muscle of both old and younger adults (Larsson *et al*., 1997; D'Antona *et al*., 2003). Krivickas *et al*. (2001) add that the effect of ageing differs between the sexes, with MHC-IIA fibres V_o being predominantly affected in older men whereas MHC-I fibres V_o are predominantly affected in older women. In addition, the V_o of MHC-I and MHC-IIA are significantly lower in older women than older men.

Hence, as aging is also associated with a selective atrophy and total loss in number of more powerful muscle fibres containing MHC-IIA, and an increase in co-expression of MHC-I-IIA (dominated by MHC-I), the changes in biochemical properties of a muscle leads to not only single fibres changing towards a slower phenotype, but in these phenotypes V_o also become slower with age. Combining these factors with a decline in whole muscle force production inevitably leads to a

muscle becoming less powerful, especially in older women (Larsson *et al*., 1979; 1997; Lexell *et al*., 1988; De Vito *et al*., 1998; Izquierdo *et al*., 2001; Lanza *et al*., 2003; D'Antona *et al*., 2003; Narici *et al*., 2003; Macaluso and De Vito 2004; Thom *et al*., 2005; Short *et al*., 2005; McNeil *et al*., 2007; Rauge *et al*., 2009).

Since V_o is found to increase in accordance with MHC-I, IIA to IIX in both younger and older men and women (Larsson *et al*., 1997; Bottinelli *et al*., 2001; Höök *et al*., 2001; D'Antona *et al*., 2003; Trappe *et al*., 2003; Canepari *et al*., 2005), the MHC isoform cannot account for the differences in V_o between single fibres containing the same MHC isoform between older and younger sedentary subjects, and thus other mechanisms must be responsible (D'Antona *et al*., 2003). This is also supported by the fact that V_o varies noticeably between fibres containing the same MHC isoform (Larsson *et al*., 1997; Bottinelli *et al*., 2001; D'Antona *et al*., 2003). The difference in V_o between fibres of the same MHC isoform was initially thought to be caused by changes in myosin light chains (MLC), but has now been disputed by D'Antona *et al*. (2003), as no differences in MLC were found with age even though V_o declined. Larsson *et al*. (1997) indicates that a decline in myosin ATPase activity may contribute to the lower V_o with age, which is supported by Höök *et al*. (2001). Due to the cause of the decline on V_o being unclear D'Antona *et al*. (2003) examined the velocity of sliding of actin (V_f), from only pure myosin isoforms, to determine if 'specific kinetics' within myosin were responsible for the change in V_o, as suggested by Höök *et al*. (2001). However, they found V_f to be consistent with V_o and concluded that 'glycation' of the myosin protein is more likely to explain a slower V_o with age (Höök *et al*., 2001; Ramamurthy *et al*., 2001; Canepari *et al*., 2005; Haus *et al*., 2007). Glycation formation is attributed to the reduced myosin (protein) turnover with age that makes myosin vulnerable to attacks from free radicals. Ultimately, this leads to an 'irreversible' formation of glycation on myosin protein (Ramamurthy *et al*., 2001). In addition, as myosin is an important molecule that converts energy from ATP hydrolysis, which produces specific muscular contractile characteristics, glycation has been found to cause functional and morphological changes within the muscle fibres from both animal and human studies with advancing age (Höök *et al*., 2001; Ramamurthy *et al*., 2001; Haus *et al*., 2007).

More recently, Pearson *et al*. (2006) measured not only muscle volume, using MRI on the specific extensor muscles generating short-term power during cycling, but also took into account the percentage of MHC-II within the active muscle mass generating muscle power. This is due to previous studies indicating a significant association between a lower V_{opt} and maximum short-term power being likely caused by detrimental changes in muscle quality, possibly attributed to a reduction in whole muscle MHC-II content (Larsson *et al*., 1997; Martin *et al*., 2000; Kostka 2005; Thom *et al*., 2005). The results from Pearson *et al*. (2006) confirmed that V_{opt} was significantly correlated with proportion of MHC-II content, in addition to finding that short-term maximum muscle power when normalised to muscle volume and MHC-II content explained the differences in muscle power with advancing age. With regards to instantaneous muscle power, they found that the relationship between MHC-II and V_{opt} at maximal peak power did not hold true, using the modified NPR. A possible explanation is that the gluteus maximus, which is a crucial muscle for hip extension, was not included in the measurement of muscle volume and neither was its MHC content analysed (Pearson *et al*., 2006).

Similar findings have also been produced by Trappe *et al*. (2003) investigating single fibre peak power normalised against cell volume. Their initial results showed that only significant difference in absolute power from single muscle fibres was observed from older women's MHC-IIA fibres in comparison to younger women and older men. These differences were annulled once normalised with cell volume, thus concluding that the quantitative change in muscle mass accounts for majority of the decline in muscle power. Nevertheless, their reported decline in whole muscle peak power (45% from older women) would not have totally accounted for by their observed reduction in whole muscle mass (15% from older women). In addition, muscle architectural changes would have also influenced the functional characteristics of the 'whole' muscle, which cannot be determined from analysis of single fibres. The limitations of Trappe *et al*. (2003) conclusion suggesting how changes in single fibres explain the difference in muscle power at the 'whole muscle level' is supported by Haus *et al*. (2007).

Martin *et al*. (2000: p M311) state that the generation of "muscle power represents the integration of neural and muscular function, and serves as an indicator of the integrity of the neuromuscular system", highlighting that the effects of ageing on MU behaviour may have negative consequences on the generation of muscle power. Furthermore, in order to generate muscle power optimally the remaining MUs have to innervate muscle fibres with a wide range of V_o, and therefore the decline in V_{opt} may also be attributed to changes in MU behaviour (Larsson *et al*., 1997; Kostka 2005; Thom *et al*., 2005). This is supported by Thom *et al.* (2005) as they found that the loss of muscle volume does not fully explain the decline in muscle power with age.

There are few studies investigating the effects of MU behaviour change with respect to the generation of muscle power, which can be attributed to the limitations in electrophysical techniques ability to measure MU behaviour during dynamic movements especially in older people (McNeil *et al*., 2007; Klass *et al*., 2008). However, results from Klass *et al*. (2008), using intramuscular EMG, showing MU discharge rates were lower in relation to the rate of torque development suggest that changes in MU behaviour may adversely affect the performance of explosive movements generating muscle power in the older population. Recent advancements in linear array sEMG, devised by Farina *et al*. (2004), now provide a method of studying central control strategies of neuromuscular control to determine changes in MU behaviour during the generation of muscle power from explosive dynamic movements (Pozzo *et al*., 2004). Linear array sEMG specifically estimates the conduction velocity of a MU action potential (MFCV), and as the MFCV is regarded as a size principle parameter (Andreassen Arendt-Nielsen 1987), which increases in accordance with recruitment of higher threshold MUs, thus differences in MU behaviour strategies with age can be inferred (Farina *et al*., 2004; 2004a; Pozzo *et al*., 2004; Macdonald *et al*., 2008). To date, the affects of ageing on MFCV have only been studied during fatiguing static contractions, discussed previously regarding 'fatigue paradox'. Whereas, Pozzo *et al.* (2004), using young adult males have shown that MFCV increased with force during single leg explosive contractions,

indicating that higher threshold MUs were recruited and an increase in MU discharge rate.

Other neural changes that may possibly affect muscle shortening is due to a reduction in the diameter and number of motor axons found after the 7th decade at the ventral roots of the spinal cord. This has been found to cause axonal conduction velocity to decline by approximately 10-15%, which may detrimentally affect muscle contraction and a reduced rate of force development due to the excitation-contraction coupling being affected (Wilmore 1991; Vandervoort 2002; Macaluso and De Vito 2004). In addition, the increase in connective tissue within the muscle belly may also contribute to a slower shorting velocity due to the muscles becoming stiffer (Narici and Maganaris 2006).

THE EFFECTS OF RESISTANCE TRAINING ON MUSCLE STRENGTH AND POWER

Training for Muscle Strength in Older Adults

Early training studies primarily focused on increasing maximal muscle strength (static and dynamic) and muscle mass in older people through heavy resistance training (HVRT) methods, as successfully demonstrated in younger adults (Fiatarone *et al*., 1990; Komi 1992; Newton and Kreamer 1994; McArdle *et al*., 1996; Hunter *et al*., 2004; Macaluso and De Vito 2004). Resistance training theories that exercising the muscles against high resistance loads would recruit the larger sized MUs (in accordance with the size principle), which are important for force development, due to the muscles working at the high 'strength' portion of their F-V relationship (Frontera *et al*., 1988; Jones and Round 1990; Komi 1992; Wilson *et al*., 1993; Kawamori and Haff 2004; Symons *et al*., 2005). The effectiveness of HVRT has been demonstrated by a reversal of sarcopenia (regarded as the primary cause of functional disability) in the older population through significant increases in muscle strength and mass (Larsson *et al*., 1979; Frontera *et al*., 1988; Fiatarone *et al*., 1990; Harridge *et al*., 1999; Hakkinen *et al*., 2002; Aagaard 2004; Hunter *et al*., 2004; Suetta *et al*., 2004; Macaluso and De Vito 2004).

In order to produce increases in muscle strength and mass HVRT programs have adopted the 'overload' principle of training, where high external load (intensity) are progressively increased in accordance with gains in the individuals maximal strength (McArdle *et al.*, 1996; Wilmore and Costill 1999; Symons *et al*., 2005). The exercise intensity is calculated relatively as a percentage of the individual's MVC or 1RM. These heavy resistance training programs typically adopt an intensity of 80% 1RM, whereas, explosive resistance training programs can adopt intensities between <50% to 80% 1RM (Kaneko *et al*., 1983; Komi 1992; Newton and Kreamer 1994; McArdle *et al*., 1996). Each training session involves exercising against the training load over a predetermined range of movement, repeatedly over a number of times, termed as repetitions (Newton and Kreamer 1994; McArdle *et al*., 1996). When a predetermined number of repetitions are completed this is referred to as a set. Rest periods follow both completion of a prearranged number of repetitions and completion of a set. The number of training sessions performed each week is described as the frequency, and the total number of weeks of training termed as the duration. Resistance training studies vary in number of repetitions, sets, intensity and duration, e.g. 3 sets of 8 reps using 80% 1RM twice weekly for 12 weeks. Furthermore, the number of times per week the training session is performed and the duration of the training program influences the training volume, which also varies between studies, as each resistance training program aims to produce specific physiological adaptations in muscle size and function.

Previously, Frontera *et al*. (1988) found that HVRT performed 3 days week^{-1} for 12 weeks, using 3 sets of 8 repetitions with a load of 80% 1RM, produced significant gains in quadriceps 1RM (107%), isokinetic strength (10-16%), whole muscle CSA (9%) and single muscle fibre CSA in both type I (33%) and II fibres (27%) of men aged 60-72 years. They crucially found that older men's muscles maintain their ability to hypertrophy and that sarcopenia is a reversible phenomenon. These findings influenced subsequent resistance training studies to investigate the benefits of this type of resistance training on muscle functioning in the older population. One of the most influential training studies carried out in the elderly population was conducted by Fiatarone *et al.* (1990), which utilised HVRT with a population of institutionalised older men and women in their 9th and 10th decades.

These participants performed 3 sets of 8 repetitions at an intensity of 80% 1RM, 3 days per week for 10 weeks. This resulted in an average increase in quadriceps strength (1RM) of 174%, 11.4% increase in muscle mass and a 48% increase in tandem walking speed. Surprisingly, some older participants who could not initially perform functional tasks such as rising out a chair unaided where now able to perform the task without assistance. However, these increases in absolute terms where small but in some cases significant to improve functional ability in frail older people above the threshold for functional independence. Hence, results from Fiatarone and co-workers highlight the significant benefits of resistance training as a method of improving functional capacity and reversing the effects of ageing on muscle mass and strength (Astrand 1992; Aagaard 2004; Barry and Carson 2004; Hunter et al 2004; Macaluso and De Vito 2004; Symons *et al*., 2005).

The exaggerated increases in maximal strength reported for increases in 1RM (107 to 174%) can be attributed more to a learning affect, which refers to an increase motor skill, than the smaller disproportionate increases in muscle mass or neural drive (Frontera *et al*., 1988; Harridge *et al*., 1999; Macaluso and De Vito 2004). To overcome this issue HVRT studies have focused on measuring MVC and isokinetic torque as a measure of maximal strength instead of the 'amount of weight lifted', which have shown to be smaller in magnitude but still significant with increases in KE and LP MVC ranging from approximately 17-37% using moderate to high resistance training (40-80% 1RM) in both older men and women in their 7^{th} to 10^{th} decade (Hunter *et al*., 2004; Latham *et al*., 2004; Macaluso and De Vito 2004; Reeves *et al*., 2004; Parente *et al*., 2008). In addition, these increases in maximal muscle strength can be explained more through neural and muscular adaptations (Aagaard 2004; Hunter *et al*., 2004; Reeves *et al*., 2004; Narici *et al*., 2005; Parente *et al*., 2008).

The results from these HVRT studies regarding improvements in muscle strength and CSA vary considerably, which is attributed to the different training program designs adopted and methods of measuring maximal strength (Harridge *et al*., 1999; Latham *et al*., 2004; Macaluso and De Vito 2004). In addition, these studies recruited different age groups, sexes, baseline fitness and health of participants, thus making it difficult to make comparisons between studies (Latham

et al., 2004; Macaluso and De Vito 2004). However, Latham *et al*. (2004) meta-analysis indicates that the most significant factor for improving strength is the training load (intensity) and not the duration of the training program. This is attributed to approximately 75% of the increases in muscle strength occurring during the first 12 weeks of HVRT (Rhodes *et al*., 2000; Hakkinen *et al*., 2001b; Macaluso and De Vito 2004; Latham *et al*., 2004; Parente *et al*., 2008). For a more detailed review of the effects of HVRT on muscle strength refer to Macaluso and De Vito (2004). Importantly, these training studies highlight the plasticity of muscle in the older population to resistance type training (Aagaard 2004; Macaluso and De Vito 2004; Narici *et al*., 2004; 2005; Suetta *et al*., 2004; Parente *et al*., 2008). The physiological mechanisms responsible for the improvements in muscle strength following resistance training can be divided into 3 phases of training (Sale 1988; Komi 1992; Aagaard 2004; Clarke 2004; Macaluso and De Vito 2004).

The first training phase, approximately weeks 1-2, can produce rapid significant gains in muscle strength, such as seen with increases in 1RM, which mainly relates to a 'learning effect' (Almåsbakk and Hoff 1996; Frontera *et al*., 1988; Harridge *et al*., 1999; Ivey *et al*., 2000; Hakkinen *et al*., 2002; Macaluso and De Vito 2004). During the second training phase (weeks 3-4) gains in muscle strength are accompanied by an increase in voluntary neural activation without parallel increase in muscle CSA. The increase in strength is attributed to a greater activation of the agonist muscle as indicated by a higher sEMG RMS, which is associated with an improvement in neural drive through either an increase in recruitment of higher threshold MUs (FT MUs) and/or MU discharge frequency or even a greater MU synchronization (Moritani and de Vries 1980; Macaluso *et al*., 2000; Hakkinen *et al*., 2001a; 2001b; 2002; Aagaard 2004; Macaluso and De Vito 2004; Reeves *et al*., 2004; Suetta *et al*., 2004; Narici and Maganaris 2006). Conversely, no changes in MDF have been demonstrated by HVRT in the older population, which indicates that either older people are unable to bring about further increases in stimulating higher FT MUs or that the sEMG technique is not sensitive enough to detect changes in MDF with resistance training (Macaluso *et al*., 2000; Farina 2008). The third training phase refers to improvements in muscle strength after 6 weeks of continuous resistance training that are accompanied by increases in

whole and single fibre muscle CSA (muscle hypertrophy). The research shows that older people aged 65-94 can increase their whole muscle CSA from the quadriceps by approximately 7 to 12% (Frontera *et al.*, 1988; 2003; Fiatarone *et al*., 1990; Hakkinen *et al*., 1998; Tracy *et al*., 1999; Izquierdo *et al*., 2001; Hakkinen *et al*., 2002; Bamman *et al*., 2003; Reeves *et al*., 2004; Suetta *et al*., 2004; Parente *et al*., 2008). These increases in whole muscle CSA are brought about through hypertrophy of type I and IIa fibre types ranging from 25-30%, following 10-24 weeks of progressive HVRT (Frontera *et al*., 1988; Hakkinen *et al*., 1998; 2001b; 2002; Trappe *et al*., 2000; Frontera *et al*., 2003). Conversely, Bamman *et al*. (2003) add that it appears that adaptations in older women's single fibre CSA to 26 weeks HVRT may be blunted in comparison to older men of the same age, as older men produced an average 40% increase in single fibre CSA of type I and II fibre types whereas older women only increased by an average of 7%. They also state that MHC-IIA content increased significantly in both age groups, which is supported by a Hakkinen *et al*. (2002) 24 week explosive resistance training study using ATPase histochemical analysis. Although this is disputed by Canepari *et al*. (2005) adopting SDS-PAGE over the 12 week progressive resistance training program using 4 older men, aged between 68 and 82 years. Parente *et al*. (2008) investigated if a longer term one year resistance training program, utilising a 60% 1RM, would overcome the blunted training responses in MHC isoform from older women aged in their 8th decade, as observed by Bamman *et al*. (2003) and Trappe *et al*. (2003). They found a highly significant bi-directional shift in muscle fibres containing MHC-I and IIX isoform towards MHC-IIA, leading to a 77% increase type IIA fibres and a decline in type I fibres that approached statistical significance. The specific force of type I and IIA single muscle fibres were also significantly increased without any change in their CSA, which is contrary to that reported by Trappe *et al*. (2003) and possibly attributed to the differences in training intensity and duration. More recently Raue *et al*. (2009) has demonstrated that older women aged in their 9th decade and above have a limited capacity to increase whole muscle and single fibre CSA and peak force, utilising a HVRT consisting of 12 weeks of training at 70-75 % 1RM every three days. These results suggest that older women above the 9th decade, who make up about 70% of the population older than 80 years, are even more vulnerable to

ageing due to limitations in their muscles ability to hypertrophy with HVRT (Raue *et al*., 2009). In general, it does appear that HVRT can reverse the effects of a 'selective atrophy' of type II fibres with ageing, together with an increase in fibres containing MHC-IIA isoform content in older women in their 7th and 8th decades (Parente *et al.*, 2008).

Neural factors may also contribute to the increase in muscle strength during the third training phase, as Hakkinen *et al*. (2001b) found that after 6 months explosive resistance training program, twice weekly with an intensity of 10RM for 10 repetitions over 5 sets, produced a significant reduction in KE co-activation from older adults in their 8th decade. Hence, the increase in muscle strength may also be explained through a reduction in opposing force produced by the antagonist muscle, which may also be part of a learning effect (Hakkinen *et al*., 2001b). Conversely, de Boer *et al*. (2007) found that plantarflexor co-activation increased after one year of resistance training using older adults in their 8th decade. This led de Boer and co-workers to conclude that as co-activation between opposing muscle groups is crucial for joint stability thus an increase in co-activation occurs as a consequence of gains in both agonist and antagonist muscle strength.

Moreover, Reeves *et al*. (2004) investigated the effects of a 14 week progressive resistance training program, performed 3 times per week, on the specific strength of the vastus lateralis (quadriceps) taking into account changes in muscle architecture and neural drive from older men and women in their 8th decade. They highlight that previous studies may not have measured whole muscle specific strength appropriately as they did not take into account maximal isometric strength, maximal neural activation and the muscle's PCSA. Their post-training results show that muscle specific strength increased significantly by 19% without an increase in PCSA or a reduction in co-activation. However, as the anatomical CSA was significantly increased after training, whereas the PCSA did not change, this may indicate errors in their method of determining PCSA. Reeves and co-workers also found a significant increase in neural drive, indicated by a 40% higher RMS, that may be regarded as the main contributing factor in the improvement in older adult's whole muscle specific strength after 14 weeks of resistance training.

Training for Muscle Power in Older Adults

Over the last decade numerous studies emphasize the importance of developing exercise strategies that produce optimal gains in muscle power due to establishing that HVRT produces significantly greater increases in muscle strength than muscle power, HVRT not always producing improvements in functional ability and the augmented decline in muscle power in comparison to muscle strength having a greater association with functional disability (Earles *et al*., 2000; Evans 2000; Bean *et al*., 2004; Macaluso and De Vito 2004; de Vos *et al*., 2005; Hazell *et al*., 2007; Henwood *et al*., 2008). The improvements in older adults muscle power produced from HVRT ranges from 18 to 25%, which are disproportionate to the larger increases observed in muscle strength (Macaluso and De Vito 2004; de Vos *et al*., 2005; Hazell *et al*., 2007; Henwood *et al*., 2008). de Vos *et al*. (2005) add that only a relatively small number of studies have been specifically designed to improve muscle power optimally in the older population. Hence, in more recent times the direction of exercise research has focused more on improving muscle power optimally in older population (Fielding *et al*., 2002; Macaluso *et al*., 2003; de Vos *et al*., 2005; Signorile *et al*., 2005; Caserotti *et al*., 2008; Henwood *et al*., 2008). A number of these studies theories that in order to produce optimal gains in muscle power the training velocity has to be specific that that producing maximal power, as determined by the muscle's F-V and P-V relationships (Kanehisa and Miyashita 1983; Kaneko *et al*., 1983; Newton and Kraemer 1994; Earles *et al*., 2000; Macaluso *et al*., 2003; Macaluso and De Vito 2004; de Vos *et al*., 2005; Hazell *et al*., 2007; Henwood *et al*., 2008). Therefore, training muscle power requires recruiting faster speeds of movement than performed during HVRT (high load-low velocity movement), and thus to improve muscle power resistance-training regimes should utilise a lower intensity that enables a higher velocity of movement to be achieved in accordance with the muscle's F-V relationship (Newton and Kraemer 1994; Earles *et al*., 2000; Macaluso *et al*., 2003; de Vos *et al*., 2005; Hazell *et al*., 2007; Henwood *et al*., 2008).

Previously, Earles *et al*. (2000) compared the effects of 12 weeks resistance 'power training' using an intensity of 50-70% 1RM while adopting rapid movements

against a specific walking training program on muscle power, strength and functional tasks with older adults in their 8th decade. Earles and co-workers found that leg peak power increased 150% (at the higher muscle force portion of the F-V relationship) and maximal power increased 22% in the power-training group with no changes observed in the walking group utilising pneumatic resistance machines. In addition, the power training group's MVC increased by 22% compared to 12% in the walking group. Nevertheless, the significant increase in muscle power did not result in significant improvements in functional ability, which may have been attributed to the high initial fitness of the participants at the onset of the training program. However, they conclude that high-velocity resistance training produces significant gains in muscle power, and state that "Because it is clear that training to improve strength will affect muscle function differently than training for velocity and power, it is important to determine if one mode of training is superior to another in maintaining physical performance and functional independence in older adults." (Earles *et al*. 2000, p877).

Table 1.1 summarizes the effects of numerous training studies over the last decade that have investigated the effects of resistance training on muscle power in the older population. However, only a relative few of these studies to the author's knowledge have specifically investigated the effects of a high-velocity low-load versus a low-velocity high-load training program on muscle power in the older population (Fielding *et al*., 2002; Macaluso *et al*., 2003; de Vos *et al*., 2005; Signorile *et al*., 2005; Henwood *et al*., 2008). These studies indicate a wide range of increase in muscle power (9% to 97%) that may be attributed to using different training intensities, duration, movement velocities, methods of testing muscle power and movement specificity between training and testing modes (Macaluso and De vito 2004; Hazell *et al*., 2007). Among these studies, Fielding *et al*. (2002) compared the effects of low and high velocity resistance training over 16 weeks on healthy older adults in their 7th and 8th decade. Participants trained with the same relative intensity (70% 1RM) but with one group exercising with a higher velocity of movement and all groups performing the same amount of mechanical work during each session. The results showed significantly greater gains in muscle peak power in the high-velocity training group compared to low-velocity resistance training in relation to the leg

press but not during knee extension peak power (97% vs. 45% and 33% vs. 25%, respectively). This is possibly due to the difference in the training power output between the high and low velocity training groups being reduced during the knee extension training than in comparison to the leg press training (Fielding *et al*., 2002). Furthermore, the increase in dynamic strength was similar between the two resistance training methods 33% vs. 35% and 41% vs.45% for leg press and knee extension, respectively.

Whereas, Macaluso *et al*. (2003) adopting cycling ergometry, as a novel approach to resistance training, utilised 3 training modes (high velocity-low load, low velocity-high load and combined loads) with older women in their 7^{th} decade to investigate their effects on maximal peak power, MVC and selected functional abilities. They tested the hypothesis that training with a high-velocity low-load would produce greater increases in muscle power than training with a low-velocity high-load, which would in-turn produce greater increases in maximal strength. Their results showed that the 3 training regimes produced similar improvements in muscle peak power, MVC and functional ability. Hence, questioning the theory that high velocity training produces optimal gains in muscle power and functional abilities, which is supported by Almåsbakk and Hoff (1996) using untrained females in their 3^{rd} decade. Macaluso and co-workers add that the amount of mechanical work done was the same between the 3 training modes and by their estimations approximately the same training average power output. In addition, conclude that the increase in strength may have been related to the amount of mechanical work and the increase in muscle power possibly dependent upon the power output generated during training session, or that to produce greater improvements in muscle power participants may have to work closer to their muscles V_{max}.

The results from Fielding *et al*. (2002) and Macaluso *et al*. (2003) infer that the stimulus for improvements in maximal power and functional ability would seem to be the training power output. For instance, Fielding *et al*. (2002) found that the high velocity-training group, which produced the greatest increases in muscle power, trained at a higher power output than the low velocity-training group. Hence, training at a higher power output i.e. at maximum power that occurs with a load that enables V_{opt} to be achieved, should in theory produce greater gains. Nonetheless, Fielding

and co-workers did not mention if their participants were training at or near their maximal power output. While Macaluso *et al.* (2003) found that their modes of resistance training resulted in approximately the same training power output per session, using high and low velocities of movement, produced similar improvements in muscle power and strength. Therefore, optimising the training intensity to produce maximal power during the training session may have produced significantly greater increases in muscle power and possibly functional ability. This is a unique training hypothesis that has not been investigated in relation to older people. These findings are however supported by Mastropaolo's (1992) 'maximum-power stimulus theory', which has been shown to the most effective stimulus for improving muscular strength, power and hypertrophy in college aged students. Mastropaolo compared the effects of a resistance training intensity slightly greater than that producing maximal power output against HVRT at 75-95% 1RM. The findings showed that the 'power training' significantly increased maximum power and that the increase in strength was correlated with the increase in maximum power. Therefore, concluded that the strengthening stimulus is maximal power output, as it appears that the closer individual's exercises to their maximal power output the greater the increases in maximum power, strength and muscle hypertrophy (Mastropaolo 1992).

More recently, de Vos *et al.* (2005) carried out a 'systematic dose-response' to identify the optimal load that maximizes increases in muscle power using pneumatic resistance machines in older adults aged 69 to 75 years. The older participants were resistance trained using one of three intensities 20, 50 or 80% 1RM over 8-12 weeks. de Vos and co-workers results indicate that the gains in peak power were higher with the 50% 1RM group than in the 20% 1RM group, with the gains in 80% 1RM group equivalent to the 50% 1RM group. This led to the conclusion that resistance training with a heavier intensity may be more beneficial for improving both muscle power and strength in the older population. In contrast, Signorile *et al.*

Table 1.1 Effect of resistance training on muscle power in older adults. (BP bench press, CB combined intensity, F female, FEW flywheel weight training, G training group and load as a % 1RM, GW gravitational weight training, HE hip extension, HI high velocity training, HS half squat, KE knee extension, LE leg extension, LO low velocity training, LP leg press, M male, NPR Nottingham power rig, PRM pneumatic resistance machine, PP load that produces maximum peak power, PW power training group, SP speed training group, ST strength training group, 1RM one repetition of the maximum weight lifted, VJ vertical jump).

Authors	Participants			Training Program						Testing	Power gain	Measurement
	Age	Sex	N	Exercise movement	Duration (weeks)	Sessions per week	Sets	Repe-titions	Intensity	move-ment		apparatus
Skelton et al. 1995	76-93	M/F	20	Dynabands	12	3	3	4-8		LP	18% (NS)	NPR
De Vito et al. 1999	60-70	F	11	Low intensity general conditioning	12	3	-	-	-	VJ	24%	Force Platform
Izquierdo et al. 2001	64 (2)	M	11	KE, HS, BP	16	2	3-4	8-15	50-80% 1RM	KE, HS, BP	21-37%	Instrumented weight-stack
Earles et al. 2000	77 (5)	M/F	18	LP	12	3	3	10	50-70% 1RM	LP	22%	PRM
Fielding et al. 2002	73 (1)	F	30	LP	16	3	3	8-10	70% 1RM	LP	HI: 97% LO:45%	PRM
Macaluso et al. 2003	69 (2)	F	38	Cycling as fast as possible	16	3	8 4x4	SP: 16 ST: 8 CB: 16/8	40% 2RM 80% 2RM As above	LP	19% all groups	Isotonic dynamometer (optimisation)

Ferri et al. 2003	65-81	M	16	Calf raise, KE	16	3	1	8-10	80% 1RM	KE	24%	Isokinetic dynamometer
Miszko et al. 2003	65-90	M/F	65	LP, LE	16	3	3	6-8	SP: 40% ST: 50-70% 1RM	Cycling	SP: 8% ST: 10%	Peak anaerobic cycle power 30s Wingate
Ramsbottom et al. 2004	70 +	M/F	22	General conditioning and dynabands	24	2	-	-	-	LP	40%	NPR
de Vos et al. 2005	68 (5)	M/F	112	LP, BP, bilateral LE	8-12	2	3	8	G20: 20% G50: 50% G80: 80%	LP LE	9-12% 14-18% all groups	PRM
Delmonico et al. 2005	50-74	M/F	62	KE PRM	10	3	5	5-20	50-85% 1RM	KE	M:10% (NS) F: 5%	PRM
Signorile et al. 2005	61-75	F	17	KE Isokinetic training	9	3	1-3	4-10	PW: 4.73 ST: 1.05 rad/s	KE	PW: av 50% ST: av 36%	Isokinetic dynamometer
Caserotti et al. 2008	60-65 80-89	F	65	KE, LP	12	2	4	8-10	75-80% 1RM	LP	60-65: 12% 80-89: 28%	NPR
Henwood et al. 2008	65-84	M/F	67	BP, LP, LE	24	2	3	8+	HI: 45-75 ST: 75 % 1RM	LP, LE	HI: 50.5% ST: 33%	Instrumented weight-stack
Onambele et al. 2008	69 (1)	M/F	24	KE: Isoinertial training vs. weight training	12	3	1-4	8-12	GW: 80% 1RM FEW: PP	KE	GW: NS FEW: 28%	Isokinetic dynamometer

(2005) adopting isokinetic resistance training compared training velocities of 4.73 rad/s, termed power training, versus a strength training velocity of 1.05 rad/s. The older women aged between 61-75 years performing with the power training velocity produced the highest gains in average isokinetic power and the strength velocity group revealed a greater increase in peak torque. Hence, suggesting that movement velocity is more beneficial to improving muscle power in older women, as highlighted by Earles *et al*. (2000). Henwood *et al*. (2008) compared different movement velocities on muscle power through using traditional HVRT with a load 75% 1RM against a power training protocol that required participants to perform rapid movements against intensities of 45% to 75% 1RM over 3 sets, using older adults aged in their 7^{th} and 8^{th} decades. Their results showed similar significant increases in peak power and maximal strength from both training modes, but the power training group produced gains in muscle power and strength using less total mechanical work done. The power training group also produced larger gains in muscle power but this did not reach significance in comparison to the strength training group. These results concur with de Vos *et al*. (2005) and Macaluso *et al*. (2003) indicating that medium and high intensities can increase muscle power to a similar extent.

Furthermore, de Vos *et al*. (2008) have shown that the gains in peak power are more dependent on increases in force than velocity, which is supported by Macaluso *et al*. (2003) using isotonic dynamometry to investigate increases in muscle power from a novel cycle resistance training program. Therefore, it may be more beneficial to train with a high intensity (high force portion of the F-V relationship), however high velocity training has also been shown to increase muscle power and may relate more to the performance of numerous functional tasks and prevention of falls (Earles *et al*., 2000; Signorile *et al*., 2005; Onambele *et al*., 2009). The differences in the gains in muscle power produced by these studies may be explained through the training power output produced during each training session, which as stated earlier may be the stimulus for the optimal gains in older women's muscle power that to the author's knowledge has not been previously carried out. Nonetheless, Macaluso *et al*. (2003, p 2544) add that " (it) is still controversial whether high-velocity low resistance training is comparable or superior to low-

velocity high-resistance training to improve strength, power, and functional abilities in older individuals", which highlights the need for further research into developing effective training programs for older people. This suggestion is repeated by Hazell *et al*. (2007), as to date the most effective training protocol for improving muscle power and functional ability has not been established and thus "warrants continued greater research efforts."

Resistance training utilising either a high intensity-low velocity or high velocity-low intensity specifically involves recruiting type IIA muscle fibres (Henwood *et al*., 2008). Since these fibres appear to experience a selective atrophy and reduced proportion within the muscle with ageing, which crucially develop force and velocity 5-6 times greater than type I fibres, any resistance training mode that brings about increases in type II fibres size, proportion or quality will thus in turn improve muscle power through a rightwards shift in the muscle's F-V relationship. The physiological mechanisms discussed previously that bring about an increase in muscle strength with HVRT may also be applicable to resistance training for muscle power, as increases in muscle power may be predominantly due to gains in muscle force generation than movement velocity. These physiological factors explain the increase in muscle torque for a given velocity of shortening.

With regards to muscle velocity, Canepari *et al*. (2005) found that after progressive resistance training the single muscle fibres from the vastus lateralis increased their V_o. They showed that the actin sliding velocity (V_f) of type IIA fibres was significantly increased by 19% in older men, in agreement with Trappe *et al*. (2000). However, Trappe *et al*. (2001) found no change in V_o from older women, which concurs with Raue *et al*. (2009). Whereas, Parente *et al*. (2008) demonstrated a significant increase in V_o of type I fibres after a longer term 12 months resistance training with an intensity of 60% 1RM in older women. The mechanism responsible for an increase and decrease in V_o are still not fully understood (Canepari *et al*., 2005; Parente *et al*., 2008). Hence, it appears that the participant's sex and age and the training duration may have a significant bearing on the older population's ability to improve the muscles maximum shortening velocity (Trappe *et al*., 2001; Parente *et al*., 2008; Raue *et al*., 2009).

Neural adaptations may also be applicable to gains in muscle power, with RMS increasing rather than MDF in older people (Hakkinen *et al*., 1998; Sale 1988; Izquierdo *et al.*, 2001; Macaluso *et al.*, 2003; Ferri *et al*., 2003). Specifically, it has been demonstrated that older women have a blunted response in their ability for muscular hypertrophy in comparison to older men, and thus may rely more on neural factors and muscle architectural changes to bring about increase in muscle peak power with resistance training (Delmonico *et al*., 2005; Narici and Maganaris 2006). The neural mechanisms that result in an increase in muscle power are difficult to assess due to limitations in sEMG recorded during dynamic contractions (Macaluso and De Vito 2004). However, with the advancements in linear array sEMG technique, discussed previously, this technique may provide a unique insight into the effect of resistance training on MU behaviour responsible for the increase in muscle power, V_{opt} and/or F_{opt}. Previously, Van Cutsem *et al*. (1998) found that 12 weeks 'explosive resistance training' with an intensity 30-40% 1RM, similar to high-velocity low-load power training, produced significant increases in MU discharge rate that accompanied significant gains in contraction velocity and the rate of torque development. Whereas, Klass *et al*. (2008) have recently shown that MU behaviour (discharge frequency) was associated with a lower rate of force development in older adults, which is also a crucial factor in the development of explosive muscle power. Therefore, an increase in neural drive may explain the improvements in muscle power following resistance training. Another possible mechanism for increased muscle power in highlighted by Reeves *et al*. (2003) showing a stiffer musculo-tendon unit after resistance training, where an increase the transfer of force from the muscle to the limb action may assist in increasing explosive muscle power. Furthermore, Onambele *et al*. (2008) found an increase in isokinetic peak power (28%) from iso-inertial resistance training was accompanied by a 136% increase in tendon stiffness.

RATIONALE AND AIMS OF THE EXPERIMENTAL CHAPTERS

The inevitable decline in muscle strength and power that occurs after the 7th decade of life has significant detrimental consequences on older individuals' ability to remain functionally independent, especially in women. More recently, muscle power has been shown have a greater association with the performance of dynamic movements that occur more often in the activities of daily living than strength per se, and declines to a greater extent than muscle strength with advancing age. Therefore, muscle power is regarded as being more relevant to assessing functional ability in the older population than muscle strength per se. Several physiological mechanisms are responsible for the decline in muscle power and strength, which include neural, muscular and tendinous factors. All of which have been extensively reviewed in chapter 2 of this thesis, as have the effects of resistance training on these physiological parameters. The review indicates that there is limited literature on the neural factors responsible for the decline in muscle power, as well as identifying the most effective mode of resistance training to improve muscle power optimally in older people. Hence, the overall aim of this thesis was to investigate the effect of ageing on the neural activation and strategies during the development of muscle power, using linear array sEMG, in addition to determining the resistance load that produces optimal gains in older women's muscle power and the neural adaptations that may accompany increases in their muscle power.

Chapter 3

Rationale: Performance based laboratory test are limited in their ability to assess 'real world' performance of activities of daily living, which is crucial for assessing functional disability in the older population. Producing an 'all-out' effort on a friction-loaded cycle ergometer from repeated muscular contractions uniquely enables the assessment of maximum peak power with the muscles experiencing similar conditions to those encountered during the performance of everyday activities of daily living. This is due to the muscle's force generating capacity changing in accordance with its force-velocity relationship, such as would be encountered during stair climbing. However, the measurement of maximal power from the cycle

ergometer's flywheel acceleration is commonly conducted using commercially incremental encoders. An alternative method of measuring power output, and its determinants, could be achieved using stereophotogrammetry determined from motion analysis.

Research questions: Is the assessment of muscle power output and its determinants from motion analysis performed on the flywheel of a friction-loaded cycle ergometer, during short-term 'all-out' cycling, valid and reliable in older women?

Chapter 4

Rationale: The decline in short-term maximal muscle power with advancing age has severe detrimental consequences on older women's functional ability. Several physiological mechanisms including muscular and tendinous factors are responsible for a decline in both muscle force and movement velocity that detrimentally affects muscle power. However, the effects of ageing on the neural factors during muscular contractions generating power are less well understood due to limitations in measurement techniques. Advancements in linear array surface electromyography now provide a non-invasive method of studying neural activation and strategies, which govern muscular contractions, during the performance of explosive dynamic contractions, either through sEMG amplitude, median frequency or through the measurement of muscle fibre conduction velocity (MFCV). MFCV is regarded as a size principle parameter and thus can be used to infer changes in MU behaviour that accompany the decline in muscle power during short-term 'all-out' cycle ergometry, which replicates the muscular contractions encountered during the performance of everyday activities of living.

Research question: Is the decline in short-term muscle power and its determinants, across all loads tested, associated with detrimental changes in neural activity? Are there any differences in the neural strategies between young and older women during the development of muscle power?

Chapter 5

Rationale: Lower leg explosive peak power reflects the important functional gesture of rising from a chair, the performance of which has a significant bearing on older population's ability to remain functionally independent. However, the literature is limited in determining the impact of the effects of ageing on neural drive, which govern explosive single muscular contractions, during the generation of explosive peak power. In addition, muscle contractile properties, investigated using single-twitch electrical stimulation, are known to decline with age and thus provide an indication of the peripheral changes affecting explosive muscular contractions independent of neural factors. Therefore, measuring neural activity and contractile properties allow an estimate of the contribution of both neural and muscular factors on the development of explosive peak power, and its determinants.

Research question: Is the decline in older women's explosive peak power and its determinants, after an optimisation of the load, associated with changes in both neural activation and muscle contractile properties?

Chapter 6

Rationale: Exercise resistance training studies have been shown to reverse or attenuate the decline in muscle power with advancing age. However, the load and speed of movement that produces optimal gains in muscle power in the older population still remains elusive. The stimulus for optimal gains in muscle power may be training at maximal power. Uniquely, all-out cycling on a friction-loaded cycle ergometer results in maximal power generated across a wide range of friction loads. Hence, comparing two different friction loads that allow the generation of maximal power output, but with two significantly different rates of pedalling velocities, can be adopted to determine if training at maximal power is the stimulus for optimal gains in older women's muscle power. With the advancements in linear array surface electromyography neural activity and control strategies can be investigated to determine if neural factors contribute to the gains in muscle power with cycle resistance training.

Research question: Is the training stimulus for optimal gains in older women's muscle power the friction-load that allows the development of maximum

peak power using cycle ergometry? Do changes in neural activation accompany increases in muscle peak power? And are neural control strategies affected by the changes in motor unit behaviour?

CHAPTER 3

The validity and reliability of motion analysis in measuring power output during ‘all-out’ 6 second cycling on a friction-loaded cycle ergometer

ABSTRACT

The purpose of this study was to determine the validity and reliability of motion analysis as a method of measuring maximum peak power output during 'all-out' 6 s cycling on a friction-loaded cycle ergometer. The validity and reliability of motion analysis in measuring the power output from the acceleration of a flywheel, using friction-loaded cycle ergometry, was assessed against a proven direct measure of power output using pedal strain gauges. Eight healthy older participants (aged 70-80 years) performed 'all-out' 6 s cycles against loads of 20% and 60% of the maximum load they could turn through two complete pedal revolutions (2RM). A sub-group of 5 participants performed an additional two within-day 6 s 'all-out' cycles using a 40% 2RM load. The same procedure was repeated on a further 2 trial days with 14 days between trials. During each 'all-out' cycle, measurements were recorded simultaneously from motion analysis and pedal strain gauges. Motion analysis produced a systematic non-significant overestimation of both average 6 s and maximum power. The correlation coefficients where high between motion analysis and pedal strain gauges for both within and between-day measurements (ranging from; r=0.951-0.985). The intraclass correlation coefficients, ranging from 0.882-0.996, indicate motion analysis as a suitable method of measuring mechanical power output during an 'all-out' sprint cycling on a friction-loaded cycle ergometer. Hence, motion analysis can be utilised as an alternative method in measuring the mechanical power output from the flywheel acceleration on a cycle ergometer.

Introduction

The generation of maximal muscle power (Pmax) requires maximal effort over a short duration of approximately 4-6 seconds, involving repeated muscular contractions, which in turn leads to a rapid acceleration of whole body or body segments such as the lower limbs (Sargeant *et al.*, 1981; Newton and Kraemer 1994; Pearson *et al*., 2004). The assessment of lower limb Pmax in the older population is regarded as a more crucial measurement for predicting functional status than maximal strength per se (Margaria *et al*., 1966; Martin *et al*., 2000; Foldvari *et al*., 2000; Cuoco *et al*., 2004; Macaluso and De Vito 2004; Kostka 2005; McNeil *et al*., 2007). The importance of measuring Pmax in the older population is attributed to muscle power's greater association with the performance of dynamic movements, which occur more often in activities of daily living, than maximal strength that is determined through maximal isometric contractions (Bassey *et al*., 1992; Foldvari *et al*., 2000; Lauretani *et al*., 2003; Macaluso and De Vito 2004). In addition, as the effects of ageing are exacerbated in older women, making women more vulnerable to loss of functional independence and reduced quality of life than men, they are thus chosen as the target group for this study (Skelton *et al*., 1994).

Foldvari *et al*. (2000) highlight limitations between performance based laboratory tests aimed at assessing functional abilities and the actual muscular conditions experienced during performance of 'real world' activities of daily living. In order to successfully perform activities of daily living, such as walking and stair climbing, individuals are required accelerate their body's centre of mass over a distance, which causes the muscles of the lower limbs to perform along their force-velocity (F-V) relationship. However, most laboratory tests do not assess the ability of the muscle to work along the F-V curve 'naturally' with the exception of sprint cycling using cycle ergometry, which allows the muscles to accelerate a sub-maximal inertial flywheel on a cycle ergometer over a short duration of approximately 6-seconds (Sargeant *et al*., 1981; Seck *et al*., 1995; Martin *et al*., 1997; Pearson *et al*., 2004). Hence, 'all-out' sprint cycling on a friction-loaded cycle ergometer involves the muscles experiencing similar conditions to those encountered during the performance of activities of daily living, where the contracting muscles

follow their F-V relationship (Sargeant *et al*., 1981; MacIntosh *et al*., 2000; Pearson *et al*., 2004).

Various investigators have utilised strain gauges for accurately measuring pedal forces and power output during cycling (Daly and Cavanagh 1976; Sargeant and Davies 1977; Sanderson and Cavanagh 1985; Boyd *et al*., 1996; Sanderson *et al*., 2000; 2003; Mornieux *et al*., 2006). Other numerous investigators have measured Pmax from the angular velocity of the flywheel using a friction-loaded cycle ergometer (Arsac *et al*., 1996; Martin *et al*., 1997; Morin and Belli 2004; Pearson *et al*., 2004). These investigations relied on the use of commercially available incremental encoders, with a recording resolution of approximately 100 Hz, to measure the acceleration of the cycle's flywheel. An alternative method utilising motion analysis (five camera Vicon system), with a recording resolution of 250Hz, has been devised to measure the change in angular velocity using stereophotogrammetry. Thus, the purpose of this study is to determine the reliability and validity of motion analysis as a method of estimating power output during cycling, against a proven direct measure of power output from utilising strain gauges incorporated into standard bicycle pedals.

Methodology

Participants

With University ethics approval, 8 "medically stable" older participants, aged 75.5 ± 3.2 yrs (mean ± SD); stature 1.58 ± 0.06 m; body mass 64.7 ± 10.8 kg, were selected according to the criteria proposed by Greig *et al.* (1994) for participating in an exercise study. A sub-group of 5 participants performed the within-day trials of mechanical power output measurements. Participants provided written informed consent and were instructed to maintain their usual levels of physical activity throughout the duration of the study. After an initial familiarisation period participants re-visited the laboratory on a further three occasions with at least 14 days between exercise trials. The exercise trials consisted of maximal dynamic strength and power output assessments on a cycle ergometer. All participants completed the study.

6 second All Out Cycle test

All measurements involving motion analysis and the instrumented pedals (force transducer) were carried out simultaneously on a mechanically-braked cycle-ergometer (Monarch 823e, Sweden). Seat height was adjusted so that the participants could cycle with their heels touching the pedals and no sideways movement of the hips during pedalling. Each participant wore cycling shoes with cleats (Shimano® SPD) that ensured a secure fitting to the instrumented pedal during the required 'all-out' sprint efforts.

After an initial warm up period of 5 minutes, with a sub-maximal load (50-60 W), participants were tested for the maximum load each individual could pedal through 2 complete revolutions while remaining seated, referred to as 2 revolution maximum (2RM) (Macaluso *et al.*, 2003). During each attempt participants were asked to remain seated at all times whilst pedaling. Starting from the load at which the participants could not turn the pedals, the load was then reduced by 0.5 kg decrements until the participants were able to turn the pedals through two full revolutions. To ensure that the load was effectively the maximum, participants were required to perform further attempts by increasing the load in 0.5 kg increments. Each attempt was followed by a 2 minute recovery period, and once the 2RM was established the participants rested for an additional 5 minutes. Each volunteer was then asked to cycle as fast as possible for at least 6 seconds against 3 loads corresponding to 20%, 40% and 60% of their 2RM, in a random order. Verbal encouragement was given during each trial, and after each 'all-out' cycle trial a 5 minute recovery period was carried out before attempting the next load. The standardised stationary starting position was with the right knee bent at 90 degrees for all cycle trials.

Instrumented Pedals (force transducers)

Instrumented pedals, as shown in Figure 2.1, were attached to the left and right hand crank arms on the cycle ergometer in order to measure torque and power output applied to the flywheel, as developed by Bibbo (2008). The instrumented pedals measure the forces applied to each pedal independently, in a perpendicular (Fz) and anterior-posterior (Fx) direction (medio-laterial forces are not measured as

they are regarded as being negligible in comparison to Fz and Fx), in addition to the relative change in crank angle with respect to the pedal (θp). The forces exerted on pedals during cycling are directly applied to the load cell, which transmits the force to the crank through the stirrup and the spindle, thus enabling the participant to cycle as though using a standard pair of pedals. The measurement of crank angle θp, using the 'smart encoder', is necessary for determining the tangential or 'effective' force (*Ft*) causing crank rotation. The load cell is based on a strain gauge system connected to an electronic box containing two Wheatstone bridges (Figure 2.2). The electronic box from each pedal also contains 3 BNC coaxial connectors, two channels for the force components and one channel for the pedal angle, which were attached to an analog-digital converter on the Vicon Workstation (Vicon 612, California, USA) with the data recorded at 1000Hz. Data was then saved onto the hard drive of a personal computer (PC, Dell, Texas, USA).

The instrumented pedals were calibrated according to Bibbo (2008), which involved applying a series of known loads to each pedal in the Fz and Fx direction. For instance, to measure the Fz component the instrumented pedal was positioned on a hard flat surface and secured before known weights were added. The corresponding voltage outputs were then recorded from the Fz and Fx channel outputs from the electronic box. The same procedure was repeated for the Fx components, in both anterior and posterior directions, with the pedal securely positioned at a 90° angle with respect to the horizontal. Four known loads varying from zero to 200N were used. Voltage signals were measured from both output channels (Fz and Fx) when a load was applied in order to assess the cross-talk between channels and to determine the affect of the applied force on the overall channel voltage output. All the procedures described above were repeated for consistency, and performed prior to the 1st day of testing and after the last day of testing.

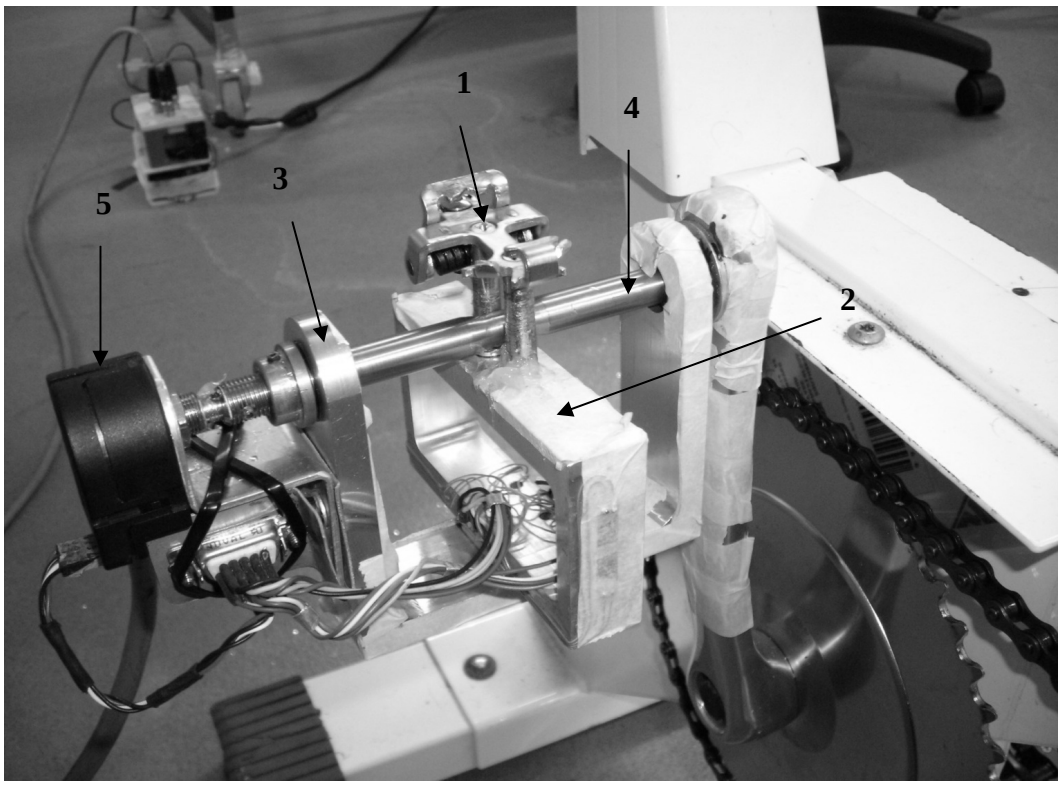

Figure 2.1 Force Transducer Pedal (Instrumented Pedal). 1. A clipless fastening system (Shimano®). 2. Specially designed load cell. 3. Fixed to a "U" shape stirrup for load cell. 4. Transmission-shaft fixed to the crank of the bicycle. 5. Smart encoder to measure crank angle with respect to pedal.

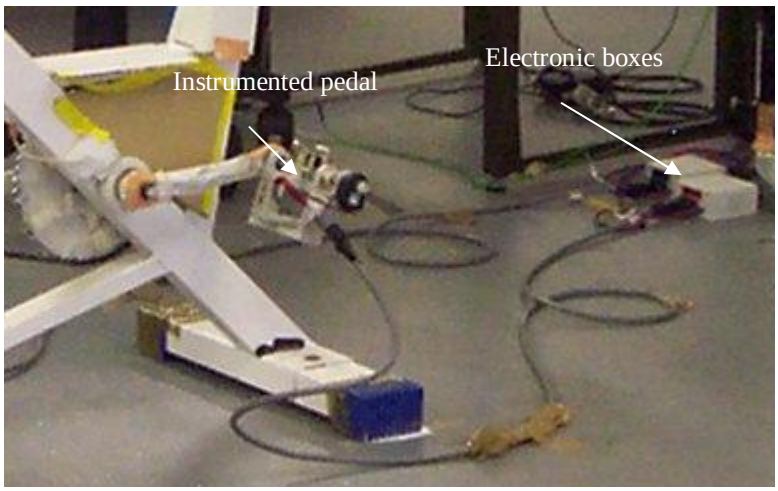

Figure 2.2 Instrumented pedal connected via the 'electronic box' containing 'Wheatstone bridges' and BNC coaxial connectors for the analogue output.

Motion Analysis

Four reflective reference markers were attached to the left lateral side of the cycle ergometer's flywheel (Figure 2.3). Three markers were placed equidistant

around the outer edge of the flywheel and one was placed closer to the flywheel's centre to assist in identifying each reflective marker's trajectory during motion analysis reconstruction. Two further reflective markers were placed on the cycle ergometer's left hand crank arm, one at the centre of the crank arm's rotation and the other at the pedal centre. All markers were 1cm in diameter.

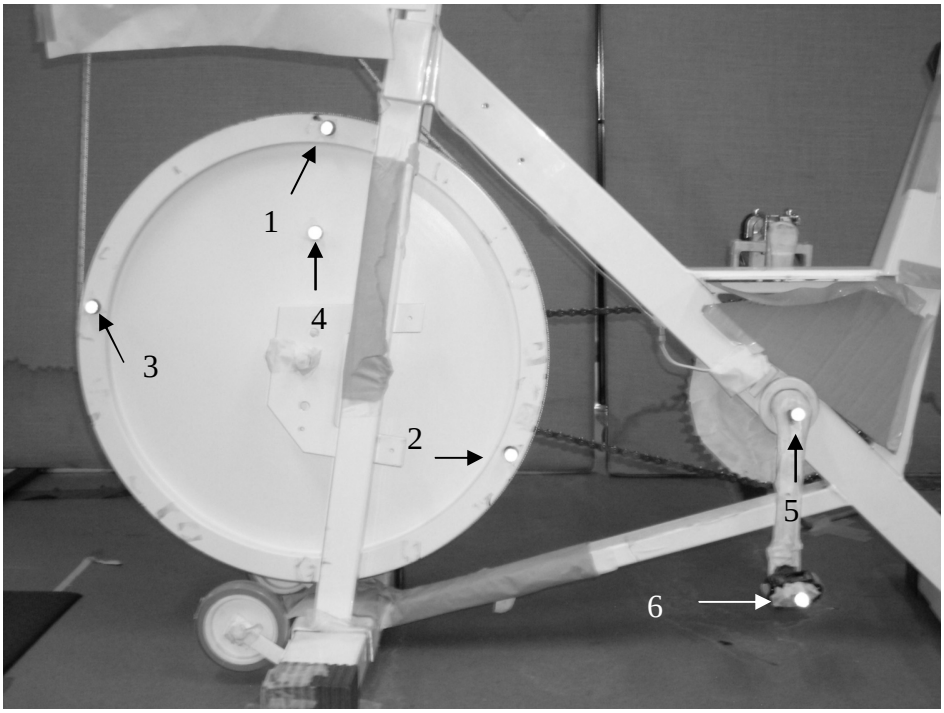

Figure 2.3 Reflective marker placement for motion analysis.

Kinematic data were recorded during each 6-s 'all-out' sprint cycle using a 5 camera system (Vicon M^2, California, USA), with a sampling rate of 250 Hz (Figure 2.4). Data were sent to an amplifier and analogue to digital (AD) converter in the Vicon work station (Vicon 612, California, USA) and saved onto the hard drive of a personal computer (PC, Dell, Texas, USA). Motion analysis cameras were calibrated both statically and dynamically on a daily basis according to the procedure described by the manufacturer, with a reference of <1.5mm mean residual and a static reproducibility of <0.5%.

Figure 2.4 Experimental set up using motion analysis.

All calculations were made using Matlab 7.0.1 (Mathworks, MA, USA). A 4th order Butterworth filter, with a cut-off frequency of 6 Hz, was applied to the reflective marker's Cartesian coordinates, reconstructed from motion analysis, and to the averaged angular velocities from each marker using stereophotogrammetry. The measurement of flywheel angular velocity was crucial in determining the overall measurements of the highest peak power output during each 'all-out' 6 s cycle, termed the maximum peak power (PPmax) and the corresponding torque (Mh) and pedal velocity (pedV).

Power output calculation from motion analysis

The amount of angular acceleration (α) experienced by the cycle ergometer's flywheel, around its axis of rotation, is proportional to the resultant moment and inversely proportional to its moment of inertia (I). The resultant moment is determined by the 'acting' moments, which can be defined as that produced by human effort (M_h), the friction due to the belt (M_b), and unwanted friction due to rotating parts including the chain and bearings (M_o). Therefore the effect of the acting moments on the flywheel's angular acceleration can be shown as:

$$M_h - M_b - M_o = I\alpha$$

As the friction force (μ) produced by the belt is linearly proportional to the applied friction load, and independent of flywheel velocity, thus the friction moment (M_b) of the belt can be determined from:

$$M_b = \mu L r$$

where L is the applied friction load and r the radius of the flywheel. The constant proportionality of μ and M_o, also assumed to be independent of velocity, can be determined by a dynamic calibration as proposed by Lakomy *et al*. (1986). The dynamic calibration involves measuring the flywheel's deceleration after reaching a constant pedalling velocity of 120 rpm (where pedalling is immediately stopped) until the flywheel reaches a stationary position, in the absence of M_h, for different friction loads (L). The gradient of each velocity-time graph was plotted against load and the constants of the linear regression analysis provided μ and M_o (r^2= 0.9991; y=1.04 x 0.0552).

The Cartesian coordinates from the reflective markers placed on the flywheel in the x, y, and z directions were reconstructed to firstly determine the change in angle. The change in angle with respect to time was then used to measure the angular velocity from each reflective marker and thereafter individual marker's angular velocities were averaged. From the flywheel's averaged instantaneous angular velocity (ω_f), at each time point, the rate of change in angular velocity (angular acceleration [α]) could be determined with respect to the change in time. This enabled the moment produced from human effort (M_h) or torque applied to be calculated from:

$$M_h = I\alpha + \mu L r + M_o$$

or

$$M_h = 1\ \alpha + 1.04 \text{ x } L \text{ x } 0.27 + 0.0552$$

where I = 1, α determined from motion analysis, L = friction load applied (N), r = radius 0.27 cm. Hence, the power output (P) was equated as:

$$P = M_h \text{ x } \omega_f$$

where ω_f corresponds to M_h with respect to time. The measurement of pedal velocity (pedV) was taken from the flywheel's angular velocity, then converted to revolutions per minute before being adjusted by the gear ratio of 52 (chain ring sprocket) x 12 (sprocket on flywheel).

Power calculation from instrumented pedals

From the measure of the force components F_z and F_x, and the angular position of crank in relation to pedal (θ_p), the tangential force (F_t) and parallel force to the crank (F_n) were determine in order to measure the total force (F_{tot}) from the two pedals according to Bibbo (2008) from:

$$F_t = F_z \text{ x } sin\ \theta_p + F_x \text{ x } cos\ \theta_p$$

$$F_n = F_z \text{ x } cos\ \theta_p + F_x \text{ x } sin\ \theta_p$$

$$F_{tot} = \sqrt{F_x^{\,2} + F_z^{\,2}} = \sqrt{F_t^{\,2} + F_n^{\,2}}$$

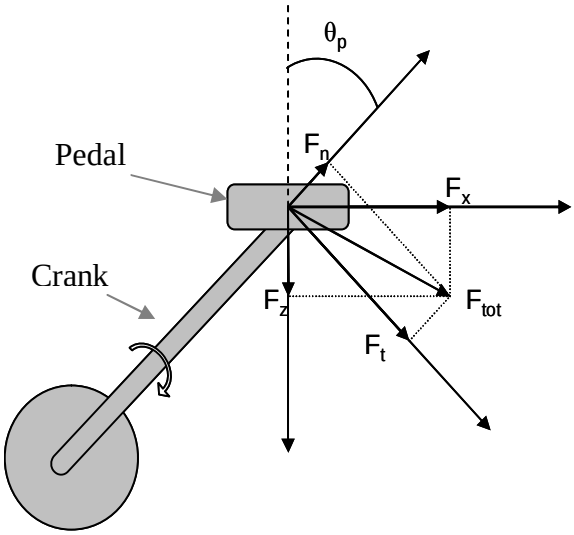

Figure 2.5: Force components on the pedal and on the crank.

Then, the F_{tot} for both left and right pedals was used to determine the effective force (F_{effect}) causing motion to the crank arm:

$$F_{effect} = F_{tot} \text{ Left} + F_{tot} \text{ Right}$$

The rate of change in θ_p with respect to time provided the measure of the crank arm angular velocity, ω_c, and given the length of the crank arm (170mm), d, the power applied (P) to the chain ring on the crank arm could thus be determined from;

$$P = F_{effect} \times d \times \omega_c$$

All calculations were made using Matlab 7.0.1 (Mathworks, MA, USA). An example of the power output determined from the two methods discussed is shown in Figure 2.5 using the same friction load.

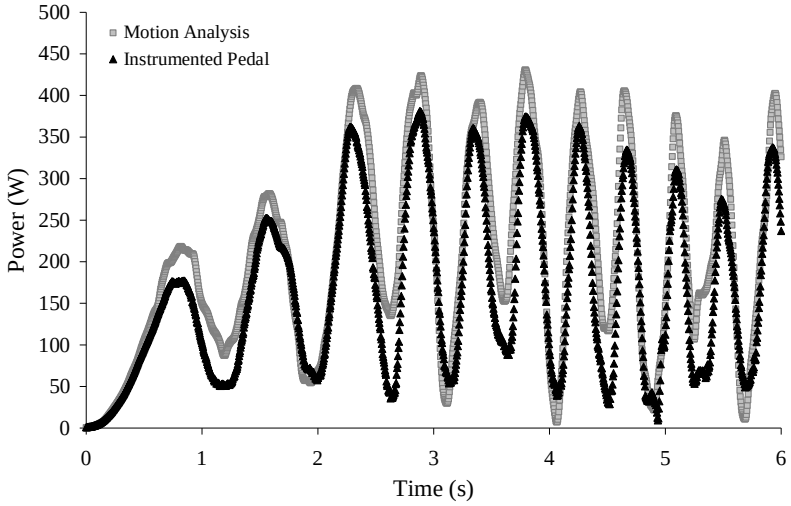

Figure 2.6 A typical trace of mechanical power output (Power) from one participant during an all-out 6 s sprint cycle, using a load 60% MVC, from motion analysis and the instrumented pedal.

Statistics

Descriptive statistical analysis including means and standard deviations (SD) were calculated for participant's characteristics, average 6 second power output and maximum peak power output. All data were normally distributed in terms of skewness and kurtosis (values less than ±2). The validity of the power output measurement using motion analysis (MA) was assessed using paired Student's t-tests (two tailed), in conjunction with Pearson's correlation coefficients. The reliability of the MA measurement was assessed using Intraclass Correlation. Statistical

significance level was set at P<0.05 and all statistical calculations were performed using SPSS software (SPSS, Chicago, USA).

Results

Validity

The absolute measurements of the maximum peak power output (PPmax) generated at 20% 2RM from motion analysis (MA) were higher than that measured with the instrumented pedal on average by 51.5 ± 10.9W on day 1, 63.9 ± 16.5W on day 2, and 53.6 ± 28.5W on day 3 (Figure 2.6a). The PPmax produced from MA were also higher using a 60% 2RM load by an average of 54.1 ± 25.7W on day 1, 57.5 ± 19.8W on day 2, and 58.7 ± 21.4 on day 3 (Figure 2.6b). Similar trends of a higher power output measurement produced by MA were also observed when power output was averaged over 6 seconds in both 20% and 60% 2RM during all trial days tested (Figure 2.7). However, the power outputs produced using MA, with 20% and 60% 2RM from all trials, were not significantly higher than those produced with the instrumented pedal (p>0.05) except the average 6 second power on trial day 2 with a 20% 2RM load (P<0.05; Figure 2.7a).

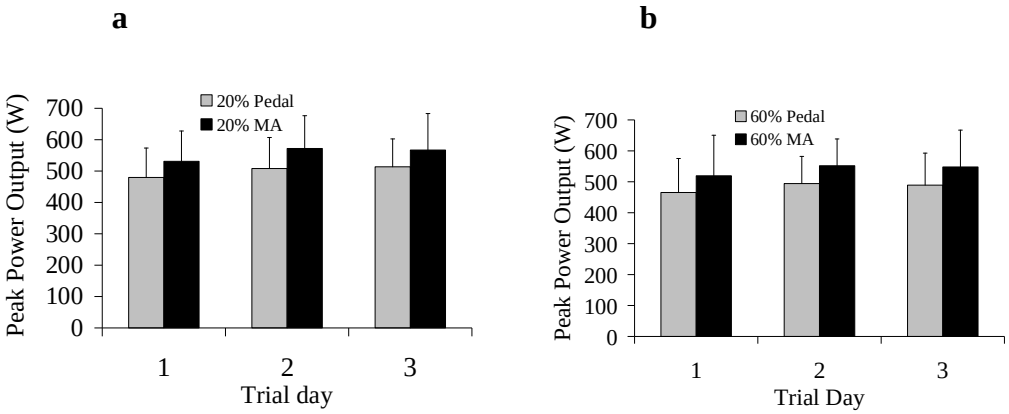

Figure 2.7 Maximum peak power (Peak Power) output in the 3 trial days using 20% 2RM (a) and 60% 2RM (b), from motion analysis (MA) and instrumented pedal (Pedal). Values are mean ± SD. No significant differences were observed (P>0.05).

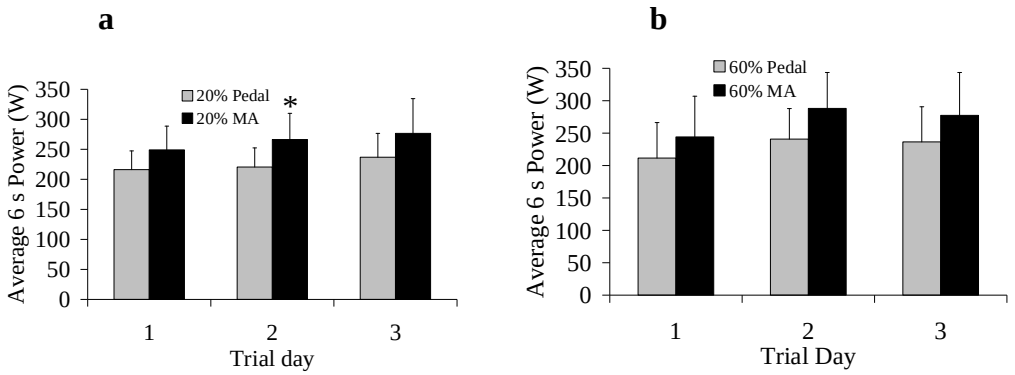

Figure 2.8 Average 6 s power output in the 3 trial days using 20% 2RM (a) and 60% 2RM (b), from motion analysis (MA) and instrumented pedal (Pedal). Values are mean ± SD, * denotes MA significantly different from instrumented pedal ($P<0.05$).

In the sub-group of five participants used to analyse the within-day trials, conducted with a 40% 2RM load, PPmax produced using MA were higher than those produced using the instrumented pedal on average by 75 ± 11.2W in trial A and 74 ± 19.6W trial B. In addition, the average power output produced over 6 seconds was higher, but not significantly, on average by 59.2 ± 8.3W in trial A and 57.1 ± 10.2W in trial B using MA (Figure 2.8).

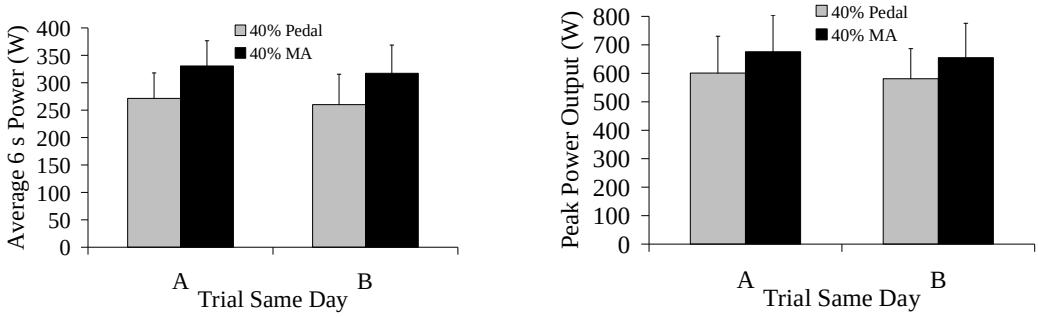

Figure 2.9 Average 6 s power and maximum peak power (Peak Power) output taken over two trials performed on the same day using 40% 2RM load, from motion analysis (MA) and instrumented pedal (Pedal). Values are mean ± SD. No significant differences were observed ($P>0.05$).

Pearson correlation coefficients indicate a high degree of correlation between MA and the instrumented pedal on all between-day trials tested (Figure 2.9) and from the within-day trials (Figure 2.10), for both PPmax and average 6 second power during all loads tested.

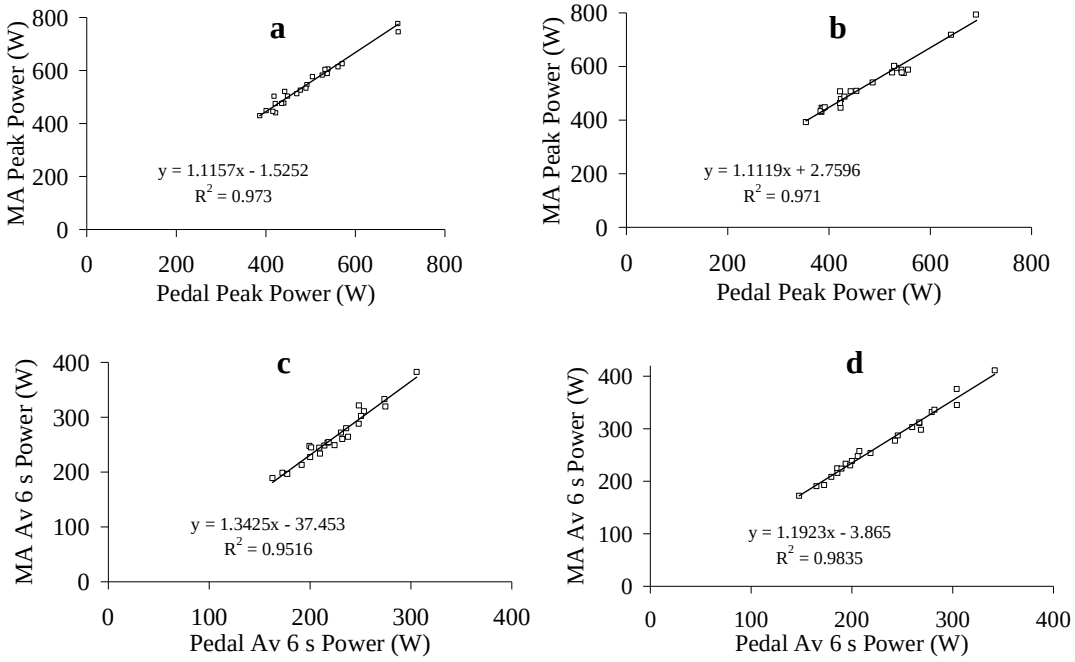

Figure 2.10 Pearson's Correlation Coefficients between motion analysis (MA) and instrumented pedal (Pedal) from the three between-day trials; a. Maximum peak power (Peak Power) 20% 2RM; b. Maximum peak power (Peak Power) 60% 2RM; c. Average 6 s Power 20% 2RM; d. Average 6 s Power 60% 2RM.

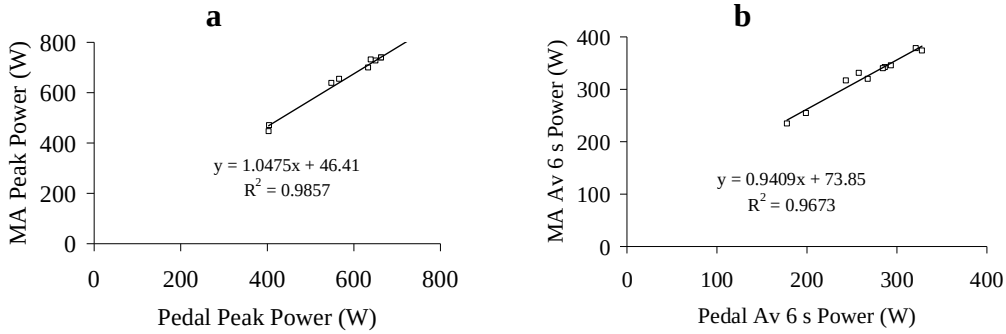

Figure 2.11 Pearson's Correlation Coefficients between motion analysis (MA) and instrumented pedal (Pedal) from the two within-day trials; a. Maximum peak power (Peak Power) 40% 2RM and b. Average 6 s Power 40% 2RM.

Reliability

The Intraclass Correlation Coefficients (ICC) was used to assess the test-retest reliability measurement of power output using MA. The PPmax and average 6 second power for the between-day trials (T1, T2, and T3) and the within-day trials (A and B) were utilised for the ICC assessment (Table 2.1). The ICC's indicate a high degree of reliability for all between-day (average ICC's from all between-day

trials; 0.905 ± 0.02, p<0.001) and within–day (average ICC's from all within-day trials; 0.917 ± 0.07, p<0.05) power output measurements using motion analysis.

Table 2.1 Intraclass Correlation Coefficients from MA power output measurements.

	Between-day Trials		Within-day Trials
		2RM Load	
Power	20%	60%	40%
Average 6 s	0.882 95% CI 0.664-0.973	0.919 95% CI 0.757-0.982	0.869 95% CI 0.196-0.986
Peak	0.931 95% CI 0.790-0.984	0.890 95% CI 0.683-0.975	0.966 95% CI 0.836-0.998

Correlation coefficient values with a confidence interval (CI) set at 95%. Intraclass Correlation performed using a 'two-way' mixed model.

Discussion

The findings of this investigation demonstrate the novel method of motion analysis, using a 5 camera Vicon system, can be utilised as an alternative method for measuring mechanical maximal peak power output during an 'all-out' 6-second sprint cycle on a friction-loaded cycle ergometer, against varying resistance loads. Both PPmax and average 6-second mechanical power output were systematically overestimated using motion analysis, by an average 10 ± 0.2% and 14 ± 0.9% respectively. However, only the average 6-second power generated with a 20% 2RM load was found to be significantly higher than the direct measure using instrumented pedals.

The correlation coefficients and intraclass correlation coefficient (ICC) indicate motion analysis as an acceptable alternative method of measuring mechanical power output for both within and between-day measurements. Previous studies utilising incremental encoders to estimate the torque required to overcome the flywheel's inertia, using Lakomy's (1986) deceleration-load methodology on standard Monark friction-loaded cycle ergometer, have been shown to provide valid measures of power output during sprint cycling with correlation coefficients ranging

between 0.86 and 0.99 (Arsac *et al*., 1996; Morin and Belli 2004; Martin *et al*., 1997). However, only Martin *et al*. (1997) investigated the reliability of their measure of the highest averaged power per pedal stroke over repeated bouts producing an ICC of 0.99. More recently, Watson *et al*. (2007), using a single-leg sprint cycle manoeuvre, determined the validity and reliability of the measurement of mechanical power output from the cycle ergometer's flywheel against a direct measure of mechanical power determined at the pedals. Their results show high ICC's, ranging from 0.91 to 0.97, for average and maximum power output for within and between day trial measurements, using younger male and female participants. Similar ICC values are also produced in the findings of this study, 0.963 to 0.998, for PPmax and average 6-second power output during 'all-out' sprint cycling.

The maximum values of peak power highlighted in our study are approximately 50% lower than those reported in the literature in young active males and females on a friction-loaded cycle ergometer (Seck *et al*., 1995; Martin *et al*., 1997; Kostka 2005). The difference in participants' age between the studies can fully account for lower PPmax recorded, as the values of PPmax reported in our study (averaged from all participants; 548 ±108W) are similar to those reported in Kostka (2005, average maximum power; 419 ± 95W) in women of similar age.

The calculation of the flywheel inertia is an important factor in producing a valid measurement of mechanical power output from a friction-loaded cycle ergometer (Arsac *et al*., 1996; Martin *et al*., 1997; Morin and Belli 2004). For this study, Newton's 2nd law of motion was utilised to determine the additional moments required to overcome the flywheel's inertia and the friction due to the belt and rotating parts during cycling from the deceleration-load relationship, which was formulated into a regression equation. The flywheel's inertia was recorded as that stated by the cycle ergometer's manufacture (1 kg·m^2), and thus enabling the resultant moment acting to produce an acceleration of the flywheel to be calculated (Lakomy 1986; Martin *et al*., 1997; Morin and Belli 2004). However, our results are the first to indicate a systematic overestimation of the PPmax during 'all-out' sprint cycling when taking the flywheel's inertia into account, as indicated from the instrumented pedal's direct measurement of mechanical power (refer to figure 2.6).

There are a number of possible explanations that could account for the systematic overestimation of PPmax and peak power per pedal stroke using motion analysis. Firstly, as highlighted by Morin and Belli (2004), the linear relationship between the flywheel's deceleration rate and friction-load may not be perfectly applicable during an 'all-out' sprint, as the deceleration-load relationship may differ at high or low torques generated and thus regarded as a possible limiting factor in determining mechanical power output. Morin and Belli (2004) add that, despite this overestimation, the validity of this method still remains true due to the high correlation between the measured and calculated PPmax (r^2=0.997). Secondly, Seck *et al*. (1995) add that oscillations due to the flywheel not rotating symmetrically around the axis leads to an increase and decrease in power output as the centre of the flywheel moves up and down. However, this does not seem to apply to our data since peak power is continually overestimated, whereas the power output at the lowest point of the pedal stroke is not consistent with a possible effect of flywheel oscillations (figure 2.6). Thirdly, as the systematic overestimation of peak power occurs at the instant where torque development reaches its peak, thus it is possible that the effect of pedalling may be adding inertia into the system leading to an increase in the flywheel's acceleration that is undetectable through the pedal strain gauges. However, this consideration is only speculative. Lastly, Watson *et al*. (2007) conclude that using the flywheel's inertia determined from the manufacture's calculation may be the major contributing factor in the systematic overestimation of mechanical power output using motion analysis.

In conclusion, the validity and reliability of motion analysis has been demonstrated as being a satisfactory method of measuring of Pmax and average power output during an 'all-out' sprint cycling on a friction-loaded cycle ergometer. Hence, laboratories where motion analysis is more ready available our method can be adopted as a suitable alternative to incremented encoders in the measure of the flywheel velocity for muscle power output using cycle ergometry.

CHAPTER 4

Comparison between young and older women in explosive power output and surface EMG during a 6 second all-out cycling effort at different loads

Data from this chapter were presented at the following conference:

1st Annual Conference of HEPA (European network for the promotion of health-enhancing physical activity) Europe; September 2008, Glasgow, Scotland.

To be submitted as:

Duffy CR, Pecoraro F, Riches PE, Farina D, and Macaluso A. (2008). Comparison between young and older women in explosive power output and surface EMG during a 6 second all-out cycling effort at different loads, *European Journal of Applied Physiology*.

ABSTRACT

The purpose of this study was to investigate the effects of ageing on the neural activity governing 'all-out' sprint cycling on a friction-loaded cycle ergometer, which generate maximum peak power, between young and older women. Ten old (OL, aged 70-83 years) and 8 young (YO, aged 19-35 years) healthy female participants were compared for peak power (PP) during 6 s 'all-out' cycles against varying load of resistance, ranging from 20% to 80% of the maximum load turned through 2 complete pedal revolutions. Advancements in surface electromyography (sEMG) linear array provided a method of inferring neural activation and strategy changes during dynamic cyclic movements, which were synchronised with mechanical power output. The 43% lower PP and 33% lower maximal dynamic strength produced by OL in comparison to YO ($p<0.001$) were accompanied by significantly lower muscle CSA (34%; $p<0.001$) and overall neural activation (45%; $p<0.05$), determined from sEMG signal amplitude during all loads tested. No differences were observed in any other parameter from sEMG between the groups ($p>0.05$). The lower sEMG signal amplitude indicates that a lower neural drive can be regarded as a contributing factor in the older women's lower mechanical power output. The differences in muscle peak power, unlike maximal dynamic strength, with age are not fully accounted for by the decline in muscle CSA and neural drive. Hence, other physiological factors affecting both muscle torque and movement velocity with advancing age have to be considered with regards to explaining the lower peak power in older women.

Introduction

The detrimental effects of the decline in muscle power within the older population are demonstrated by the reduction in their ability to perform everyday tasks unaided, which ultimately threatens their functional independence and quality of life (Bassey *et al*., 1992; Daily and Spinks 2000; Evans 2000; Foldvari *et al*., 2000; Lauretani *et al*., 2003; Macaluso and De Vito 2003; 2004). Since the effects of ageing are exacerbated in older women, this makes women more vulnerable to loss of functional independence and reduced quality of life than men and thus should be regarded as a target group for intervention studies (Skelton *et al*., 1994).

The generation of muscle power involves the production of muscle force during the execution of a dynamic contraction, with the amount of force a muscle generates declining with increasing contraction velocity, as described by the muscle's force/torque-velocity relationship (F/T-V; AV Hill 1938; Sargeant *et al*., 1981). With advancing age, the amount of force a muscle generates declines for any given speed of movement due to a leftwards shift in muscle's T-V relationship, which inevitably leads to a decline in muscle power (Lieber and Friden 2000; Hortobagyi *et al*., 2003; Lanza *et al*., 2003; and Narici *et al*., 2003; Pearson *et al*., 2006). Several interacting physiological mechanisms are responsible for this phenomenon affecting both force production and speed of movement, which include muscular, tendinous and neural factors (Roos *et al*., 1997; Connelly *et al*., 1999; Frontera *et al*., 2000a; Vandervort 2002; Doherty 2003; D'Antona *et al*., 2003; Narici *et al*., 2003; Maganaris *et al*., 2004; Narici and Maganaris *et al*., 2006; Pearson *et al*., 2006). Among the neural factors, the effects of ageing on motor unit (MU) behaviour and neural drive, which govern muscular contractions, on the performance of dynamic movements are less well understood (Farina *et al*., 2002; Vandervort 2002; Doherty 2003; McNeil *et al*., 2007). Advancements in surface electromyography (sEMG) linear array technique, devised by Farina *et al*. (2004), now provide a method to study neural activation and the central strategies of the neuromuscular control that may be utilised to determine changes in MU behaviour, during the performance of explosive dynamic muscular contractions (Farina *et al*., 2002; 2004; 2004a; Pozzo *et al*., 2004). The linear array sEMG technique estimates

the conduction velocity of a MU action potential (or muscle fibre conduction velocity [MFCV]) and as the MFCV is regarded as a size principle parameter that increases in accordance with recruitment of higher threshold MUs (Farina *et al.*, 2004; 2004a; Macdonald *et al.*, 2008), thus differences in MU behaviour strategies with age can be investigated. To our knowledge, previous ageing studies adopting linear array sEMG have only been carried out during fatiguing static contractions, which have shown that MFCV declines at a lower rate and neural activation is lower in both older women and men compared to younger women and men indicating higher resistance to fatigue, a process which is referred to as "fatigue-paradox" (Merletti *et al.*, 2002; Bazzucchi *et al.*, 2004). Merletti *et al.* (2002) indicates that in older men the main neural mechanism responsible for muscle weakness and "fatigue paradox" may possibly be attributed to a significantly lower MU firing rate. The relevance of such results in relation to investigating the neural factors affecting functional ability in the older population may be questioned, as MU behaviour strategies may differ to govern dynamic movements.

Pearson *et al.* (2004) found that accelerating a sub-maximal inertial load on a cycle ergometer, similar to a mechanically-loaded cycle-ergometer, during a 6-s sprint cycle resulted in the muscles performing along their T-V curve, which produces similar conditions to those encountered during the performance of functional tasks involving the lower limbs, i.e. climbing stairs. Therefore, 'all-out' 6-s sprint cycling on a mechanically-braked cycle-ergometer in conjunction with sEMG can be adopted as a 'novel' method for studying the effects of ageing on MU behaviour during muscular actions commonly encountered during activities of daily living. Whereas previously, assessment of the muscle's T-V relationship was reconstructed from a series of single explosive muscular isotonic or isokinetic contractions against varying loads (Lanza *et al.*, 2003; Macaluso and De Vito 2003; 2004).

The present study was designed to compare muscle power output and sEMG parameters between young and older women during 'all-out' 6-s sprint cycling on a mechanically-braked cycle-ergometer, which can be regarded as a functional action, against a series of loads to identify the optimal load that produces the highest power output. It is hypothesised that the leftwards shift in the muscle's T-V relationship,

lower peak power and corresponding torque and velocity, with advancing age, are accompanied by changes in MU behaviour and neural drive detected through sEMG, which include not only a lower magnitude of sEMG parameters (average MFCV, median frequency (MDF) and root mean square (RMS)) but also differences in sEMG parameters varying across time during each 6-s sprint, from all loads tested. In particular, it is hypothesised that sEMG parameters from younger women may experience a steeper decline in MFCV and MDF, which would reflect differences in MU recruitment strategies.

Methodology

Participants

With approval of the Ethics Committee of the University of Strathclyde, 18 "medically stable" female participants were selected, according to the criteria proposed by Greig *et al*. (1994) for participating in exercise studies, 10 older women (OL) and 8 young women (YO). Participants' characteristics are presented in Table 4.1 Volunteers provided written informed consent and were instructed to maintain their usual levels of physical activity throughout the duration of the study. After an initial familiarisation period participants re-visited the laboratory after at least 7 days between exercise cycle trials.

Table 3.1 Summary of participants' characteristics by age group.

Participant's characteristics	*OL Group*	*YO Group*
Age (yr)	77.8 ± 3.9	23.6 ± 5.8*
Stature (m)	1.58 ± 0.04	1.65 ± 0.08*
Mass (kg)	63.3 ± 8.7	63.1 ± 8.6

Values are means ± SD for 10 OL and 8 YO. * Significantly different from OL ($P < 0.05$).

6 second All Out Cycle test

All measurements were carried out on a mechanically-braked cycle-ergometer (Monarch 823e, Sweden). Seat height was adjusted so that the participants

could cycle with their heels touching the pedals and no sideways movement of the hips during pedalling. Each participant wore cycling shoes with cleats that ensured a secure fitting to the pedal during the required 'all-out' effort. After an initial warm up period of 5 minutes, with a sub-maximal load (50-60 W), participants were tested for the maximum load each individual could pedal through 2 complete revolutions while remaining seated, referred to as 2 revolution maximum (2RM) (Macaluso *et al.,* 2003). During each attempt participants were asked to remain seated at all times whilst pedaling. Starting from the load at which the participants could not turn the pedals, the load was then reduced by 0.5 kg decrements until the participants were able to turn the pedals through two full revolutions. To ensure that the load was effectively the maximum, participants were required to perform further attempts by increasing the load in 0.5 kg increments. Each attempt was followed by a 2 minute recovery period, and once the 2RM was established the participants rested for an additional 5 minutes. Each volunteer was then asked to cycle as fast as possible for at least 6 seconds against 7 different loads corresponding to 20% to 80% of their 2RM, with increments of 10%, in a random order. Verbal encouragement was given during each trial, and after each 'all-out' cycle trial a 5 minute recovery period was carried out before attempting the next load. The standardised stationary starting position was with the right knee bent at 90 degrees for all cycle trials.

Measurement of Power, Torque and Pedal Velocity

Four reflective reference markers were attached to the left lateral side of the cycle ergometer's flywheel, as shown in Figure 3.1, three placed equidistant around the outer edge of the flywheel and one other placed randomly nearer the flywheel's centre to assist in identifying each reflective marker's trajectory during motion analysis reconstruction. Two further reflective markers were placed on the cycle ergometer's left hand crank arm, one at the centre of the crank arm's rotation and the other at the pedal centre. All markers were 1cm in diameter.

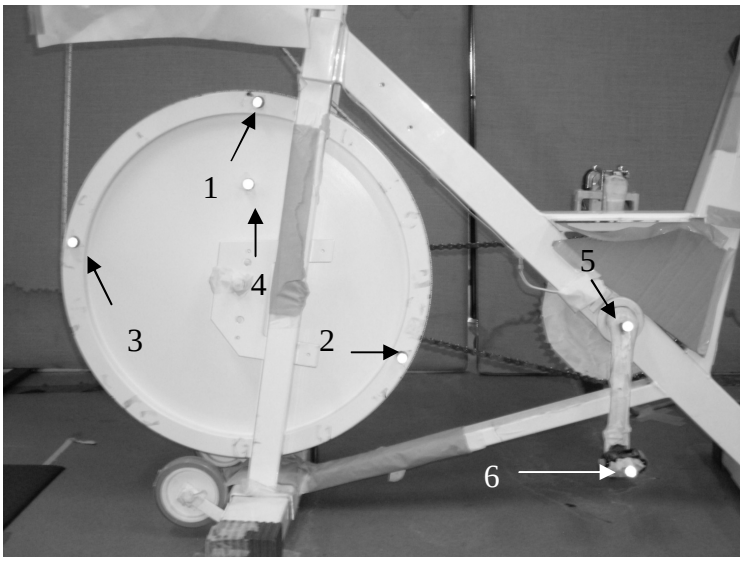

Figure 3.1 Reflective marker placement on cycle ergometer.

Kinematic data were recorded during each 6-s 'all-out' sprint cycle using a 5 camera system (Vicon M^2, California, USA), with a sampling rate of 250 Hz. Data were sent to an amplifier and analogue to digital (AD) converter in the Vicon work station (Vicon 612, California, USA) and saved onto the hard drive of a personal computer (PC, Dell, Texas, USA). Motion analysis cameras were calibrated both statically and dynamically on a daily basis according to the procedure described by the manufacturer, with a reference of <1.5mm mean residual and a static reproducibility of <0.5%.

All calculations were made using Matlab 7.0.1 (Mathworks, MA, USA) with a 4^{th} order Butterworth filter set with a cut-off frequency of 6 Hz (Martin *et al.*, 1997), which produced a typical power output trace as shown in Figure 3.2, from each 6-s cycle trial. Power output, torque (Mh) and pedal velocity (pedV) were calculated from the reconstruction of reference reflective markers trajectories on the cycle ergometer's flywheel and a gearing differential between the crank and flywheel (52x12), as described in chapter 3. Power output was calculated as the sum of the friction load placed on the cycle ergometer (N) and the 'addition load' (N) to overcome the cycle ergometer's flywheel, multiplied by the velocity of the flywheel. The 'addition load' was calculated from a regression equation using the correlation between friction load and the rate of decline in flywheel velocity, as described by

Lakomy (1983). Five different loads varying in resistance were each separately pedalled to a velocity of approximately 120 rpm, and when a steady pedalling rate was achieved pedalling was stopped and the flywheel's deceleration was recoded using motion analysis. The flywheel's rate of deceleration from 100 rpm to rest was measured and correlated against the 5 resistance loads to form the regression equation. This allowed the 'additional load' the participants would have to overcome for any given change in flywheel velocity to be calculated.

The highest power output produced during each 6-s sprint cycle was termed the peak power output (PP), and corresponding Mh and pedV recorded as the optimal Mh and pedV at PP. The time it took participants to pedal until PP, from time point zero, was taken as the time to peak power (TPP). The rate of power development to PP (RPD) was calculated as PP divided by the corresponding time to PP.

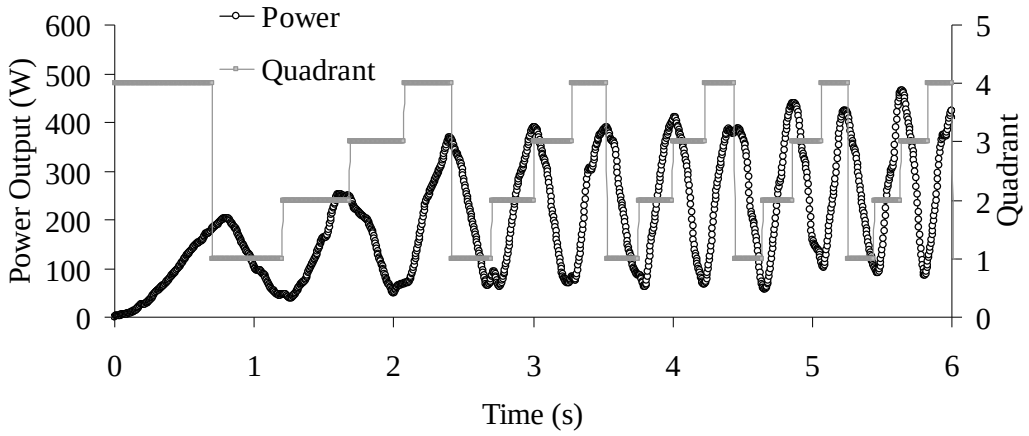

Figure 3.2 Power output during a 6 s 'all-out' sprint cycle trial. The quadrants represent the 360° pedal stroke divided into 4 segments. Pedal down-stroke from top to bottom dead centre of the right leg refer to quadrants 4 to 1 and left leg down stroke refers to quadrants 2 to 3.

Surface Electromyography

Multi-channel surface EMG signals were detected from the vastus lateralis muscle of the dominant lower limb using a linear adhesive array (model ELSCH008, SPES Medica, Salerno, Italy) consisting of 4 electrodes with a 10 mm inter-electrode distance, in bipolar configuration. The EMG signals were amplified (16 channel surface-EMG amplifier, EMG-16, LISiN – Prima Biomedical & Sport, Treviso, Italy), band-pass filtered (10 Hz to 500 Hz) and fed into a 12-bit acquisition board sampled at 2048 samples/s per channel, which was displayed on a PC. The 'optimal'

adhesive electrode placement on the vastus lateralis activity was performed from a series of short duration static muscular contractions with a moistened solid array of 8 electrodes (silver bars, 5 mm long, 1 mm diameter, 10 mm inter-electrode distance). During each static contraction, the array was placed in-line with the estimated fibre orientation, and from visual inspection of the EMG signals on the PC the muscle innervation zone and correct fibre orientation could be detected and marked on the skin (Farina *et al.*, 2004). The detection of the innervation zone location was performed with a knee bend of 90° and 170° knee joint angles (the total change in knee angle during pedalling). This was due to the position of the innervation zone changing as the muscle length shortens and elongates between the two joint angles under the skin during limb movement (Farina *et al.*, 2004; Pozzo *et al.*, 2004). Adhesive array placement was between the most distal location of the innervation zone detected and the distal tendon region. This procedure of electrode placement allowed the detection of signals between the innervation zone and tendon at all joint angles involved during cycling. Prior to adhesive array placement, the skin was slightly abraded with abrasive paste (Meditec–Every, Parma, Italy) and cleaned with a paper towel. Once the adhesive array was orientated within the marks placed on the skin, conductive gel (20-30 μl) was inserted in each electrode grove of the array for proper electrode-skin contact using a dispenser (Eppendorf AG – Multipette plus, Hamburg, Germany). The quality of the signal was then checked before proceeding to the cycle sprint test.

Surface EMG acquisition was synchronised with motion analysis via a 'switch' that simultaneously sent a change in voltage signal to the Vicon work station and sEMG signal acquisition board. Finally, the sEMG and kinematic data were aligned via the change in switch voltage using Matlab 7.0.1 (Mathworks, MA, USA).

Signal Analysis

For each pedal down stroke of the right thigh the same 6 crank arm angles (divided equidistant between top dead centre and bottom dead centre of the pedal stroke) were identified from motion analysis, ensuring that the muscle length recorded at each of the angles was the same between pedal strokes, as the participant's pedalling continually accelerated during each cycle trial. The average

MFCV was estimated using Matlab 7.0.1 (Mathworks, MA, USA), according to the method of Farina *et al*. (2002) and Pozzo *et al*. (2004), as the average of the conduction velocities of the action potentials occurring close to the selected pedal angles, weighted by a Gaussian window whose standard deviation (set to 50ms) could be tuned according to the desired pedal angle of the MFCV estimation. The 6 average MFCV velocities measured during each pedal down stroke of the right thigh were averaged over each 6-s 'all-out' sprint cycle trials and termed the avMFCV. The amplitude and frequency of the EMG burst from each pedal down stroke, estimated between the initial and last pedal angle, were also determined to produce a value of RMS and MDF using the same Matlab program for average MFCV. The RMS and MDF per pedal stroke were also averaged over each 6 s cycle trial and termed avRMS and avMDF. The delta (Δ) changes in sEMG variables during each 6-s sprint cycle were calculated from the ratio between first and last pedal strokes from each trial and the differences reported as a percentage to determine change in sEMG variables (ΔRMS, ΔMDF, and ΔMFCV), which were crucial for analysing differences in motor control strategies between the groups over time.

Ultrasonography

Quadriceps' mid-thigh cross-sectional area (CSA) and subcutaneous fat depth was estimated using ultrasonography (Diagnostic Scanner Ultrasonic System HS-2000, Honda Electronics CO. LTD. Japan). All measurements were performed on the right leg after participants lay quietly for approximately 10 minutes, with their leg relaxed. A 'reference template' containing a series of 15 plastic flat strips 100mm in length and 5mm in diameter (2mm thickness) with their lengths lying parallel and spaced out approximately 45mm apart, was wrapped around the width of the quadriceps muscle group of the mid-thigh using adhesive tape. The mid-thigh was taken as the distance halfway between the greater trochanter portion of the hip joint to the proximal portion of the patella. Participants were instructed to relax their leg muscles during each measurement.

Gel (Aquasonic 100 ultrasound gel) was directed onto the linear ultrasound probe (HLS-475M, 50mm 7.5MHz) approximately 10-15mm thick and the probe lightly placed onto the skin, with the probe's length running along the transverse

plane of the mid-thigh. Initially, the lateral edge of the vastus lateralis, along the transverse plane of the mid-thigh, was identified and marked on the skin. Thereafter the reference template was wrapped around the upper thigh with the 'skin mark' visible between the first 2 plastic strips. The re-gelled ultrasound probe was then lightly placed on top of the plastic grid resulting in 2 plastic strips lying underneath the ends of the probe. The shadows created on the monitor's picture from the reference template were crucial in the measurement of muscle CSA, as it provided a method of identifying each adjacent picture's start/end point. The ultrasonography probe was moved along in a medial direction between the adjacent strips, resulting in a series of pictures until the medial edge of the vastus intermedius. Each picture was recorded on the Diagnostic Scanner and then transferred to a PC. 'Image J' imaging process program (NIH, USA) was used to calibrate each picture in order to measure the total sum of CSA from each picture. The subcutaneous fat depth was measured as the distance between the vastus lateralis fascia to the dermis underneath the skin, at the region where the vastus lateralis reached a constant thickest along the lateral plane of the mid-thigh.

Statistics

Descriptive statistical analyses including means, and standard deviations were calculated for participants' characteristics (Table 3.1), maximum dynamic 2RM strength, quadriceps CSA, specific strength, and subcutaneous fat. All data were normally distributed in terms of skewness and kurtosis (all values less than 2) except MFCV and MDF. The effects of age and 2RM % load on PP, corresponding Mh and pedV, TPP, RPD, avMFCV, avRMS and avMDF were analysed using 2-way repeated measures ANOVA, with group as a between–subject factor and 2RM % load being a within-subject factor. Where relevant, a Post Hoc one-way ANOVA, with Bonferoni adjustment was performed to identify significant differences between 2RM % loads within each of the two age groups and a Student t-test to look at differences between the two age groups for each of the percentage loads. To statistically analyse the Δ change in sEMG variables during each 6-s all-out cycle three univariate repeated measures ANOVAs were performed on the ΔMFCV, ΔRMS

and ΔMDF, with 2RM % load as a within-subject factor and group as a between-subject factor. Statistical significance level was set at $P < 0.05$, and all statistical methods were performed using SPSS software.

Results

Strength and Quadriceps Muscle Cross Sectional Area

Muscle CSA from the OL was 34% significantly lower than the YO (51.29 ± 5.59 cm^2 vs. 78.3 ± 17.67 cm^2, $p<0.001$). Maximum dynamic muscle strength, estimated using the 2RM, in the OL was also significantly lower than the YO by 33% (55.8 ± 7.3 N vs. 83.9 ± 19.7 N, $p<0.001$). However, differences in specific dynamic strength, calculated as the ratio between 2RM maximum dynamic strength (N) and CSA (cm^2), were not significant between the groups (OL 1.09 ± 0.21 $N \cdot cm^2$ vs. YO 1.12 ± 0.07 $N \cdot cm^2$, $p>0.05$).

6 s 'all-out' Sprint Cycling

Mechanical PP, Mh and pedV were all significantly lower in the OL compared to the YO in all relative loads tested (Figure 3.3; average from all loads tested: 511 ± 41.8 W vs. 969 ± 46.7 W; 20.9 ± 1.2 N·m vs. 29.1 ± 1.4 N·m; 6.7 ± 0.2 $rad \cdot s^{-1}$ vs. 9.2 ± 0.3 $rad \cdot s^{-1}$, $p<0.001$). Maximum peak power and corresponding optimal Mh and pedV were also significantly lower in the OL than YO, with declines similar in magnitude to those reported above for average PP, from all loads tested (46% for maximum PP ($p<0.001$), 23% for optimal Mh ($p<0.01$) and 29% for pedV ($p<0.01$)). When the averaged PP from all loads or maximum PP where normalised for body mass or muscle CSA significant differences in muscle power were maintained ($P<0.001$ and $p<0.05$, respectively).

There was a significant effect of 2RM % load on the PP, Mh and pedV, with the effect of 2RM % load being significantly different between groups (Figure 3.3). For instance, PPs from each 2RM % load tested in the OL were equivalent for loads from 20% to 70% and from 70% to 80% ($p>0.05$), whereas PPs for loads from 20% to 60% were all significantly higher than 80% ($p<0.05$). In the YO, PP was equivalent for loads from 20% to 60% and from 70% and 80% ($p>0.05$), whereas PP

for loads from 20% to 60% were significantly higher than 80% ($p<0.05$). The corresponding Mh at PP from each 2RM % load tested in the OL showed that 20% to 50% and 70% to 80% were all equivalent ($p>0.05$), and Mh produced at 70% and 80% were significantly higher than those produced for loads from 20% to 60% ($p<0.05$). Whereas, YO's corresponding Mh at PP were equivalent for loads from 20% to 60% and from 60% to 70%, but only the Mh produced at 80% was significantly higher than that produced for loads from 20% to 70% ($p<0.05$). The corresponding pedV at PP for the OL were equivalent for loads from 20% to 50% and from 70% to 80% ($p>0.05$), whereas the pedV at PP produced for loads from 20% to 60% were all significantly higher than those produced at 80% ($p<0.05$). In contrast, YO's pedV at PP were equivalent for loads from 20% to 50% and from 50% to 60% ($p>0.05$), and the pedV at PP using loads from 20% to 70% were all significantly higher than those at 80% ($p<0.05$).

The time to peak power (TPP) output was not significantly affected by age ($p>0.05$; Figure 3.4a), but was significantly influenced by 2RM % load in both YO and OL groups, with the higher the 2RM % load the longer the time to PP ($p<0.001$). Conversely, the rate of power development (RPD) was significantly affected by age ($p<0.001$) and load ($p<0.001$), with the RPD declining significantly in accordance with increasing load (Figure 3.4b).

Surface EMG measurements

All sEMG parameters averaged over each 6-s sprint cycle, from each 2RM % load used, are presented in Figure 3.5. Due to motion artefacts only 4 OL vs. 7 YO participants data are presented for avMFCV, 5 OL vs. 6 YO presented for avMDF, and 8 OL vs. 8 YO presented for avRMS. The 2-way RM ANOVA demonstrated that there was no significant effect of age or 2RM % load on avMFCV or avMDF ($p>0.05$), whereas only age had a significant effect on the avRMS ($p<0.05$).

The 3-way ANOVA performed on the Δ changes in sEMG variables over 6 s demonstrated that only ΔRMS significantly changed during the each 'all-out' sprint cycles ($p<0.05$) in both groups (Table 3.2). Conversely, no significant changes in ΔMDF and ΔMFCV were observed between groups or loads ($p>0.05$).

Subcutaneous fat thickness measured below the vastus lateralis was not significantly different between OL and YO (0.95 ± 0.38 cm vs. 1.25 ± 0.28 cm, $p>0.05$, respectively).

Table 3.2 Delta percentage change (ratio between First pedal stroke and Last pedal stroke with differences calculated as a percentage) in sEMG variables (RMS, MFCV and MDF) during each 6 s 'all-out' sprint cycle.

Δ change sEMG	Group	2RM % LOAD						
		20%	30%	40%	50%	60%	70%	80%
ΔRMS (%)	OL	119 ± 94*	184 ± 170*	117 ± 96*	126 ± 128*	135 ± 132*	121 ± 94*	64 ± 79*
	YO	159 ± 102*	182 ± 111*	293 ± 137*	262 ± 180*	230 ± 159*	164 ± 124*	192 ± 145*
ΔMFCV (%)	OL	2.7 ± 7	8.3 ± 6	5.2 ± 22	7.2 ± 15	9.2 ± 14	7.5 ± 2	3.8 ± 10
	YO	-0.1 ± 6	-0.1 ± 7	-1.0 ± 9	4.0 ± 11	-1.5 ± 5	0.1 ± 7	6.0 ± 9
ΔMDF (%)	OL	-4.5 ± 15	-6.4 ± 9	-1.6 ± 10	2.8 ± 18	-3.9 ± 13	9.9 ± 25	8.0 ± 18
	YO	-8.1 ± 5	-3.7 ± 5	-5.0 ± 11	6.5 ± 13	1.7 ± 7	5.7 ± 10	8.0 ± 12

Data expressed as mean ± SD. * significantly different from ΔMFCV and ΔMDF ($p < 0.05$).

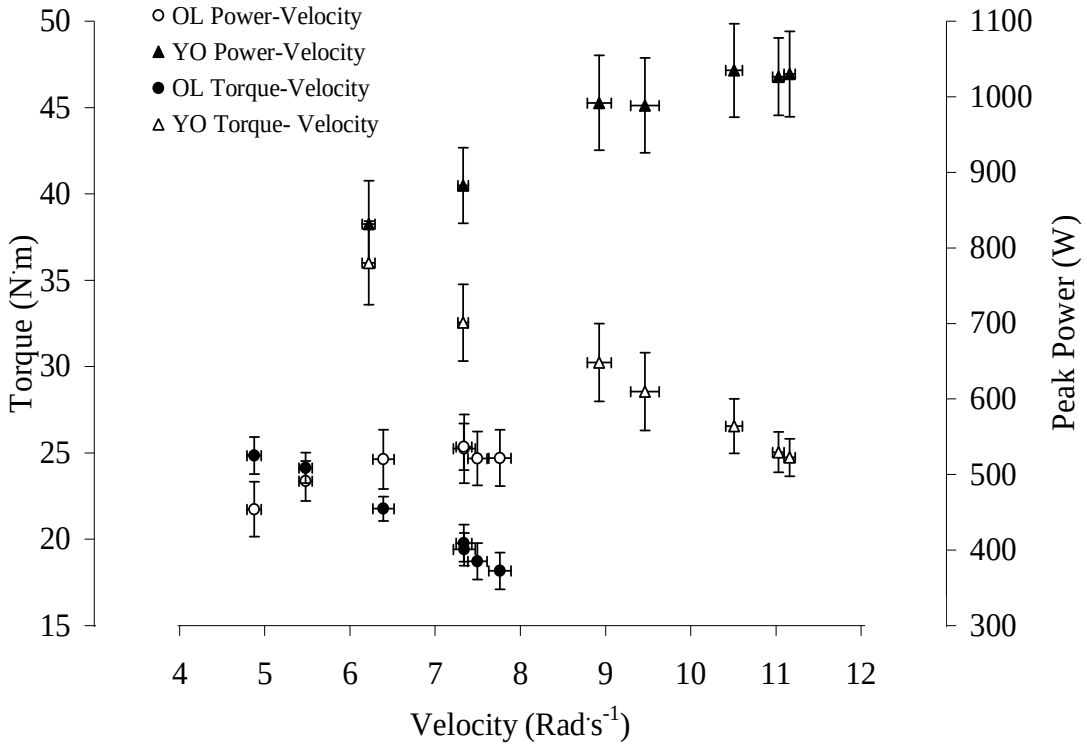

Figure 3.3 Peak Power- Velocity and Torque- velocity relationships constructed from loads 20, 30, 40, 50, 60, 70 and 80% of the maximal resistance to complete 2 revolutions (2RM) during the 6 s 'all-out' sprint cycles. Values are mean ± SE. The Peak Power-Velocity relationship is represented with open circles (○) in the old (OL) and closed triangles (▲) in the young (YO). The Torque-Velocity relationship is displayed with closed circles (●) in the OL and open triangles (Δ) in the YO. All values are significantly lower in the OL ($p < 0.001$). Refer to the text for statistical analysis.

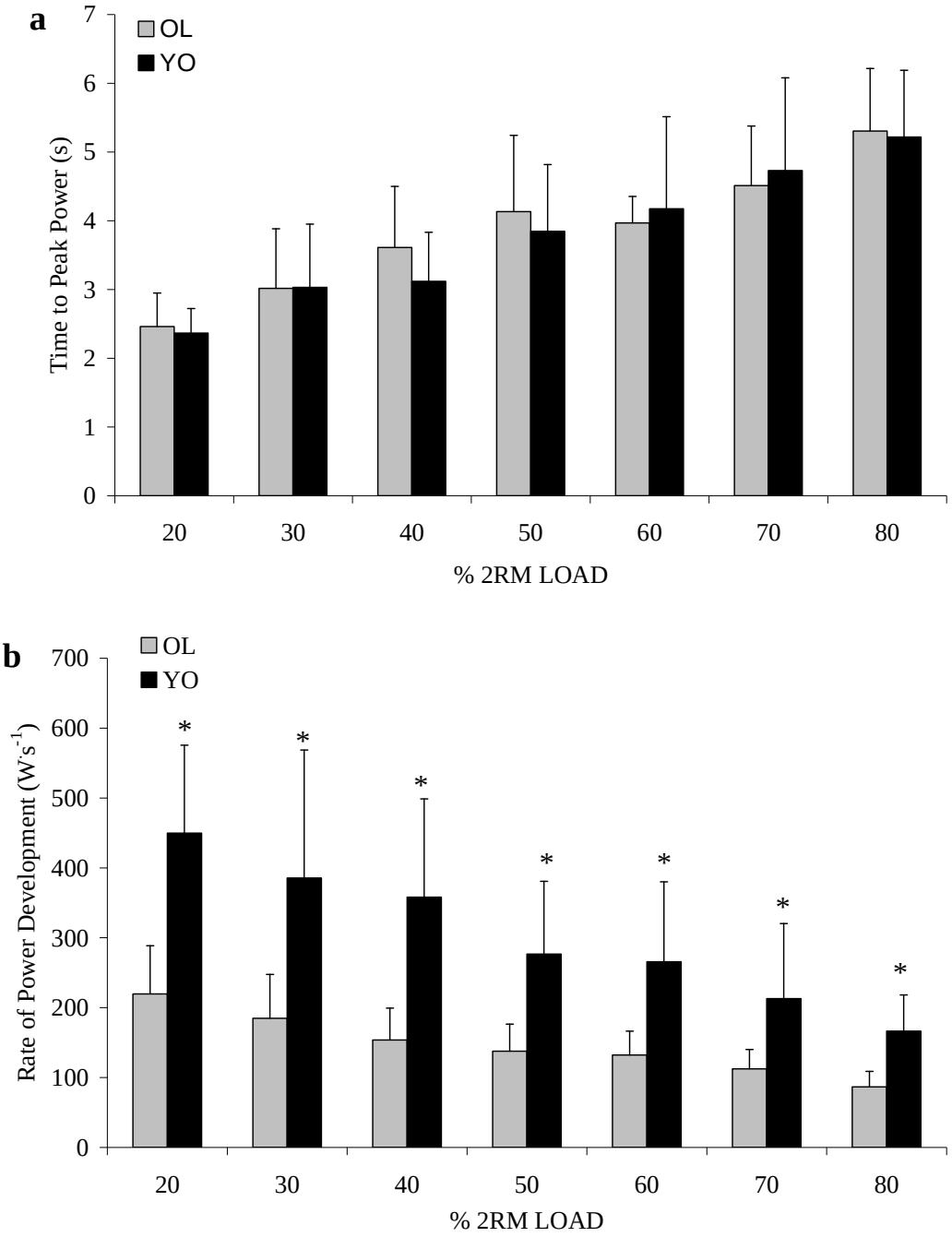

Figure 3.4 a. Differences in time to peak power between groups. **b.** Differences in rate of power development between groups. Values expressed as mean ± SD from each % 2RM load tested. ■ column represent OL group ■ column represents YO group. * Significantly different from OL ($p<0.05$).

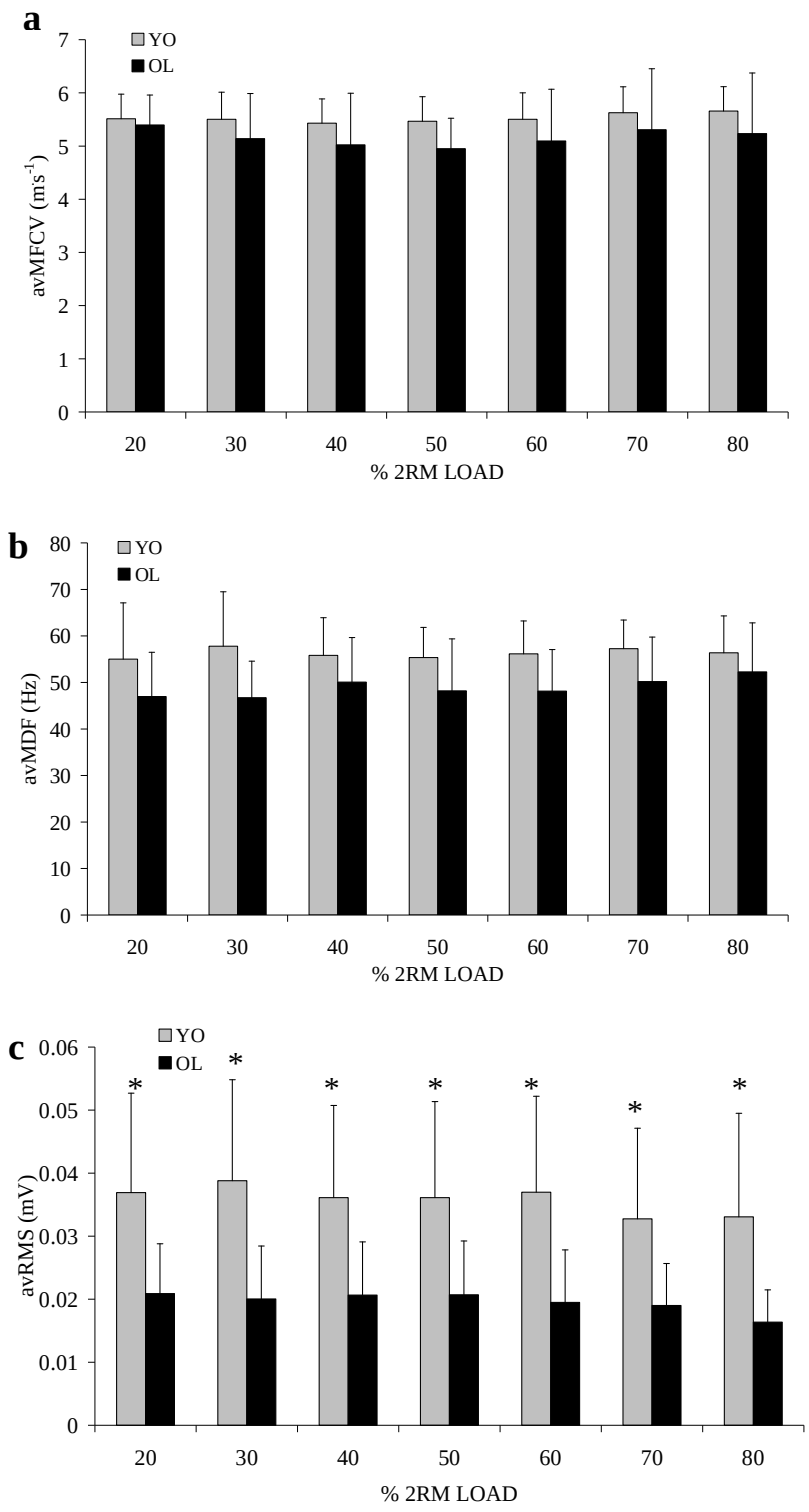

Figure 3.5 Average sEMG data over each 6 s 'all-out' sprint cycle **a**. Average Muscle Fibre Conduction Velocity (avMFCV), **b**. Average Median Frequency of the power spectrum (avMDF) and **c**. Average Root Mean Square (avRMS), from each % 2RM load tested. Values expressed as mean ± SD. ■ Column represent OL group ■ column represents YO group. * Significantly different from OL ($p<0.05$).

Discussion

The main finding of this study is that healthy older women's lower peak power and leftward shift in the Torque-Velocity (T-V) relationship, produced during all-out sprint cycling (from all loads tested) was accompanied by a lower muscle CSA and an overall lower neural activation, expressed as avRMS. Whereas unexpectedly there were no differences between young and older women in how sEMG parameters vary across time during each 6-s sprint, per each load, expressed as delta percentage changes in RMS, MFCV and MDF. Hence, it appears that there are no significant differences in motor control strategies to govern each 6-s sprint with advancing age.

The 33% lower dynamic strength in the older women in the current study, measured as 2RM, is consistent with the finding of others measuring 1RM concentric dynamic strength on resistance training machines (Frontera *et al*., 1991; Larson *et al*., 1979; Lindle *et al*., 1997; Hakkinen *et al*., 2001; Lanza *et al*., 2003). Since muscle torque production is a function of muscle CSA (Roubenoff and Hughes 2000), the smaller muscle mass recorded from OL appears to totally account for their decline in maximal muscle strength, termed sarcopenia. This is supported by the specific strength being equivalent between OL and YO women, which is consistent with a number of studies (Kent-Braun Ng 1999; Klitgaard *et al*., 1990; Frontera *et al*., 2000a), but contradicts the more recent findings of other authors (Narici *et al*., 2004; Morse *et al*., 2005). The lack of differences in specific strength could be attributed to the amount of non-contractile tissue within the muscle, which increases with advancing age, being included in the measurement of muscle CSA (Kent-Braun *et al*., 2000). Therefore, ultrasonography may possibly have led to an overestimation of OL's muscle CSA and specific strength (Kent-Braun *et al*., 2000; Macaluso *et al*., 2002). Nonetheless, results from Reeves *et al*. (2004a) demonstrated ultrasonography as a valid and reliable method of assessing muscle CSA.

The 48% lower PP (average from all loads) produced during the 'all-out' sprint from the OL group, as opposed to their 33% reduction in dynamic strength, provides support towards the ageing process having a significantly greater effect on muscle power output than muscle strength (Skelton *et al*., 1994; Macaluso and De

Vito 2003; Kostka 2005). The lower PP in the OL group can be attributed to significant reductions in both optimal Mh [torque] (22%) and pedV [velocity] (28%) at PP, with the magnitudes of decline in PP and optimal velocity being similar to those reported by Kostka (2005) and Pearson *et al*. (2006) using short-term cycle ergometry comparing older and younger men in their 8^{th} and 3^{rd} decades.

Muscle volume has been found to be proportional to muscle peak power, and the ratio between these factors termed specific power has been used to establish if physiological factors other than changes in muscle mass and muscle architecture are contributing to the decline in peak power with ageing (Thom *et al*., 2005; Pearson *et al*., 2006). On the other hand, Miyatani *et al*. (2002) have demonstrated that muscle CSA is a valid method of producing an estimation of muscle volume of the knee extensors in humans, thus specific power was estimated using muscle CSA. The results show that significant differences in muscle power between the age groups still remained, indicating that physiological factors other than muscle mass are responsible for the decline in muscle peak power. This is supported by Thom *et al*. (2005) using a measure of muscle volume, but disputed by Pearson *et al*. (2006), whose study took not only muscle volume but the MHC-IIa content of the vastus lateralis into account when normalising peak power between young and older participants.

The decline in older women's maximal muscle torque production (indicated by maximal dynamic strength) produces a leftward shift in their T-V relationship that also contributes to the observed lower Power-velocity relationship, and ultimately lowers their peak muscle power (refer to Figure 3.3). Since the magnitude of the decline in muscle CSA does not fully explain the total loss in muscle power experienced by the OL group, the physiological factors affecting movement velocity can be regarded as significant contributory factors in the older women's lower peak power. Pearson *et al*. (2006) found that the lower optimal velocity at peak power, during all-out sprint cycling, was significantly correlated with the muscle's Myosin Heavy Chain (MHC)-IIa isoform content in older and younger participants. This supports previous findings that ageing muscle experiences a preferential loss in the number of fast twitch fibres with a MHC-IIa content that are 5-6 times more powerful than fibres containing MHC-I, and all muscle fibre phenotypes

experiencing a change towards a slower contraction velocity (D'Antona *et al.*, 2003). The effect of which can also be observed in the decline in values of RPD, with the OL experiencing a greater rate of decline for all relative loads. These results highlight that even in the healthy older female individuals muscle peak power is vulnerable to the effects of ageing, which potentially has a severe negative consequence towards their functional independence in the future.

The overall lower neural activation, demonstrated through a significant decrease in the avRMS from OL during 'all-out' dynamic conditions from the vastus lateralis, can be used to regard a lower neural drive as a contributing physiological factor for the decline in muscle torque production, movement velocity and peak power with advancing age (Roos and Rice 1996; Larrson *et al.*, 1997; Macaluso *et al*., 2002; Merletti *et al*., 2002; Thom *et al*., 2005). A lower neural drive has been demonstrated previously during static muscular contractions, which is attributed to either a decline in number of active MUs or MU firing rate (Stegeman *et al*., 2000; Macaluso *et al*., 2002; Merletti *et al*., 2002). Nonetheless, skin conductivity and the volume conductor (amount of subcutaneous fat between electrode and muscle being voluntary activated) have a significant influence on the sEMG amplitude. The subcutaneous fat measured in our study from all participants showed that the average subcutaneous fat thickness was similar between the groups, thus indicating that differences in tissue properties between individuals are unlikely to fully explain the magnitude of difference in sEMG amplitude produced between the age groups, as supported by Macaluso *et al*. (2002) and Merletti *et al*. (2002). One possible limitation in interpreting the RMS data from this study may be regarding the use absolute values of RMS. However, since our EMG data were recorded using loads relative to the individual's strength it may be argued that the EMG data have been normalised (Rouffet and Hautier 2008). Alternatively, sEMG data normalised against those recorded during a maximal voluntary muscular contraction (MVC), may not be appropriate to older individuals due to the possibility that older individuals may not be able to fully activate their pool of MUs, and thus lead to a situation where normalisation may falsely show no differences (Staudenmann *et al*., 2009). Hence, absolute values of RMS are used for comparison between age groups, as performed

by the studies of Häkkinen *et al*. (1998), Merletti *et al*. (2002), Macaluso *et al.* (2000a), Pearson *et al.* (2002) and Bazzucchi *et al*. (2004).

Consequently, the most likely explanation for the decline in avRMS or neural drive is the 'MU remodelling process', which describes a process of apoptosis of large alpha MUs with advancing age leading to a dennervation of muscles fibres that are re-innervated by smaller surviving MUs leading to an increased MU 'innervation ratio' (Brooks and Faulkner 1994; Doherty 2003; McNeil *et al*., 2007). This leads to the remaining active MUs having to stimulate a greater number of fibres bringing about an increased 'load' placed on the MU, which may possibly in turn bring about a decline in MU firing rates (Doherty and Brown 1997; Roos *et al*., 1997; McNeil *et al*., 2005). Even though studies adopting intramuscular EMG show there is a loss of MU numbers with age the general consensus is that lowering of RMS is the result of a decline in MU firing rates (Merletti *et al*., 2002; Roos and Rice 1996; Klass *et al*., 2008). Additional support for this conclusion may be provided by our results that show non-significant changes in MFCV (a size principle parameter) and MDF with age (Merletti *et al*., 2002; Bazzucchi *et al*., 2004; McNeil *et al*., 2005).

Taking our results of the delta change in YO and OL's MDF and MFCV as they stand, no significant differences were found when compared over time during each sprint cycle and between all loads tested. This could be interpreted as the effects of ageing on the OL's percentage of fast twitch muscle fibres being non-significant. This is due to MFCV being influenced by the recruitment of high threshold MUs (Solomonow *et al*., 1990; Pincivero *et al*., 2001). Furthermore, the lack of change in MFCV during dynamic cycling movements has been reported previously by Farina *et al*. (2004b), who concluded that the non-significant changes in MFCV with increasing power output while maintaining a constant high pedal frequency 120 rpm (pedalling velocities that are equivalent to that produced in our study), may be attributed to the small limited effect of increasing muscle force (torque) production on the recruitment of MUs during high pedal velocities. Hence, possibly MFCV did not increase in our study due to the recruitment of additional high threshold MUs not being possible at high pedal velocities.

A significant increase in ΔRMS occurred in parallel with power output in both groups, which has also recently been shown by Macdonald *et al*. (2008), during

a graded incremental aerobic cycle test, and MacIntosh *et al*. (2000) investigating cadence during different sub-maximal power outputs using sEMG. One possible explanation for the observed increases in ΔRMS may be attributed to an increasing neural drive i.e. number of MUs recruited and/or MU firing rate, but it is not possible to determine which central factor/s led to an increase in neural drive using sEMG amplitude alone (Farina *et al*., 2004a). It maybe worth noting that during sprint cycling the muscle groups were continually being recruited and de-recruited to produce the pedalling action, and as the motor tasks was conducted over a short duration of approximately 6-s, this may have led to a situation where the active high threshold MUs had enough time to recover to a sufficient degree between pedal strokes before fatigue would have resulted in myoelectrical manifestations of the sEMG signals.

The finding of previous studies showing that non-significant differences in PP are experienced when sprint cycling against loads varying in resistance, on either a friction-loaded or an inertial-loaded cycle ergometer (Linossier *et al*., 1996; Martin *et al*., 1997; Pearson *et al*., 2004), have been confirmed by our study in both young and older women. Importantly, our results have enabled the identification of an 'upper-load' from utilising a series of loads relative to the individual's maximal dynamic strength, which produces a series of PPs equivalent in magnitude before a significant decline in PP is observed with increasing load (Figure 3.3). The 'upper-load' was found to be dependent on age, with OL maintaining their highest PP using 2RM % loads from 20% to 70%, compared to 20% to 60% 2RM for YO. The differences in the range of relative loads that produced the highest PP may be attributed to the fact that the younger women were exercising against significantly heavier loads even though the same relative loads were being utilised. It has been shown that the load cycled against during sprint cycling determines the portion of the T-V relationship the muscles work along (Sargeant *et al*., 1981; Seck *et al*., 1995), thus using a heavier load, but same relative load, would have caused the YO's muscles to follow a higher relative portion of their T-V relationship in comparison to the OL. This would possibly explain why the significant decline in PP occurred at a lower relative load in the YO.

In conclusion, to the author's knowledge, for the first time it has been demonstrated that a lower neural activation accompanies the decline in peak power with age during 'all-out' sprint cycling on a mechanically-braked cycle ergometer. The decline in muscle CSA has been shown to have a significant effect on the decline in peak power, muscle strength and leftwards shift in the muscles T-V curve with advancing age. However, the changes in muscle CSA alone do not account for the decline in muscle power and its determinants, and thus the observed lower neural drive may be regarded as a contributory factor. The significantly greater decline in muscle peak power than strength confirms the importance of the need to develop effective training methods to improve muscle power optimally in older women in order to combat the threat to their loss of functional independence.

CHAPTER 5

Comparison between young and older women of surface EMG during single leg extensions and electrically evoked mechanical contractile properties

ABSTRACT

The purpose of this study was to investigate the influence of the age-related changes in neural and muscular factors, between young and older women, on the generation of lower leg explosive peak power, which is an important movement gesture for remaining functionally independent. Nine older (OL, aged 71-83 yrs) and 9 younger (YO, aged 19 to 35 yrs) healthy women were asked to perform a series of single leg extensions against five loads, calculated as a percentage of their maximal isometric strength (MVC), to determine differences in peak power output (PP). Surface electromyography (sEMG) was simultaneously recorded during the generation of PP from the vastus lateralis muscle. Muscle contractile properties were recorded using an evoked single maximal muscle twitch from the quadriceps muscle. OL's maximum PP ($p<0.001$), MVC and muscle CSA ($p<0.05$) were all lower than the YO. The decline in MVC with age was accounted for by differences in muscle CSA. SEMG activity indicates signal amplitude and median frequency were significantly lower in the OL at loads corresponding to 60% MVC and above ($P<0.05$). Single muscle twitch characteristics were also slower (13%; $p<0.001$) and weaker (37%; $p<0.01$) in the OL than YO. Hence, a lower neural drive can only be regarded as a contributing physiological factor when PP generated requires a high torque development. Whereas, the physiological factors affecting muscle twitch characteristics appear too have a significant influence on PP generated for any given movement velocity.

Introduction

Older people's ability to perform functional tasks such as standing from a seated position has a significant bearing on their ability to remain functionally independent (Foldvari *et al*., 2000; Ploutz-Snyder *et al*., 2002; Hurda *et al*., 2003; Runge *et al*., 2004). This type of functional manoeuvre requires the generation of muscle force over a short period of time, referred to as explosive muscle power, which has been shown to decline at a greater rate and be a significantly greater predictor of functional disability than maximal strength per se (Bassey *et al*., 1992, De Vito *et al*., 1998; Foldvari *et al*., 2000; Lanza *et al*., 2003; Macaluso and De Vito 2003; 2004; Thom *et al*., 2005). Previously, Macaluso and De Vito (2003) developed an optimisation procedure in order to determine the optimal force and velocity for achieving maximum explosive power output during the execution of a single leg extension, which demonstrated that the decline in power output in 65 to 74 year old individuals was due to a lower ability to generate both muscle force and contraction speed.

Several age-related physiological mechanisms interact to affect both muscle contraction speed and force generating capacity, which include muscular, tendinous and neural factors (Roos *et al*., 1997; Connelly *et al*., 1999; Frontera *et al*., 2000a; Vandervort 2002; Doherty 2003; D'Antona *et al*., 2003; Narici *et al*., 2004**;** Maganaris *et al*., 2004**;** Klass *et al*., 2005; 2008; Thom *et al*., 2005; Pearson *et al*., 2006). Previously, limitations in surface electromyography (sEMG) only allowed the study of neural factors during static muscle contractions (Merletti *et al*., 2002; Vandervoort 2002; McNeil *et al*., 2007). Advancements in sEMG, devised by Farina *et al*. (2004), now make the study of central and peripheral neural control possible during functionally relevant explosive dynamic movements using a linear array technique estimating the average conduction velocity of motor unit action potentials (MFCV). Since, MFCV is regarded as being a size principle parameter, which increases with the recruitment of higher threshold motor units (Andreassen Arendt-Nielsen 1987), it can thus be used to infer age-related changes in motor unit behaviour on explosive muscle power (Farina *et al*., 2002; Pozzo *et al*., 2004; Farina *et al*., 2004). Pozzo *et al*. (2004) successfully utilised EMG linear arrays during the

generation of explosive movements lasting a fraction of a second, using leg extensions, which relates to the execution of several functional tasks of daily living. However, there is no information in the literature on the effects of ageing on neural activation estimated from sEMG during this functionally relevant movement.

Electrical stimulation can be utilised to evoke a single muscle twitch by replacing the voluntary neural drive governing the muscle contraction, which allows an investigation of the muscle contractile properties independent of the central neural influences that affect the muscle performance with age (Davies *et al*., 1983; Roos *et al*., 1997; Pääsuke *et al*., 2000; Thom *et al*., 2005; Klass *et al*., 2008). Studies adopting single twitch electrical stimulation have found that ageing ultimately leads to a weaker and slower muscle contraction (Davies *et al*., 1983; Duchateau and Hainaut 1987; Pääsuke *et al*., 2000; Klass *et al*., 2008). Davies *et al*. (1983), in particular, showed a significant correlation between mechanical contractile characteristics and the generation of muscle power output during fast functionally relevant movements in older men.

The purpose of this study was to compare sEMG parameters between young and older women during a series of single explosive muscular contractions utilising varying loads of resistance in order to determine the maximum peak explosive muscle power and its two determinants, i.e. the optimal force and velocity, in addition to comparing muscle contractile characteristics, evoked through single twitch muscle electrical stimulation. Since, ageing is associated with a decline in motor unit number and firing rate, it is hypothesised that the expected decline in explosive peak power decline will be accompanied by a concomitant decline in neural drive detected through sEMG, together with detrimental effects on muscle contractile characteristics.

Methodology

Participants

With University ethics approval, 18 "medically stable" participants were selected, according to the criteria proposed by Greig *et al.* (1994) for participating in exercise studies, 9 older women (OL) aged 78.3 ± 3.8 yrs (mean ± SD); stature 1.58 ± 0.05 m; body mass 64.7 ± 8 kg and 9 younger women (YO) aged 23.6 ± 5.5 yrs; stature 1.64 ± 0.08 m; body mass 63.0 ± 8.7 kg. Volunteers provided written informed consent and were instructed to maintain their usual levels of physical activity throughout the duration of the study. After an initial familiarisation period participants re-visited the laboratory on a further two occasions with at least 3-4 days between exercise trials. The exercise trials included strength and power measurement on a dynamometer using the lower limbs, a single twitch electrical stimulation test and functional ability tests. All participants completed the study.

Dynamometric Measures

Isometric maximal voluntary contraction (MVC) and muscle power output were measured from the dominant lower limb using a dynamometer (Biodex, Medical Systems Inc, NY). The leg participants use to kick a ball was considered as the dominant leg. As shown in Figure 4.1, participants adopted a slightly reclined position with a seat angle of 110^{o}, and then placed their foot of the dominant leg to a custom made pedal on the dynamometer's crank arm (set at 90^{o}). Once the participant's trunk was secured to the seat, using three belts crossing over the participant's chest and waist, the seat height and distance was adjusted until participants' knee angle reached 90^{o} and the centre of the knee was horizontal with the pedal axial. The non-active leg was placed comfortably in a neutral position. The dynamometer's crank arm rotation resulted in a leg extension range of motion similar to that produced during the cycle pedal down-stroke.

Once the initial position was determined the dynamometer crank arm was brought back until the participants' knee angle was set at 80^{o}, which was determined as the starting position for the MVC and muscle power trials. Before undertaking maximal strength participants performed a series of warm-up leg extension exercises

against varying loads of resistance. Participants were asked to try and push the pedal forward as fast and as hard as possible on a given signal and continue until instructed to rest, resulting in a contraction time of 2-3 seconds. Verbal encouragement was given during each attempt. The MVC trial consisted of 3 attempts with 3 min recovery in between attempts, with the MVC considered as the highest torque averaged over 1-s from any attempt. Further attempts were only permitted if the 3rd attempt was higher than previous MVC values recorded.

Figure 4.1 Experimental set up on chair dynamometer.

Power output trials were conducted approximately 5 minutes after the completion of the last MVC. After adopting the same starting knee angle, as the MVC trial, the dynamometer was set to 'isotonic' mode. The loads used were 40-80% of the participants' MVC, administered in 10% increments, with 3 attempts performed against each of the 5 loads in a random order. Participants were given verbal encouragement to push the pedal forward as fast and as hard as possible during each leg extension, with a 2 min rest period between each attempt. Power output was calculated for all attempts, which was determined as the product of torque

at the instant at which it reached the pre-selected level and the corresponding velocity (Macaluso and De Vito 2003). The highest power output from each load was then recorded.

Surface Electromyography

Multi-channel surface EMG signals were detected from the vastus lateralis muscle of the dominant lower limb using a linear adhesive array (SPES Medica, Salerno, Italy) consisting of 4 electrodes with 10 mm inter-electrode distance, in bipolar configuration. The EMG signals, along with synchronised dynamometric recordings of torque and change in knee angle angle, were amplified (16 channel surface-EMG amplifier, EMG-16, LISiN – OT Bioelettronica, Italy), band-pass filtered (10-500 Hz) and fed into a 12-bit acquisition board sampled at 2048 samples/s per channel, which were all displayed on a PC. The location for placement of the electrodes on the vastus lateralis muscle was chosen during a series of short duration static muscular contractions during which EMG signals were recorded with a moistened solid array of 8 electrodes (silver bars, 5 mm long, 1 mm diameter, 10 mm inter-electrode distance). The array was placed in-line with the estimated fibre orientation between the innervation zone and tendon, as identified from visual inspection of the EMG signals (Farina *et al.*, 2004). The detection of the innervation zone location was performed with a knee flexed at 80° and 170° joint angles (knee angle range of motion), as previously described in chapter 4. Prior to adhesive array placement, the skin was slightly abraded with abrasive paste (Meditec–Every, Parma, Italy) and cleaned with a paper towel. Once the adhesive array was orientated within the marks placed on the skin, conductive gel (20-30 μl) was inserted in each electrode grove of the array for proper electrode-skin contact using a dispenser (Eppendorf AG – Multipette plus, Hamburg, Germany). The quality of the signal was then checked before proceeding with the dynamometric measurements.

Signal Analysis

For each dynamic leg extension and MVC trial the MFCV estimation was weighted by a Gaussian window whose standard deviation (set to 50ms) could be tuned according to the desired area of torque generated. The average MFCV was

estimated using Matlab 7.0.1 (Mathworks, MA, USA), according to the method described by Pozzo *et al.* (2004). The amplitude and frequency of the EMG burst from the same area of torque development was also determined to produce a value of RMS and MDF using the same Matlab program for average MFCV.

Ultrasonography

Quadriceps' mid-thigh cross-sectional area (CSA) and subcutaneous fat depth were estimated using ultrasonography (Diagnostic Scanner Ultrasonic System HS-2000, Honda Electronics CO. LTD. Japan), as described in chapter 4.

Stimulation Test

An electrical stimulator (Constant Current Stimulator, Model DS7A, Digitimer LTD, UK) was utilised to electrically evoke a single muscle twitch from the quadriceps, whilst the corresponding elicited isometric torque was measured by a dynamometer (Biodex, Medical Systems Inc, NY). Participants were seated in an upright position with a knee bend of 90^{o} before being securely fastened to the seat using 3 cross straps over the chest and waist. The participant's lower leg was securely attached to the dynamometer's lever arm using an ankle strap, and then the lever arm's centre of rotation was aligned to the lateral femoral epicondyle. The dynamometer was then set to 'isometric' mode and adhesive stimulation electrodes (VERSA-STIM, 76mm x 127mm, CONMED, USA) were attached proximally and distally above the quadriceps muscles. The anode was connected to the distal electrode and the cathode connected to the proximal electrode. Twitches were evoked using a single rectangular pulse of 50 μs duration. An initial current of 50 mA was evoked and increased in increments of 50 mA every 30s until an increase in current resulted in a plateau of peak torque. The trial producing the highest peak torque was recorded for further analysis and converted into an ASCII file. The trace of torque generated against time (refer to Figure 4.2) was analysed to measure values of Peak Torque (P_t), time to peak torque (TPT), and ½ relaxation time (½RT), which the time taken for the peak torque to return to half its value (Pääsuke *et al.*, 2000).

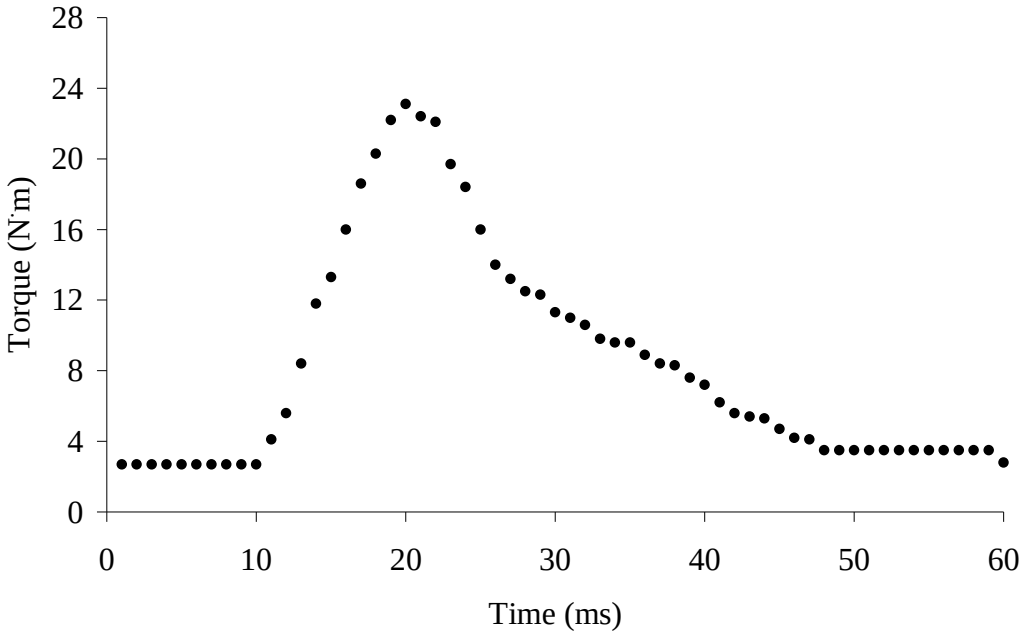

Figure 4.2 A typical trace of an evoked single twitch muscle contraction of one participant from the OL group.

Statistics

Descriptive statistical analyses including means, and standard deviations were calculated for participant's characteristics, MVC, quadriceps CSA, specific strength, P_t, TPT, ½RT, and subcutaneous fat. All data were normally distributed in terms of skewness and kurtosis (all values less than ±2). The effects of age and % load on PP, corresponding torque and velocity, MFCV, RMS and MDF were analysed using 2-way repeated measures ANOVA, with age as a between–subject factor and % load being a within-subject factor. When relevant, a Post hoc one-way ANOVA, with Bonferroni adjustment was performed to identify significant differences between % loads within each of the two age groups and a Student's *t* test to look at differences between the two age groups for each of the percentage loads. Comparisons between young and older subjects for participant's characteristics, MVC, quadriceps CSA, specific strength, P_t, TPT, ½RT, and subcutaneous fat were made using a two-sample Student's *t* test. A Student's t test was also carried out to compare maximum peak power, corresponding optimal torque and velocity and sEMG parameters produced at

maximum peak power between young and older subjects. Statistical significance level was set at $P<0.05$ and all statistical calculations were performed using SPSS software.

Results

Strength and Quadriceps Muscle Cross Sectional Area

Muscle CSA and MVC from the OL were significantly lower than the YO (50.34 ± 4.99 cm^2 vs. 76.6 ± 17.3 cm^2, $p<0.01$ and 101.7 ± 11.8 N·m vs. 155.9 ± 65.5 N·m, $p<0.05$, respectively). However, no differences in strength were found between the groups once strength was normalised by muscle CSA (OL 2.03 ± 0.22 Nm·cm^{-2} vs. YO 2.01 ± 0.42 Nm·cm^{-2}, $p>0.05$).

Dynamometric testing

Figure 4.2 shows peak explosive power and optimal angular velocity of movement, as a function of Torque, at 40, 50, 60, 70 and 80% of the maximal isometric force (MVC). The repeated measures ANOVA showed that there was a significant effect of % load on peak power output (PP) and the corresponding optimal torque and velocity, in addition to a significant interaction between % load and age. Post-hoc analysis reveals that the PP generated at 60% MVC was significantly higher than 70% and 80% MVC in the YO ($p<0.05$), with PP generated from all other % loads being equivalent ($p>0.05$). Whereas, the effect of % load on OL PP showed that PP produced at 80% MVC was significantly lower than 40% and 50% MVC ($p<0.05$), with all other % loads being equivalent ($p>0.05$). Both the corresponding torque and velocity generated at PP for each load were significantly different between all % loads tested in either YO or OL ($p<0.01$).

When the maximum values of PP (as highlighted in Figure 4.3 by the dotted lines) were compared between YO (obtained at 50% MVC) and OL (obtained at 60% MVC), PP was 46% lower in the OL than YO ($p<0.001$) due to a 25% lower optimal torque ($p>0.05$) and a 34% lower optimal velocity ($p<0.001$). However, when individual values of highest PP output were selected and averaged from all loads tested in each group, the significantly lower PP in the OL than YO (245 ± 62 W vs.

449 ± 118 W; $p<0.001$) was due to both a significantly lower optimal velocity (4.55 ± 0.64 rad·s^{-1} vs. 5.83 ± 0.42 rad·s^{-1}; $p<0.001$) and optimal torque (54.3 ± 12.9 N·m vs. 77.8 ± 22.9 N·m; $p<0.05$). The differences in maximum PP generated still remained significant lower in the OL than YO after normalising PP with muscle CSA (average from all loads tested: OL; 3.3 ± 0.9 W kg^{-1} vs. YO; 6.3 ± 1.3 W kg^{-1}, $p<0.001$).

Surface EMG measurements

The sEMG parameters for both MVC and explosive power are shown in Figure 4.3. Due to signal propagation problems only MFCV data from 3 YO and 4 OL are presented. The repeated measures ANOVA showed that there was no effect of age or % load on MFCV ($p>0.05$), whilst there was a significant effect of age on MDF and RMS ($p<0.05$), but no effect of % load nor an interaction between % load and age ($p>0.05$). Post hoc analysis on the effect of age revealed that the OL's MDF was only significantly lower at loads 60% to 80% MVC and RMS was only significantly lower at loads 70% and 80% MVC than in the YO. The MDF and RMS analysed during MVC were also found to be significantly lower in the OL than in the YO by 14% and 58%, respectively ($p<0.05$). Conversely, no significant differences in sEMG parameter were observed at maximum peak power ($p>0.05$).
The subcutaneous fat thickness measured above the vastus lateralis muscle was not significantly different between OL and YO (0.95 ± 0.38 cm vs. 1.25 ± 0.28 cm, $p>0.05$, respectively).

Single twitch electrical stimulation

Due to the discomfort of the muscle stimulation 2 YO participants were unable to complete the measurements and data are therefore presented on 7 YO and 9 OL. The evoked muscle contractile characteristics of P_t were higher (30.7 ± 8.7 Nm vs. 19.4 ± 3.0 Nm, $p<0.01$) and TPT lower (8.9 ± 0.8 ms vs. 10.2 ± 1.1 ms, $p<0.001$) in the YO than the OL, whereas no differences were observed for ½RT (OL: 9.0 ± 2.1 ms vs. YO: 8.1 ± 2.2 ms, $p>0.05$). When P_t was normalised by muscle CSA the significant differences in P_t were cancelled ($p>0.05$).

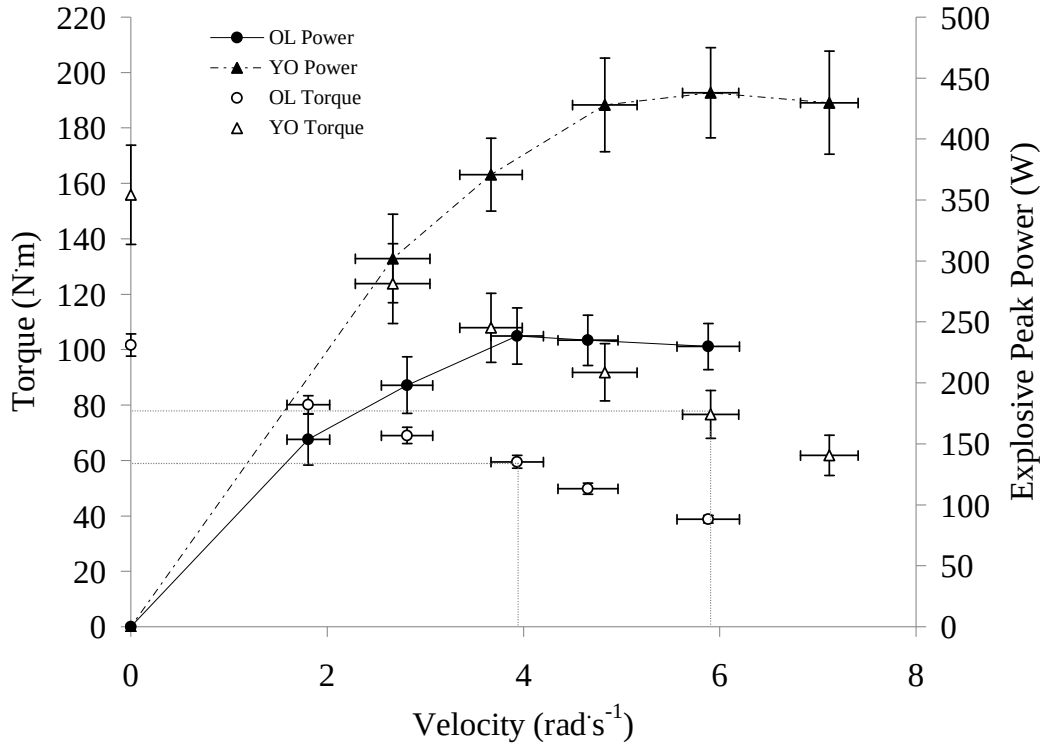

Figure 4.3 Explosive Peak Power-Velocity and Torque-Velocity relationships constructed from relative loads 40, 50, 60, 70 and 80% of the maximal isometric force (MVC). Values are mean ± SE. The Torque-Velocity relationship is displayed with open triangles (Δ) in the young (YO) and open circles (○) in the older (OL) women. The Peak Power-Velocity relationship is represented with closed circles (●) from the OL and closed triangles▲ from the YO. The dotted lines (⋯⋯) represents the optimal torque and velocity corresponding to each group's highest average peak power. Refer to the text for statistical analysis.

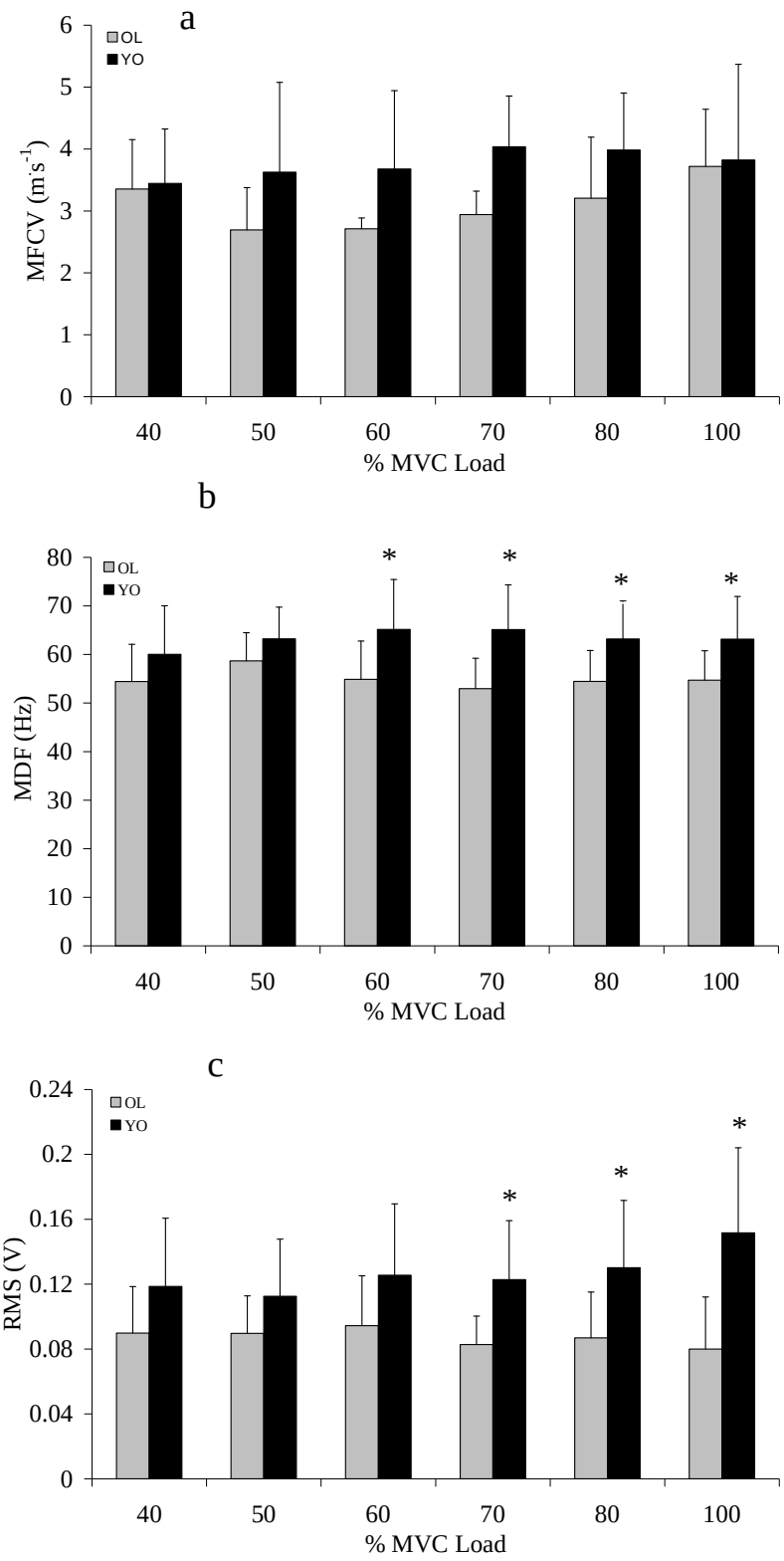

Figure 4.4 Surface EMG **a**. Muscle Fibre Conduction Velocity (MFCV), **b**. Median Frequency of the power spectrum (MDF) and **c**. Root Mean Square (RMS), from each % MVC load tested. Values are expressed as mean + SD. ■ OL group ■ YO group. * Significantly different from OL ($p<0.05$).

Discussion

For the first time, to the author's knowledge, we have shown that the decline in explosive peak power produced during a single leg dynamic extension in healthy older women was accompanied by an overall lower neural activity, expressed as RMS and MDF, but only at % loads exceeding 60% MVC. These changes were accompanied by significant detrimental changes in the muscle CSA and peripheral muscle contractile properties, evoked through single twitch muscle electrical stimulation.

The 35% decline in MVC reported in our study is consistent with that of previous findings adopting a single leg extension protocol (Macaluso and De Vito 2003; Yamauchi *et al*., 2009), which was accompanied by an equivalent 34% decline in muscle CSA. Since muscle CSA is proportional to maximal muscle torque generated (Roubenoff and Hughes 2000) the differences in MVC between the age groups can be fully explained through the decline in muscle mass, termed sarcopenia. This is further supported when maximal muscle torque is normalised with muscle CSA, termed specific strength, using either MVC or P_t (detected from electrical stimulation) showing that there were no significant differences with age. These results contradict recent studies measuring muscle quality, where maximal muscle strength is normalised with corresponding muscle CSA (Macaluso *et al.*, 2002; Narici *et al*., 2003; 2005; Morse *et al*., 2005). One possible explanation for the lack of difference in muscle quality indicated by our study is due ultrasound being unable to differentiate between the increasing amount of non-contractile tissue with contractile tissue within ageing muscle (Kent-Braun *et al*., 2000; Macaluso *et al*., 2002).

The maximum peak power output generated from a single explosive leg extension was on average 45% lower in the older women compared to younger women, results that are similar to those reported in studies adopting a single leg extension (Bassey *et al*., 1992; Macaluso and De Vito 2003; Pearson *et al*., 2006; Yamauchi *et al*., 2009). The decline in the maximum explosive peak power was attributed to a 30% and 22% lower optimal torque and velocity, respectively, which are similar to the findings of Pearson *et al*. (2006) using a modified Nottingham

Power Rig, between men in their 3rd and 8th decade. Our results confirm those obtained in previous studies indicating that for a given percentage of maximal strength (or for a given torque) older women exhibited a slower speed of contraction than younger women, which resulted in a lower peak power and highlights that ageing affects muscle power to a significantly greater extent than muscle strength per se (Macaluso and De Vito 2003; 2004; Lanza *et al*., 2003; Trappe *et al*., 2003; Pearson *et al*., 2006).

The amount of muscle power generated by the quadriceps muscle is proportional to its muscle volume (Thom *et al*. 2005; Pearson *et al*., 2006). However, Miyatani *et al*. (2002) state that there is a significant correlation between muscle CSA and muscle volume and thus in our study muscle peak power was normalised using muscle CSA, which showed that significant differences in specific power still remained with age. Studies by Thom *et al*. (2005) and Pearson *et al*. (2006) also concluded that differences in explosive muscle power with age were not fully accounted for by the differences in muscle volume. Hence, the physiological factors affecting muscle velocity appear to have an additional significant influence on the decline in explosive peak power. Furthermore, since in our study specific strength, in contrast with specific power, appears to be maintained with age, the physiological factors relating to the decline in optimal velocity and velocity of movement for a given load need to be highlighted.

Lexel *et al*. (1988) found using histochemical methods based in the characterisation of the ATPase that ageing muscle experiences a significant increase in the percentage of slow twitch fibre types and a selective atrophy of type II fast twitch fibres within the quadriceps. This has been confirmed by D'Antona *et al*. (2003) using the more sophisticated electrophoretic technique SDS-PAGE, hence ageing muscle appears to change towards a slower phenotype combined with a reduction in high force generating fibres. D'Antona and co-workers also found that the velocity of shortening from all muscle fibre phenotype types were slower with age, possibly due to glycation that causes functional and morphological changes within the muscle fibre that effects the 'cross-bridge mechanics'. The consequence of combining the findings from these studies provides an explanation for the lower optimal velocity and detrimental changes in muscle twitch contractile properties

shown by the older women. However, Trappe *et al.* (2003) disputes the findings that 'cross-bridge mechanics' are affected with age in both men and women, due to the effects of ageing on single muscle fibre peak power being fully accounted for after normalising with cell volume. Conversely, Thom *et al*. (2005) clearly demonstrated that quantitative changes in muscle volume at the 'whole' muscle level only accounted for approximately 50% of the decline in maximal peak power observed between young and older men in their 3rd and 8th decades. Therefore, sarcopenia alone does not account for the decline explosive peak power and that factors relating to a slower muscle contraction speed, as highlighted by detrimental changes in muscle twitch characteristics, appear to have a significant influence on the decline in older woman's explosive peak power (Thom *et al*., 2005; Pearson *et al*., 2006).

Since changes in neural activation affect both force production and movement velocity (Larsson *et al*., 1997; McNeil *et al*., 2007; Klass *et al*., 2008), the lower neural activation detected from the vastus lateralis muscle in healthy older women, demonstrated by declines in both RMS and MDF (but only when torque production was above 60% MVC), can also be regarded as a contributing physiological factor influencing the decline in older women's explosive peak power. The lower RMS can be attributed to a lower neural drive that includes either a reduction in the number of active motor units, motor unit discharge rate or synchronization, which cannot be differentiated using sEMG (Macaluso *et al*., 2000; 2002). The factors that significantly affect the interpretation of sEMG data have to be taken into account when determining if neural drive can be regarded as the most likely cause for the lower sEMG amplitude (RMS), which include the amount of subcutaneous fat located under the electrode placement area (volume conductor) and skin conductivity (Macaluso *et al*., 2002; Farina et al 2004a; Klass *et al*., 2008). Furthermore, one limitation with regards to the sEMG data from this study may arise from not adopting a normalisation procedure, which can add validity to the influence of neural drive being accountable for the lower signal amplitude (Farina *et al*., 2004a; Rouffet and Hautier 2008; Arabadzhiev *et al*., 2009). However, normalising sEMG data may be disadvantageous in regards to older participants, according to Staudermann *et al*. (2009), who state that normalisation of sEMG recordings from older participants may falsely lead to a situation showing no differences in RMS when there are.

Therefore, the absolute values of RMS were used for comparison as carried out by the studies of Merletti *et al*. (2002) and Macaluso *et al*. (2002). In addition, since the average subcutaneous fat thickness recorded between the groups was found to be similar thus differences in tissue properties are unlikely to fully account for the significant decline in RMS with age in older women.

The decline in RMS observed at higher % loads in our study may be explained in relation to the findings of Klass *et al*. (2008), which showed that motor unit discharge rates (using intramuscular EMG) during rapid contractions requiring maximal rate of torque development were significantly lower in older participants aged 71-84 yrs. Hence, the decline in discharge rate can be regarded as a significant contributing factor in the lower ability to generate torque with advancing age, findings that are supported by Merletti *et al*. (2002) and Roos and Rice (1996). These findings when applied to our results may possibly be used to indicate that the lower RMS produced with high loads, which require a high development of torque, can be attributed to a lower motor unit discharge rates. This ultimately inhibites the generation of high muscle torques where the muscle's contraction speed declines in accordance with the muscle's force/torque-velocity relationship. The detrimental impact of which is highlighted by Ferri *et al*. (2003), who state that the older population require an ability to generate power using slow movement speeds in order to perform functional tasks, e.g. generate high torques during slow dynamic movements. Therefore, the decline in neural drive can be regarded as a contributing factor for the reduced ability to generate torque at slower contraction speeds and explosive peak power. Whereas, the non-significant changes observed in sEMG amplitude below loads 60/70% MVC suggest that factors relating to the lower muscle CSA and detrimental changes in muscle twitch characteristics are more likely to explain the decline in explosive peak power in healthy older women.

The lower MDF observed in our study during generation of peak power above 60% MVC may be used to infer a lower MFCV, and also the possibility that older women's muscles appear to have a greater relative proportion of slower twitch muscle fibres than in the younger women (Macaluso and De Vito 2004). On the other hand, recently Farina (2008) questioned the use of MDF in estimating MFCV, especially during explosive dynamic muscular actions, so criticising the use of

spectral frequency of the sEMG signal as a tool for the estimation of the recruited muscle fibres. Unfortunately in the present study, the difficulty in achieving proper signal propagation from sEMG in all participants may be responsible for our observed change in MDF not being supported through the subsequent measure of MFCV.

The observed declines in neural drive, detected through sEMG, are possibly the result of the 'motor unit remodelling process', which describes the process where apoptosis of large alpha motor neuron causes a denervation of fast twitch muscle fibres that are re-innervated by smaller threshold motor units to prevent a total loss of its muscle fibres. This leads to increased innervation ratio and explains the decline in neural drive combined with a greater proportion of slow twitch fibres within aging muscle (Brooks and Faulkner 1994; Doherty 2003; Trappe *et al*., 2003; D'Antona *et al*., 2003; McNeil *et al*., 2005).

In conclusion, for the first time, to the author's knowledge, a lower neural activation was found to accompany the decline in peak power produced during explosive dynamic contractions requiring a high development of torque in healthy older women. Together with a decline in muscle CSA and a weaker and slower evoked muscle contraction, central neural and peripheral factors may be regarded as contributing to the decline in peak power during functional manoeuvres requiring the generation of high torques. Conversely, the generation of peak power requiring faster contraction speeds, as described by the muscles torque-velocity relationship, peripheral changes within the muscle appear to be the most influential factor in lowering peak power.

CHAPTER 6

The effects of cycle resistance training at peak power using different pedalling velocities on muscle power and strength in older women

Data from this chapter were presented at the following conference:

13th Annual Congress of the European College of Sport Science; July 2008, Estoril, Portugal. The effects of cycle resistance training at peak power using different velocities on muscle power in older women.

ABSTRACT

Cycle resistance-training on a friction-loaded cycle ergometer utilising two different loads, which resulted in significantly different pedalling velocities but all loads enabling the development of maximal peak power, were adopted to investigate if training at maximum peak power is the stimulus for optimal gains in 24 older women's muscle power aged 70-80 years. Linear array surface electromyography (sEMG) from the vastus lateralis muscle was synchronised with power output during cycling to investigate central and peripheral neural adaptations. The 8-week training program consisted of twice weekly sessions of 3 sets of eight 'all-out' 8-s supervised sprint cycles, with rest periods of 3 min between sets. The speed training group (SP) utilised a load 20% of the maximum load pedalled through 2 complete revolutions (2RM), whereas strength training group (ST) utilised a 60% 2RM load. All participants were tested pre and post-training for power output from a series of 6-s 'all-out cycles' against loads of 20, 40, 60 and 80% 2RM. The delta increase in peak power, measured during the first pedal stroke (average of all loads tested), was significantly greater in ST (ΔP 106 ± 59 W; mean ± SD) than compared to the SP (12 ± 45 W) and CON (14 ± 16 W; $p<0.017$). ST delta increases in peak power was accompanied by a significant gains in optimal torque only (ST: ΔT 7.6 ± 3.7 N·m) than in comparison to SP (0.08 ± 2.7 N·m) and CON (0.44 ± 2.3 N·m, $p<0.017$). Controversially, no changes in any sEMG variable were observed ($p>0.05$). Since, only ST improved peak muscle power, accompanied by a greater ability to generate torque, training at maximal peak power cannot be regarded as the optimal training load, whilst the training stimulus appears to be the intensity of load utilised.

Introduction

Resistance training interventions for the older population have recently focused primarily on improving maximal muscle power, which is the product of force and velocity of movement. This is due to acknowledgment that ageing is associated with a greater rate of decline in muscle power than muscle strength per se (Skelton *et al*., 1995; Evans 2000; Macaluso and De Vito 2004; Henwood *et al*., 2008). In addition to muscle power's greater association with the ability to perform dynamic movements, which occur more often in performing activities of daily living, than muscle strength per se (Foldvari *et al*. 2000; Evans 2000; Macaluso and De Vito 2004). Nonetheless, the training load and speed of movement that produce 'optimal' gains in older people's maximal muscle power still remains elusive (Macaluso *et al*., 2003; de Vos *et al*., 2005; Hazell *et al*., 2007).

Explosive resistance training adopting high-velocity movements against a medium intensity resistance-load is currently recommended for improving muscle power in the older population, which has been shown to produce similar increases in muscle power as traditional low-velocity high-intensity resistance training (Earles *et al*., 2000; de Vos *et al*., 2005; and Henwood *et al*., 2008). Conversely, Fielding *et al*. (2002) reported a significantly greater improvement in muscle power from a training group adopting a high movement velocity compared to training with a slow movement velocity. Nonetheless, the significant gains in muscle power produced by the 'high-velocity' resistance training group were likely to be explained through training at a higher power output, as both training groups adopted the same relative load. Hence, the optimal training stimulus may be peak power output rather than movement velocity per se, as supported by Mastrapaolo's (1992) maximum-power stimulus theory and Caserotti *et al*. (2008). In order to achieve a training stimulus that produces maximum peak power the training load has to correspond with the portion of the participants' muscles Force-Velocity relationship that allows optimal velocity to be achieved, approximately 30% of maximal value (Hill 1938).

Macaluso *et al*. (2003) devised a novel method of resistance training using friction-loaded cycle ergometry, which compared high-load low-velocity versus low-load high-velocity training and found similar increases in muscle peak power for

both training modes. However, Macaluso *et al*. (2003) may have not adopted the best combination of training load and speed of movement to exercise at maximal power output. In addition, Duffy *et al*. (2009) have recently shown that there are non-significant differences in maximum peak power generated utilising friction-loads between 20% to 60% 2RM (the maximum load turned through two complete revolutions, as devised by Macaluso *et al*. 2003) using friction-loaded cycle ergometry, but differences are observed in corresponding rates of power development and pedalling velocity. Therefore, the resistance cycle-training program adopted two different friction-loads that produced a unique situation where all participants were able to achieve maximal peak power but involving different rates of power development and movement velocities, in order to determine if training at maximum peak power output is the stimulus that produces optimal gains in older women's peak power output and not the level of training intensity. The loads chosen were a 20% 2RM friction-load, as it allowed participants to generate maximal peak power using their fastest rate of power development since it is regarded as a crucial physiological factor for the successful completion of numerous functional tasks (Bassey *et al*., 1992; Narici *et al*., 2005; Caserotti *et al*., 2008). The 20% 2RM load was compared against a heavier 60% 2RM friction-load, which can be regarded as a 'strength training stimulus', but results in maximum peak power achieved through a significantly slower rate of power development and lower pedalling velocity as determined by the muscles' F-V relationship.

Neural mechanisms are thought to be responsible for the early gains in movement velocity and force production with resistance training (Van Cutsem *et al*., 1998; Patten *et al*., 2001). Nonetheless, the neural mechanisms responsible for gains in muscle power due to resistance training have not yet been identified due to limitations in the investigation techniques. Advancements in surface EMG (sEMG), using linear arrays, now provide a method of studying the central and peripheral strategies of the neuromuscular control during explosive dynamic movements through the estimation of the conduction velocity of the action potential travelling along the muscle fibre (MFCV), root mean square and median frequency (Pozzo *et al*., 2004). The estimation of MFCV is regarded as a size principle parameter that increases with recruitment of higher threshold motor units (MUs), and therefore can

be used to infer changes in MU behaviour responsible for increases in peak muscle power (Farina *et al.*, 2004; Pozzo *et al.*, 2004).

This study aims to determine if the training stimulus that produces optimal gains in peak power output in older women is maximal peak power by comparing the effects of two different resistance training loads, which result in significantly different rates of power development and pedalling velocity, but with all training loads allowing the generation of maximal peak power. In addition, the study aims at investigating the changes in neural activation that accompany gains in maximal peak power output through adopting sEMG linear arrays. We hypothesize that the two loads utilised will produce similar gains in maximal peak power output, and because increases in overall neural activation are though to bring about early gains in muscle force and movement velocity, increases in muscle power will be accompanied by a higher MFCV and neural drive.

Methodology

Participants

With approval of the Ethics Committee of the University of Strathclyde, 24 "medically stable" older female participants (aged 74 ± 3.4 yr (mean ± SD); stature 1.58 ± 0.07 m; body mass 68 ± 9.6 kg) were selected, according to the criteria proposed by Greig *et al.* (1994) for participating in exercise studies. Volunteers provided written informed consent and were instructed to maintain their usual levels of physical activity throughout the duration of the training study. After completing a familiarisation period, all participants were tested approximately 2 weeks before the onset of training (week 0) and then re-tested after training (week 8). At week 0, participants were matched for age and dynamic strength before being randomly assigned to one of three training groups: Speed (SP), Strength (ST) or Control (CON).

6 second 'all-out' cycle test

All measurements of peak power output (PP), corresponding optimal torque (Mh) and optimal pedalling velocity (pedV) were carried out on a friction-loaded cycle-ergometer (Monarch 823e, Sweden), as described in Chapter 4.

Power output, Mh and pedV calculations

All calculations of PP, Mh and pedV from each 6-s 'all-out' cycle, on a friction-loaded cycle-ergometer, were carried out as described in chapters 3 and 4. The time to reach PP, from time point zero, was taken as the time to peak power (TPP). The rate of power development (RPD) to PP was calculated as the highest PP produced during each cycle divided by the time to PP.

Surface Electromyography

Multi-channel surface EMG signals were detected from the vastus lateralis muscle of the dominant lower limb using a linear adhesive array (model ELSCH008, SPES Medica, Salerno, Italy) consisting of 4 electrodes with a 10 mm inter-electrode distance, in bipolar configuration. Values of root mean square (RMS), median frequency (MDF) and MFCV were calculated as described in Chapter 4.

Ultrasonography

Quadriceps' mid-thigh cross-sectional area (CSA) and subcutaneous fat depth were estimated using ultrasonography (Diagnostic Scanner Ultrasonic System HS-2000, Honda Electronics CO. LTD. Japan), as described in chapter 4.

Cycle-Resistance Training Program

The SP and ST groups' training consisted of two training sessions per week for 8 weeks on a friction-loaded cycle ergometer (Monark, model 824E). Each participant's training workload was determined as a relative percentage of their 2RM cycle test pre-training. The 2RM cycle test was repeated at week 4 with the training load increasing in accordance with gains in 2RM to maintain the correct training workload. The training sessions were designed so that there was a minimum of 3 days between sessions, and during each session the SP and ST performed 3 sets of

eight ‘all-out’ 8-s supervised sprint cycles. Rest periods were set at 1.5 min after each ‘all-out’ 8-s sprint cycle followed by a 3 min recovery period after each set. The SP group utilised a light workload, 20% 2RM, whereas the ST utilised a heavier workload, 60% 2RM, resulting in significantly different pedV at maximum PP and RPD to maximum PP between the training groups. Conversely, the CON did not participate in any exercise training program and were asked to carry on with their normal levels of daily activity.

Compliance

Of the 24 volunteers initially starting the training program 19 completed the final testing session (79%). The 5 dropouts withdrew at different points during the training program, one due to health problems and four due to lack of time caused by personal commitments and re-scheduled hospital appointments. Compliance with the training program was determined by the number of sessions attended divided by the number of sessions conducted. Exclusion criteria from the training study was set at missing more than two consecutive sessions or undertaking less than 75% of the required training session. No participants were excluded. The mean compliance rate was 100% for CON, 96% for SP and 95% for ST.

Statistics

All data were normally distributed in terms of kurtosis and skewness (all values <2). Statistical comparisons of the parameters (2RM, quadriceps CSA, specific strength, PP, Mh, pedV, RPD, RMS, MDF and MFCV) between training group (ST; SP; CON) across time (pre and post training) were carried out using two-way Repeated Measures ANOVA, followed by Post-hoc Bonferroni-adjusted one-way ANOVAs when appropriate. Statistical significance level was set at $p<0.05$ and all statistical procedures performed using SPSS software. Unless otherwise specified, data were presented as mean ± standard deviation.

Results

There were no significant differences in any variable measured between the three training groups at week 0 ($P>0.05$).

Quadriceps Muscle Cross Sectional Area and Strength

The two-way ANOVA showed that the 8-week training program had a significant effect of time on muscle quadriceps CSA ($p<0.05$), which increased by 8.2 ± 10.1% in ST, 3.8 ± 4.4% in SP and 0.4 ± 2.5% in CON, but no significant effect of group was observed ($p>0.05$). Whereas, a significant effect of time and group by time interaction occurred for maximum dynamic strength, estimated using 2RM ($p<0.001$) and whole muscle specific strength, calculated as the ratio between 2RM and muscle CSA ($P<0.05$). Post hoc analysis showed that the 33.1 ± 17.9% increase in 2RM of ST was significantly greater than both the 7.4 ± 9.1% increase of SP and the 3.9 ± 8.6% increase of CON ($p<0.017$). The 21.1 ± 13.4% increase in specific strength of ST was significantly greater than the 3.5 ± 9.4% increase of CON ($p<0.017$). There was a tendency, however, towards ST producing a greater increase in specific strength than SP ($p=0.034$).

6-s 'all-out' sprint cycle test

The two-way ANOVA for PP (Figure 5.1a) and Mh during the first pedal stroke, averaged from all loads tested, showed a significant effect of time and a significant group by time interaction ($p<0.001$). Whereas, only time had a significant effect on the pedV during the first pedal stroke ($p<0.05$). Comparison of the delta changes across time between the training groups revealed that the 27.4 ± 2.1% increase in PP during the first pedal stroke, averaged from all loads tested, from ST was significantly greater than both the delta increases of 3.6 ± 5.7% from SP and 4.6 ± 2.7% from CON ($p<0.017$). The 23.2 ± 1.9% delta increase from ST of the corresponding Mh, averaged from all loads tested, was significantly greater than both the delta increases of 0.4 ± 3.7% and 0.3 ± 5.4% from SP and CON, respectively ($p<0.017$). Figure 5.1b reports the delta changes of PP during the first pedal stroke for each individual load tested. The ST delta increases of PP and Mh, produced

during the first pedal stroke, were significantly greater than CON at loads 20, 40 and 80% 2RM ($p<0.017$), and significantly greater than SP at 80% 2RM ($p<0.017$).

The two-way ANOVA for the highest PP generated during each 6-s 'all-out' cycle test and corresponding Mh, averaged from all loads tested, also demonstrated a significant effect of time ($p<0.001$ and $p<0.05$, respectively) and a significant group by time interaction ($p<0.05$). There was no effect of time or group on corresponding pedV ($p>0.05$). Comparison of the delta changes across time between the training groups revealed that the 16.3 ± 2.1% increase in the highest PP, averaged from all loads tested, from ST was significantly greater than the average delta increase of 3.3 ± 2.1% from CON. The corresponding Mh, averaged from all loads tested, delta increase of 15.9 ± 5.8% from ST was significantly greater than the delta decrease of -2.2 ± 2.1% from CON ($p<0.05$). The ST delta increase of the highest PP and corresponding Mh produced at each load tested was only significantly greater than CON at 80% 2RM ($P<0.017$).

The two-way ANOVA showed a significant effect of time and group by time interaction for RPD, averaged from all loads tested, during both the first pedal stroke (Figure 5.2a) and to the highest PP generated during the 6-s 'all-out' cycles ($p<0.001$ and $p<0.05$, respectively). Comparison of the delta increase of RPD during the first pedal stroke, averaged from all loads tested, of 39.6 ± 3.8% from ST was significantly greater than the average delta increase of 8.0 ± 7.7% and decrease of -1.8 ± 10.3% from SP and CON, respectively ($p<0.005$ and $p<0.001$, respectively). The average delta increase in RPD to the highest PP, from each 6-s cycle trial, of 25.9 ± 4.9% from ST was greater than the delta decreases of -9.1 ± 16.6% and -0.7 ± 5.7% from SP and CON, respectively ($P<0.05$). Figure 5.2b reports the delta increase in RPD produced during the first pedal stroke, which show that the ST was significantly greater than the CON at all loads tested, and significantly greater than SP at 20% 2RM ($p<0.017$). The delta changes in RPD to the highest PP showed no significant differences across time between groups for any load tested ($p>0.017$).

Surface EMG Linear Arrays

Due to motion artefacts only 3 SP, 3 ST and 2 CON participants were used in two-way ANOVA, which demonstrated that no sEMG variable measured using

linear arrays changed over time pre and post training from any group($p>0.05$; Figure 5.3).

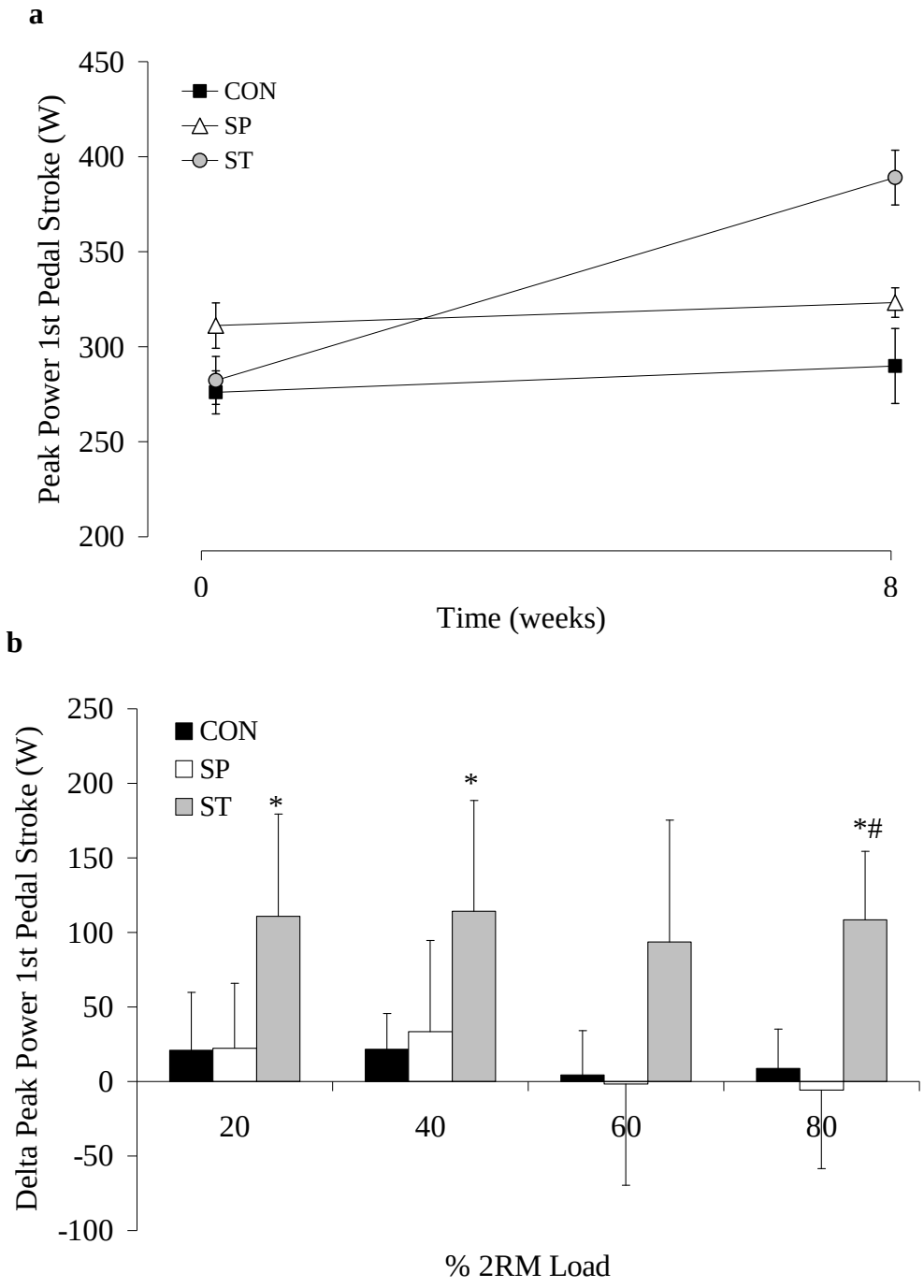

Figure 5.1 a. Mean (±SD) peak power generated during the first pedal stroke across time (week 0 to week 8). **b.** Mean (±SD) delta changes in peak power at each load tested across time. The three training groups are represented as CON: control group; SP: speed training group; ST: strength training group. * significantly different from control and # significantly different from SP group ($p<0.017$).

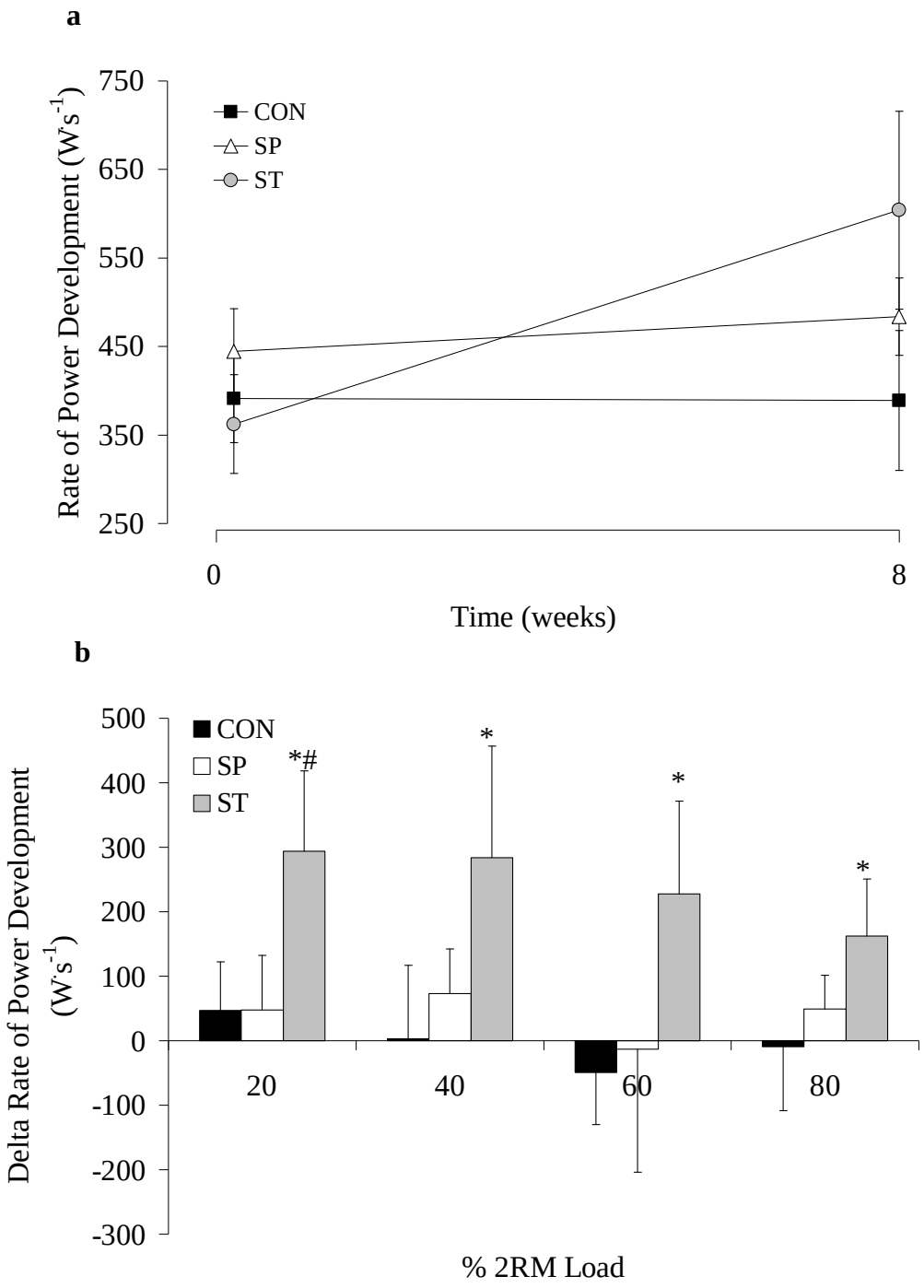

Figure 5.2 a. Mean (±SD) rate of power development during the first pedal stroke across time (weeks 0 to week 8) **b.** Mean (±SD) delta changes in rate of power development during the first pedal stroke at each load tested. The three training groups are represented as CON; control group, SP; speed training group, ST; strength training group. * significantly different from CON and # significantly different from SP.

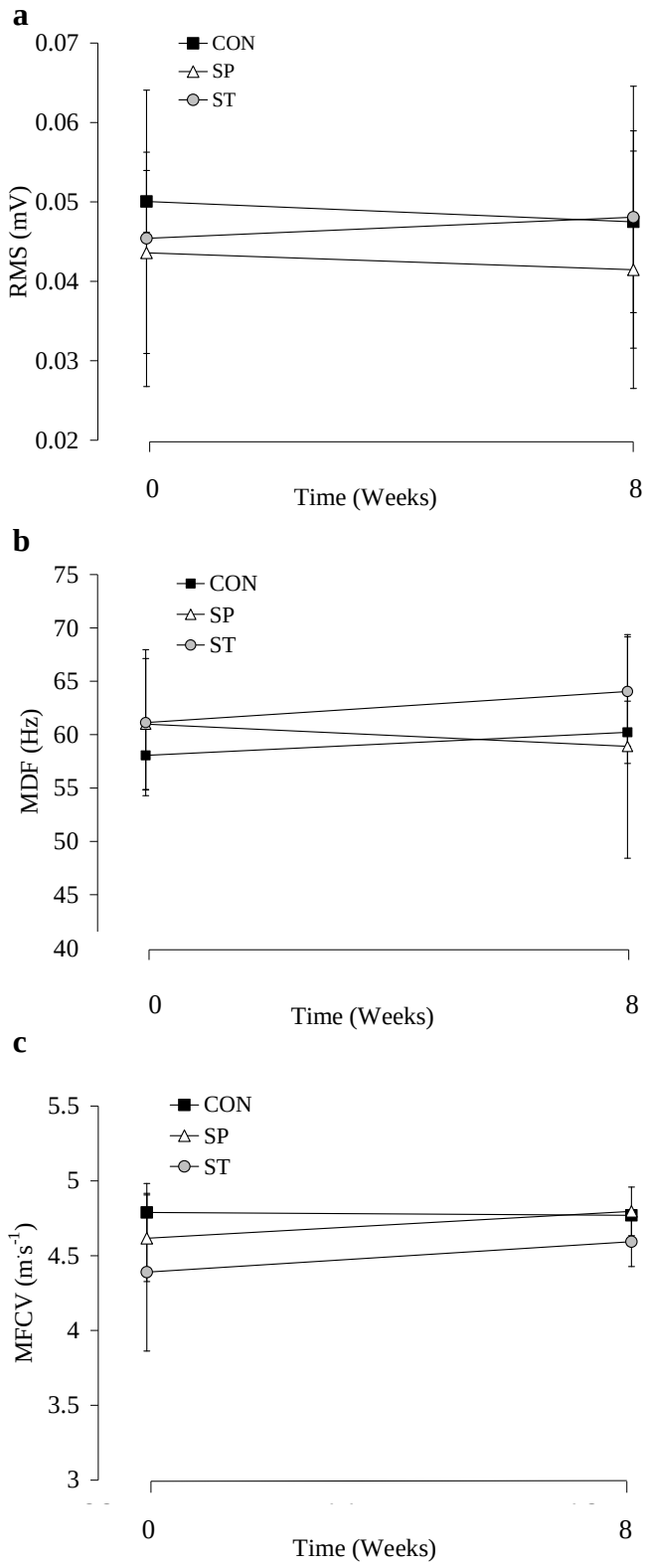

Figure 5.3 a. Mean (±SD) root mean square (RMS) **b.** Mean (±SD) median frequency of the power spectrum density (MDF) **c.** Mean (±SD) muscle fibre conduction velocity (MFCV) from all loads tested across time (week 0 to week 8). There were no significant differences between any group at any time point ($p>0.05$).

Discussion

The main finding of this study is that cycle resistance training at maximum peak power using a resistance load of 60% 2RM (ST training group) produced significantly greater gains in healthy older women's peak muscle power and dynamic strength, than training at a similar peak power using a 20% 2RM resistance load (SP training group). The improvements in peak power were accompanied by significant gains in muscle torque only. Conversely, sEMG variables measured using linear arrays did not mirror the gains in peak muscle power with training.

Maximal dynamic strength, measured using the 2RM cycle test, significantly improved only in the ST group after the 8-week training program. Previously, Macaluso *et al*. (2003) demonstrated increases in 2RM of a similar magnitude over the same training period utilising training loads of 40 and 80% 2RM. Single knee extension maximal dynamic strength, measured using the maximum load moved through 1 complete repetition (1REPmax), has been shown to increase by 30% over a 16-week training period (Ferri *et al*., 2003), and by 20% over a training period of 8-12 weeks (de Vos *et al*., 2005), with both studies adopting a resistance load of 80% 1REPmax. Therefore, the magnitude of increase in dynamic strength described by this study, using 2RM, is consistent also with the findings of previous studies adopting 1REPmax.

The ST group's gains in dynamic strength were accompanied by an 8% increase in quadriceps anatomical CSA, which is consistent with that reported by Reeves *et al*. (2004) and Ferri *et al*. (2003), although these studies utilised longer-term resistance training programs of 14 and 16 weeks with a resistance load of 80-85% 1REPmax. Controversially, Frontera *et al*. (2000a) reported increases in whole muscle CSA after only 2 weeks of progressive resistance training at 65-75% 1REPmax with no significant increases in single muscle fibres observed. Nonetheless, the authors stated that after 12 weeks of training single fibre CSA and specific force had significantly improved. This led to the conclusion that possibly muscle oedema may have been responsible for the early increase in whole muscle CSA, which can not be ruled out as a possible explanation for increases in whole muscle CSA in our study.

Training increased specific strength by 21%, which concurs with the previous findings of Reeves *et al*. (2004). Nonetheless, limitations may arise from our method of measuring specific strength as the ratio between dynamic strength (2RM) and whole muscle CSA. Previous authors have indicated that specific strength should be calculated as the ratio of maximal voluntary isometric strength and the physiological cross sectional area, which takes into account muscle pennation angle, as it is more representative of a muscle's force generating capacity (Reeves *et al*., 2004; Narici *et al*., 2005). In addition, Reeves *et al*. (2004) reported that the gains in specific strength were accompanied by significant increases in pennation angle from older individual's vastus lateralis muscle aged between 70 and 75 years following 14 weeks of resistance training. Therefore, as these changes are not detectable through measuring muscle CSA our results may have overestimated the change in specific strength with cycle resistance training.

The ST group's magnitude of improvement in muscle peak power, generated during the first pedal stroke, is approximately 10% higher than that reported by Macaluso *et al*. (2003). Nevertheless, Macaluso and co-workers found no significant differences in the magnitude of increase in maximal peak power between the training groups, from adopting resistance loads of 40% and 80% 2RM, at any time point during their 16 week training program. Similarly, de Vos *et al*. (2005) found no significant differences in the increase in muscle peak power between 3 explosive resistance training modes, which utilised loads of 20, 50 or 80% 1REPmax, over a training period of 8 to 12 weeks twice weekly. The magnitude of increase from the de Vos study (average 14%) was lower in comparison to that reported by other resistance training studies. Studies conducted by Fielding *et al*. (2002) and Henwood *et al*. (2008) observed larger increases in leg extension peak power (33 and 35%, respectively), using a load 70% 1REPmax, but over longer training periods of 16 and 24 weeks using three and two training sessions per week, respectively. The larger increases in peak muscle power reported by these authors are slightly higher than those of the present study, which may be attributed to the longer training duration. However, our study shows significant improvements after performing 8-weeks training twice weekly, which supports the findings of Hakkinen *et al*. (2001).

One possible factor explaining the differences in magnitude of peak muscle power between our study and Macaluso *et al*. (2003), utilising cycle resistance training, is that they utilised a single-leg extension test to measure peak power, which was not specific to the cycle resistance training gesture. For this reason a short-term (6-second) 'all-out' cycle test, devised in chapter 4, was utilised to assess specific changes in maximal peak power output and corresponding torque and velocity from the cycle-resistance training program. In our study, significant increases in muscle peak power from the ST training group occurred over the range of resistance loads tested, which appears to dispute previous finding of a velocity-specific training adaptation highlighted by Earles *et al*. (2000) in a group of older individuals aged over 70 years. For instance, our study shows that training with a 60% 2RM load produced greater gains in muscle peak power at a specific training resistance load of 20% 2RM adopted by the SP training group, in comparison to the controls.

The improvements in ST group's muscle peak power were accompanied by a significantly greater torque generating capacity than movement velocity, which confirms the results of de Vos *et al*. (2008) but contradicts those of Macaluso *et al*. (2003). The differences between these studies may be attributed to the non-specific testing procedure adopted by Macaulso and co-workers to assess peak muscle power from their novel cycle resistance training program. From our results, it appears that the older participates increased their peak power through a greater ability to generate torque without a change in optimal velocity. Previous studies have either shown no change or a decrease in optimal velocity at peak power in older adults (Earles *et al*., 2000; Macaluso *et al*., 2003; Delmonico *et al*., 2005; de Vos *et al*., 2008). de Vos *et al*. (2005) state that training studies appear to show that older adults generate peak power against a higher load with training and thus generating peak power at a higher portion of their muscles' torque-velocity relationship, which can negatively affect optimal velocity and provides an explanation of why optimal velocity can either decrease or remain unchanged with training. These conclusions are supported by the findings of this study, and highlight that the detrimental effects of ageing on optimal velocity are unlikely to be reversible through resistance training (de Vos *et al*., 2008). Further support is provided at the cellular level, as Trappe *et al*. (2001) found no change in the contraction velocity from any fibre type after a 12 week heavy

resistance training program. Conversely, Parente *et al*. (2008) have shown that older women after a longer training period of 12 months muscles may experience an increase in contraction speed from slow twitch fibres only. The impact of such a change from slow twitch muscle fibres on functional ability can only be speculated.

The increased rate of power development reported in our study may also be explained by improvements in tendon mechanical properties, which has important implications for improved balance and fall prevention in the older population (Onambele *et al*., 2008). Maganaris *et al*. (2004) found that a 14-week resistance training program utilising a load 80% 1REPmax, 3 times per week, produced significant improvements in older men and women's *in vivo* tendon mechanical properties, leading to a significant increase in tendon stiffness and a 27% greater rate of torque development. More recently, Onambele *et al*. (2008) found significantly greater increases in tendon stiffness (136%) using 'flywheel' resistance training, utilising an 80% 1REPmax load three times per week for 12 weeks.

The greater increase in muscle peak power produced utilising a resistance load of 60% 2RM than a 20% 2RM load suggests that factors other than the training power output and the total mechanical work performed (with the total amount of mechanical work being equivalent for each training group) influences the gains in muscle peak power. One possible explanation why the 60% 2RM training load produced significant increases in muscle peak power compared to a 20% 2RM training load may relate to the length of time the muscles exercised at producing higher torques, i.e. the length of time each training load allowed the muscles to exercise along the higher (strength) portion of the muscle's torque-velocity relationship curve. Another factor could be that the lighter the friction-load adopted resulted in a faster the rate of decline in torque generated per pedal stroke as the cycle ergometer's flywheel is accelerated. Therefore, the training stimulus may not only be the amount of torque generated (training load) during the repeated 'explosive' muscular contractions during each training bout but also the length of time the muscles exercise at generating higher torques. Possibly, if taking the results of Macaluso *et al*. (2003) into account, these findings suggest that there is a minimum resistance training load that can be adopted to produce significant

increases in muscle peak power, crucial for improving or maintaining older women's functional ability into later life.

Unfortunately in the present study, the difficulty in achieving proper signal propagation in the majority of older female participants may possibly explain the lack of change in any sEMG variable measured and the lower statistical power of the results. Furthermore, this suggests that the use of linear array sEMG as a tool to monitor changes in neural activity due to resistance training in older women may not be suitable, as supported by Klass *et al*. (2008). Previously, Hakkinen *et al*. (2001) demonstrated that the voluntary neural activation from the vastus lateralis muscle increased after a 6-month progressive resistance training program, which has also been demonstrated by Macaluso *et al*. (2003) using a shorter-term training programs of 16 weeks. In our study, the neural activation from the vastus lateralis increased in the training groups but did not reach statistical significance (Figure 5.3). However, the reported lack of change in MDF confirms that of Macaluso *et al*. (2003). In contrast to previous studies, the speculative lack of change in MFCV suggests that motor unit firing rate and MU number recruited did not change with resistance training (Hakkinen *et al*., 2001; Macaluso *et al*., 2003). Another possible neural factor that may have influenced the increase in muscle peak power and the ability to generate torque refers to a decrease in muscle co-activation between the vastus lateralis and bicep femoris muscles (Hakkinen *et al*., 2001). Moreover, Delmonico *et al*. (2005) found that older women responded differently to strength resistance training than older men, as improvements in older women's muscle quality were not dependent on muscle hypertrophy. Therefore, it is possible that neural adaptations, such as an increase in the number of MUs recruited and/or MU firing rate (neural drive), can not be ruled out as a possible mechanism for increasing the torque generating capacity and muscle peak power from our training study.

In conclusion, cycle resistance training at 60% 2RM was more effective in improving muscle peak power and strength than training at 20% 2RM. Therefore, the training stimulus for improving peak muscle power optimally during cycle resistance training is not the peak power output generated during training, but possibly may relate to the training load during each pedal stroke of the 8-s 'all-out' exertion. Hence, training programs designed for the ageing population may focus on adopting

high training loads due to the increase in peak power being attributed to a greater torque generating capacity. Controversially, changes in sEMG variables did not parallel the improvements in muscle peak power.

CHAPTER 7

GENERAL DISCUSSION

The purpose of this thesis was to investigate some of the neural mechanisms underlying the decline in older women's muscle power and to determine the neural adaptations to a specific mode of resistance training that aims to produce optimal gains in their muscle power. Older women in their $7^{th}/8^{th}$ decade were the chosen population for the studies of this thesis due to their greater vulnerability to the effects of ageing than men of a similar age.

The first study (chapter 3) demonstrated that stereophotogrammetry constructed from motion analysis of the cycle ergometer's flywheel, during 'all-out' cycling on a friction-loaded cycle ergometer, can be utilised as an alternative method for measuring of short-term muscle power and its determinants in older women.

In the second study (chapter 4) for the first time, to the author's knowledge, it has been shown that the decline in older women's short-term muscle power, in comparison to younger women, was accompanied by an overall lower neural activation. This was accomplished using advancements in linear array sEMG, which can be used to speculate changes in motor unit behaviour that govern muscular contractions during the performance of explosive dynamic movements. The older women's lower signal amplitude (RMS) in comparison to younger women, from all cycle loads tested, indicates that the decline in neural drive may be regarded as a contributing factor in their decreased ability to generate peak power.

The third study (chapter 5) investigated for the first time, to the author's knowledge, the effect of ageing on sEMG and muscle contractile properties during the development instantaneous peak power, which reflects the functional gesture of rising from a seated position. SEMG demonstrated that an overall lower neural activation (signal amplitude and frequency) was only significant corresponding to the generation of peak power requiring a high torque/force development. Hence, the peripheral mechanisms, reflected by detrimental changes in muscle contractile properties within older women's muscles, are regarded as the main contributing factor for the decline in muscle peak power requiring high movement velocities.

The last study of the thesis (chapter 6) employed cycle resistance training using two modalities that uniquely produced maximal peak power adopting two different loads that produced significantly different pedalling velocities (low velocity: strength training group vs. high velocity: speed training group) to determine

if the ‘elusive’ stimulus for optimal improvements in older women’s muscle power was training at maximal peak power. The strength training group increased their peak power to a significantly greater extent than the speed training group, with the increase in muscle power being accompanied by a greater ability to generate torque. Therefore, as improvements in muscle power are brought about by a greater ability to generate torque it appears that older women’s muscle power benefits to a greater extent by adopting a cycle resistance training with a high-load. Conversely, no changes in neural activity were observed after training from linear array sEMG recordings taken from the vastus lateralis, which may be attributed to problems attaining good sEMG signals during dynamic movements between trials leading to a low statistical power with regards to sEMG data analysis.

The measurement techniques and procedures used to test the hypotheses in the experimental chapters from this thesis have to be remarked on for their originality. The first study highlights the versatility of motion analysis in its ability to be used as an alternative method of measuring mechanical power from the flywheel of a cycle ergometer, which previously required using incremented encoders. The development of a test to measure short-term maximal muscle power, using cycle ergometry, was crucial for a number of reasons that included; the ability to assess muscle power during movements that replicated the natural physical conditions experienced by the muscles during activities of daily living, as a result producing a valid measure of muscle power relating to functional ability, in addition to developing a test to measure muscle power that was specific to the movement gestures produced during the cycle resistance training used in chapter 6.

In chapters 4 and 5 linear array sEMG provided an insight into the underlying neural mechanisms that can be associated with the decline in muscle power in older women. Specifically, in chapter 4, the leftwards shift in older women’s torque-velocity and power-velocity relationships (Figure 3.3) was likely influenced by a lower number and/or discharge rate of active motor units (neural drive), which can not be differentiated using sEMG (Macaluso and De Vito 2004). A review of the literature can be used to make speculative conclusions on the influence of the two parameters of motor unit behaviour on the generation of muscle power, as discussed in chapter 4. For instance, Klass *et al*. (2008) state that the maximal discharge rates

of active motor units can be fully activated at the onset of a maximal muscle contraction 'performed as fast as possible', a situation that most likely was encountered during the onset of all of the methods for testing muscle power in this thesis. Hence, the lower signal amplitude (RMS) produced by older women may be explained through a decline in motor unit discharge rate (rate coding). Since RMS increased with the generation of short-term muscle power, rate coding may have been the neural mechanism governing the muscular contractions, and thus possibly changes in motor unit discharge rate limits older women's ability to generate muscle power. This has been confirmed by Klass *et al*. (2008) in relation to the maximum rate of force development during isometric contractions.

The amount of subcutaneous fat between the electrode and muscle influences the signal amplitude, as it acts like a 'low-pass spatial filter' and decreases the myoelectrical signal amplitude and frequency (Macaluso *et al.*, 2002; Farina *et al.*, 2004a; Merletti and Parker 2004). However, the average subcutaneous fat between the age groups was not significantly different, thus suggesting that the neural factors discussed are more likely responsible for the lower RMS in older women.

The results from sEMG during the cycle trials, in both the young and older women, demonstrate that RMS increased in accordance with the generation of muscle power whereas MFCV and MDF appeared to remain constant. Importantly, this highlights that the neural strategies governing repeated explosive contractions where the muscles perform along their F-V relationship appear to be unaltered with age in healthy women. Furthermore, since the sEMG parameters either increased or remained constant, indicating that no myoelectrical manifestations of the signal occurred, muscle fatigue may be ruled out as a possible influence on the lower short-term muscle power and neural drive in the older women (Merletti *et al.*, 2002).

The extent of the impact the changes in 'neural drive' have on the generation of muscle power in older women may be indicated by the results from Pearson *et al*. (2006). They found that the differences in maximal short-term power between young and older men were 'eliminated' when normalised with muscle volume and the proportion of type IIA muscle fibres. Therefore, the consequences of the observed detrimental changes in neural drive from the older women may not be regarded as great an influence on the decline in muscle power as the physiological factors that

are found to occur within the muscle with advancing age discussed in chapter 2, e.g. a selective atrophy of type II fibres, reduction in number of type IIA fibres and higher proportion of muscle containing type I fibres, coupled with detrimental changes in muscle volume (reduction in muscle fibre PCSA and length).

The magnitude of decline in maximal peak power and its determinants reported in chapters 4 confirm the findings of previous studies using similar testing methodologies (Macaluso and De Vito 2003; Kostka 2005; Pearson *et al*., 2006). Moreover, the finding that maximal short-term peak power was not significantly different across a number of resistance-loads during 'all-out' cycling, using a friction-loaded cycle ergometer, was confirmed in older women. This unique phenomenon, explained by Pearson *et al*. (2004), enabled the development of a novel training program to test the hypothesis that training at maximal peak power is the stimulus for optimal gains in muscle power in older women, and not the velocity of movement per se.

In chapter 5 instantaneous peak power generated from a single leg extension was investigated as it relates to a different but an important functional gesture than that produced during 'all-out' cycle ergometry, e.g. the ability to rise from a seated position (Bassey *et al*., 1992). An overall lower neural activation (sEMG signal amplitude and frequency) was observed but only where instantaneous peak power required a high generation of torque, as would be experienced during functional tasks that required body weight to be overcome. Therefore, peripheral mechanisms, as highlighted by detrimental changes in older women's muscle contractile properties, suggest that muscular and possibly tendinous factors are more likely to fully explain the decline in instantaneous muscle peak power during very fast explosive muscle contractions, as required in the prevention of falls (Narici and Maganaris 2006; Onambele *et al*., 2008).

The cycle resistance training study, devised in chapter 6, is the first training study to the author's knowledge that has studied the effects of training at maximal peak power using different velocities of movement, in order to investigate if the training stimulus for improving muscle power optimally in older people is training at maximal peak power rather than the velocity of movement per se. This was due to the current literature indicating that the optimal training mode for improving muscle

power in older population still remains elusive and thus warranting further research (Macaluso *et al*., 2003; de Vos *et al*., 2005; Hazell *et al*., 2007). The results show that the stimulus for improving muscle power appears to be more related to the resistance-load, as the strength training group improved their peak power greater than the speed training group. This may be explained through the length of time the muscles worked at the high portion of their F-V relationship.

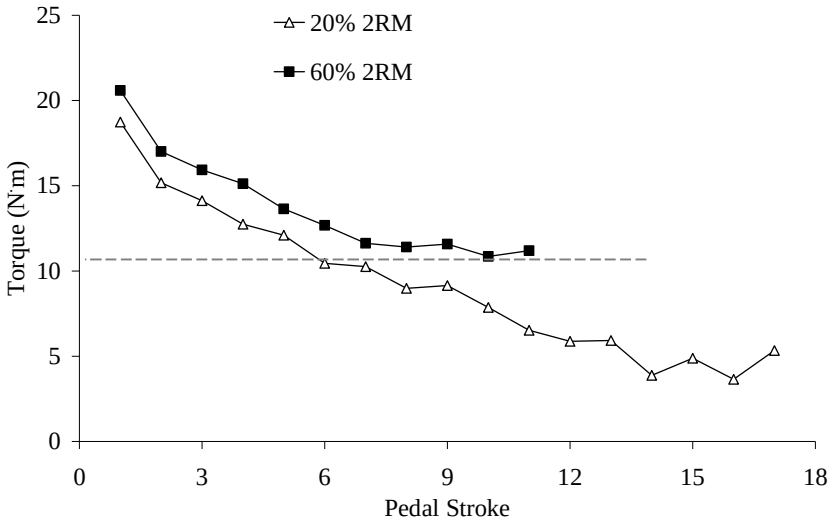

Figure 6.1 The amount of torque produced per pedal stroke during a 6 s 'all-out' cycle trial, pre-training from the same participant, from the two different resistance loads adopted for the training program (20% 2RM: speed training vs. 60% 2RM: strength training). The dotted line represents an arbitrary torque value of approximately 11 N·m.

Figure 6.1 shows that the time it took for the torque pre pedal stroke to decline to approximately 11 N·m, while the individual's pedal rate was continually being accelerated, using a 20% 2RM load was approximately 2.8 s and required 6 pedal strokes. Conversely, cycling against the 60% 2RM load required almost 6 s and involved performing 10-11 pedal strokes for the torque generated to decline to a similar level. Hence, training with the 60% 2RM would have resulted in the participants exercising at a higher torque for a longer duration. This is confirmed by the rate in decline of torque per pedal stroke during the 6-s cycle trials, 1.7 N·m s^{-1} for the 60% 2RM vs. 4.04 N·m s^{-1} for 20% 2RM, relating to the results of figure 6.1.

These results appear to suggest that there is a 'critical' portion and length of time to which the muscles have to be exercised along their F-V curve in order to stimulate gains in torque from all velocities and subsequent muscle power.

Another important finding of our study was that the most trainable determinant of muscle power in older women was the amount of torque generated for a given movement velocity rather than movement velocity, as supported by de Vos *et al.* (2008). Therefore, the most effective method of improving muscle power may be adopting a medium to high resistance training intensity, as the improvements in muscle power are brought about by a greater ability to generate torque. Nevertheless, there appears to be a limit in the training intensity, since resistance training with loads of 80% 1RM or greater do not always produce optimal gains in muscle power (chapter 2). Moreover, it may be further speculated that cycle resistance training may have an advantage over conventional resistance training in improving functional ability, as performing repeated muscular contractions against an inertial load (ergometer's flywheel) results in the muscles exercising along a greater portion of their F-V relationship in comparison to using traditional weight training that only allows the muscles to exercise along a smaller portion of its F-V relationship. This has recently been shown by Onembelle *et al*. (2008) comparing inertial resistance training versus traditional resistance training.

Unfortunately, the neural adaptations to cycle resistance training were difficult to assess due to problems attaining proper signal propagation in older women using multi-channel linear array sEMG. This may relate to both the amount of subcutaneous fat above the muscle and the additional amount of connective tissue within ageing muscle, which possibly causes disruption of the action potential travelling along the muscle fibre. As a consequence it may be inferred that sEMG may not be suitable for estimating motor unit behaviour in older people requiring repeated measurement recordings pre and post-training. Moreover, Delmonico *et al*. (2005) have demonstrated that older woman's muscles appear to have a blunted response for muscle hypertrophy, and thus sEMG spectral analysis should have resulted in significant increases in neural activation in order to explain older women's greater ability to generate torque after 8 weeks of cycle resistance training.

The physical fitness and health status of the 'healthy' older women recruited in all the experimental studies of this thesis need to be commented upon. Participants were recruited only if they could be defined as being 'medially stable', according to a strict criteria devised by Greig *et al*. (1994) for the inclusion in exercise studies. Most of the participants performed recreational physical activity no more than twice weekly, and had no background of regular strength training or competitive sports. Once volunteers were recruited they were required to maintain their normal levels of physical activity throughout the duration of the experimental studies. However, it should be highlighted that the people (young and old) answering to the advertisements of calling for volunteers to undertake exercise studies may already have or had an interest in physical activity, thus possibly not representing the general population. Another important issue highlighted by Macaluso and De Vito (2004), is that comparative studies recruiting young and older individuals may be limited by the fact that older people would have had a totally different experience of physical activity, health and nutrition to that of today's younger population.

The results from chapter 4 and 5 emphasise that even healthy older women experience significant decline in muscle power from the lower limbs, indicating their potential vulnerability to functional disability with advancing age. Since the older women can significantly improve their ability to generate muscle power and strength after 8 weeks of cycle resistance training, this highlights the benefits of regular resistance training in order to attenuate or reverse the effects of ageing with the aim of improving their functional ability and quality of life into later life.

GENERAL CONCLUSIONS AND RECOMMENDATIONS FOR FUTURE STUDIES

The main findings of this thesis are:

1. Motion analysis can be adopted as an alternative technique to measure mechanical power output and its determinants from the flywheel of a friction-loaded cycle ergometer during 'all-out' cycling.
2. For the first time, to the authors' knowledge, linear array sEMG spectral analysis revealed that the decline in older women's short-term muscle power

and it determinants, using cycle ergometry, was accompanied by a lower overall neural activation. However, the neural strategies controlling the muscle contractions were unaffected between the age groups.

3. The decline in instantaneous muscle power from a single explosive leg extension, which relates to rising from a seated position, was accompanied by an overall lower neural activation, but only against loads requiring a high generation of torque. In addition, the muscle contractile properties of older women were significantly slower and weaker in comparison to younger women. Therefore, the decline in muscle power from explosive muscle contractions requiring a fast movement velocity, where torque generation is low, may be related more too peripheral changes in the muscle than neural factors.
4. Cycle resistance training at maximal peak power was not found to be the stimulus for optimal gains in older women's muscle power. This was attributed to resistance training with a medium/high load producing significantly greater increases in muscle power than training with a low intensity load, even though both training intensities resulted in all participants training at their maximal peak power. The stimulus for improvements in muscle power appears to be related to the training load and length of time the muscles exercised at the higher portion of their F-V relationship. The gains in muscle power were accompanied by significant increases in ability to generate torque than movement velocity, and thus highlights that the most trainable determinant of muscle power is torque.

Recommendation for future research:

1. The benefits of resistance training in improving muscle functioning in the healthy older population have been well established. However, further research is required to be conducted with regards to older and frailer populations who may be restricted by health issues in conducting physical activity.
2. Cycle resistance training is a unique exercise as the resistance load can be adjusted to a level relative to the individual's dynamic strength, and is safe in

that the participants do not have risk factors relating to balance while lifting heavy loads. Nonetheless, accessing the high saddle position, as seen on a normal cycle, may be difficult for some older frail individuals, thus future cycle resistance training methods may be made easier by adopting a recumbent cycling position. In this setting the elderly individuals can be easily placed in a normal seated position while still having access for the legs to produce the pedalling action.

3. Resistance training studies may also think about including exercises that resemble functional tasks, i.e. stair climbing and rising from a chair, however this may be inappropriate with the frail elderly population but may be regarded as a factor in developing suitable resistance training programs in relation to healthy older populations.

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PUBLICATIONS AND PRESENTATIONS

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