The Effects of Age- and Training-Related Changes in Tendon Stiffness on Muscular Force Production and Neuro-Motor Control during Childhood

A thesis submitted for the degree of Doctor of Philosophy

by

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"Some of the world's greatest feats were accomplished by people not smart enough to know they were impossible" – *Doug Larson*

Thesis Abstract

The research described in this thesis examined age- and strength training-related changes in Achilles tendon stiffness and plantarflexor force production in prepubertal children. The measurement of both Achilles tendon stiffness and muscular force production requires *in vivo* moment arm lengths to be known. Currently, this is possible only by using expensive and time-consuming medical imaging methodologies. Therefore, the predictability of the Achilles tendon moment arm from surface anthropometric measurements was assessed in the first experimental study (Chapter 3). The results demonstrated that a combination of foot length and the distance between the calcaneal tuberosity and 1st metatarsal head could explain 49% of the variability in Achilles tendon moment arm length in 5 – 12 year-old children. This was considered to be unacceptable for further use, thus an ultrasound-based method was decided upon for obtaining moment arm length in subsequent experimental studies. In the second and third experimental studies (Chapters 4 and 5), age-related changes in tendon mechanical and structural properties were documented and their relationship with changes in force production ability were examined in prepubertal children (5 – 12 years) and adult men and women. In Chapter 4, Achilles tendon stiffness was shown to increase with age through to adulthood, and that changes in tendon stiffness were strongly and independently associated with body mass (R^2 = 0.58) and peak force production capacity (R^2 = 0.51),which may provide the tendon with an increasing mechanical stimulus for growth and microadaptation. These increases in tendon stiffness were associated with a greater increase in tendon CSA (~105%) than that found for tendon length (~60%), in addition to an increase in Young's modulus (~139%), suggesting that gross increases in tendon size as well as changes in its microstructure underpinned the increase in stiffness. In Chapter 5, the relationships between Achilles tendon stiffness and both electro-

mechanical delay (EMD) and rate of force development (RFD) were determined during maximal isometric plantarflexion contractions. Moderate correlations were found between tendon stiffness and both EMD (r = -0.66) and RFD (r = 0.58). RFD was significantly better predicted when muscle activation (estimated as the rate of EMG rise) was included in a regression model. These data clearly show that increases in tendon stiffness with age through to adulthood are associated with decreases in EMD and increases in RFD, and that the rate of muscle activation has an additional influence on RFD during growth. Given that 1) Achilles tendon stiffness was lower in children than adults, 2) this lower stiffness was associated with a longer EMD and slower RFD, and 3) that strength training in adults had previously been shown to increase tendon stiffness and RFD, the adaptability of the developing Achilles tendon to a resistance training programme, and consequence of the potential changes on force production capacity were examined in the final experimental study (Chapter 6). Significant increases in Achilles tendon stiffness and Young's modulus were found after 10 weeks of twice-weekly plantarflexor strength training in 8-9 year-old boys and girls, which demonstrates that the larger muscle force production provided a sufficient stimulus for tendon microadaptation. The training also resulted in a decrease in EMD, which was moderately correlated with the change in tendon stiffness (r = 55), but no change in RFD. Thus, the increasing tendon stiffness with training was associated with a decreasing EMD, but had no detectable effect on RFD. This would likely have a significant effect on the performance of tasks requiring rapid muscle force production. Together, the results of the present series of investigations demonstrate that the tendon loading experienced from both normal ageing and overloading (strength training) can increase tendon stiffness in children, and that these changes have a detectable effect on rapid force production.

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List of Abbreviations

CG_{5-6}	child group age 5 – 6 years
CG_{7-8}	child group age 7 – 8 years
CG_{9-10}	child group age 9 – 10 years
CONT	control group
COR	centre of rotation
CSA	cross-sectional area
CV	coefficient of variation
$d_{calc-met}$	distance between the calcaneus and 1st metatarsal
ECM	extracellular matrix
EMD	electromechanical delay
EMG	electromyography
EXP	experimental group
GM	gastrocnemius medialis
k	stiffness
MA_{AT}	Achilles tendon moment arm
MAFD	mean average fibril diameter
MA_{PT}	patellar tendon moment arm
MRI	magnetic resonance imaging
MT	musculo-tendinous
MTJ	muscle-tendon junction
MTU	muscle-tendon unit
QR	quick-release
RER	rate of EMG rise
RER_{norm}	normalised (to peak EMG amplitude) rate of EMG rise
RFD	rate of force development
RFD_{norm}	normalised (to peak force) rate of force development
SEC	series elastic component
TA	tibialis anterior
TE	tendon excursion
VL	vastus lateralis

CHAPTER 1: General Introduction

1.1 Introduction

Motor skill acquisition is a complex, progressive and multifactorial process in typically developing children (For review, see Thelen, 1995). When new skills are first acquired, children exhibit markedly different movement patterns and large movement variability compared to adults (Clark *et al*., 1988; Sutherland *et al*., 1988; Lasko-McCarthey *et al*., 1990). With practice, movements become more coordinated and refined (Turvey, 1990; Shumway-Cook and Woollacott, 2007) as children acquire the ability to develop and apply task-appropriate forces. As a result, children's movements become more adult-like as they develop (Sutherland *et al.*, 1980b). However, even when movement kinematics in children appear adult-like, differences in the mechanical construction of a task can still remain. More specifically, due to the redundancy of the human system, the same movement kinematics can be produced with a different set of muscular forces or inter-muscular coordination patterns (Bernstein, 1967). Within this context, researchers have shown that children use different inter-muscular coordination patterns than adults to achieve the same movement outcome (Shiavi *et al*., 1987; Sutherland *et al*., 1988; Chao *et al*., 2002; Korff and Jensen, 2007; Korff *et al*., 2009).

Traditionally, differences in movement kinematics and kinetics have often been attributed to an 'immaturity' of the neuromotor system (McGraw, 1943; Forssberg, 1985). The contribution of the developing neuromotor system to motor skill acquisition is irrefutable (Sutherland *et al.*, 1980a; Forssberg, 1999), however, maturation of the nervous system is only one amongst many factors contributing to motor skill acquisition during childhood. In order to gain a better understanding of the process of motor skill acquisition it is necessary to have an understanding of all the factors contributing to the construction of movement

and how they interact within the context of child development. Thus, researchers have recently begun to consider the role of non-neuromotor factors within the acquisition of movement skills during childhood (Wang *et al.*, 2004; Brown and Jensen, 2006; Korff and Jensen, 2008; Korff *et al.*, 2009a).

One example is that a substantial redistribution of mass occurs between body segments during growth (Jensen, 1989). Such an alteration of inertial properties can result in age-related differences in muscular force and power production (Brown and Jensen, 2003, 2006; Korff and Jensen 2008). In fact, adding mass to children's limbs has been shown to produce a more adult-like construction of movement kinematics (Clark and Phillips, 1993) and muscular forces (Brown and Jensen, 2006). Thus, age-related differences in muscular force production may not always be reflective of an 'immature' neuromotor system, but can be reflective of functional adjustments essential in accounting for differences in body size and mass distribution between children and adults.

Such findings also raise an interest as to what role the mechanical properties of the muscle-tendon complex play in motor skill acquisition. It is well established that the mechanical properties of the muscle-tendon complex change during childhood(Kubo *et al*., 2001; Lambertz *et al*., 2003; Cornu and Goubel, 2001; O'Brien *et al*., 2010). As major physiological and mechanical determinants of strength, increases in muscle size (Ikai and Fukunaga, 1968; Malina, 1969) and moment arm length (Wood *et al*., 2006; O'Brien *et al*., 2009) during childhood contribute significantly to age-related changes in force production capacity (Blimkie, 1989; Blimkie and Sale, 1998). Less obvious are the potential influences of age-related increases in the stiffness of the muscle-tendon complex (Kubo *et al*., 2001; Lambertz

et al., 2003; Cornu and Goubel, 2001; O'Brien *et al*., 2010). From research in adults we know that the mechanical properties of the tendon, in particular its stiffness, are important factors influencing force production. More specifically, tendon stiffness governs time delay for the transfer of muscular forces to the skeleton(Hill, 1950; Wilkie, 1950; Cavanagh and Komi, 1979; Muraoka *et al.*, 2004b; Bojsen-Moller *et al.*, 2005; Grosset *et al.*, 2009). Furthermore, tendon stiffness is strongly associated with peak muscular force in adults (Muraoka *et al*., 2005), and it is able to adapt to changes in loading intensity (Kubo *et al*., 2010; Kongsgaard *et al*., 2007; Seynnes *et al*., 2009).

Such findings raise the question about the role of tendon stiffness in muscular force production capacity within motor development/motor skill acquisition. This role is of particular interest as tendons provide the link between muscle and bone and therefore largely govern the rate and mechanical efficiency with which force is transferred during movement. In fact, increases in tendon (Kubo *et al.*, 2001b; O'Brien *et al.*, 2010) and whole muscle-tendon stiffness (Cornu, 2001; Lambertz, 2003) have been reported with increasing age during childhood. Two mechanisms have been proposed for age-related increases in tendon stiffness. First, tendon stiffness depends largely on its dimensions (Proske and Morgan, 1987) such that a greater quantity of material in parallel (i.e. increased cross-sectional area) increases its stiffness whilst more material in series (i.e. increased length) decreases stiffness; both parallel and series tendon growth occurs during normal childhood development (Elliott, 1965; O'Brien *et al*., 2010). In addition to the tendon's physical size, a tendon's stiffness is also influenced by its material properties (Parry *et al.*, 1978a; 1978; Bailey *et al.*, 1998). Young's modulus is defined as a tendon's stiffness normalised to its cross-sectional area and length, and can therefore be used to compare the material

properties of different tendons. Young's modulus is largely determined by the underlying arrangement of the tendon's collagen (Parry *et al.*, 1978; Bailey *et al.*, 1998), which show marked changes with both biological ageing (Vogel, 1980; Nakagawa *et al*., 1996; O'Brien *et al*., 2010) and mechanical loading (Michna, 1984; Michna and Hartmann, 1989). Microstructural changes with both developmental ageing and mechanical loading are also associated with increases in Young's modulus (Kubo *et al*., 2001; Reeves *et al*., 2003; Kongsgaard *et al*., 2007; O'Brien *et al*., 2010). Changes in a tendon's Young's modulus during childhood may result from the tendons maturation, its response to mechanical loading, or both (O'Brien *et al*., 2010). Nonetheless, increases in both tendon cross-section and Young's modulus contribute to age-related increases in tendon stiffness (O'Brien *et al*., 2010). In adults, similar increases have been documented as a result of chronic tendon loading (Kubo *et al*., 2001; Kongsgaard *et al*., 2007). To better understand typical tendon growth, it is of interest whether changes in stiffness or Young's modulus during childhood are the result of actual tissue 'maturation' or simply a by-product of the increased loading with age. Increases in muscular strength (as a result of muscular, neural and mechanical changes) as well as body mass with age would both contribute to the chronic daily loading of a tendon and have both been postulated as factors causing age-related increases in absolute and relative tendon stiffness (O'Brien *et al*., 2010). Whilst it is known that increases in both dimensions and Young's modulus of the patellar tendon occur between childhood and adulthood, there is still a substantial gap in our knowledge of typical tendon development young children and its impact on force development and, ultimately, movement performance.

The potential role of a changing tendon stiffness in the development of force production capacity in children raises the question as to whether increasing tendon loading through means of strength training could increase tendon stiffness and directly (and positively) influence force production in children. It is well established that resistance training improves strength by increasing muscle size and enhancing neural activation capacity in adults (Moritani and deVries, 1979; Van Cutsem *et al.*, 1998; Aagaard and Thorstensson, 2003), and increasing muscle activation in children (Ramsay *et al.*, 1990; Ozmun *et al.*, 1994). Furthermore, resistance training is known to increase tendon stiffness(Reeves *et al*., 2003; Kongsgaard *et al*., 2007; Seynnes *et al*., 2009; Kubo *et al*., 2010) and to improve force production capacity in adults (Kubo *et al.*, 2001a; Reeves *et al.*, 2003b; Grosset *et al.*, 2009). Little is known about the adaptability of the developing tendon to training-related increases in tendon loading. From a developmental perspective, it is of interest to determine whether the children's tendons respond to resistance training in a similar way to that found in adults, given that children are capable of gaining muscular strength with training. Moreover, strength training is becoming an increasingly popular form of exercise amongst children (Faigenbaum *et al*., 1996; Guy and Micheli, 2001).Therefore knowledge of the effects of chronic loading on tendon adaptation is also of significant practical importance. Finally, it is of interest whether the relationship between training-induced increases in stiffness and force production characteristics in children are similar to those observed in adults (Kongsgaard et al., 2007; Kubo et al., 2010). Such findings would further our understanding of how the typically developing tendon may influence movement production in children.

The overall purpose of this research was to gain a clearer understanding of the complex interactions between tendon mechanics and muscular force production during childhood. In

particular, it was of interest to examine the role of tendon stiffness within the context of age-related increases in force production capacities during childhood. In order to achieve this, four experiments were conducted. The first two experimental chapters are descriptive in nature and geared at understanding dimensional aspects of age-related increases in tendons stiffness. The purpose of the first experiment (Chapter 3) was to ascertain whether age-related changes in moment arm length (an essential measure used for deriving tendon stiffness and muscular force) scales as a function of body anthropometry. The purpose of the second experiment (Chapter 4) was to document changes in the mechanical and structural properties in order to differentiate between the contributions of physical tendon growth and tendon maturation on the tendon's mechanical properties during childhood. The final two experiments were aimed at understanding the link between tendon stiffness and the acquisition of muscular force production capabilities during childhood. The third experimental study (Chapter 5) was designed to examine the relationship between force production and tendon stiffness in children, and more specifically to differentiate between neural and mechanical contributions to age-related changes in muscular force production characteristics. The purpose of the final experiment (Chapter 6) was then to examine the adaptability of the developing Achilles tendon to chronic increases in loading, and examine the effect that training-induced changes in tendon stiffness and muscle activation have on the force production capabilities in children.

Before the experimental chapters are presented, the literature relevant to the background of the experimental research will be critically reviewed. Based on the overarching theme of this thesis, the topics of interest have been broadly sectioned into those relating to muscular force production, structure, function and mechanical properties of tendons, and

tendon adaptations with training. These topics are discussed within both a mature and developmental context.

CHAPTER 2: Critical Review of the Relevant Literature

2.1 Determinants of Force Production

2.1.1 Somatic Determinants of Strength in Adults

Strength, defined here as the peak moment or force achieved during muscular contractions under a specific loading condition, is often used as a key parameter in assessing functional performance. The physiological determinants of strength include both neural and muscular influences. Strength is also influenced by a leverage system (i.e. the moment arm) that provides a mechanical advantage to enhance the force applied to the skeleton.

2.1.1.1 Muscle

The basic function of a muscle is to produce force to cause movement or for stabilisation. The functions of muscle are achieved using a well-defined hierarchical filament organisation. The contractile unit of muscle is the sarcomere, composed of myosin and actin filaments that produce muscle shortening and extension according to the sliding filament theory (Huxley and Niedergerke, 1954; Huxley and Hanson, 1954). Repeating sarcomere units form myofibrils (muscle cells) and are enclosed by a sarcolemmal membrane which receives and conducts electrical signals to initiate myofibril contraction. Large numbers of myofibrils assemble together to form muscle fibres, and fibres are bound together into fascicles by perimysium which forms the muscle. Determinants of muscular strength include the muscles size and fibre composition, although the main influencing factor is thought to the muscle's cross-sectional area (CSA; Ikai and Fukunaga, 1968; Close, 1972; Maughan *et al.*, 1983) which defines the number of sarcomeres in parallel. Most muscles have fibres that run at an angle (pennate) to the longitudinal axis of the muscle (i.e. direction of force transmission). Fibre pennation allows a greater number of fibres to run in parallel, thus

increasing the active CSA and peak force potential (Woittiez *et al*., 1984). Muscle pennation also allows a greater quantity of contractile tissue to attach to the tendon (or aponeuroses).

In humans, skeletal muscle is made up of different fibre types that can be distinguished from each other based on their contractile and metabolic properties (Engel, 1962; Pullen, 1977; Schiaffino *et al.*, 1989). In humans, skeletal muscle fibres are classified as being type I (slow oxidative fibres; SO), type IIa (fast oxidative fibres; FOG) or type IIb (fast glycolytic fibres; FG) which are present in different proportions depending on the muscle's function (Pette and Staron, 1997) and typically regarded as being genetically determined (Komi *et al*., 1976; Bouchard *et al*., 1986). There is some evidence to suggest that different fibre types have different specific tensions (Harridge *et al*., 1996; Larsson *et al*., 1997; Bottinelli *et al*., 1999) and, thus, the relative population of each fibre type may influence muscular strength. Differences between populations in their gross muscle size, architectural characteristics or fibre type may contribute to significant differences in force production capacity.

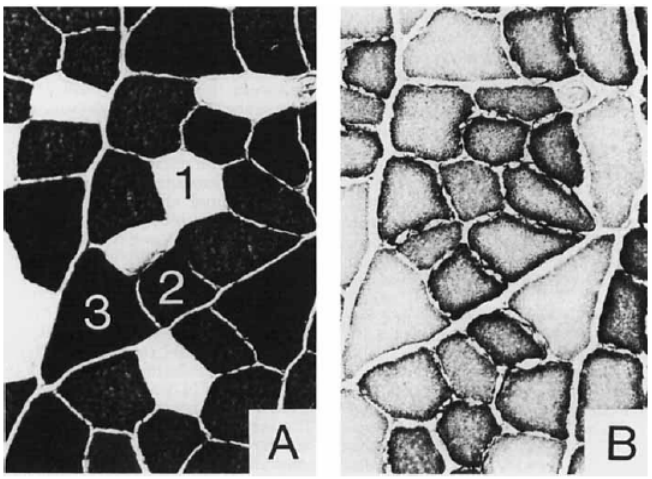


Figure 2-1. Typical photograph of serial cross-sections of rat skeletal muscle showing 1) slow oxidative (SO), 2) fast oxidative glycolytic (FOG) and 3) fast glycolytic (FG) fibres. Fibres were identified by staining for mATPase activity using A) alkaline incubation and B) NADH-t reductase activity (from Armstrong and Phelps, 1984).

2.1.1.2 Neural

Muscle fibres are organised into groups depending on their characteristics, which are innervated by the axonal projections of a single motor neuron (MN). Collectively, this is known as a motor unit (MU). The number of axonal projections, and hence fibres innervated, is positively related to the axonal diameter of a MN, which in addition also dictates action potential conduction velocity (Hursh, 1939; Henneman *et al.*, 1965b). Its conduction velocity is matched to the contractile speed of the innervated fibres (Buller *et al.*, 1960), thus small diameter MNs synapse with a small number of type I fibres to produce small forces and large diameter MNs synapse with a large number of type II fibres to produce larger forces. Like muscle fibres therefore, MUs can be grouped depending on their contractile speed, force producing attributes and sensitivity to fatigue. Henneman *et al.* (1965a, 1965b) also found that MN excitability was an inverse function of their diameter. As such, MUs were recruited in a size dependent, allowing incremental increases in force development with each additionally activated MU. Large MUs, recruited last, are necessary for movements requiring rapid or large forces to be developed, but due to their greater fatigability, only allow such forces to be maintained for short durations. Figure 2-2shows a representation of the innervation of muscle fibres by MNs to make up MUs of varying sizes and contractile properties.

In the adult human, the nervous system can increase a muscle's force output largely by (1) increasing the number of MUs active at a given time (Harrison, 1983) and/or (2) by increasing MN firing rate (Person and Kudina, 1972; Kernell, 2006). It is thought that all MUs are active at around 60% of a muscle's maximum isometric force, with the remaining 40% achieved almost exclusively by increasing MU firing rate (Kukulka and Clamann, 1981; Van

Cutsem *et al*., 1998; Conwit *et al*., 1999; Herzog, 2000). Depending on the muscle, this can typically average 25 – 60Hz during maximal voluntary contractions (review; Enoka and Fuglevand, 2001). Thus, the neural influences on strength are substantive, so differences in the ability of different populations to activate muscle will strongly affect their force production capabilities.

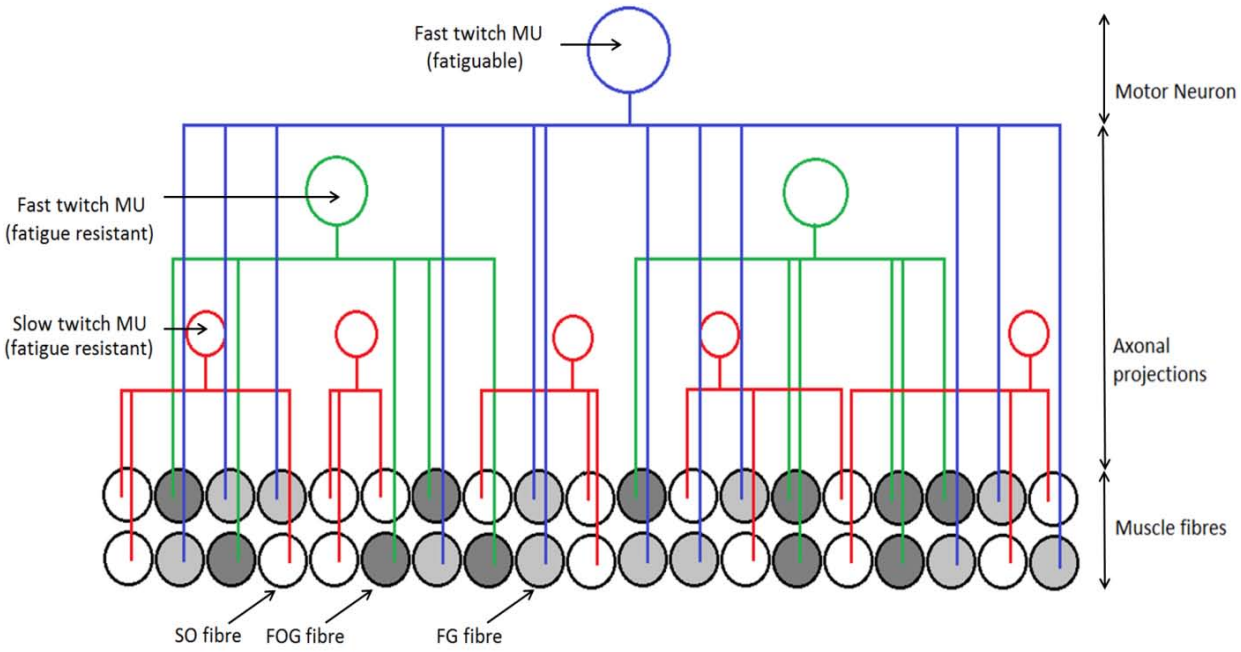


Figure 2-2. Innervation of heterogeneous skeletal muscle. The number of fibres innervated by a motor neurone (MN) is related to its axonal diameter. Axonal diameter also dictates its conduction velocity, which is matched to the contractile speed of the innervated fibres. Small diameter MNs therefore typically innervate a small number of slow, type I fibres (red MU) whilst large diameter MNs innervate a large number of fast, type II fibres (blue MU).

2.1.1.3 Moment Arm

In addition to the physiological determinants of muscle strength, the moment arm, defined as the perpendicular distance between the joint centre of rotation and the line of action of the muscle/tendon (Spoor and van Leeuwen, 1992), is an essential measure from which the

calculation of muscular forces from a joint moment is highly sensitive(Tsaopoulos *et al*., 2007). The muscle gains a mechanical advantage from its tendon inserting onto the skeleton at a distance from the joint centre of rotation by reducing the force required to perform a task. Increasing the moment arm length increases the muscles mechanical advantage for maximum moment production, thus the apparent strength of the muscle will be greater. However, it will also reduce the joint angular excursion for a given muscle shortening distance, or reduce angular velocity for a given muscle shortening velocity. Therefore, differences in muscle-tendon moment arms between populations will influence the active joint moment (i.e. strength) generated for a given muscular force.

2.1.2 Determinants of Strength in Children

Strength increases dramatically with age such that a 2-fold increase is observed between the ages of 5 and 10 years (Blimkie, 1989). This can be attributed to changes in muscle size and architecture, muscle activation and moment arm length observed during childhood.

2.1.2.1 Muscle

Skeletal growth stimulates muscle lengthening during childhood. An overload in muscle tension developed as a result of bone growth prompts sarcomeres to be added in series in order to maintain the resting length of the existing sarcomeres for force production(Goldspink, 1964). Increases in muscle cross-sectional area(CSA) are also seen during childhood(Ikai and Fukunaga, 1968; Blimkie, 1989). As fibre number is thought to be fixed shortly after birth (Gollnick *et al*., 1981), increases in muscle size with development are mostly caused by fibre hypertrophy. In fact, fibre diameter increases three-fold from the age of one through to adolescence (Aherne *et al*., 1971; Colling-Saltin, 1980; Oertel, 1988). Both

the increase in length and CSA lead to an increase in muscle mass. Between the ages of 5 – 17 years, muscle mass increases by 3.5-fold and 5-fold in girls and boys, respectively (Malina, 1969), which have a large influence on strength gains in childhood (Blimkie and Sale, 1998a).

2.1.2.2 Hormonal Influences

From the onset of puberty, boys exhibit large gains in strength to adulthood (Blimkie, 1989). This is largely due to a surge in levels of circulating androgens that promote muscle mass development. Moderate increases in testosterone levels are observed in early puberty, followed by a 20-fold further increase during mid- to late-stages of puberty. Circulating testosterone binds to androgen receptors in the myofibres to stimulate anabolic processes, such as protein synthesis, for muscle growth. Increases in muscle mass occur via the addition of sarcomeres in parallel. In contrast, strength gains in girls past the onset of puberty are minimal and plateau during post-puberty (Blimkie and Sale, 1998a). This is because increased levels of circulating oestrogens associated with the onset of puberty matures the skeleton, thus removing the stimulus provided by skeletal growth for muscle growth (Jones and Round, 2000).

2.1.2.3 Architecture

Fibre pennation angle has been found to increase as a function of age to adulthood (Blimkie and Sale, 1998a; Binzoni *et al*., 2001; Kannas *et al*., 2010), which should allow a greater number of fibres to fit in parallel within the muscle's volume. As CSA is strongly correlated with strength in children also (Ikai and Fukunaga, 1968; Blimkie, 1989), increases in strength with development appear to be partly attributable to increases in pennation angle. An

increased pennation might also increase the amount of tissue connecting to the tendon and ultimately increase muscle force.

2.1.2.4 Fibre type

A considerable differentiation of muscle fibre type occurs between birth and one year of age (Oertel, 1988). By one year of age, fibre type proportions resemble that of an adult. Myotypology is deemed to be complete by the age of three in humans (Colling-Saltin, 1980; Elder and Kakulas, 1993), thus muscle-fibre composition in children is believed to be similar to that of adults. This is supported by the fact that muscle contractile characteristics, such as muscle contraction time and half-relaxation time, examined during electrical twitch stimulation, are similar across age groups (McComas *et al*., 1973; Davies *et al*., 1983; Belanger and McComas, 1989; Paasuke *et al*., 2000). Therefore, it is not considered that changes in muscle fibre type are a significant factor influencing differences in force production or movement behaviour between childhood and adulthood.

2.1.2.5 Neural

Maturation of the nervous system was first suggested as factor influencing force development with age by Asmussen and Heeboll-Nielsen (1955), who found that strength in children increased beyond that expected from muscle growth alone. The evidence for neurological influences on muscle strength in children has grown in recent years (albeit to a somewhat limited extent) and it is now widely accepted that neuro-motor maturation is an important parameter in the acquisition of strength with childhood development (Asmussen, 1973; Blimkie, 1989).

Specifically, it has been postulated that children are not readily able to activate large motor units. Neuromuscular activation under both voluntary and evoked contractile conditions is less in children than adolescents and adults (Belanger and McComas, 1989; Stackhouse *et al*., 2005). For example, Blimkie (1989) found that 11-year-old boys activated a smaller percentage of their available MUs than 16-year-old boys when performing a maximal voluntary contraction (MVC). Similarly, Paasuke *et al.* (2000) reported an increase in the ratio between peak force and muscle CSA with age to adulthood and concluded that children did not fully recruit their MU pool. Halin *et al.* (2003) reported a greater relative decrease in muscular force and contractile velocity in men than boys in response to a 30 s sustained maximal contraction, concluding that maximum force development in boys involved less fast fatigable units than men. A similar suggestion was made by Falk *et al.* (2009) who noted a greater delay in children's force development when compared to adults.

Maximum MN firing rate does not appear to differ significantly between children and adults, although mean firing rate has been shown to be marginally greater in children (Piotrkiewicz *et al*., 2007). This is interesting as children also demonstrate a longer residual latency (delay in transmission at the neuromuscular junction;Thomas and Lambert, 1960). A slower motorneurone conduction velocity is exhibited in children when compared to adults (Thomas and Lambert, 1960; Oh, 1984). Possible differences in nerve fibre diameter (Jacobs and Love, 1985) and nerve myelination (Gutrecht and Dyck, 1970; Webster and Favilla, 1984), may be attributable to this finding. These characteristics may influence muscle recruitment for force production.

2.1.2.6 Moment Arm

The moment arm length of a muscle-tendon unit (MTU) increases with skeletal and muscular growth associated with childhood (Wood *et al*., 2006; O'Brien *et al*., 2009). This has a substantial effect on maximum moment generation and is a key factor underlying age-related gains in muscular strength with development (Wood *et al*., 2006; Morse *et al*., 2008; O'Brien *et al*., 2009). From a developmental context, knowledge of muscle moment arms are important for understanding the mechanisms underpinning age-related increases in strength (Denis and Korff, 2009).

2.1.2.7 Muscle-Specific Force

To compare strength between individuals, muscular force is often expressed relative to muscle size, termed muscle-specific force, which provides a useful measure of intrinsic muscle strength. However, it is influenced *in vivo* by the neural drive activating the agonist muscle, antagonist coactivity levels and moment arm length. These variables must be accounted for to ensure accurate estimations of muscle-specific force (Maganaris *et al*., 2001). Child–adult differences have been reported for specific muscle force (Blimkie, 1989; Paasuke *et al*., 2000). As the physiological determinants of muscle-specific force include a number of neural and muscular influences, differences in factors other than muscle size or fibre architecture must be responsible. During an MVC, antagonistic coactivation creates a negative moment which reduces the resultant recorded MVC by opposing the positive agonistic moment (Kellis, 1998; Maganaris, 1998). Some studies have demonstrated greater coactivation in children (Frost *et al.*, 1997; Lambertz *et al.*, 2003; Grosset *et al.*, 2008) whilst others have not (Kellis and Unnithan, 1999; Bassa *et al*., 2005). The effect of coactivity on muscle-specific force is therefore likely to be dependent on the individual or muscle pair. In

addition, muscle density appears to be relatively constant, irrespective of anatomical site or age of the individual (Lohman, 1986) and as such is not considered to affect specific strength between children and adults either. Based on these findings together, it is likely therefore that neural factors are responsible for the differences in muscle-specific force between children and adults.

2.1.3 Adaptation of Strength with Training in Adults

2.1.3.1 Neural

It is well documented that early gains in muscular strength with resistance training are attributable to improved muscle activation in adults(Moritani and deVries, 1979; Hakkinen and Komi, 1983; Figure 2-3). Changes in neural drive are associated with an increase in EMG activity (Hakkinen and Komi, 1983; Davies and Young, 1985), which may be reflective of, or at least associated with, the earlier activation of MUs in the development of force (Van Cutsem *et al*., 1998; Aagaard *et al*., 2002), a greater maximal firing frequency and the introduction of double motor unit discharges (Van Cutsem *et al*., 1998), an improved MU firing synchronisation (Milner-Brown *et al*., 1975; Moritani *et al*., 1987), a decrease in antagonist activity (Carolan and Cafarelli, 1992; Amiridis *et al*., 1996), an enhanced excitation-contraction coupling (Duchateau and Hainaut, 1984) and increased motor unit excitability (Sale *et al*., 1983). There are a number of reviews detailing these findings, therefore they will not be explored further here (Moritani, 1993; Aagaard, 2003; Duchateau *et al*., 2006). As force production is reliant on motor unit activation, variations in neural drive will affect force production characteristics, and thus influence movement capacity.

2.1.3.2 Morphological

Gross changes in muscle architecture have been shown to occur remarkably early after the onset of a resistance training program (Blazevich *et al.*, 2003; Blazevich *et al.*, 2007a; Blazevich *et al.*, 2007b; Seynnes *et al.*, 2007), followed by the potentially slower process of fibre hypertrophy (Moritani and deVries, 1979; Hakkinen and Komi, 1983; Figure 2-3). Such changes in muscle structure are considered the primary adaptation to long-term training. Preferential hypertrophy of type II fibres is commonly reported after strength training (Thorstensson *et al*., 1976a; Tesch, 1988; Staron *et al*., 1990; Campos *et al*., 2002). Due to a greater specific tension (Harridge *et al*., 1996; Larsson *et al*., 1997; Bottinelli *et al*., 1999) and contractile velocity (Edstrom and Kugelberg, 1968; Burke *et al*., 1971), a greater relative area of type II fibres could have consequences for whole muscle strength and RFD. Variations in muscle size, architecture and relative area assumed by each fibre type may also influence force production characteristics, and thus influence movement capacity. There are a number of reviews available that discuss morphological adaptations to strength training and their relative influences on force production (Blazevich and Sharp, 2005; Folland and Williams, 2007).

2.1.4 Adaptation of Strength with Training in Children

Resistance training also produces gains in muscular strength in children. Previous studies have highlighted the importance of sufficient intensity and volume over training duration (Blimkie and Sale, 1998b; Faigenbaum *et al*., 2007). For example, strength gains have been documented after a period of weekly training (Faigenbaum *et al*., 2007), although individuals training more frequently experience significantly greater strength gains still

(Faigenbaum *et al.*, 2007). Faigenbaum (1999) found significant strength gains in children after 8 weeks of low repetition-high load and high repetition-low load resistance training.

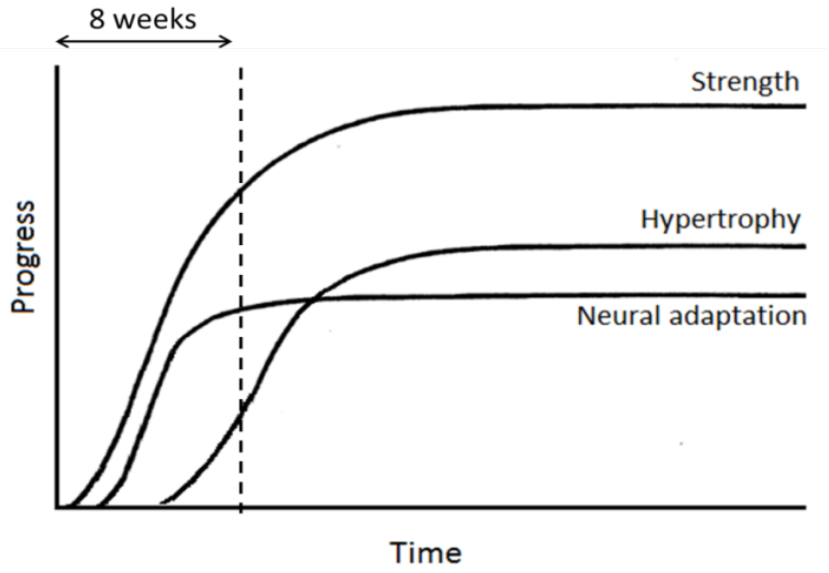


Figure 2-3. Classic model by Moritani and DeVries (1979) presenting the contributions of neural and hypertrophic factors in the time course of strength gains with strength training in adults

These improvements appear to be down to improved coordination of the involved muscle groups via lowered co-contraction and an increase in MU activation ability, rather than structural changes in the muscle. Ramsey *et al.* (1990) did not find an increase in muscle CSA with an increase in maximal force production after a 20-week resistance training plan in 9 - 11 year old boys. The authors conclude that strength gains found were a consequence of increased motor unit activation. A number of studies undertaking a shorter training duration have presented similar results and drawn similar conclusions (Weltman *et al.*, 1986; Blimkie *et al.*, 1989b; Faigenbaum *et al.*, 1993; Ozmun *et al.*, 1994; Damiano *et al.*, 1995). It may be

that a longer training period is necessary to observe muscle hypertrophy in children, although it is clear that an improved ability to recruit muscle is a primary adaptation underpinning the strength increases. The lack of circulating androgen-type hormones in prepubescent children could be a reason that there is little evidence of fibre hypertrophy or increased muscle CSA after a period of resistance training in this population. Nonetheless, the evidence that strength training is effective for promoting strength gain in a prepubescent population provided by the results of previous studies is overwhelming.

2.1.5 Characteristics of force development

The ability to produce movement relies on the ability to produce muscular forces of requisite magnitude and with appropriate timing. The ability to react quickly to a stimulus (e.g. when tripping or falling) or to produce forces rapidly are often important factors in movement success, impacting on daily task (Pijnappels *et al.* 2005; Thelen *et al.* 1996) and sporting performance (Moritani 2002). Two important variables commonly used to characterise the ability to produce fast force are the electromechanical delay and the maximum rate of force development.

2.1.5.1 Electromechanical Delay

Each component of the neuromuscular force development process introduces a delay in the course of tension development. The accumulative delay between the onset of muscle excitation and the onset of muscle force has been termed the electromechanical delay (EMD; Norman and Komi, 1979). EMD is influenced by the transmission of an action potential (AP) across the muscle membrane, mechanisms associated with the excitation-contraction coupling process, velocity of muscle contraction and the efficiency of force

transmission to the skeleton (Hill, 1950; Cavanagh and Komi, 1979; Norman and Komi, 1979; Muraoka *et al.*, 2004b).

The first significant delay is introduced by the transmission of the depolarising signal across the muscle. The motor end plate of the innervating motor neuron (neuromuscular junction) is situated approximately at the level of the muscle belly, thus this is the site that first depolarises. Sarcolemma depolarisation occurs between 3 – 5 m/s (Basmaijan and De Luca, 1985; Zhou *et al*., 1995; Moritani *et al*., 2004). For a distance of 10 cm therefore (e.g. to the distal ends of the muscle), the propagation of the action potential (AP) could introduce a 25 ms delay. The wave of electrical signal initiates a sequence of processes that result in the release of calcium (Ca^{2+}) from the sarcoplasmic reticulum (SR), a structure with the muscle cell that regulates cytosolic levels of Ca^{2+} (Endo, 1970; Ford and Polodosky, 1968). This also introduces a small delay of 3 – 5 ms (Vergara and Delay, 1986). Finally, a major portion of EMD is thought to be the time taken in stretching the elastic structures (tendon) of the MTU to a suitable for tension for force transmission (Cavanagh and Komi, 1979; Muraoka *et al.*, 2004b). Figure 2-4 provides a more detailed overview of the processes underlying these influences of force production.

EMD duration varies considerably between muscles (Komi, 1984; Zhou et al., 1995) and between children and adults (Asai and Aoki, 1996; Grosset et al., 2005). It has been correlated with fibre type proportion, such that muscles with a greater proportion of fast type II fibres have a shorter EMD (Nilsson *et al.*, 1977). As a consequence, EMD decreases with training (Kubo *et al*., 2001; Grosset *et al*., 2009) and also increases with levels of fatigue

MOTOR CORTEX EXCITATION

- Excitation and depolarisation of axons initiated in the motor cortex
- Synapse in the spinal cord with motor neurones directly linked to skeletal muscle
- Excitation and depolarisation of motor neurone (MN)
- Propagation of action potential (AP) along MN axon to neuromuscular junction
- Release of Acetylcholine (ACh) transmitter into synaptic cleft

↓

EXCITATION-CONTRACTION COUPLING

- Binding of ACh to receptors on post-synaptic membrane to increase membrane resting potential
- Depolarisation of muscle membrane (plasmalemma)
- AP propagation across muscle membrane at 3 – 5 m/s
- AP propagation down transverse tubules to activate voltage-sensing dihydropyridine receptors (DHP-R)
- Voltage-mediated signal transmitted from DHP-R to ryanodine receptors (R-R) on the sarcoplasmic reticulum (SR) via projections spanning cytoplasmic gap
- R-R activation causes Ca^{2+} channels on SR to open, releasing Ca^{2+} into the cytoplasm.
- Ca^{2+} binds to Troponin C on actin filaments, moving tropomyosin filament covering cross-bridge binding sites on the actin filament.

↓

DEVELOPMENT OF FORCE

- Head of myosin filament attaches to cross-bridge binding site on actin filament of sarcomere
- Shortening of the sarcomere via sliding of filaments requiring hydrolysis of Adenotriphosphate (ATP) energy.
- Contraction of muscle transmits force longitudinally
- Tendon crimp uptaken
- Force transmission to skeleton

Figure 2-4. Outline of the sequence of events underlying the production of muscular force for movement.

(Zhou *et al*., 1996; Yavuz *et al*., 2010), where fatiguable type II fibres are the first to be lost from muscle recruitment. EMD strongly influences the rapid generation of muscular forces necessary for successful movement and movement reaction times (Wilkie, 1950; Aagaard *et al.*, 2002; Reeves *et al.*, 2003a; Bojsen-Møller *et al.*, 2005; Pijnappels *et al.*, 2005; Holtermann *et al.*, 2007; Blazevich *et al.*, 2008; Pijnappels *et al.*, 2008; Grosset *et al.*, 2009; Nordez *et al.*, 2009)and is necessary to account for when synchronising EMG and kinetic or kinematic data (Muraoka *et al*., 2004). Therefore, EMD should be accounted for on an individual basis when assessing muscle function and force production.

2.1.5.2 Rate of Force Development

In addition to the influence of EMD, the maximum rate of force development (RFD), defined as a change in force with a change in time, has implications for movement generation. RFD is an important parameter describing an individual's capacity to develop explosive force. The ability to develop appropriate forces within a minimal time interval may be prerequisite for successful task completion. For example, the average reported time taken for a muscle to reach its maximum force is 300 - 400 ms (Sukop and Nelson, 1974; Thorstensson *et al*., 1976b), whilst many fast movements may involve muscle contraction times considerably shorter than this (Aagaard *et al*., 2002). Although the importance of RFD is obvious for athletes who have a need to develop large forces rapidly (Moritani, 2002), it also has important consequences for the prevention of falls (Pijnappels *et al*., 2005). Level of neural activation, or efferent motor drive (Nelson, 1996; Van Cutsem *et al*., 1998; Aagaard *et al*., 2002), the muscle's size and physiological properties (Buller and Lewis, 1965; Harridge *et al*., 1996; Andersen and Aagaard, 2006), and the mechanical properties of the MTU's elastic

structures (Wilkie, 1950; Wilson *et al.*, 1994; Bojsen-Møller *et al.*, 2005) are all factors influencing RFD.

Peak strength, associated with muscle size, influences the slope of the force-time curve (Hakkinen *et al*., 1985; Aagaard *et al*., 2002) if peak strength is attained in the same time interval. In order to directly compare RFD between individuals therefore, RFD is commonly normalised to peak force, as this removes peak muscle strength as a factor influencing RFD (Aagaard *et al*., 2002; Blazevich *et al*., 2008). Normalised RFD provides an insight into the neural and physiological determinants of maximal RFD.As previously discussed, muscular force development is dependent on the activation levels of the innervating motor neurones (Adrian and Bronk, 1929). The rate at which MUs can be activated is a key factor influencing rapid force development (Corcos *et al*., 1989; Nelson, 1996), i.e. a rise in RFD is accompanied by an increased MU firing rate (Grimby *et al*., 1981).

Slower, type I, fibres activated early in the force production process have a slower shortening velocity than type II fibres (Edstrom and Kugelberg, 1968; Burke *et al*., 1971), probably due to a lower cross-bridge cycling rate (Metzger and Moss, 1990), and a slower RFD than fast type II fibres. The functional importance of this was demonstrated by Harridge *et al.* (1996) who found that a muscle's maximum contractile RFD was related to its fibre type composition. There is also a growing body of literature demonstrating that large motor units (innervating type II fibres) may be recruited at a lower firing threshold when faced with a task requiring rapid force development (Feiereisen *et al*., 1997; Linnamo *et al*., 2003; Wakeling, 2004; Hodson-Tole and Wakeling, 2009). The implications of these two processes

are particularly relevant when examining force development in children, due to their lower capacity for large MU recruitment.

Children demonstrate a significantly slower RFD than adults (Belanger and McComas, 1989; Grosset *et al*., 2005), even when normalised to peak strength (Asai and Aoki, 1996; Falk *et al*., 2009). This suggests that children may have a significantly lower skeletal muscle maximal shortening velocity than adults (Fuchimoto and Kaneko, 1981; Belanger and McComas, 1989) or a lesser ability to rapidly recruit fast type II fibres (Asai and Aoki, 1996; Grosset *et al*., 2005). Fibre type is determined early in life (Oertel, 1988) and is not thought to differ substantially between children and adults(not performing specific exercise training).As adults may have a greater potential for fibre hypertrophy - and there is some evidence that fast-twitch fibre hypertrophy is more achievable (Hakkinen *et al.*, 1985a; Fry, 2004) - they may possess a greater relative area of fast twitch fibres.

Another factor that has been shown to substantially influence muscular force production is the mechanical properties of elastic components within the muscle-tendon complex. They influence RFD by affecting the efficiency of muscular force transmission to the skeletal (Wilkie, 1950; Hill, 1950; Bojsen-Moller *et al*., 2005). Although there are several elastic bodies within the MTU, the major elastic structure is the tendon. Thus, a detailed review of the structure and function of the tendon and its importance in force production is necessary.

2.2 Tendon Properties

2.2.1 Tendon Ultrastructure

The tendon's basic function is to transmit tensile forces from a muscle to a bone in order to produce a movement. Tendons are highly organised, hierarchical structures that are mainly composed of water and collagen. Collagen, synthesised by specialised fibroblasts called tenocytes, self-associate to form fibrils that run longitudinal to its axis. Several parallel fibrils embedded together within a tendon's extracellular matrix (ECM) form fibres (Benjamin and Ralphs, 1996; O'Brien, 1997; Vogel, 2003), which group together to form fascicles and make up the gross tendon structure (Kastelic *et al.*, 1978; Figure 2-5). Macroscopically, the parallel arrangement of collagen bundles gives the tendinous tissue a wavy configuration, known as crimp (Diamant *et al*., 1972; Dale *et al*., 1972). Straightening this waveform by applying a load allows tendons to elongate by ~4% (Rigby *et al*., 1959), thus providing a buffer against mechanical loading. In addition to this characteristic, the complex biological structure of the tendon provides it with a large inherent tensile strength. The extensibility and tensile strength of a tendon allows it to withstand forces generated through muscular loading whilst maintaining its structural integrity.

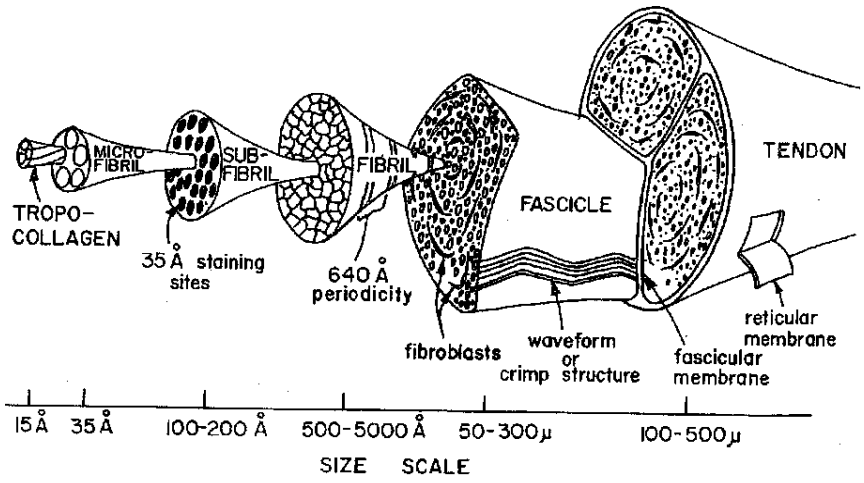


Figure 2-5.Hierarchical structure of tendon (from Kastelic et al., 1978).

Many consider the collagen fibril to be the basic force transmitting unit of the tendon (Patterson-Kane *et al*., 1997b; Magnusson and Kjaer, 2003; Kannas *et al*., 2010). In fact, it was recently suggested that fibrils within mature tendon are continuous structures, after a lack of fibril ends were observed (Provenzano and Vanderby, 2006). Tendon fibrils vary in diameter as a function of anatomical location, age, species and exercise history (Diamant *et al*., 1972; Parry *et al*., 1978a; Strocchi *et al*., 1996; Patterson-Kane *et al*., 1997a; Patterson-Kane *et al*., 1997b; Liao and Vesely, 2003; Edwards *et al*., 2005) and can be broadly separated into distinct groups depending on their size (small diameter fibrils = ~20 – 40 nm, large diameter fibrils = ~165 – 215 nm) rather than displaying a continuous spectrum of diameter sizes (Figure 2-6). Tendons may exhibit a unimodal or multimodal distribution of fibril diameters (Dyer and Enna, 1976; Parry *et al*., 1978a; Patterson-Kane *et al*., 1997b; Edwards *et al*., 2005). The population distribution of fibril diameter size within a tendon largely determines its strength and mechanical properties (Parry *et al*., 1978a). Tendons with a high proportion of large diameter fibrils are associated with a greater overall tendon stiffness and strength as a result of having a greater (1) number of mature inter-fibrillar covalent (strong) cross-links (that have been converted from immature inter-fibrillar cross-links) and (2) density of intra-fibrillar non-covalent (weak) cross-links. These bonds prevent the fibrils from sliding past each other when under stress (Parry *et al.*, 1978a; Bailey *et al.*, 1998; Curwin, 2007). Tendons with a high proportion of small diameter fibrils have fewer intra-fibrillar and inter-fibrillar (covalent) cross-links but a greater fibril surface area provided by a large number of small diameter fibrils increases the chances for inter-fibrillar non-covalent (weak) crosslinks to form between fibrils and components of the ECM. As such, they are associated with a greater compliance (Parry and Craig, 1988; Derwin and Soslowsky, 1999; Battaglia *et al*., 2003; Liao and Vesely, 2003).

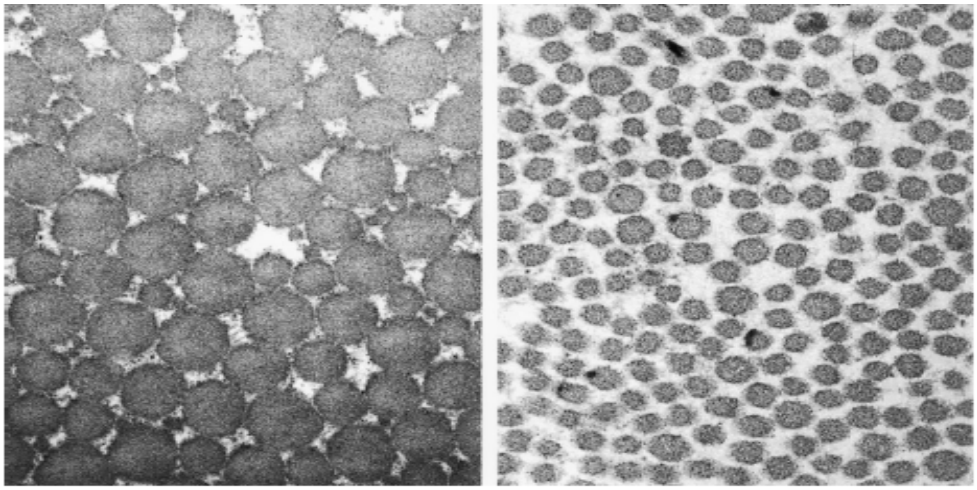


Figure 2-6. Micrograph of tendon cross-section showing the large (left) and small (right) diameter collagen fibrils. Images taken with a scanning electron microscope (Waugh, unpublished data)

2.2.2 Tendon Development

The fibril morphology within a tendon is important to its function. As such, it varies widely between tendons from different anatomical locations and between species (Parry *et al*., 1978a) in addition to changing with maturation and ageing. Young tendons are primarily formed from uniformly sized small collagen fibrils (Greenlee and Ross, 1967; Parry *et al*., 1978) which increase in diameter by laterally incorporating new microfibrils manufactured from ongoing fibrillogenesis (For review, see Zhang *et al.*, 2005). As a result of this process, the mean average fibril diameter (MAFD) shows an increase until maturation (Schwartz, 1957; Parry *et al*., 1978b; Scott *et al*., 1981; Cetta *et al*., 1982; Strocchi *et al*., 1991; Strocchi *et al*., 1996). The processes that regulate collagen synthesis are mechano-sensitive (For review, see Kjaer, 2004), therefore the increase in MAFD with age might suggest that tendon is adapting to increase its strength in response to greater muscular loading or change in functional demand with age. However, there is also evidence to suggest that

unloaded developing tendons have similar mechanical properties to loaded tendons and thus the processes underlying their development with maturation occurs irrespective of mechanical loading history (Eliasson *et al*., 2007). Tendon development must, therefore, involve other influencing factors (For review, see Zhang *et al.*, 2005).

2.2.3 Mechanical Properties

The mechanical properties of tendon are used to describe its behaviour under loading conditions and have been the subject of extensive investigation (for reviews: Alexander, 1981; Woo *et al*., 1986; Proske and Morgan, 1987; Zajac, 1989). Properties commonly reported include stress (σ; the magnitude of force [F] per unit area [A]; Equation 1) and strain (ε; a tendon's displacement [x] with respect to its resting length [L]; Equation 2). The ratio between these characteristics provides information regarding the magnitude of material displacement that can be expected for a given deforming load and is termed Young's modulus (E; Equations 3 and 4). Young's modulus is a measure of a material's stiffness relative to its dimensions. As a normalised measure, it provides a means of comparing material stiffness between different tendons and may allow inferences to be made regarding a tendon's underlying microstructure. For example, a greater fibril diameter or density, improved collagen arrangement or greater fibrillar crosslinking, all of which are associated with a stiffer tendon (Parry *et al*., 1978a; 1978; Bailey *et al*., 1998), may be suggested as factors contributing to a tendon's greater Young's modulus. The most important property concerning force transmission is tendon stiffness. The mechanical stiffness of the tendon can be described as a tendon's resistance to elongation when a muscular force is applied, and is measured as a tendon's displacement attributable to a change in force (Equation 5). It is governed by both its material stiffness (i.e. Young's

modulus) and dimensional characteristics (Proske and Morgan, 1987). According to Hookean law (that states the deformation of an elastic body is proportional to its deforming load) longer tendons are more compliant for a given Young's modulus, because stiffness decreases as more material is arranged in series ($k = 1/[k_1 + k_2 + k_3…]$). Conversely, tendons with a greater CSA are stiffer because stiffness increases as more material is arranged in parallel ($k = k_1 + k_2 + k_3…$).

$$\text{Stress } (\varepsilon) = \frac{\text{force } (F)}{\text{area } (A)} \qquad \text{Equation 1}$$

$$\text{Strain } (\varepsilon) = \frac{\text{length } (L)}{\text{displacement } (x)} \qquad \text{Equation 2}$$

$$\text{Young's modulus } (E) = \frac{\text{force } (F) \times \text{length } (l)}{\text{displacement } (x) \times \text{area } (A)} \qquad \text{Equation 3}$$

$$\text{Young's modulus } (E) = \frac{\text{Stress } (\sigma)}{\text{Strain } (\varepsilon)} \qquad \text{Equation 4}$$

$$\text{Stiffness } (k) \frac{\text{force } (F)}{\text{displacement } (x)} \qquad \text{Equation 5}$$

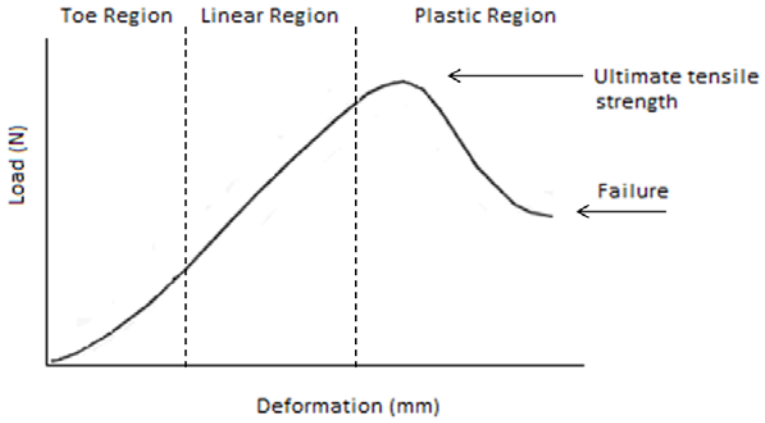
2.2.4 Viscoelastic Properties

Like many biological materials, collagenous tissues display non-linear elastic properties. These characteristics allow the tissue to elongate when subjected to a deforming load and return, unchanged, to its original resting length after the load is removed. The deformation

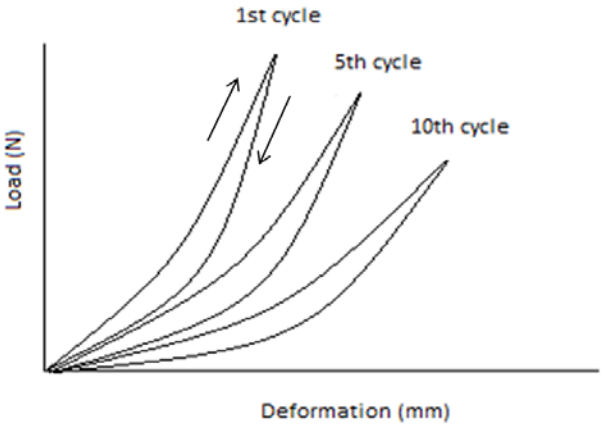
exhibited by a tissue is dependent upon its stiffness and the load (force) imposed on it. Tendons show nonlinear elasticity when placed under load (Figure 2-7a). More specifically, their stiffness increases as they are stretched (Gratz, 1931; Rigby *et al*., 1959). The reason for a lower stiffness at relatively low stresses is due to the un-crimping of the wavy collagen fibrils causing considerable tendon extension (Rigby *et al*., 1959; Abrahams, 1967; Diamant *et al*., 1972; Woo, 1982). Once the collagen fibrils have been straightened, the load-deformation relationship becomes relatively linear, representing the physical stretching of the collagen fibrils (Diamant *et al*., 1972). Repeated loading within this force region poses no threat to permanent deformation, allowing collagen fibrils to maintaining a reproducible load-deformation relationship and to return to their original length once a deforming load has been removed (Rigby *et al*., 1959; Abrahams, 1967). Hence, this region is termed the elastic region. It is within this region that measures of tendon stiffness are typically made. Beyond this region, additional loading causes individual fibrils to fail and the tendon can be considered permanently lengthened, with a reduction in stiffness Abrahams, 1967 (Rigby *et al*., 1959). Microdamage accumulating from loading in this force region also reduces the functional CSA of the tendon, jeopardising the integrity of the tendon and predisposing it to rupture. The ultimate tensile strength (UTS) represents the maximum stress that a tendon can withstand before failing and has been positively correlated with both its MAFD (Parry *et al*; 1978) and CSA(Woo, 1982). The UTS of mature tendons are reportedly as much as 100 MPa (Elliott, 1965; Butler *et al*., 1978; Bennett *et al*., 1986).

In addition to its elastic properties, tendon exhibits viscous characteristics. Such properties mean that the tendon's mechanical properties are dependent on its loading history, or have time-dependent mechanical behaviour (Butler *et al*., 1978; Taylor *et al*., 1990; Fung, 1993;

Magnusson *et al*., 1995). If a load is applied to a tendon repeatedly, an initially stiff tendon can become more compliant (Graf *et al*., 1994)until a steady state of stiffness is reached after a short period of cyclic loading, seen as a right-ward shift of the stress-strain curve (Figure 2-7b). The time-dependent effects of viscoelasticity should therefore be considered when assessing a tendon's mechanical properties to minimise the variation in results (Taylor *et al*., 1990; Fung, 1993; Magnusson *et al*., 2001). The time-dependent effect is relatively short lived with no apparent effect on maximal load (Woo, 1982; Graf *et al*., 1994).



a)



b)

Figure 2-7. Representative load-deformation curve showing a) non-linear tendon elasticity and b) the history-dependent effects of repetitive loading on tendon mechanical properties.

2.2.4.1 Hill's Muscle Model

Tendons lie in series with muscle. Collectively, with other structures that aid the production and transfer of forces to the skeleton, they are termed the muscle-tendon unit (MTU). The MTU is commonly modelled as a three-component structure comprising of a contractile component (CC), representing the muscle's myofilament interaction, in series with an elastic component (SEC). The SEC is mainly comprised of the tendon (passive SEC element) and the tendon aponeuroses and myofilament cross-bridges (active SEC element). These two components are situated in parallel with a second elastic component (PEC), made up of the connective tissues that surround the muscles fibres and fascicles, the sarcolemma and cytoskeletal elements (Enoka, 1994). Thus, according to this model, tendon properties will substantially affect the magnitude and rate of muscular force transferred to the skeleton.

The majority of human movements require muscles to operate at a shortened length for force production. At these lengths, the PEC is slack and therefore its contribution to a muscle's stiffness is often ignored (Jewell and Wilkie, 1958; Arnet, 1967). When the muscle is at rest, the PEC represents the muscle's stiffness (Latash and Zatiorsky, 1993). When the muscle is active, it stiffens considerably because of the strong cross-bridge bonds between the myofilaments. Despite the SEC being stiff enough to resistance some stretch, when a muscle is active, its stiffness can be greater than that of the SEC, causing the SEC (as the more compliant of the two structures) to stretch. This stretching of the elastic components has implications for movement efficiency, discussed below.

2.2.4.2 Importance of Tendon Mechanical Properties on Force Production

Tendon mechanical properties substantially influence human movement performance. In activities where MTUs are stretched rapidly before shortening, such as running or jumping, stiffer tendons may be associated with decreases or increases in movement speed or economy(Ker *et al*., 1987; Ettema, 1996; Lichtwark and Wilson, 2007). The optimum stiffness of the tendon will depend greatly on the force-time characteristics of the movement to be performed (Blazevich, 2011). However, tendon properties also impact on simple, isometric or concentric-only muscle actions. Compliant tendons stretch more for a given muscle force, thus the time required to stretch the tendon to a point where force can be effectively transferred to bone will be longer. This will impair rapid force production. Moreover, the greater time taken for force transmission will warrant a longer muscle activation period, which will likely increase the metabolic cost of performing a task (Hill, 1970; Muraoka *et al.*, 2004b).

Essentially, the optimum stiffness of a tendon varies according to the force production requirements of the movement in question, and since effective stretch of a tendon during loading requires muscle forces to be applied with appropriate magnitude and timing, the tendon's mechanical properties will also partly dictate the optimal muscle activation pattern (Lambertz, 2003).

The mechanical properties of the muscle-tendon complex have been shown to influence force production characteristics as well as contributing to movement efficiency and performance in adults. Hill (1950) first postulated that the presence of elastic elements in series with the muscle were the cause of delay before slow production of tension whilst

Wilkie (1950) demonstrated the importance of the stiffness of an in-series elastic structure on the rate of moment development (RMD; Figure 2-8). Since this pioneering work, a clearer picture of the role of the tendon on force production has been constructed. Recently, Muraoka *et al.* (2004) found a significant negative relationship between tendon slack length (i.e. tendon tension) and EMD in adults whilst paired changes in EMD and stiffness of the elastic MTU components have been found after isometric (Kubo *et al*., 2001a), endurance and plyometric training in adults (Grosset *et al*., 2009). In addition, several authors postulated that increases in RFD observed after a training intervention were the result of increases in stiffness (Reeves *et al*., 2003). Bojsen-Møller *et al.* (2005) confirmed this relationship by demonstrating a significant correlation between stiffness of the vastus lateralis (VL) tendon and contractile RFD *in vivo*. Tendon stiffness has therefore been recognised as a key influencing factor in determining EMD and RFD.

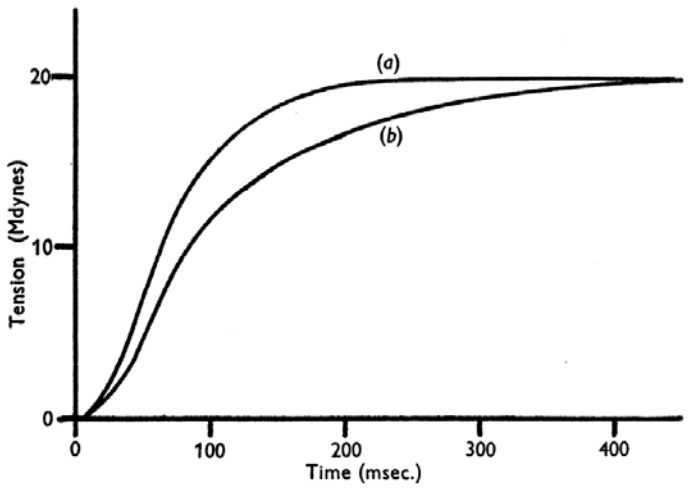


Figure 2-8. Representation of the rate of rise of isometric tension experience by a resistance strain gauge when being pulled by a) the arm alone and b) the arm with an inert compliant structure inserted between the arm and the resistive load (Wilkie, 1950).

As a consequence of this association, it may be suggested that tendon stiffness directly influences movement performance, as delay in muscle tension development may impact on movement regulation and coordination (Rack and Ross, 1984; Proske and Morgan, 1987; Mora *et al.*, 2003; Pijnappels *et al.*, 2005; Pijnappels *et al.*, 2008; Granacher *et al.*, 2010). It has been previously shown that an increased tendon stiffness is associated with greater joint power in adults (Bojsen-Møller *et al.*, 2005; Arampatzis *et al.*, 2007). This relationship may result of the greater recoiling force associated with stretching a stiffer tendon (see Wilson 1994) or from the more efficient transfer of muscular force. Bojsen-Møller *et al.* (2005) found that stiffness of the vastus lateralis (VL) tendon was positively correlated with maximum jump height, power, force and velocity (at peak power) obtained from squat and countermovement jumps in adults, and a similar relationship was shown for the Achilles tendon by Kubo *et al.* (2007). In addition, Wu *et al.* (2010) found that increases in Achilles tendon stiffness after 8 weeks of plyometric training in young adults were positively correlated with counter-movement jump height. Although little is known regarding the relationship between tendon stiffness and joint power production in children, age-related increases in tendon stiffness have been suggested as a factor underlying increases in both leg stiffness and jump performance with age (Wang *et al.*, 2004; Korff *et al.*, 2009a).Based on the relationships pointed out previously in regards to tendon stiffness, force production and movement performance in adults, it is possible that tendon stiffness impacts on movement performance in children This would have implications for movement efficiency.

2.2.5 Methods of Measuring Muscle-Tendon Stiffness

Stiffness can be described from the level of a single tendon fibril, to a multi-joint system encompassing the stiffness of all of its components. The stiffness of the limbs determines a

person's movement outcomes and is a reflection of the combined stiffness of the tendons, ligaments, cartilage, and muscle that they are assembled from (Latash and Zatsiorsky, 1993). Understanding the individual contributions of these components is therefore valuable in assessing or predicting movement outcomes. In order to make inferences about previous findings, it is essential that we have some knowledge of the different methodologies used in measuring different aspects of stiffness (e.g. series elastic component stiffness, musculo-tendinous or musculo-articular stiffness). In addition to *in vitro* methodologies, there are several *in vivo* techniques that are commonly used to measure tissue-specific stiffness.

2.2.5.1 Estimating the Mechanical Properties of Soft Tissues from Mechanical Testing

Prior to the 1990s, the elastic properties of biological tissues could only be estimated by means of mechanical testing on excised tissue (Rigby *et al*., 1959; Abrahams, 1967; Butler *et al*., 1978; Ker, 1981; Bennett *et al*., 1986), or on anaesthetised animals (Morgan *et al*., 1978; Rack and Westbury, 1984; Baratta and Solomonow, 1991), thus most of our knowledge of the mechanical properties of tendon comes from isolated material testing.

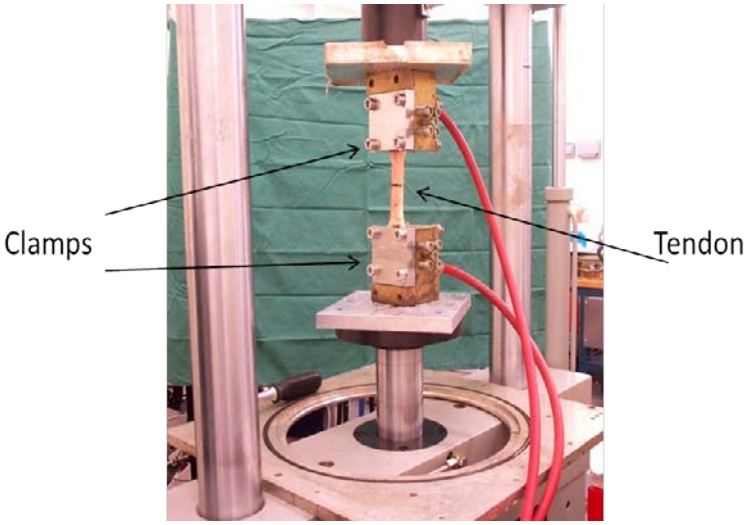


Figure 2-9. Example materials testing machine for compression and tensile testing.

In vitro or mechanical testing involves mounting the tissue of interest securely into the clamps of a materials testing machine (Figure 2-9). The tissue is subjected to mechanical loading and the tension and deformation of the specimen measured. In order that the *in vitro* testing best replicates the *in vivo* physiological conditions, whole soft tissue units (i.e. soft tissue and bony attachments) undergo testing (Hukins *et al*., 1990; Leung *et al*, 2008). Limitations to this method include difficulties in securely fixing biological soft tissue without (1) the tissue slipping and (2) damaging the tissue in contact with the clamp, both of which influence the accurate determination of mechanical properties. In addition, different storage methods and handling of soft tissue prior to testing may affect its mechanical properties (Matthews and Ellis, 1968; Smeathers and Joanes, 1988).

2.2.5.2 Estimating Tendon-Specific Stiffness using Ultrasonography

Advances in imaging technology now allow accurate, non-invasive methods of assessing muscle and tendon properties *in vivo* (Henriksson-Larsen *et al*., 1992; Fukashiro *et al*., 1995; Maganaris and Paul, 1999). For estimating tendon stiffness using ultrasonography, the change in tendon length attributable to an applied muscular load (Equation 1) is usually measured by tracking the displacement of the muscle-tendon junction (MTJ) during a voluntary [typically ramped] maximal isometric muscle contraction. The corresponding tendon force is then calculated as the ratio between joint moment, obtained simultaneously (typically via dynamometry), and a tendon's moment arm (An *et al*., 1983; Spoor and van Leeuwen, 1992; Ito *et al*., 2000; Maganaris *et al*., 2000). Tendon stiffness is calculated as the gradient of the slope produced from plotting the relationship between tendon force and MTJ displacement (Fukashiro *et al.*, 1995; Maganaris and Paul, 1999; Figure 12).

Despite several methodological considerations that must be taken into account when using this method (Magnusson *et al.*, 2001; Reeves *et al.*, 2003a; Arampatzis *et al.*, 2005; Maganaris, 2005), ultrasound-based measurement of tendon stiffness *in vivo* in humans has greatly enhanced our understanding of tendon stiffness and its adaptation with growth, aging, loading and immobilisation (Kubo *et al.*, 2001a; Kubo *et al.*, 2001b; Reeves *et al.*, 2003a; Maganaris *et al.*, 2006; Seynnes *et al.*, 2008; O'Brien *et al.*, 2010).

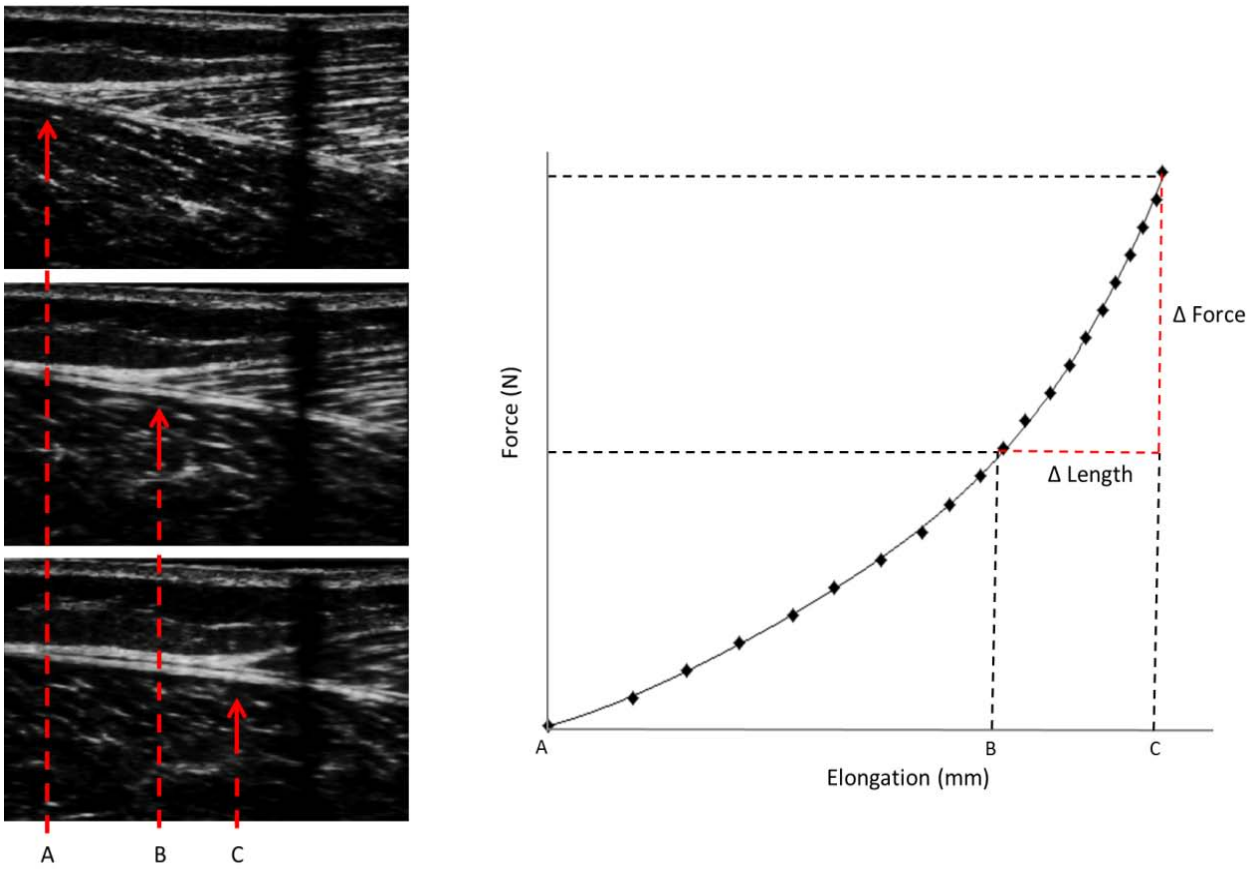


Figure 2-10. Ultrasonographic images visualising displacement of the muscle-tendon junction (MTJ) during a ramped isometric muscle contraction at rest (A), mid contraction (B) and maximal force (C). MTJ displacement, indicated by the red arrow, is plotted against the corresponding force to produce a force-elongation graph. Tendon stiffness is measured as the gradient of the linear region of the graph (between points B and C here), or the ratio between the Δ force and Δ length.

2.2.5.3 Estimating Muscle-Tendinous Stiffness using the Quick Release Method

The quick release (QR) method characterises the stiffness of the SEC, achieved by releasing the moveable parts of an ergometer at a higher angular velocity than the maximal shortening velocity of the muscle in question (Pérot *et al*., 1999; Lambertz *et al*., 2001). The joint recoil velocity in the period immediately after the release of the ergometer, before reflex changes in muscle activity are initiated, is related to the stiffness of the SEC of the muscle-tendon unit (Angel *et al*., 1965; Goubel and Pertuzon, 1973). Angular muscle-tendon stiffness can then be estimated from the limb's inertia, angular acceleration and angular displacement attained in the period when the SEC is supposed to recoil. Based on the concept that, due to covariance, there is a linear relationship between the activation levels of synergistic muscles (and that this relationship is independent of the velocity and inertia of the movement), angular stiffness can be transformed into linear stiffness (Bouisset, 1973). Stiffness index is then calculated from the slope of the relationship between musculo-tendinous stiffness and joint moment or EMG amplitude (Cornu and Goubel, 2001; Lambertz *et al*., 2003).

2.2.5.4 Estimating Musculo-Articular (Muscle-Joint) Stiffness from Sinusoidal Perturbation

Based on the concept of mechanical impedance (i.e. how much a body resists motion when a force is applied), musculo-articular stiffness can be quantified from the limb-system's response to sinusoidal perturbations (Cannon and Zahalak, 1982; Winters and Stark, 1988). As mechanical impedance varies with angular velocity, or frequency, of an applied force, this implies that impedance of the joint also changes with the frequency of an applied force. The passive elastic properties of the joint can be quantified by plotting the ratio between the joint displacement and the joint moment developed in resisting motion, against the

imposed oscillation frequency to a establish a frequency-response relationship (i.e. bode diagrams; Tognella *et al*., 1997). The resonant frequency of the system is identified as the frequency displaying the least phase lag between joint position and moment generated (indicating the lowest impedance) as less force is required to move a body at this frequency (Cannon and Zahalak, 1982). As the resonant frequency is directly related to stiffness, stiffness can be estimated from the frequency-response relationship of the system (Kearney and Hunter, 1982; Cornu and Goubel, 2001).

Musculo-articular stiffness can also be estimated by externally perturbing a limb weighted with varying loads and recording the oscillation response due to its own elasticity. This technique is based on the assumption that the muscle-tendon unit (MTU) can be modelled on a damped spring system (Shorten, 1987) and thus stiffness is calculated directly from the oscillation frequency. This technique is useful in allowing the stiffness of a system to be estimated under specific loading conditions.

2.2.5.5 Estimating the Active and Passive Components of the SEC from the Alpha Method

The alpha method was developed to differentiate between the stiffness of the tendinous components and stiffness of the muscle-tendon complex cross-bridge attachments within the SEC of the MTU, based on the assumption that these components can be modelled as two springs in series (Morgan, 1977). This method hypothesises that tendon stiffness is constant (i.e. linear) at forces exceeding 20% maximal isometric muscle force. In addition, the force-extension relationship of a single cross-bridge is assumed to be linear (Ford *et al*., 1977), and that the total muscle force is directly related to the number of formed cross-bridges. However, in demonstrating that tendon stiffness increases non-linearly after a

threshold of muscular force, Ettema and Huijing (1994) suggested instead that the alpha method distinguished between the force dependent and force independent components (i.e. active and passive) of SEC stiffness. Despite its adaptation for use *in vivo*, the alpha method has not been widely used to determine active and passive components of SEC stiffness (Cook and McDonagh, 1995; Svantesson *et al*., 2000; Foure *et al*., 2010).

2.2.6 Muscle-Tendon Stiffness in Children

There is a body of evidence demonstrating a positive relationship between musculo-tendinous stiffness and the performance of movements utilising a stretch shorten cycles (SSC) such as running and jumping in adults (Belli and Bosco, 1992; Wilson *et al*., 1991; 1992; Kyrolainen and Komi 1995, Kubo *et al*., 1999). Due to its influence on movement performance in adults, it is of substantial interest for researchers to document the development of musculo-tendinous stiffness in children. For example, it has been suggested previously that differences in stiffness between children and adults are likely to contribute to the significantly longer EMD and lower RFD found in the elbow flexor (Asai and Aoki, 1996) and plantarflexor muscle groups (Grosset *et al*., 2005) of children compared to adults. These relationships are important to establish in children in future research in order to further our understanding of movement performance differences and efficiency between children and adults.

2.2.6.1 Development of Musculo-Tendinous Stiffness during Childhood

Cornu and Goubel (2001) used the QR method to quantify musculo-tendinous (MT) stiffness of the elbow flexors in children aged 9 and 15 years and found stiffness to be similar to values published for adults. This suggests that elastic tissue stiffness increases in parallel

with the gains in force production capacity associated with upper body growth during development. Interestingly, the relationship between peak strength and tendon stiffness has been identified in the lower body in adults (Muraoka *et al.*, 2004b) but this relationship has not been examined in children. Conversely, Lambertz *et al.* (2003) identified increases in plantarflexor MT stiffness with age in typically developing prepubertal children (aged 7 – 10 years) using the QR method. To address the contrast in results found for development of MT stiffness in different limbs, the author suggests that the upper and lower limbs might be influenced by different neuromuscular activation capacities in the muscle groups concerned.

2.2.6.2 Development of Tendon-Specific Stiffness during Childhood

Musculo-tendinous (MT) stiffness characterises both the active (Huxley and Simmons, 1971) and passive (Shorten, 1987) components of the SEC. In many cases, the tendon (passive SEC) comprises a substantial component of MT stiffness and in addition, has previously demonstrated a greater stiffness than its aponeuroses (Ettema and Huijing, 1989; Lieber *et al*., 1991). Because of this, it is common to regard the tendon as a separate entity, thus quantify the mechanical properties of the tendon separately is of interest.

Animal-based studies show that tendon stiffness increases with both age (Elliott, 1965; Woo *et al*., 1980; Danielsen and Andreassen, 1988; Shadwick, 1990; Nakagawa *et al*., 1996) and body mass (Pollock and Shadwick, 1994) during growth. The limited data on human tendon development is consistent with these previous findings in mammals. Kubo *et al.* (2001) was the first to document changes in tendon-specific stiffness as a function of age during development in humans using the ultrasound method. They compared stiffness of the VL tendon in young (~11 years) and old (~15 years) boys to adults and found stiffness increased

significantly as a function of age. Following these findings, O'Brien *et al.* (2010) compared the stiffness and Young's modulus of boys and girls (~9 years) to those of adult men and women. Stiffness and Young's modulus was calculated from a force region common to all participants, as a method of normalising for individuals producing markedly different forces. Both stiffness and Young's modulus were found to be greater in adults compared to children but were not significantly different between boys and girls or women and men. Thus, it appears that, at least for the patellar tendon, stiffness increases with age from late childhood through to adulthood.

There are numerous gaps in our understanding of normally developing young tendons. First, little is known in regards to the stimulus for tendon growth and microadaptation. The influence of the tendon loading from body mass(Pollock and Shadwick, 1994)or muscle strength(Muraoka *et al.*, 2005), rather than ageing *per se*, might be a more important factor underpinning the age-related increase in tendon stiffness. This is important as the identification of 'normally developing' tendons might require this knowledge. Second, it is not yet known whether other tendons (including those important for locomotion, such as the Achilles) develop similarly to the patellar tendon (O'Brien et al., 2010), so a substantial research effort is required to document the development of other tendons. Third, there is a lack of data detailing the development of tendons in children younger than 9 years of age, thus the temporal response from early childhood through to adulthood is incomplete. Given these shortcomings in our knowledge, more research is required to fully understand the normal development of tendon stiffness in childhood.

2.2.6.3 Development of Musculo-Articular Stiffness during Childhood

Another type of stiffness is that exhibited by joints. Active joint stiffness is commonly referred to as musculo-articular stiffness as it depends on levels of muscle activation, whereas passive joint stiffness is measured when all associated muscles are relaxed and can thus be considered to represent the combined stiffness of all of the structures spanning it. Knowledge of musculo-articular/joint stiffness is of functional importance as it helps in determining resistance to external perturbation (Akeson *et al*., 1987) as well as a possible role in efficient movement control movement (Hasan, 1986) via active stabilisation of the limb-joint system using co-activating agonist- antagonist muscle pairs (Ghez *et al*., 1983).

Lebiedowska and Fisk (1999) assessed knee joint stiffness in 87 children ranging between 5 and 18 years of age. Using the free oscillation technique, joint stiffness was found to increase as a function of body stature, and thus age. The joint stiffness of the oldest children were comparable to previously published values for adults (Mansour and Audu, 1986). Normalising stiffness to body stature (Lebiedowska *et al*., 1996) removed the relationship with age, thus joint stiffness was considered to be invariant with growth. Cornu and Goubel (2001) used sinusoidal perturbations to find musculo-articular stiffness of the elbow flexors was also lower in children than adults, but did not find like differences in MT stiffness. This finding suggests an increase in the stiffness of structures that cross the joint with development that were not examined, whilst also demonstrating the complexity of joint stiffness. Increases in passive joint stiffness with age may be related to an improved force producing ability.

Results from the literature generally demonstrate that stiffness of the elastic structures within the MTU increase with age during childhood. It has been suggested that mechanical loading from body mass and increased muscle force generating capacity with maturation may provide a progressive, dual stimulus for tendon stiffness adaptation with age (O'Brien *et al*., 2010), as have been identified for animals and adults, respectively (Pollock and Shadwick, 1994; Muraoka *et al*., 2005). Development of joint stiffness may also be associated with simultaneous development of elastic tissue stiffness but further research is required to quantify this relationship.

2.3 Responsiveness of Tendon to Adaptation from Mechanical Loading

2.3.1 Adaptation of the Mature Tendon to Chronic Heavy Loading

It is widely accepted that chronic loading or unloading results in an adaptation of tendon mechanical properties (Review: Kannus *et al*., 1997). Such adaptations have been shown to include an increase in stiffness (Woo *et al.*, 1980; Narici *et al.*, 1996; Kubo *et al.*, 2001a; Reeves *et al.*, 2003a; Wu *et al.*, 2010), ultimate tensile strength (Woo *et al*., 1980; Vilarta and Vidal Bde, 1989) and CSA (Woo *et al*., 1980; Michna and Hartmann, 1989; Birch *et al*., 1999; Rosager *et al*., 2002; Kongsgaard *et al*., 2007), and decrease in strain (Urlando and Hawkins, 2007). In humans, Kongsgaard *et al.* (2007) found a ~15% increase in patellar tendon stiffness after 12 weeks of heavy knee extension exercise, the findings of which were replicated by Seynnes *et al.* (2009), who found a 24% increase in patellar tendon stiffness after a 9-week resistance training intervention. In both studies, the increase in stiffness was attributed to an increase in patellar tendon CSA, which indicates that changes in the tendon dimensions rather than microstructural adaptations underpinned the change in stiffness. Kubo *et al.* (2010) found a ~30% increase in patellar tendon stiffness in young adults after an intensive resistance training program performed 5 days per week. Interestingly, none of these studies using young untrained adult subjects reported increases in patellar tendon stiffness of the magnitude reported in an elderly population. Reeves *et al*.(2003a) found an increase of ~65% in patellar tendon stiffness after 14 weeks of thrice-weekly resistance training in older (74.3 ± 3.5 years) men and women. With such variation in results, it is clear that a variety of factors must determine the magnitude of tendon adaptation to a loading regime, possibly including training intensity, duration and volume, the degree of tendon strain, age of the individual, tendon function and anatomical location (Kongsgaard *et al*., 2007; Arampatzis *et al*., 2010; Kubo *et al*., 2010).

2.3.2 Adaptation of the Mature Tendon to Chronic Habitual Loading

In addition to strength training, the chronic tendon loading that occurs as a result of habitual movement performance has been shown to be sufficient for inducing adaptation of tendon mechanical properties. The effects of chronic loading were first documented by Conkrite (1936), who noticed that tendons in the dominant hand were thicker than those in the non-dominant hand. Increases in tendon stiffness have been reported by a number of authors in various animals in response to running training (rabbit, Viidik, 1967; swine, Woo, 1982; guinea fowl, Buchanan and Marsh, 2001), the changes attributed to qualitative rather than quantitative changes in collagen arrangement. Conversely, a number of studies have not found changes in tendon mechanical properties after running training (Swine, Woo *et al.*, 1981; Rat, Huang *et al.*, 2004; Legerlotz *et al.*, 2007). In humans, adaptations to tendon mechanical properties in response to chronic habitual loading have often been examined retrospectively in individuals who have undertaken long-term running training, with mixed results. Kubo *et al.* (2000) found stiffness of the VL tendon was ~20% greater in runners than non-runners whilst Rosager *et al*.(2002) reported a greater Achilles tendon CSA in men who habitually ran than those who did not, and without differences in tendon stiffness between groups. Westh *et al.* (2008) also failed to find differences in patellar tendon stiffness or CSA between habitual women distance runners and non-runners. It appears that years of habitual training may be required for significant tendon adaptation to habitual loading (Hansen *et al*., 2003). This may be due to tendons of the lower extremity having adapted sufficiently to the significant loading experienced through daily activities such as walking and stair climbing or that the forces experienced are not great enough to stimulate tendon adaptation.

The positive relationship found between peak muscular force capacity and tendon stiffness (Scott and Loeb, 1995; Muraoka *et al*., 2005) has been hypothesised to reflect a mechanism by which substantial tendon elongation, resulting from an increase in muscular loading, prompts tendon adaptation. An increase in tendon stiffness in response to greater muscular loading is probably required to avoid excessive tendon strain and subsequent rupture (Muraoka *et al*., 2005) whilst maintaining a muscle fascicle length optimum for efficient force development and transfer (Zajac, 1989; Reeves *et al*., 2004).

2.3.3 Adaptation of the Developing Tendon to Loading

There have been surprisingly few studies attempting to document the effect of chronic, exercise-induced loading on a developing tendon's mechanical properties, despite the potential influence an increases in tendon stiffness on the rate of force development and transfer during movement. Curwin *et al.* (1988) examined the effects of an 8-week treadmill-based running intervention on tendon properties in 3-week old roosters. The authors found a 46% increase in the rate of collagen deposition and a decrease in levels of pyridinoline, an indictator of the maturational status of collagenous tissue, when compared to non-running controls. Together, these results suggest an increase in collagen turnover which is indicative of an accumulation of younger collagen and may result in reduced tendon stiffness. Birch et al.(1999) examined the effects of 5 months of high-intensity treadmill running on the properties of the superficial digital flexor (SDFT) and common digital extensor tendons (CDET) in the horse, as weight-bearing and positional tendons, respectively. Interestingly, the CDET showed significant hypertrophy post-training whereas the SDFT, as the equine equivalent of the human Achilles tendon, did not differ to the control group. Although an element of tendon hypertrophy could be a result of normal

growth with development, Kasashima *et al.* (2002) also found tendon hypertrophy of the SDFT in the developing equine athlete in response to 13 months of treadmill running. Given that tendon stiffness appears to substantially influence force production characteristics, it might be hypothesised that an increase in tendon stiffness resulting from a heavy strength training programme should affect electromechanical delay and rates of force development favourably. Despite this, the receptiveness of the developing tendon to adaptation from a training intervention has not previously been examined in children.

2.4 Summary

A complex network of biological mechanisms underpin the age-related increases in force production capacity in children. A number of these factors also change dramatically during childhood development. Muscle growth and increases in muscle-tendon moment arms have substantial implications for strength gains during childhood, and thus for movement performance. In addition, maturation of the nervous system causes significant improvements in the recruitment of muscle for force production, and hence increases strength and rapid force production. The time course and extent of change of these factors are relatively well documented. The mechanical properties of the tendon, in particular, its stiffness, have been shown to be different in children than adults, although the cross-sectional development of stiffness (together with the factors that prompt stiffness development) during childhood has not been previously investigated and may be different for tendons with different functions or locations. The influence of tendon stiffness on force production during childhood is also not known, despite the importance of tendon stiffness on rapid force development being highlighted in adults. Understanding the role of tendon stiffness in force production may provide valuable insight the mechanisms underpinning

age-related differences in movement performance. Lastly, the plasticity of the developing tendon to different loading regimes is largely unknown. Determining the adaptive potential of the developing tendon's mechanical properties to chronic loading, unloading or different levels of cyclic strain may have important implications movement performance.

CHAPTER 3: Can Achilles Tendon Moment Arm be predicted from Anthropometric Measures in Prepubescent Children?

3.0 Abstract

Muscle-tendon moment arm length is an essential variable for accurately calculating muscle forces from joint moments, however their accurate measurement requires specialist knowledge and expensive resources. Recent research has shown that the patellar tendon moment arm length is related to leg anthropometry in children. Here, we asked whether the Achilles tendon moment arm (MA_{AT}) could be accurately predicted in prepubescent children from surface anthropometry. Age, standing height, mass, foot length, inter-malleolar ankle width, antero-posterior ankle depth, tibial length, lower leg circumference, and distances from the calcaneus to the distal head of the 1st metatarsal and medial malleolus were determined in 49 prepubescent children. MA_{AT} was calculated by differentiating tendon excursion, observed using ultrasonography, with respect to ankle angle change over an angular displacement of 20°. Pearson's product-moment correlation coefficients and coefficients of determination were calculated and a backwards stepwise regression analysis was performed to identify predictors of $MA_{AT.}$ When all variables were included, the regression analysis predicted 49% of MA_{AT} variability. Foot length and the distance between calcaneus and 1st metatarsal were the only significant predictors, accounting for 49% of MA_{AT} variability (p<0.05). The regression model predicted moment arm values with 3.8 ± 4.4 mm absolute error, which would result in significant error (mean = 14.5%) when estimating muscle forces from joint moments. It was concluded that MA_{AT} cannot be accurately predicted from anthropometric measures in children, as they account for less than half the variation in moment arm length.

A paper relating to this chapter is now in print and can be found at:
http://dx.doi.org/10.1016/j.jbiomech.2011.03.023

3.1 Introduction

Muscular strength, which can be defined as the maximum moment developed by a muscle group about a joint during a voluntary contraction, increases during childhood (Blimkie, 2001). The mechanisms underpinning the development of age-related gains in muscular strength include neuro-maturation (Savelsburgh, 2003), increases in muscle size (Kanehisa *et al*., 1995; Morse *et al*., 2008), changes in muscle architecture (Binzoni *et al*., 2001) and increases in moment arm length (Wood *et al*., 2006; Morse *et al*., 2008; O'Brien *et al*., 2009).

The moment arm of a muscle-tendon unit is defined as the perpendicular distance between the line of muscle/tendon force action and the rotational centre of the corresponding joint (Spoor and van Leeuwen, 1992). Its measurement is vital for quantifying changes in muscle force production during growth (Wood *et al*., 2006), where the changing moment arm impacts on joint moment magnitude. In addition, moment arm is both an essential parameter in many musculoskeletal models (Delp *et al*., 1990) and is also used in the calculation of other important parameters such as tendon stiffness and Young's modulus (O'Brien *et al*., 2010). Muscle moment arms can be obtained using imaging techniques such as magnetic resonance imaging (Spoor and van Leeuwen, 1992; Maganaris *et al.*, 1998; Reeves *et al.*, 2003b; Sheehan, 2007), X-ray videofluoroscopy (Baltzopoulos, 1995; Kellis and Baltzopoulos, 1999) and ultrasonography (Ito *et al.*, 2000; Fath *et al.*, 2010). Given that such techniques require specialist knowledge and resources, it is not always feasible for developmental researchers to use them to obtain accurate moment arm lengths. As a result, previous authors have estimated moment arms in children using allometric scaling (Kanehisa *et al.*, 1995; Kubo *et al.*, 2001b). This practice is based on the assumption that

moment arm length scales proportionally with stature or the geometry of the corresponding segments, and that these relationships are constant between children and adults. However, this assumption has not been explicitly confirmed.

In adults, the strength of the relationship between moment arm length and body anthropometrics depends on the muscle or tendon of interest. For example, strong relationships between moment arm length and surface anthropometry have been demonstrated for elbow (Murray *et al*., 2002) and trunk flexors and extensors (Reid *et al*., 1987; Moga *et al*., 1993; Jorgensen *et al*., 2003; Seo *et al*., 2003). However, such a relationship could not be shown for the knee extensors (Tsaopoulos *et al*., 2007; O'Brien *et al*., 2009). Despite this lack of relationship in adults, it has been demonstrated that the knee extensor moment arm in children can be accurately predicted from the segment lengths of the lower limb (O'Brien *et al*., 2009). This finding raises the question as to whether moment arm lengths of other functionally important muscle groups can be accurately predicted in children from anthropometric measures.

The plantarflexor muscle group plays a major functional role in common motor tasks such as quiet standing and balance performance (Shambes, 1976; Roncesvalles *et al*., 2001) as well as dynamic movements including walking, running and jumping (Cupp *et al*., 1999; Fukunaga *et al*., 2001; Lichtwark *et al*., 2007). The ability to predict the moment arm of this muscle group in children would allow researchers to estimate muscle forces from joint moments and develop accurate paediatric musculoskeletal models without the need for specialised equipment. In addition, simple moment arm determination is of substantial practical interest and could be helpful for further understanding the mechanisms underpinning age-

related increases in plantarflexor strength (Denis and Korff, 2009) and the developmental differences in plantarflexor moment during locomotor activities such as bicycling (Korff *et al.*, 2009b). Therefore the purpose of the present study was to determine whether Achilles tendon moment arm can be accurately predicted from surface anthropometric characteristics in prepubescent children.

3.2 Method

3.2.1 Ethics and Participant Information

Forty-nine prepubescent children aged 5 to 12 years (mean = 8.0 ± 1.7 years) volunteered to participate in this study (26 boys, 23 girls). Prepubertal status was determined for children over 8 years of age according to Mirwald *et al.* (2002). The average age from peak height velocity, an indicator for maturational offset (Malina and Bouchard, 1991), in this age group was 3.92 years (± 0.53 years). All children were free from musculoskeletal and orthopaedic disorders and were not involved in competitive sports. Ethical approval was granted by the Human Research Ethics Committee of the School of Sport and Education, Brunel University. Written parental consent was given by a guardian. Children provided written assent to participate in the study. All procedures were explained to the participants in a child-appropriate manner, and they were made aware of their right to withdraw from the study at any time without penalty.

3.2.2 Anthropometric Measurements

The surface anthropometric variables used to predict Achilles tendon moment arm were (1) standing height, (2) body mass, (3) foot length, (4) inter-malleolar ankle width, (5) antero-posterior ankle depth, (6) tibial length, (7) lower leg circumference (largest calf measure), (8) distance from the insertion of the Achilles tendon into the calcaneus to the distal head of the 1st metatarsal, and (9) distance from medial malleolus to the insertion point of the Achilles tendon into the calcaneus. These variables were measured to the nearest millimetre using bone callipers and a flexible tape measure (coefficient of variation of 0.81%). The definitions of these measures were based on the work of Norton and Olds (1996) and are

given in Table 3-1. These measurements, in addition to age (years), were used as potential moment arm predictors.

*Table 3-1. A description of the anthropometric variables collected and their method of collection. *Definitions provided by Norton and Olds (1996).*

Measure	Method
Foot length*	Linear distance from the longest toe to the most posterior point on the heel of the foot with the participant standing with weight distributed between the feet.
Inter-malleolar ankle width*	Distance between the centres of the medial and lateral malleoli, measured using bone callipers with the participant standing with feet slightly apart.
Antero-posterior ankle depth*	Distance in the sagittal plane between the Achilles and tibialis anterior tendons at the height of the medial malleolus.
Tibial length*	Distance between the lateral malleolus and the centre of the lateral epicondyle, measured with a flexible tape with the participant standing.
Lower leg circumference*	Largest record from a flexible measuring tape measure taken at the mid-belly region of the triceps surae group with the participant standing.
Calcaneal – 1st Metatarsal	Linear distance between the Achilles tendon insertion onto the calcaneus (determined by palpation) and the distal head of the 1st metatarsal on the medial side of the foot, measured with the participant standing.
Calcaneal – Medial Malleolus	Distance between the Achilles tendon insertion onto the calcaneus and the centre of the medial malleolus.

3.2.3 Measurement of Achilles Tendon Moment Arm

Achilles tendon moment arm (MA_{AT}) was determined by the tendon excursion method using ultrasonography (Ito *et al.*, 2000; Maganaris *et al.*, 2000; Fath *et al.*, 2010). Moment arms

derived from this method at a neutral (0^{o}) ankle angle have been shown to be reliable and to correlate well with those obtained from MRI measures (Fath *et al.*, 2010).

3.2.4 Procedure

Participants were seated in an isokinetic dynamometer (Biodex Multi-joint System 3, Shirley, New York). The lateral malleolus of the right ankle was aligned with the centre of rotation of the head unit. The footplate was positioned perpendicularly to the horizontal axis so the foot was perpendicular to the tibia (neutral ankle angle). The hip angle was set at 85^{o} and the knee was fully extended. Stabilisation straps were firmly tightened over the foot, thigh and chest to minimise movement of the upper body or leg. To pre-condition the triceps surae muscle-tendon unit, 5-8 sub-maximal and maximal voluntary isometric plantarflexion contractions were performed (Schatzmann *et al.*, 1998). The right foot was then passively rotated between 20° dorsiflexion and 30° plantarflexion at a constant angular velocity of $10°{\cdot}s^{-1}$ to familiarise the participant with the task. With the exception of one, all participants were comfortable with this range (the range of motion was reset to 15° dorsiflexion and 30° plantarflexion for the remaining participant). The ankle was then passively rotated through the range of motion at a constant angular velocity of $10°{\cdot}s^{-1}$ three times, starting from a dorsiflexed position. Participants were instructed to relax the muscles of their lower limbs during the passive rotations to minimise the influence of muscle forces on the moment arm calculations. This was monitored by visually inspecting real-time muscle activity from the gastrocnemius medialis (GM) and tibialis anterior (TA) muscles using electromyography (Telemyo 2400R, NorAxon U.S.A Inc, Arizona, USA).

3.2.5 Instrumentation

GM MTJ excursion during the passive rotation was visualised using 2D B-mode ultrasonography with a 45-mm linear array probe (Megas GPX, Esaote, Italy) operating at a frequency of 10 MHz. The probe was positioned perpendicularly to the skin surface and orientated to image the GM MTJ. Water-based gel (Henley's Medical, Hertfordshire, UK) was used to enhance the acoustic contact and, therefore, the contrast of the images. The probe was secured to the skin by means of a custom-made foam holder and micropore medical tape. An echoabsorptive strip was positioned on the skin immediately above the GM MTJ to provide a suitable marker on the ultrasound image for identification of any probe movement with respect to the skin during data collection (Figure 3-1).

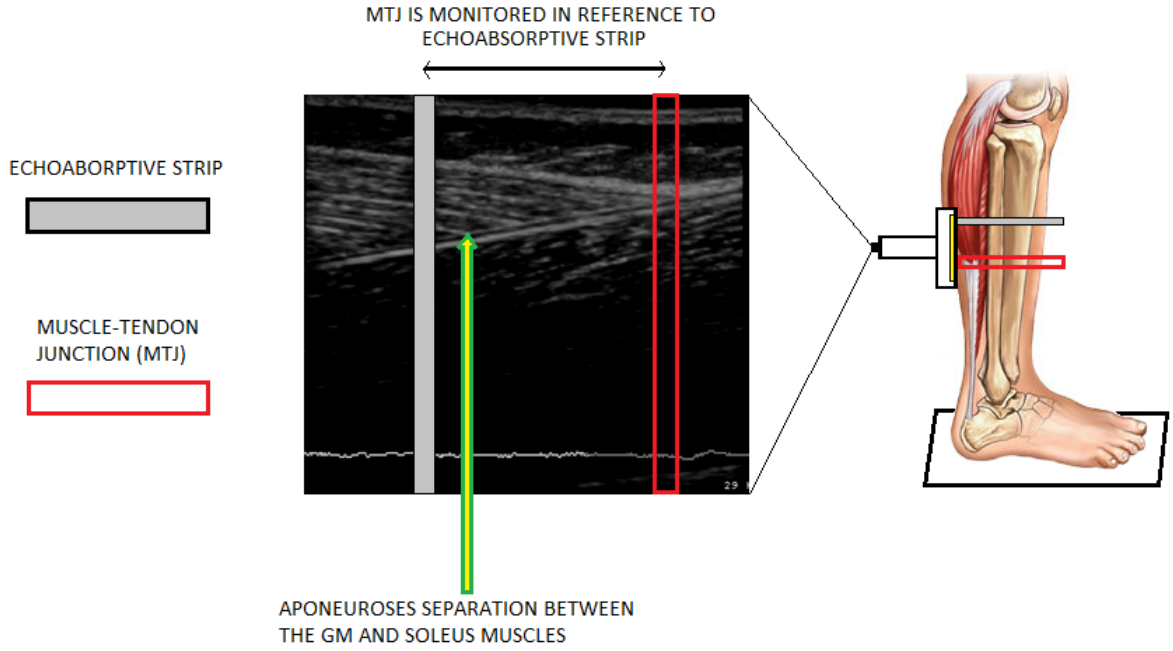


Figure 3-1. Graphical representation of the set-up and method for calculation of the Achilles tendon moment arm using the tendon excursion method. The muscle-tendon junction (MTJ) of the gastrocnemius medialis muscle (located within the red lines) is shown on a representative ultrasonographic image. Excursion of the MTJ during ankle rotation was made in reference to an echoabsorptive marker (grey line) placed on the skin to identify movement of the ultrasound probe with respect to the skin.

3.2.6 Data Processing

Data were captured using Cortex software v1.1.4 (Motion Analysis, Santa Rosa, USA). Ultrasound images were digitally captured at 25 Hz using a digital video-converting frame grabber (ADVC-55, Grass Valley, France). Peak Motus digitising software (v9, Vicon Motion Systems, Colorado, USA) was used to manually identify the GM MTJ in each video field. Scaled GM MTJ position data were low-pass filtered using a fourth-order, zero-lag Butterworth filter with a cut-off frequency of 3.25 Hz, as determined by residual analysis (Winter, 1990b). Ankle joint position data were sampled at 1000 Hz using a 12-bit A/D card (NI PCI-6071E, National Instruments, Texas, USA) and low-pass filtered at 14 Hz using a fourth-order zero-lag Butterworth filter. Joint position and ultrasound data were synchronised using an electrical trigger input (Stimulator DS7A, Digitimer Ltd., Hertfordshire, UK). The filtered joint position data were down-sampled to match the ultrasound data.

3.2.7 Calculation of Achilles Tendon Moment Arm

According to the TE method (An *et al*., 1983; Ito *et al*., 2000), moment arm can be calculated as the mathematical derivative of tendon excursion with respect to the angular displacement of the corresponding joint. Following the recommendations of Fath *et al.* (2010), a third-order polynomial was fitted to approximate the relationship between tendon excursion and ankle displacement over an angular displacement of 20° (from 10° dorsiflexion to 10° plantarflexion; 0° corresponds to the neutral ankle position). Only the data from the third passive plantarflexion were used. The mean (± standard deviation) coefficient of determination (R^2) for the approximated tendon elongation-angular displacement relation was 0.993 (± 0.0036) across the 49 participants. For each participant, the fitted polynomial was then analytically differentiated at the neutral ankle position to

obtain the MA_{AT}. The researcher's coefficient of variation for calculating MA_{AT} using this procedure was 3.66%.

3.2.8 Statistical Analysis

Coefficients of determination (R^2) were computed to describe the strength of the relationship between MA_{AT} and each of the anthropometric variables. To determine whether MA_{AT} could be predicted from a combination of the anthropometric parameters, a backwards stepwise multiple regression analysis was performed. The threshold of significance for inclusion of a parameter into the regression model was set at $p \leq 0.05$.

3.3 Results

The mean (±SD) calculated MA_{AT} length for the present participants was 30.0 ± 6.1 mm. The individual relationships between MA_{AT} and the individual anthropometric predictor variables are shown in Figure 3-2; coefficients of determination (R^2) ranged from 0.24 for leg circumference to 0.42 for both foot length and the distance between the calcaneus and 1st metatarsal ($d_{calc\text{-}met}$). The regression analysis indicated that the inclusion of all anthropometric variables accounted for 49% of MA_{AT} variability, however the only significant correlates of MA_{AT} were foot length and $d_{calc\text{-}met}$(p<0.05), which, when combined, accounted for 49% of the variability of the MA_{AT} across the 49 participants. The regression equation (equation 1) for predicting MA_{AT} from foot length and $d_{calc\text{-}met}$ was:

$$MA_{AT}\ (mm) = (0.117 \times \text{Foot Length}) + (0.180 \times d_{calc\text{-}met}) - 19.618 \qquad \text{Equation 1}$$

(NB. All measurements were made in mm).

The standard error of the estimate relating to Equation 1 was 4.57 mm (15.2%). The mean absolute difference (± SD) between actual and estimated (from equation 1) moment arms for the 49 participants was 3.8 ± 4.4 mm.

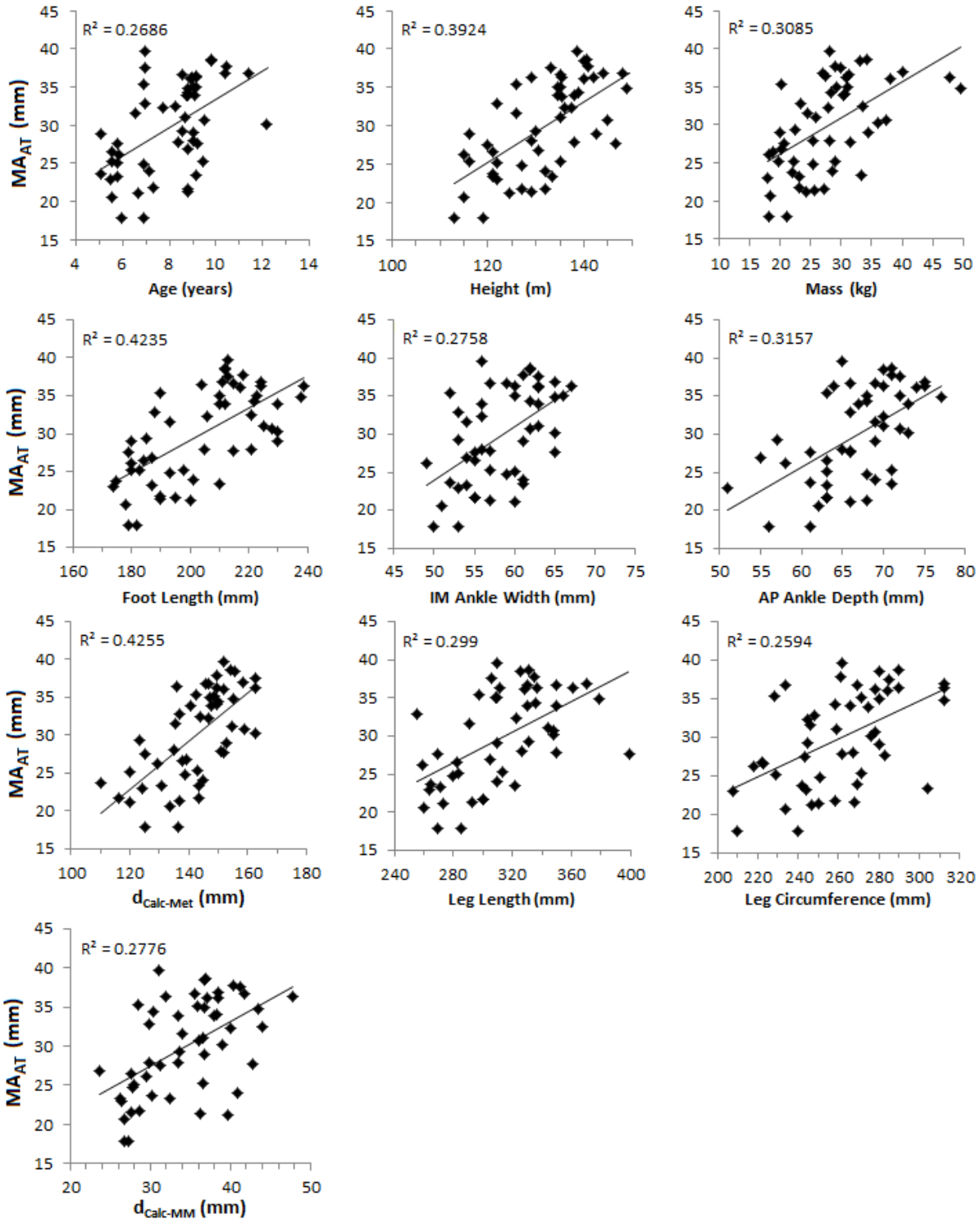


Figure 3-2. Relationship between the Achilles tendon moment arm (MA_{AT}) length and various anthropometric predictors. R2 are unadjusted values. Height (standing), IM: inter-malleolar; AP: antero-posterior; dcalc-met: distance between the calcaneus and 1st metatarsal; dcalc-MM: distance between the calcaneus and medial malleolus.

3.4 Discussion

The aim of this study was to determine whether Achilles tendon moment arm (MA_{AT}) could be predicted from surface anthropometric characteristics in prepubescent children. The results show that only half of the variability of MA_{AT} (49%) across the participants could be accounted for by variation in anthropometric measures. Foot length and $d_{calc\text{-}met}$ were the only significant predictors of MA_{AT} and accounted for the vast majority (96%) of the explained variance of the regression model that included all variables. The predictive ability of MA_{AT} is therefore poorer than that of patellar tendon moment arm (MA_{PT}) shown previously in a similar population (O'Brien *et al*., 2009). While the regression model explained less than 50% of the variance in MA_{AT}, O'Brien *et al.* (2009) found that 83% of variance in MA_{PT} could be explained by tibial length. The discrepancy in the predictability of the two moment arms raises the question as to why the MA_{AT} is less predictable than the MA_{PT} in children.

A possible explanation for the difference in predictability between MA_{PT} and MA_{AT} is the difference in the anatomical complexity of the predictors. The tibia, as the segment which is immediately distal to the knee, is a single long bone whose growth is relatively predictable (Anderson *et al*., 1963). On the other hand, the foot segment, immediately distal to the ankle, comprises numerous long and irregular bones (tarsals, metatarsals and phalanges). As a consequence, the age-related increase in the superficial measures related to foot length are likely to be less predictable than that of tibial length, which might in part explain the lesser predictability of MA_{AT} compared to $MA_{PT.}$ Based on this speculation, it is possible that

the lengths of individual bones within the foot (e.g., calcaneus) could be better predictors of MA_{AT}. However they cannot be accurately measured using surface anthropometry.

Another possible explanation for the lesser predictability of MA_{AT} compared to MA_{PT} is that triceps surae volume (or more specifically the antero-posterior muscle thickness) might influence the orientation of the Achilles Tendon and its moment arm with respect to the ankle joint. Conversely, MA_{PT} is unlikely to be affected by muscle thickness because the patellar tendon directly connects the patella to the tibial tuberosity. The assumption that muscle thickness might have influenced these results is supported by the fact that the ratio between muscle CSA and moment arm length in children is not the same as in adults (Kanehisa *et al*., 1995; Morse *et al*., 2008) and that the MA_{AT} length increases during a muscle contraction as a result of the increase in muscle thickness, altering the tendon line of action (Maganaris, 2004). In spite of these factors, the addition of lower leg circumference to the regression model did not improve the moment arm estimation. Therefore, the influence of muscle thickness on MA_{AT} length during both passive and active conditions in children warrants further study.

The practical implications of the present findings are substantial. Moment arm length is a necessary measure for the derivation of muscle-tendon forces from joint moments. Errors in the estimation of moment arms strongly influence the accuracy of muscle-tendon force estimates from joint moment measurements. The average absolute difference between the actual and predicted MA_{AT} (equation 1) was 3.8 mm. This mean difference would strongly influence the accuracy of muscle-tendon force estimates from joint moment measurements. Based on this mean difference, over- or underestimating the average actual moment arm by the mean difference between actual and predicted moment arm would result in errors of

muscle force calculation by -11.2% and +14.5% respectively. In fact, the difference between the actual and predicted MA_{AT} in one participant was large enough to overestimate muscle forces by 34.4%. Thus the errors associated with using surface anthropometry to predict MA_{AT} in children are unacceptably large.

The tendon excursion method, which is based on a principle of virtual work, assumes that any forces acting across the joint of interest during its rotation are negligible (Storace and Wolf, 1979; An et al., 1984). However, Fath et al. (2010) found an increase in joint moment during dorsiflexion due to both increases in active (reflex muscle activation due to muscle stretch) and passive forces (stretching the elastic components of the muscle-tendon unit), which may result in an underestimation of tendon elongation for a given joint rotation and subsequent calculation of a smaller moment arm. Despite the fact that this method has not yet been validated in children, the potential issues associated with its use are arguably lower in children. Firstly, children have a greater ankle plantarflexion–dorsiflexion flexibility than adults (Soucie et al., 2011). As such, the passive forces associated with stretching the elastic components of the MTU will be lower. Additionally, and theoretically, a more compliant tendon would also take up more of an imposed muscle-tendon unit length change thus reducing the likeliness of reflex muscle activity associated with muscle stretch (Liddell and Sherrington, 1924).

3.4.1 Conclusion

In summary, the Achilles tendon moment arm length at rest in a neutral ankle position could not be accurately predicted from surface anthropometric characteristics in prepubescent (5 to 12 years) children. Whilst the regression analysis indicated some moderate relationships,

the error associated with the regression model was too large to allow for accurate predictions of Achilles tendon moment arm. Thus, estimating the moment arm from surface anthropometry should not be considered when quantifying Achilles tendon moment arm in children.

CHAPTER 4: Age-related Changes in the Mechanical Properties of the Achilles tendon

4.0 Abstract

The transfer of muscular forces to the skeleton is influenced by the stiffness of the adjoining tendon. Tendons stiffness adapts in response to chronic loading, therefore it was hypothesised that growth-related increases in body mass and force production capabilities, as well as changes in tendon dimensions, would be associated with the age-related increases in tendon stiffness during childhood. Achilles tendon stiffness, Young's modulus, tendon dimensions, peak stress, strain, force and body mass were determined in fifty-two prepubertal children (aged 5 to 12 years) and19 adults. Achilles tendon elongation, measured using ultrasonography, and peak plantarflexor moment from dynamometry were obtained during maximal isometric plantarflexion contractions. Peak force was calculated from plantarflexor moment using the Achilles tendon moment arm (MA_{AT}), estimated using the tendon excursion method. Tendon stiffness was calculated as the slope of the force-elongation curve and Young's modulus was calculated by normalising stiffness to resting tendon length and CSA. The relationship between tendon stiffness and age, body mass and peak force was determined separately for children and all ages combined (i.e. children + adults) using multiple regression analyses. Variables relating to either stiffness or Young's modulus were documented descriptively as a function of age and effects sizes calculated between groups. Strong relationships were found between body mass and peak force with tendon stiffness and Young's modulus in children, indicating that age-related adaptations most likely result from chronic increases in tendon loading and are not a true 'ageing' effect. Changes in tendon stiffness during childhood were largely attributable to CSA hypertrophy and increased Young's modulus with age, both of which are likely to be influenced by force producing ability. In addition, peak stress increased from childhood to adulthood due to greater increases in strength than CSA with age; however peak strain remained constant as

a result of parallel increases in tendon length and peak elongation. The differences in Achilles tendon properties found between adults and children are likely to influence force production so determining the impact of this is an important area of future research.

4.1 Introduction

Tendons are spring-like structures that play an integral role in movement by transferring muscular forces across joints to bones. Their mechanical properties, and in particular their stiffness, affect force production and complex motor performance (Bojsen-Moller *et al*., 2005; Arampatzis *et al*., 2006). Importantly, tendon mechanical properties adapt in response to chronic increases (Kubo *et al.*, 2001a; Wu *et al.*, 2010) or decreases (Reeves *et al.*, 2003a; Maganaris *et al.*, 2006) in loading.

During childhood, the stiffness of weight-bearing tendons has been shown to increase with age from 9 years to adulthood in humans (Kubo *et al.*, 2001b; O'Brien *et al.*, 2010), the findings of which are consistent with those relating to load bearing animal tendons (Woo *et al*., 1982; Shadwick, 1990). Changes in body mass and force production capabilities, as inter-related mechanisms that increase with age, have been postulated to contribute to these observed increases in tendon stiffness with age (O'Brien *et al*., 2010) but have not been explored previously as potential underlying mechanisms. Body mass increases substantially from childhood to adulthood, requiring the weight-bearing tendons to tolerate higher loads. Although changes in body composition occur with prepubertal age (Ruxton *et al*., 1999), muscle mass tends to increase in proportion with body mass (Fomon *et al*., 1982; Malina and Bouchard, 1991; Maynard *et al*., 2001). Muscle mass is directly associated with force production capacity, but is not the only determinant of muscular strength. Age-related increases in muscular strength are also influenced by improvements in the individual's ability to recruit and coordinate the available muscle mass for force production (Stackhouse *et al*., 2005; Falk *et al*., 2009). As a result, strength does not increase in direct proportion to body mass (Asmussen and Heeboll-Nielsen., 1955; Blimkie, 1989). Therefore, both body

mass and force production capabilities may contribute independently to age-related increases in chronic loading (Proske and Morgan, 1987). Consequently, tendons may adapt based on their requirement to transmit higher forces from the muscular to the skeletal system safely and effectively.

In addition to a requirement to tolerate greater loads, a tendon's stiffness is also dependent on its dimensions. Based on Hookean law, thicker tendons (greater CSA) are associated with higher stiffness as more spring-like material is arranged in parallel, whereas longer tendons are associated with lower stiffness as more spring-like tissue is arranged in series (Proske and Morgan, 1987). Because these dimensions change with age (Jozsa and Kannus, 1997; O'Brien *et al*., 2010), increases in tendon stiffness must also be set in the context of tendon growth. By normalising tendon stiffness to its dimensions (i.e. calculating the Young's modulus) in an attempt to account for dimensional differences between individuals, it is possible to monitor changes in a tendon's material properties independent of changes in dimensions. For example, previous research has shown that Young's modulus increases with age (O'Brien *et al*., 2010), suggesting that in addition to dimensional growth, the tendon's intrinsic material properties undergo a maturation process also. In animals, this is caused by a number of processes acting to improve collagen density within the tendon (Elliott, 1965; Parry *et al.*, 1978a; Curwin *et al.*, 1988; Bailey *et al.*, 1998; Bayer *et al.*, 2010), initiated by mechanical loading (Kjaer, 2004). Increases in tendon stiffness with age have therefore been attributed to both maturational and dimensional tendon adaptations (O'Brien *et al*., 2010). Such changes have not been documented in younger children (<9 years), so the development of tendon properties throughout childhood is not yet known.

Expanding on previous research, the primary purpose of this study was to partition out the contributions of age, body mass and muscular strength on prepubertal developmental increases in tendon stiffness. Within this context, the influence of age, body mass and muscular strength on tendon stiffness irrespective of tendon size i.e. with Young's modulus, was also examined. This allowed the relative importance of tendon size and Young's modulus on age-related changes in tendon stiffness to be examined. The secondary purpose of this study was to document the development of parameters directly influencing or pertaining to either stiffness or Young's modulus (including the tendon's dimensions, elongation due to peak force and mechanical stress and strain) in order to better understand the mechanisms underpinning their change with age. Understanding such changes may help to provide an explanation into differences in movement performance (Asai and Aoki, 1996; Chao *et al.*, 2002; Ganley and Powers, 2005; Grosset *et al.*, 2005; Korff and Jensen, 2007; Falk *et al.*, 2009) and movement efficiency (DeJaeger *et al*., 2001; Schepens *et al*., 2001; 2004) between children and adults. Within this context, a final purpose of the present study was to examine the elastic energy storage capacity of the Achilles tendon.

Although some data are available to describe the age-related mechanical adaptations in the patellar tendon, a lack of data exists detailing such changes in the functionally important Achilles tendon. The Achilles tendon contributes substantially to the movement outcome of dynamic tasks such as walking, running and jumping (Fukunaga *et al*., 2001; Lichtwark and Wilson, 2005; Ishikawa *et al*., 2007) and plays a significant role in quiet standing and balance control (Roncesvalles *et al*., 2001; Loram and Lakie, 2002). Moreover, it encounters considerable loading during these everyday tasks.

4.2 Methods

4.2.1 Ethics and Participant Information

Fifty-two prepubertal children between the ages of 5 and 12 years (27 boys, 25 girls; mean age 8.12 ± 1.66 years), 10 men (25.7 ± 3.6) and 9 women (25.2 ± 3.5) volunteered to participate in this study (for participant information, see Table 4-1). Peak height velocity, as an indicator of maturational offset (Malina and Bouchard, 1991), was estimated in children over 8 years of age to confirm their prepubertal status (Mirwald *et al*., 2002). The minimum age from peak height velocity found in this subgroup of children was -1.9 years. All participants were free from neuromuscular or musculoskeletal disorders and were not involved in any competitive sports. The study was approved by the Human Research Ethics Committee at Brunel University. Testing conformed to the guidelines set out in the Declaration of Helsinki. Children provided written assent to participate in the study and parents/guardians provided written informed consent. All procedures were explained to the participants in a child-appropriate manner. All participants were made aware of their right to withdraw from the study at any time without penalty.

Table 4-1. Participant characteristics (mean ± SD).

Group	N	Age (years)[ab]	Height (cm)[ab]	Mass (kg)[abc]
$CG_{5–6}$	16	6.1 ± 0.7	120.7 ± 4.1	21.2 ± 2.4
$CG_{7–8}$	18	8.3 ± 0.7	134.9 ± 5.2	29.0 ± 6.5
$CG_{9–10}$	16	9.5 ± 0.9	138.2 ± 5.4	32.2 ± 5.3
Adult Men	10	26.8 ± 1.8	179.6 ± 6.5	78.6 ± 11.7
Adult Women	9	24.8 ± 3.2	167.6 ± 5.6	64.3 ± 7.8

CG_{5-6},CG_{7-8} andCG_{9-10} represent children aged5 – 6, 7 – 8 and 9 – 10 years, respectively. Significant difference (p< 0.05) between a) adults (M and W) and all child age groups, b) CG_{5-6} and other child age-groups and c) adult men (M) and women (W).

4.2.2 Overview

The participants visited the laboratory on one occasion during which a comprehensive familiarisation of the procedures preceded data collection. Standing and sitting height (for determining peak height velocity) and body mass were measured before familiarisation. During data collection, participants performed maximal isometric plantarflexion contractions during which Achilles tendon elongation was measured using ultrasonography. Tendon stiffness was calculated as the slope of the relationship between tendon elongation and plantarflexor force (corrected for antagonist activity). Force was calculated as the ratio of plantarflexor moment, obtained from dynamometry, and Achilles tendon moment arm, estimated from tendon excursion during passive ankle rotations (Fath *et al.*, 2010). Young's Modulus was subsequently estimated by normalizing tendon stiffness to both resting tendon length and CSA. An overview of the testing protocol is shown in Figure 4-1.

4.2.3 Familiarisation

To familiarise the participants with the equipment and the isometric plantarflexion required for testing, five to eight sub-maximal isometric plantarflexion contractions were performed with the instruction to "rotate the foot away from the body using the ball of the foot whilst keeping the heel in contact with footplate". Participants then typically performed 3 – 5 further contractions with maximal effort to ensure their maximum force was identified. These contractions also provided a task-specific warm-up, important for pre-conditioning the tendon to ensure consistency of load-deformation properties during data collection (Rigby *et al.*, 1959; Viidik *et al.*, 1982; Schatzmann *et al.*, 1998; Maganaris and Paul, 1999). A minimum of 30 s separated two consecutive contractions. A 5-minute passive rest period

was given to the participants between the familiarisation period and the testing protocol to minimise fatigue.

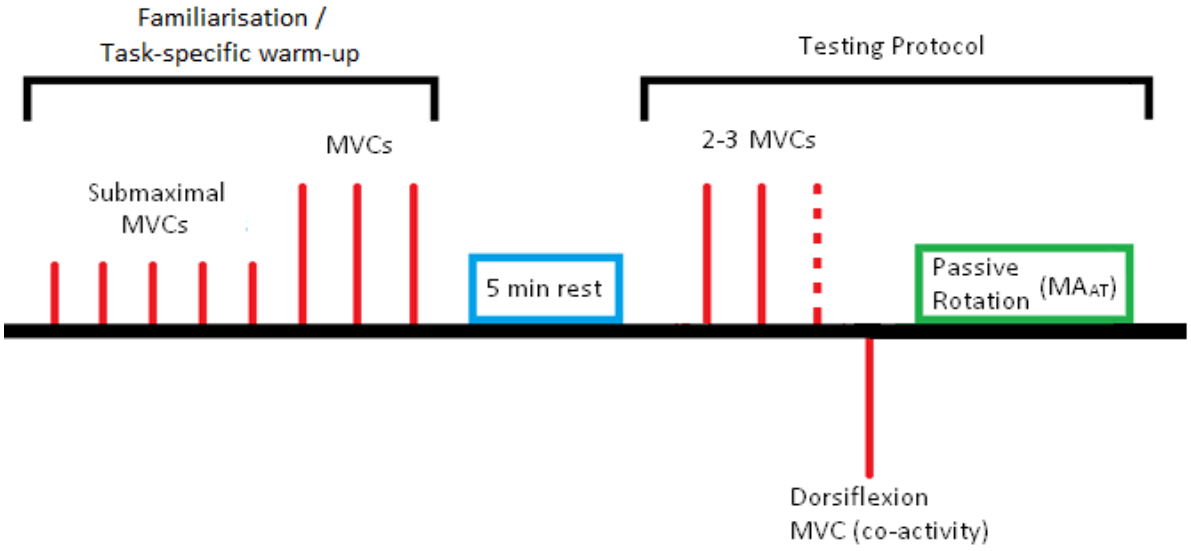


Figure 4-1. Overview of experimental protocol. Participants performed submaximal and maximal voluntary contractions (MVCs) as a task-specific warm-up. Children performed 2 – 3 MVCs whilst adults always adults performed 3 MVCs. A dorsiflexion MVC was completed to allow antagonist coactivity to be quantified from the tibialis anterior moment-EMG relationship. The ankle was then passively rotated through its range of motion in order to determine Achilles tendon moment arm (MA_{AT}).

4.2.4 Measurement of Plantarflexor Moment

The moment about the ankle joint was measured using an isokinetic dynamometer (Biodex Medical Systems, New York, USA). Participants were seated on the dynamometer chair with relative hip and knee angles set to 95^{o} (between trunk and thigh) and 0^{o} (i.e. full extension), respectively. The dynamometer footplate was positioned perpendicularly to the inertial horizontal axis to provide a relative ankle angle of 90^{o} between the foot and the tibia (neutral ankle angle). The lateral malleolus of the right fibula was aligned with the dynamometer's rotational axis by adjusting the length of the dynamometer arm. Due to a

degree of compliance within the chair and dynamometer head unit, translation of the footplate with respect to the chair was noticeable during plantarflexion contractions during pilot testing. This translation resulted in the lateral malleolus misaligning with the centre of rotation of the dynamometer head unit and changed the ankle angle considerably, a problem observed previously (Arampatzis *et al*., 2008). To minimise joint rotation due to unwanted dynamometer movement, participants were initially seated with the knee flexed, which upon straightening (i.e. performing a leg press manoeuvre) locked the knee joint and allowed the leg to act as a passive strut (see Figure 4-2). Subsequent ankle plantarflexion deformed of the dynamometer system only minimally. To reduce the risk of leg, upper body or heel movement affecting the dynamometer readings, stabilisation straps were applied tightly over the foot, thigh and torso. Furthermore, the participants were instructed to cross their arms over their chest. This protocol resulted in a mean ankle angle change of 5 - 6˚ in children and adults (for details, see the section "*Correction for muscle-tendon junction movement due to joint rotation during MVC",* pages 83-84) as opposed to ~10^{o}reported previously for adults (Magnusson *et al*., 2001; Muramatsu *et al*., 2001).

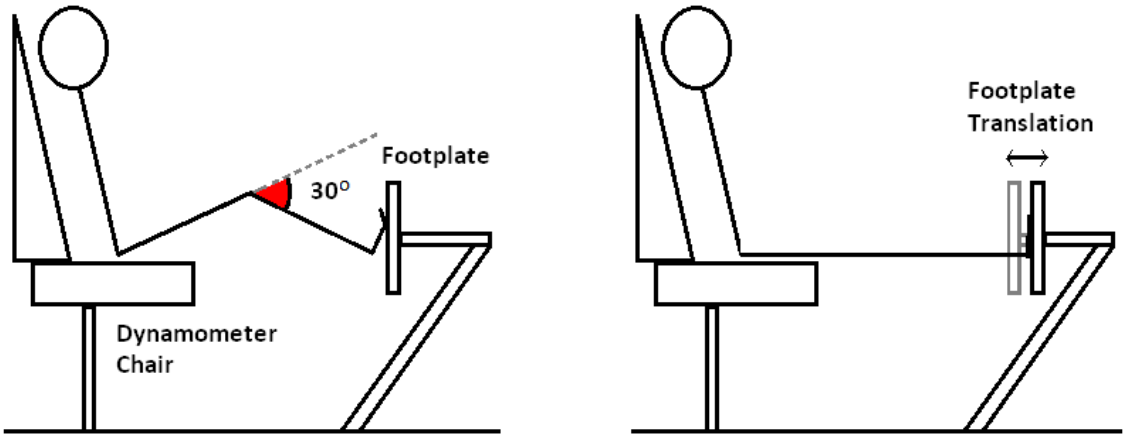


Figure 4-2. Representation of the leg press manoeuvre (pressing from a flexed to a fully extended knee angle) used to minimise dynamometer-chair movement during the maximal plantarflexor contractions.

The results from pilot testing revealed that, despite extensive practice, many of the children found it difficult to perform slowly ramped maximal plantarflexion contractions and produce a uni-modal moment-time profile. Thus children were instructed to produce their MVCs with a maximum rate of force development. The rate at which children can produce force voluntarily is markedly lower than adults (Asai and Aoki, 1996), therefore the resolution of the force-tendon elongation curves created with an ultrasound sampling frequency of 25 Hz proved adequate for reliable data capture. The rate at which adults can produce force voluntarily was too high for reliable data capture, therefore adults performed ramped contractions over 3-4 seconds. Despite different instructions, the time taken to reach maximum torque was not very dissimilar between children and adults (children - 2.27 ± 0.77-s; adults – 3.02 ± 0.87-s). Plantarflexor moment was recorded during two 4-s MVCs with verbal encouragement (McNair *et al.*, 1996). A third trial was allowed if the peak moment achieved in the first two trials differed by more than 5%. The performance of only two plantarflexion MVCs (where possible) was used to reduce the testing time for the younger children. Trials were separated by a 30 s rest period. Plantarflexor moment and ankle joint position were sampled at 1000 Hz using a 12 bit A/D card (NI PCI-6071E, National Instruments, Texas, USA) and low-pass filtered using a fourth-order, zero-lag Butterworth filter with a cut-off frequency of 14 Hz, as determined by residual analysis (Winter, 1990a).

4.2.5 Measurement of Tendon Elongation

Tendon elongation was measured as the displacement of the GM MTJ from rest during the MVC trials. The GM MTJ was visualised using B-mode ultrasonography with a 45-mm linear array probe (Megas GPX, Esaote, Italy; 10-MHz transducer scanning). Water-based gel (Henley's Medical, Hertfordshire, UK) deposited between the ultrasound probe and skin

enhanced acoustic transmission so that clear images could be taken without depressing the dermal surface. The probe was placed perpendicularly to the skin's surface above the MTJ and the scanning interface was orientated to clearly display both the separation between the aponeuroses of the GM and soleus (SOL) muscles and the GM MTJ simultaneously. Following these criteria allowed GM MTJ movement to be suitably visualised during active conditions. The probe, cupped by a custom made foam holder, was secured in position using micropore medical tape. An echoabsorptive strip placed on the skin above the GM MTJ provided a reference point to identify probe movement relative to the skin during the trials by casting a linear shadow through the images (see Figure 4-3 for a visual representation of the experimental setup relating to ultrasonography of the GM MTJ).

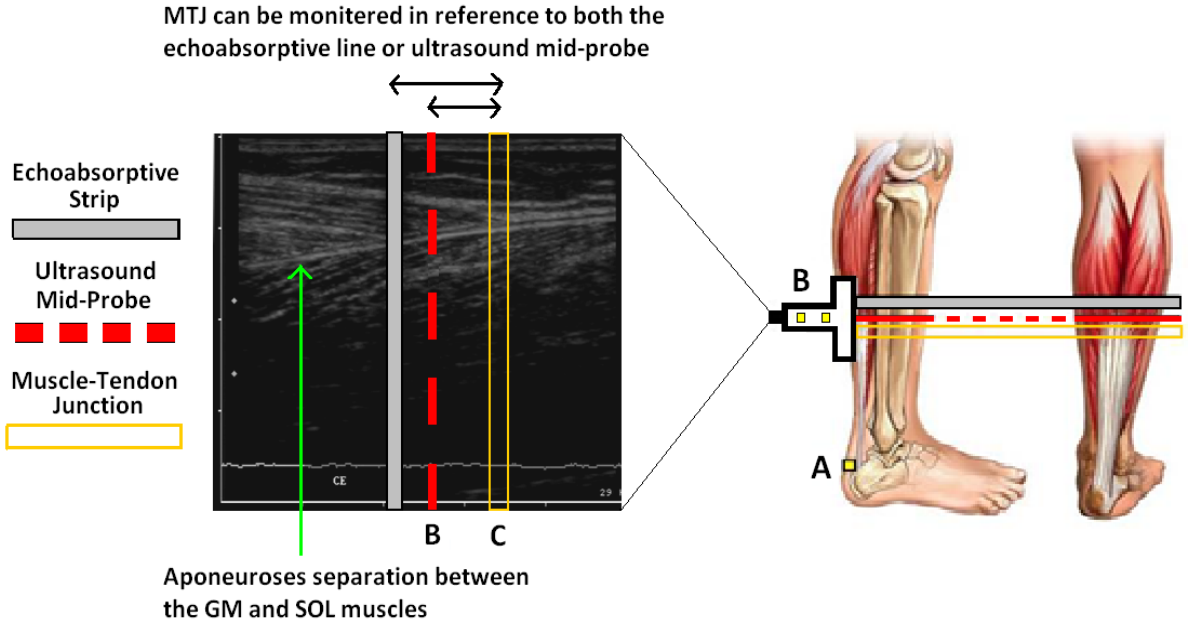


Figure 4-3. Experimental setup for measuring tendon elongation and tendon resting length. Elongation of the tendon was measured as movement of the muscle-tendon junction (MTJ) in reference to the echoabsorptive marker. The resting tendon length was calculated as the distance between the tendon insertion point on the calcaneal bone (A) and ultrasound mid-probe (B), adjusted for the distance of the MTJ from the mid-probe (Tendon Length = LengthAB + LengthBC).

Ultrasound images were digitally captured at 25 Hz using a digital video converting frame grabber (ADVC-55, Grass Valley, France). Peak Motus digitising software (v9, Vicon, Oxford, UK) was used to manually identify the GM MTJ in each video field. GM MTJ positional data were low-pass filtered using a fourth-order, zero-lag Butterworth filter with a cut-off frequency of 3.25 Hz(Winter, 1990b).

4.2.6 Correction for Muscle-Tendon Junction Movement due to Joint Rotation during MVC

Ankle joint rotation during plantarflexion results in movement of the MTJ, leading to an underestimation of tendon elongation and an overestimation of tendon stiffness (Magnusson *et al*., 2001; Arampatzis *et al*., 2008). Despite efforts to reduce heel movement, small rotations still occurred. Therefore the MTJ displacement due to ankle displacement was estimated as the product of angular rotation (rad) and moment arm length (Magnusson *et al*., 2001; Rosager *et al*., 2002), based on a linear relationship between tendon displacement and joint rotation (Fukunaga *et al*., 1996). Three infrared LED motion capture cameras (MotionAnalysis, Santa Rosa, USA) were positioned on one side of the dynamometer, focusing on the medial aspect of the participant's foot when positioned on the footplate. Two reflective markers (5 mm x 5 mm) were positioned along the length of the footplate with further markers positioned on the calcaneal tuberosity and the distal end of first metatarsal of the foot. The instantaneous change in angle between the footplate and the foot (defined as the line connecting the calcaneus and metatarsal markers) during the MVC was considered to be the change in ankle angle during the isometric contraction. The movement of the MTJ attributable to the ankle rotation was then subtracted from that recorded during the MVC to provide a corrected measure of MTJ displacement. Coordinate data were captured using Cortex software v1.1.4 (MotionAnalysis, Santa Rosa, USA). Motion

capture markers were sampled at 100 Hz and low-pass filtered using a fourth-order zero-lag Butterworth filter with a 7-Hz cut-off frequency. Filtered data were down-sampled to 25 Hz to match the frequency of the ultrasound data.

4.2.7 Moment Arm Estimation

Achilles tendon moment arm (MA_{AT}), defined as the perpendicular distance from the joint centre of rotation to the line of Achilles tendon action, was calculated using the tendon excursion method (An *et al.*, 1984; Fath *et al.*, 2010). For this purpose, the dynamometer was set to passively rotate the right foot between 20° dorsiflexion and 30° plantarflexion at a constant angular velocity of $10°\cdot s^{-1}$. The participants were encouraged to relax during the rotation to minimise muscular activity and were provided with an emergency stop device in case they experienced discomfort. With the exception of one, all participants were comfortable with this range (for the remaining participant, the range of motion [ROM] was reset to 15° dorsiflexion and 30° plantarflexion). The ankle was rotated three times through the ROM, starting with a dorsiflexion movement. The third plantarflexion movement in the trial was subsequently used for deriving the moment arm.

Ankle joint position was sampled at 1000 Hz using the dynamometer, and the data were low-pass filtered at 14 Hz using a fourth-order zero-lag Butterworth filter. A third-order polynomial was fitted between tendon excursion and ankle displacement over an angular displacement of 20° (from 10° dorsiflexion to 10° plantarflexion). The mean coefficient of determination for this approximation was $R^2 = 0.993 \pm 0.0036$ across participants. The polynomial was then analytically differentiated at the neutral ankle position to obtain the

MA_{AT} at this ankle position (Fath *et al.*, 2010). The coefficient of variation (CV) for calculating MA_{AT} from digitising each trial three times was 4.5%.

4.2.8 Electromyographic Measurement of Muscle Activity

During maximal contractions, it is common that antagonist muscle activity accompanies agonist muscle activation (Magnusson *et al*., 2001; Mademli *et al*., 2004). Both agonist and antagonist activity contribute to the net moment recorded by the dynamometer. As a result, the force derived from the moment measured by the dynamometer is an underestimation of the actual force developed by the agonist muscle. To account for this co-activation during testing, the TA EMG-moment relation was determined during a ramped dorsiflexion contraction, performed after the plantarflexion trials. Using this relationship, the antagonist moment during plantarflexion was estimated. The TA, as the major dorsiflexor muscle, was assumed to represent antagonist co-activation during plantarflexion.

In children, the electrode placement area was rubbed vigorously with an alcohol-based antiseptic using a disposable pad. This method of preparing the skin has been recommended to achieve low impedence EMG signals in children(Damiano *et al.*, 2000). In adult participants, the skin was prepared by shaving and lightly abrading the area prior to disinfecting the area (Konrad., 2005). Self-adhesive electrodes (Kendall H59P, Covidien plc, Ireland) were placed approximately in parallel with the orientation of the underlying fascicles using a bipolar setup with a centre-to-centre inter-electrode distance of 20 mm. A ground electrode was positioned on the anterior tibia. Real-time EMG signals sent via telemetric transmission to a remote receiver at 1000 Hz (Telemyo 2400R, NorAxon U.S.A Inc, Arizona) were captured synchronously with the other analogue signals. A three-way

pre-amplified lead wire connected to the ground and TA electrodes acted as a ground reference for the whole measurement system. Signals were amplified (base gain = 500, input impedance = 100 MΩ, CMRR ≤100 dB), digitally filtered (Spike2 v5.12a software, Cambridge Electronic Design, UK) using a 10-500 Hz band pass filter, and smoothed by means of calculating the root mean square over a 100 ms window.

The EMG-moment relationship for the TA was determined from a sub-maximal ramped isometric dorsiflexion trial, completed after the final plantarflexion MVC with the instruction to slowly pull the foot towards themselves. The data were adjusted for the lag between the onset of EMG and force (electromechanical delay), calculated from the point at which the EMG signal exceeded ±2 SD of the signal baseline to the point at which force signal exceeded ±2 SD of the signal baseline (Linford *et al*., 2006). A third-order polynomial was fitted to the EMG-moment data corresponding to the greatest level of TA EMG observed throughout the plantarflexion trials (R^2 of 0.96 ± 0.03 [mean ± SD]; Figure 4-4). The resulting regression equation was then used to estimate the antagonist moment present during the plantarflexion trials from the TA EMG amplitude, after adjusting the data for the same electromechanical delay found in the dorsiflexion contraction. The moment corresponding to the level of EMG was subsequently added to the net moment in each plantarflexion trial to provide a corrected plantarflexor moment.

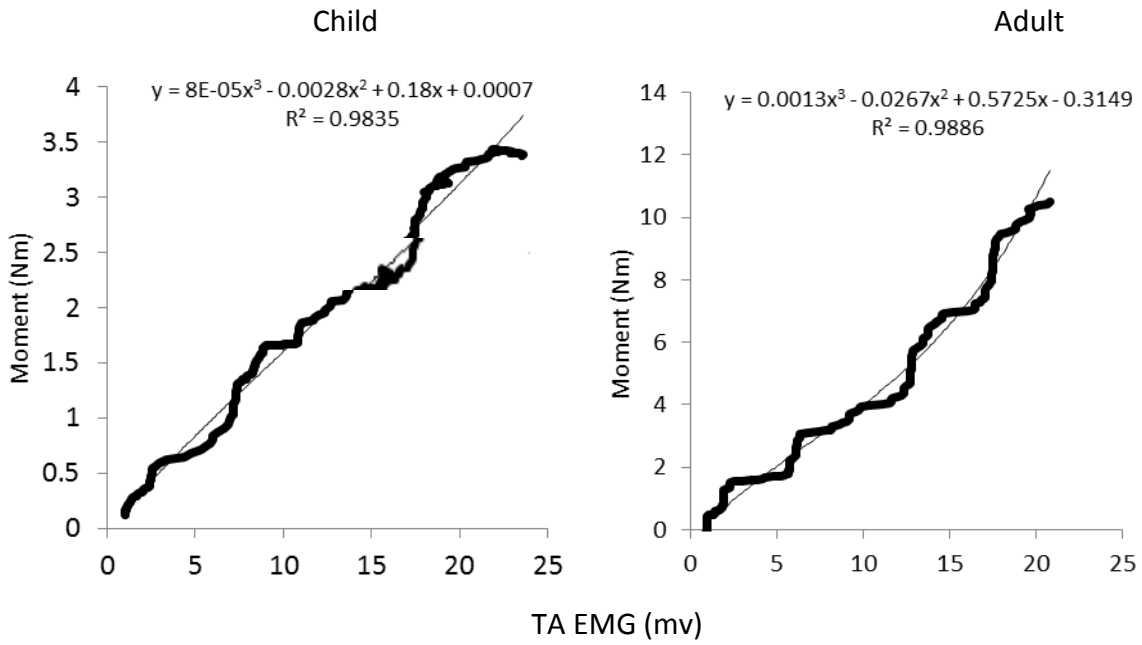


Figure 4-4. Example tibialis anterior (TA) moment-EMG relationships obtained during ramped dorsiflexion contractions for a child (8 yo; left panel) and adult (right panel), respectively. Also shown are the regression equations and R2 values.

4.2.9 Calculation of Tendon Force

Tendon force (*F*) was calculated using the equation $F = {}^{M}/_{r}$, where *M* is the corrected plantarflexor moment and *r* is the Achilles tendon moment arm length at a neutral ankle position. The tendon force calculated therefore represents the combined force of all of the plantarflexor muscles being transmitted through the tendon. The GM has been shown to contribute approximately 18% of the maximum voluntary force of the plantarflexor group (Fukunaga et al., 1992). MTJ displacement is commonly documented at the tendon's junction with the GM, due to clearer imaging of this location. As a consequence, previous studies have adjusted the transmitted plantarflexor force to approximate the contribution of the GM only, thus estimating GM-specific tendon stiffness (Mahieu et al., 2007; Reeves et al., 2005). Here, we assumed that movement of the GM MTJ was representative of that of

the Achilles tendon. As the adjustment of plantarflexor group force to GM-specific force is a ratio, the resulting relationships between tendon stiffness and other independent variables would remain unchanged.

4.2.10 Calculation of Tendon Stiffness

The slope of the line fitted to the force-elongation data points corresponding to 10% and 90% of peak force was obtained to represent the average stiffness for the trial. The final stiffness value was calculated as the mean stiffness from the MVC trials adhering to the following criteria: 1) the peak force of a trial was within ±5% of the maximum recorded value achieved, and 2) the time curve was uni-modal. Uni-modal was defined here as a force-time curve where no fall in force is greater than 10% of MVC. This interval provided the most reliable tendon stiffness data across trials in children (mean CV of 7.6%, calculated from 20 children with three MVCs adhering to the criteria outlined below).

4.2.11 Measurement of Resting Tendon Length and Peak Strain

The resting length of the Achilles tendon was defined as the linear distance from its insertion on the calcaneal tuberosity to the GM MTJ, taken at a neutral ankle angle under passive conditions. This distance was calculated using a combination of motion capture and ultrasound imaging (pages 81-82, Figure 4-3). The 2D coordinates of two reflective markers, placed in series on the midline of the ultrasound probe handle were acquired (in addition to the marker on the calcaneal tuberosity) using the motion capture cameras. In knowing these coordinates and the perpendicular distance of each of the mid-probe markers from the probe's scanning interface, 2D coordinates of the middle of the scanning interface – a point easily identifiable on the ultrasound image – were calculated. The horizontal distance

between the midline of the probe and the MTJ on the ultrasound image was then added to the newly calculated coordinates. The linear distance between the coordinates of the MTJ and the calcaneus maker was then determined by means of the Pythagorean theorem. The reliability of calculating tendon length from the start of each plantarflexion, across the three plantarflexion contractions, was 2.4%.

Tendon strain (ε; deformation of the tendon with respect to resting length) was calculated according to the following equation:

$$\varepsilon = (L_e / L_o) \times 100 \qquad \text{Equation 1}$$

where L_o and L_e represent the resting length and elongation of the Achilles tendon, respectively.

4.2.12 Measurement of Tendon Cross-Sectional Area and Peak Stress

The Achilles tendon CSA was measured in the region encompassing its narrowest point and therefore the region that experiences the greatest stress (Voigt *et al*., 1995). This location has been described to be approximately 30 mm proximal to the tendon insertion in adults (Magnusson *et al*., 2001) therefore was used as a guide only in the present study. The narrowest CSA was identified here by visual inspection and palpation of the tendon. Using a modified silicon ultrasound gel pad (Aquaflex 2x9-cm, Parker Labs Inc., NJ, USA) to provide the greatest probe-to-skin contact area, three transverse US images revealing the Achilles tendon CSA were taken at discrete intervals in the region. The tendon perimeter was traced using specialist software (Esaote, Italy). The reliability of obtaining the smallest CSA was

verified by removing and then replacing the silicon pad and probe after each set of transverse images had been obtained. The area of interest was not visible during the process of collecting the images, neither did the silicon pad leave marks on the skin. The CV calculated from 3 individuals was 4.1%. The researcher's CV for determining the CSA was 3.8%, calculated from the CSA obtained by analysing 30 images three times. An example image is shown in Figure 4-5.

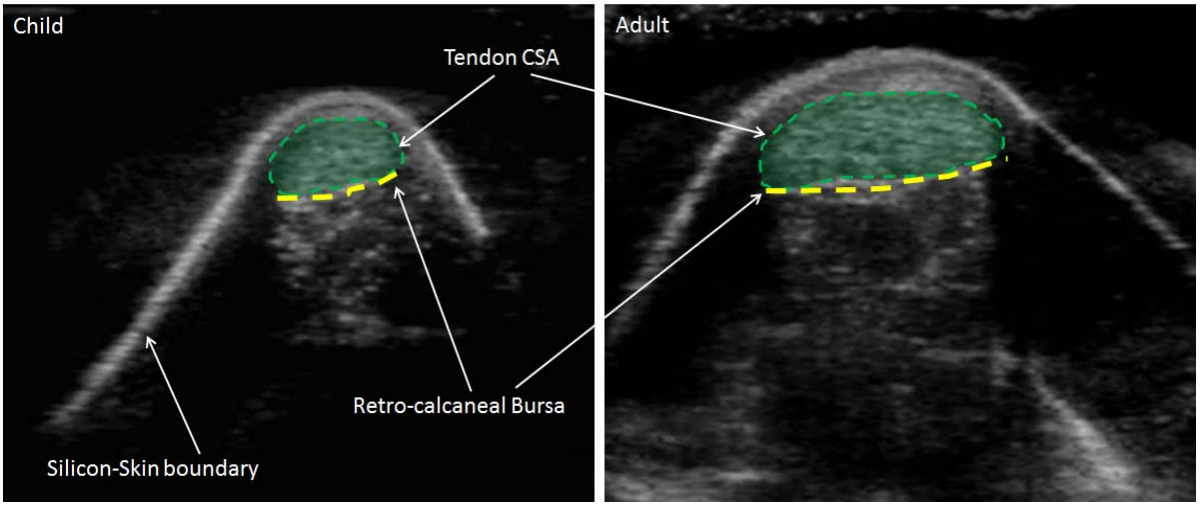


Figure 4-5 Representative images of the Achilles cross-sectional area (CSA) for a child and an adult.

Stress (σ), determined as the magnitude of force per unit area, was calculated using the smallest recorded CSA from each participant according to the equation:

$$\sigma = (F / A) \times 100 \qquad \text{Equation 2}$$

where *F* is the Achilles peak force (N) and *A* is the Achilles tendon cross sectional (mm^2) area.

4.2.13 Calculation of Young's Modulus

Young's modulus is a measure of a material's stiffness normalised to its dimensions (i.e. the magnitude of material displacement for a given load). To allow the direct comparison of the mechanical properties of the tendon between individuals, independent of tendon dimensions, Young's modulus (E) was calculated as the ratio of stress over strain between 10% and 90% MVC according to the equation:

$$E = \sigma / \varepsilon \qquad \text{Equation 3}$$

4.2.14 Statistical Analysis

All data were analysed using SPSS statistical software (v16.0, SPSS Inc., Chicago, USA). Regarding the first purpose of this study, the relationship between each independent variable (IV; age, tendon force and body mass) with each dependent variable (DV; stiffness and Young's modulus) was determined (coefficients of determination [R^2] were derived to quantify the strength of each relationship). The relationship between each IV and DV was approximated using the method of least squares (Table 4-2). The order of the polynomial chosen to represent each pair of variables was the order with the least number of terms which, when adding an addition term to, did not improve the R^2 value by more than 0.02 (2%). This kept the equation to the data restrained. For variables that were best represented by a non-linear polynomial, higher order terms of the polynomial of interest from the corresponding IV were included in the regression analysis as separate IVs (i.e. squaring or cubing data in order to represent each term of the polynomial explaining the data). Partitioning non-linear data in this manner allowed a linear regression to be performed. All IVs were then included in a stepwise multiple regression analysis ($p< 0.05$ for inclusion into

the model) to determine whether a combination of parameters provided a better prediction of Achilles stiffness for children and all ages combined. A separate multiple regression was then performed to ascertain the predictiveness of Young's modulus from the same independent variables.

Regarding the second purpose of this study, the variables relating to tendon stiffness and Young's modulus were also descriptively documented according to chronological age. To establish the practical importance of changes in variables throughout childhood, children were grouped according to ages 5 – 6, 7 – 8 and 9 – 10 years (subsequently referred to as $CG_{5\text{-}6}$,$CG_{7\text{-}8}$, $CG_{9\text{-}10}$) and the effects sizes(Cohen's d; Cohen, 1988) for each variable were calculated between neighbouring age groups. In addition, an overall effects size was calculated between $CG_{5\text{-}6}$ and adults. A multivariate analysis of variance (MANOVA) was performed to examine differences in all measured variables with respect to gender for all groups.

4.3 Results

No sex differences were found for any measured variable for any age group, therefore all data for subsequent analyses were pooled. Results reported are for pooled data. Polynomial curves fitted to the independent variables age, body mass and force for stiffness and Young's modulus can be found in Table 4-2.

Table 4-2. Polynomial equations fitted to each independent variables and stiffness or Young's modulus.

	Variable	Population	Equation	R^2
Stiffness	Age	Children	$Y = -0.6718x^2 + 29.02x - 52.436$	0.37
		All ages combined	$Y = 0.0193x^3 - 1.4228x^2 + 36.965x - 77.107$	0.68
	Mass	Children	$Y = 3.0391x - 30.084$	0.58
		All ages combined	$Y = 0.0005x^3 - 0.1361x^2 + 12.731x - 116.72$	0.75
	Force	Children	$Y = -9E\text{-}06x^2 + 0.0888x + 22.38$	0.51
		All ages combined	$Y = -7E\text{-}06x^2 + 0.0822x + 25.754$	0.76
Young's	Age	Children	$Y = 66.924x - 17.57$	0.29
modulus		All ages combined	$Y = -2.6247x^2 + 108.91x – 177.89$	0.55
	Mass	Children	$Y = 16.213x + 75.04$	0.25
		All ages combined	$Y = -0.156x^2 + 23.888x - 13.025$	0.49
	Force	Children	$Y = -4E\text{-}05x^2 + 0.3478x + 87.561$	0.39
		All ages combined	$Y = -3E\text{-}05x^2 + 0.3324x + 96.937$	0.58

The coefficients of determination (R^2) for the relationship between Achilles tendon stiffness and age, body mass and peak force in children were 0.37, 0.58 and 0.51 respectively. These values increased to 0.68, 0.75 and 0.76 when adults were included in the analyses (Figure 4-7). The regression analysis indicated that body mass and peak force were predictors of tendon stiffness in children, explaining 66% of its variability. The inclusion of age did not greatly improve the predictability interpretation (age, p = 0.805). Similarly, body mass and peak force best predicted stiffness for all ages combined, explaining 78% of the variability in Achilles tendon stiffness. The coefficients of determination (R^2) for the relationship between Young's modulus and age, body mass and peak force in children were 0.33, 0.28 and 0.42 respectively, increasing to 0.57, 0.52 and 0.61 for all ages combined (Figure 4-8). Peak force was the sole variable able to significantly predict Young's modulus in children (43%) and all ages combined (61%). Predictive equations for tendon stiffness and Young's modulus for both populations are given in Table 4-3.

Table 4-3. Regression equation constants for predicting Achilles tendon stiffness and Young's modulus from mass and force. Constants presented for a variable should be multiplied by the variable associated with it in the form of y = a + bx + cx2. Coefficients of determination (adjusted R^2) are also represented.

		Mass^2	Mass	Force^2	Force	Constant	R^2 value
Stiffness	Children	-	4.145	-	0.032	-28.423	0.66
	All ages combined	-0.036	5.026	-3.921×10^{-6}	0.047	-37.893	0.78
Young's	Children	-	-	-	0.224	169.471	0.43
Modulus	All ages combined	-	-	-3.485×10^{-5}	0.332	96.937	0.61

The mean (± standard deviation) for tendon CSA, tendon length, peak stress, strain, elongation and force, moment arm length, tendon stiffness and Young's modulus are presented descriptively by age group in Table 4-4. A representative graph of the force-elongation relationship for a subgroup of $CG_{9\text{-}10}$ children and a subgroup of adults - from which the tendon's stiffness can be obtained- is presented in Figure 4.6. Large positive effect sizes were calculated between $CG_{5\text{-}6}$ and $CG_{7\text{-}8}$, and small to medium positive effect sizes calculated between $CG_{7\text{-}8}$ and $CG_{9\text{-}10}$for all variables except peak elongation and peak strain. Large positive effect sizes were observed between children and adults for all bar one variable examined; peak strain did not differ with increasing developmental age to adulthood.

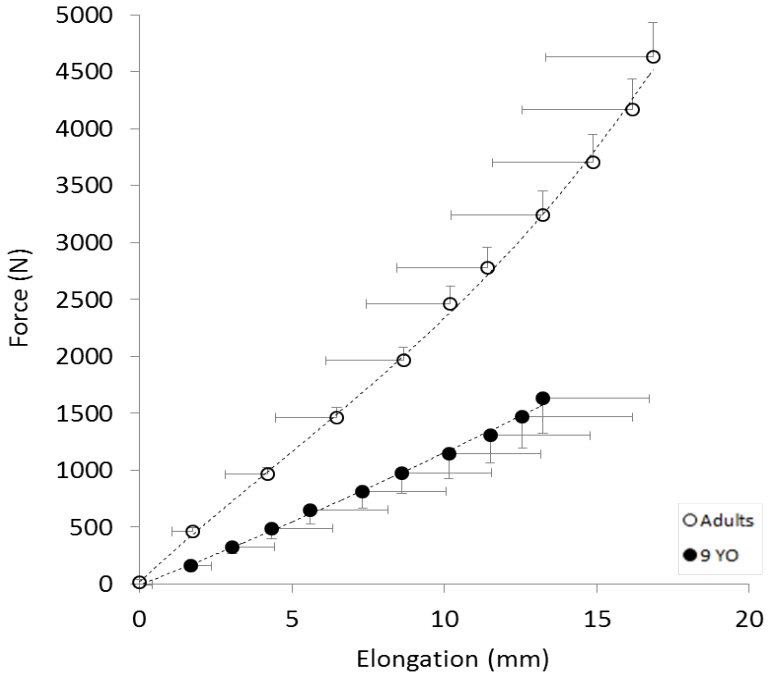


Figure 4-6. Mean force-elongation curve for five adults and five $CG_{9\text{-}10}$ children. Error bars are included *to give an indication of the range within an age group and represent the standard* deviation of the mean.

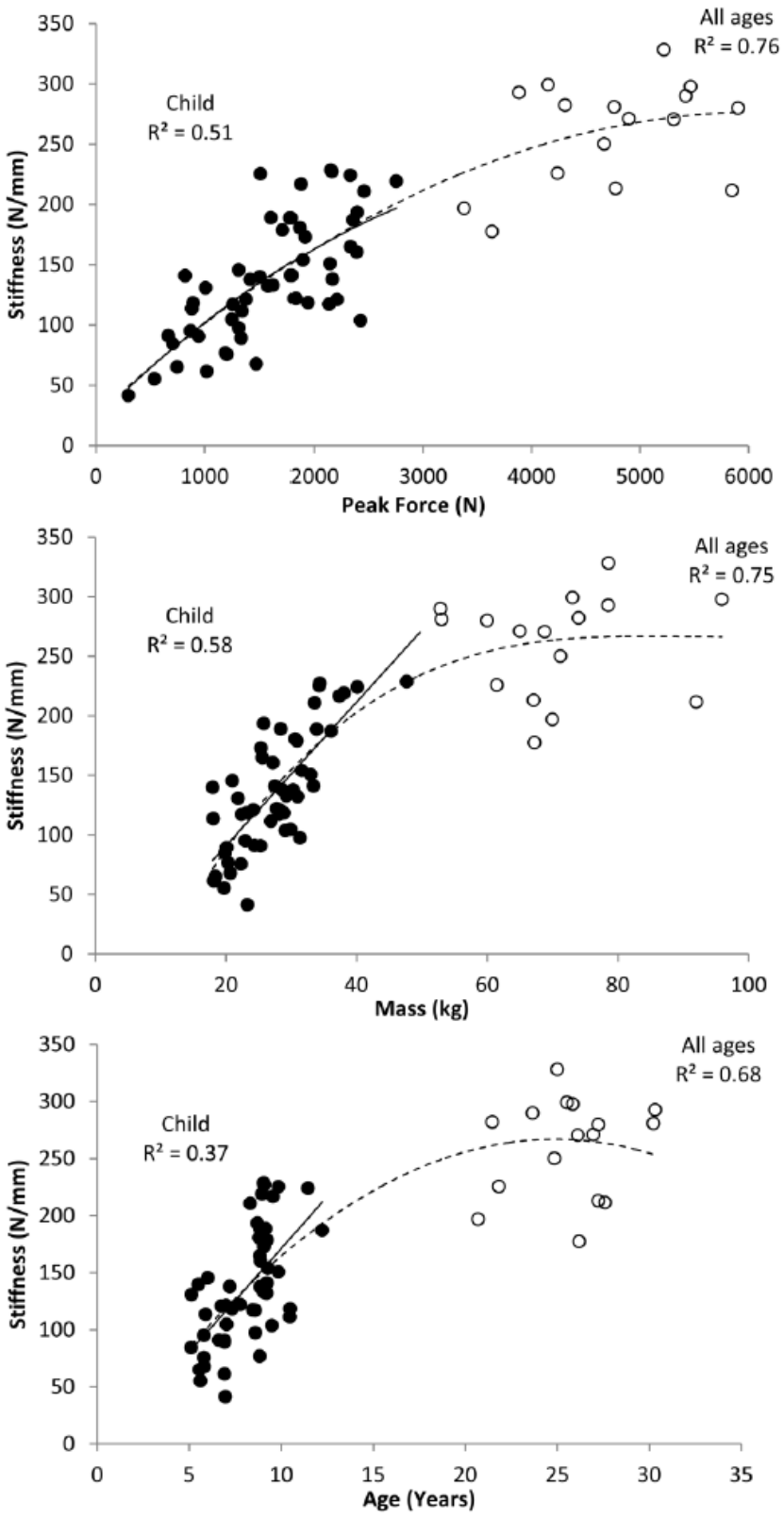


Figure 4-7. Relationship between Achilles tendon stiffness and peak force body mass and age for children (filled circles) and adults (open circles). Lines of best fit are for children (solid line) and all ages combined (dashed line).

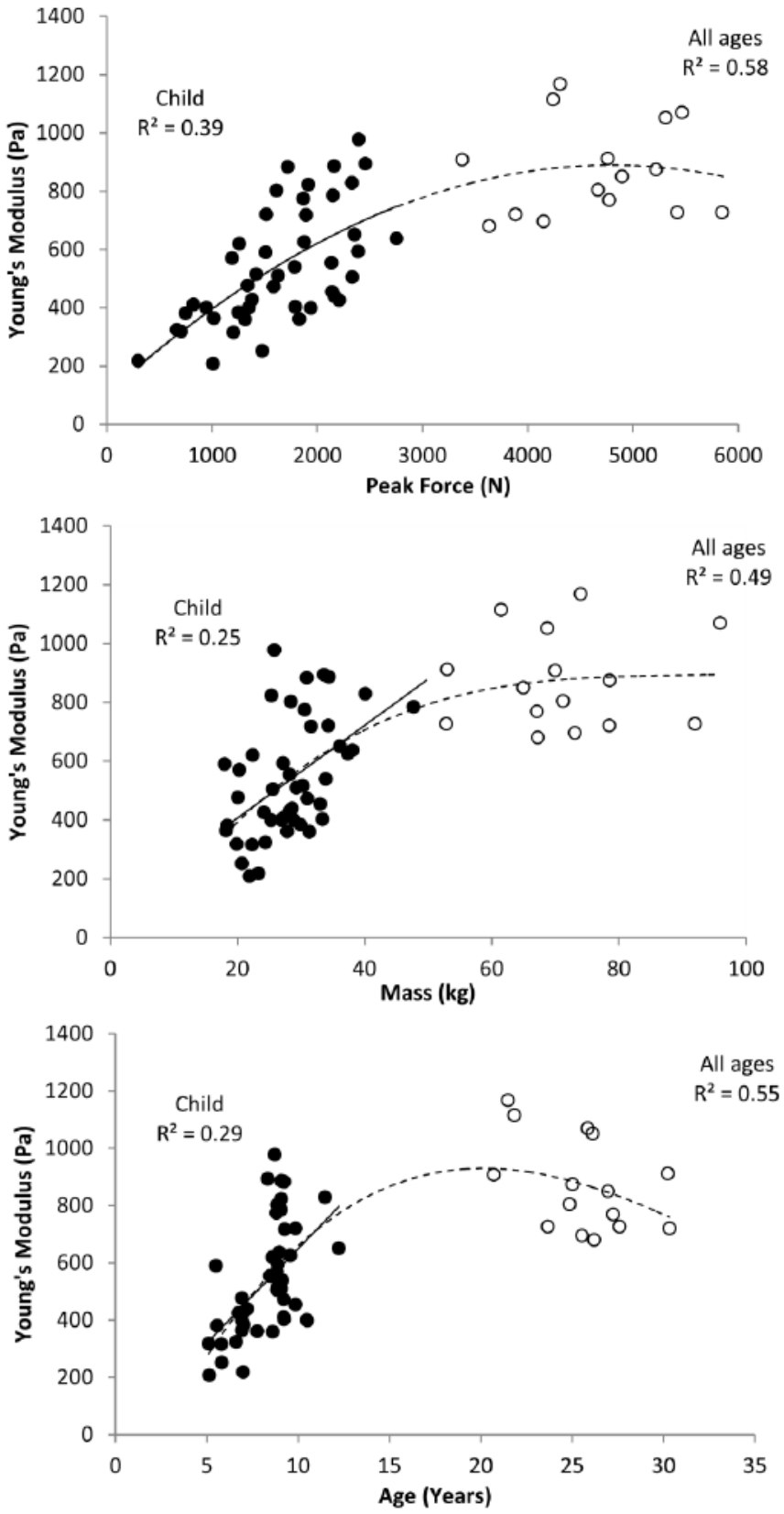


Figure 4-8. Relationship between Achilles tendon Young's modulus and peak force, mass and age for children (filled circles) and adults (open circles). Lines of best for are for children (solid line) and all ages combines (dashed line).

Table 4-4. Descriptive characteristics of the variables associated with calculating the mechanical properties of the Achilles tendon. Effects size (shown in italics)represents the difference between groups in SD units.

Variable	CG_{5-6}	CG_{7-8}	CG_{9-10}	Adults
Tendon CSA (mm^2)	29.5 ± 6.5	39.1 ± 7.0	41.6 ± 6.3	58.9 ± 11.1
	1.42	*0.38*	*2.21*	
		3.59		
Tendon Length (mm)	118.4 ± 23.4	147.3 ± 23.0	149.9 ± 26.1	192.5 ± 28.5
	1.25	*0.11*	*1.53*	
		2.90		
Peak Elongation (mm)	10.7 ± 3.8	12.0 ± 3.6	11.9 ± 3.7	17.4 ± 2.9
	0.04	*-0.03*	*1.54*	
		1.88		
MA_{AT} (mm)	25.3 ± 5.0	31.2 ± 5.5	33.5 ± 4.8	33.8 ± 3.9
	1.12	*0.45*	*0.12*	
		1.99		
Peak Force (N)	1046 ± 459	1743 ± 489	1840 ± 394	4742 ± 763
	1.47	*0.22*	*4.86*	
		5.87		
Peak Strain (%)	8.6 ± 1.9	8.4 ± 2.6	8.3 ± 3.1	9.2 ± 2.2
	-0.09	*-0.04*	*0.40*	
		0.36		
Peak Stress (N/mm^{-2})	34.6 ± 12.6	46.9 ± 11.7	44.5 ± 9.7	81.2 ± 17.0
	1.02	*-0.23*	*2.79*	
		3.20		
Stiffness (N/mm)	89.8 ± 33.4	135.2 ± 40.0	162.2 ± 40.5	259.2 ± 44.2
	1.24	*0.67*	*2.29*	
		4.25		
Young's Modulus (MPa)	342.1 ± 118.8	588.6 ± 189.1	633.2 ± 179.0	871.5 ± 162.4
	1.60	*0.24*	*1.25*	
		3.56		
Energy Storage (J/kN^{-1})	5.2 ± 1.9	5.9 ± 2.1	6.2 ± 2.1	8.1 ± 1.6
	0.34	*0.16*	*1.02*	
		1.64		

CSA, cross-sectional area; MA_{AT}, Achilles tendon moment arm; MVC, maximum voluntary contraction

4.4 Discussion

The primary aim of this study was to assess the contributions of age, body mass and muscular strength on the development of Achilles tendon stiffness. As a major weight-bearing tendon, it was not surprising to find that stiffness of the Achilles tendon was well correlated with body mass in children, based on the knowledge that mass increases with age. The relationship found between tendon stiffness and mass is consistent with previous reports in animals (Pollock and Shadwick, 1994). Increasing body mass would have provided a progressive increase in tendon loading during weight-bearing tasks such as walking, running and stair climbing (Ishikawa *et al*., 2007; Lichtwark *et al*., 2007; Spanjaard *et al*., 2008). Achilles tendon stiffness has also been shown to correlate with muscular strength in adults (Scott and Loeb, 1995; Muraoka *et al.*, 2005), and thus an increase in stiffness with age was also hypothesised to reflect increases in muscular force capacity with age. We found a moderate correlation between peak force and tendon stiffness in children, irrespective of body mass. The relationship between peak force and tendon stiffness has been postulated to reflect an injury prevention mechanism, whereby excessive tendon elongation caused by an increase in muscular loading prompts adaptation of the tendon to avoid strain and rupture (Scott and Loeb, 1995; Muraoka *et al*., 2005). Finally, we found tendon stiffness to increase with age, however this effect could largely be explained by the age-related increases in body mass and force producing ability and thus was not considered a true 'ageing' effect.

4.4.1 Age-Related Increases in Tendon Stiffness

The regression analysis demonstrated that 58% of tendon stiffness could be predicted by body mass. This proportion increased to 66% when peak force was added to the model.

From these results we might speculate that the additional 8% of variability in tendon stiffness that can be accounted for when including peak strength as a separate variable might represent the neuromuscular aspect of muscular strength improvements with age. It has been shown previously that muscular strength increases at a rate that surpasses increases in muscle mass with growth (Asmussen and Helboll-Nielsen, 1955), a phenomenon that has been subsequently explained in terms of neuro-motor maturation, rather than the muscle's microstructure (Asmussen, 1973; Blimkie, 1989; Falk *et al*., 2009). Nevertheless, based on these results, we conclude that body mass and peak strength have separate and additive effects on tendon stiffness in developing children.

4.4.2 Age-Related Increases in Young's Modulus

The regression analysis also demonstrated that 43% of Young's modulus could be predicted from peak force in children. Interestingly, the inclusion of body mass did not improve Young's modulus predictability. These results indicate that the ability to generate larger muscular forces with age provides the mechanical stimulus required for improving the structural integrity of tendon material. As a consequence of growth, greater muscular forces are required to propel segments of increasing mass with growth (Jensen and Bothner, 1998), therefore greater chronic mechanical loading is experienced during activities such as locomotion to further stimulate structural and biochemical changes in the tendon (Elliott, 1965; Curwin *et al*., 1988; Michna and Hartmann, 1989; Kjaer, 2004; Bayer *et al*., 2010).

4.4.3 Maturation or Growth?

In an attempt to differentiate between the effects of maturation and dimensional growth on changes in Achilles tendon stiffness with age, the tendon's length, CSA and Young's modulus

were determined. In the present study, both tendon length and CSA were shown to increase by ~60% and ~105% respectively between $CG_{5\text{-}6}$and adults. A greater increase in CSA than tendon length with development is evidence of a dimensional bias underpinning the age-related increase in tendon stiffness. It can therefore be assumed that tendon hypertrophy is a major adaptation influencing tendon stiffness and possible consequence of chronic loading with age. Nonetheless, Young's modulus was also found to increase (~139%) with age, which is suggestive of structural changes playing a central role in age-related increases in tendon stiffness. Increases in fibril diameter, fibril density and the relative proportions of small-to-large fibrils have been previously shown to accompany tendon maturation in mammals (Bailey and Robins, 1976; Parry *et al.*, 1978a; 1978; Bailey *et al.*, 1998). These modifications improve the tensile strength of the tendon by means of greater intrafibrillar crosslinking (Parry *et al.*, 1978a; Svendsen and Thomson, 1984) and thus can increase its stiffness without dimensional changes. In addition, mechanical loading stimulates further structural and biochemical changes in the tendon (Elliott, 1965; Curwin *et al*., 1988; Michna and Hartmann, 1989; Kjaer, 2004; Bayer *et al*., 2010). Based on the significant correlation between Young's modulus and peak force, loading in the form of an increase in force producing ability with development appears to be a potent stimulus for improving the structural integrity of tendon material. From these results, it can be concluded that both maturational and dimensional adaptations occur with age to increase Achilles tendon stiffness. Moreover, strength development with age appears to drive these adaptive mechanisms.

4.4.4 Changes in Tendon Characteristics as a Function of Age

Lastly, the parameters underlying the mechanics of the tendon were descriptively documented in order to gain an insight into their importance and role during different stages of the prepubertal period. Peak elongation was found to increase in proportion with resting tendon length with age, resulting in a consistent peak strain across all age groups. This result contrasts the findings of Kubo *et al.* (2001) who found peak elongation to be similar between boys and men in the VL tendon (although they did not report tendon strain (%) values). This discrepancy could be attributed to differences in the properties or growth rates of the Achilles compared to the patellar tendon, or between age groups. However, an important functional consequence of the greater maximum tendon elongation (~17 mm in adults vs. ~11 mm in children in the present study) and stiffness found in adults is an enhanced capacity for energy absorption, normalised to peak force (~5.2 J/kN^{-1} in $CG_{5\text{-}6}$vs. ~8.1 J/kN^{-1} in adults, calculated from the results), resulting in superior movement efficiency. In fact, this finding may partly explain the difference in movement efficiency between children and adults during activities involving the stretch shortening cycle (DeJaeger *et al*., 2001; Schepens *et al*., 2001; Schepens *et al*., 2004).

Peak stress increased with age as a result of strength gains exceeding those of tendon hypertrophy (~350% vs. ~105%). This might suggest that children are more protected from stress-related Achilles tendon injuries than adults; certainly the prevalence of such injuries is much higher in the adult population (Houshian *et al*., 1998). However, because their Young's modulus is lower, the ultimate tensile strength (i.e. the point at which the tendon would fail) will also be lower (Almeida-Silveira *et al*., 2000). Children's Achilles tendons may therefore operate very close to their physiological limits and with little safety margin,

although the risk of tendon rupture has been linked more so to age- and exercise-related microdamage accumulation over time (Kannus and Jozsa, 1991; Patterson-Kane *et al.*, 1997b; Smith *et al.*, 1999; Young *et al.*, 2009). On a separate note, the lack of sex differences found between adults in the current study probably reflects the closer matching of body mass and maximum strength between the sexes compared to previous studies (Kubo *et al*., 2003; Onambele *et al*., 2007).

The ranges of the Achilles tendon stiffness found in the present study were marginally lower than those previously reported for adults (Magnusson *et al.*, 2001; Rosager *et al.*, 2002; Muraoka *et al.*, 2004a). This is most likely due to the different peak force ranges used to determine stiffness between studies (e.g. 10 – 90% MVC in the present study vs. 90 – 100% used by Magnusson *et al*., 2001 and Rosager *et al*., 2002). The use of a wider range of forces in the present study would mean more of the less steep (early) part of the curvilinear force-elongation curve is included in determining stiffness (Baratta and Solomonow, 1991), and thus contribute to a smaller stiffness. This effect would have been enhanced differences in strength between children and adults been accounted for by calculating stiffness over a force range common to every participant, dictated by the weakest participant (O'Brien *et al*., 2010). In the present study, 10 – 90% of the force achieved by the weakest individual would have equated to 0.5 – 4.5% of the maximum force attained by the strongest individual and was thus deemed an inappropriate method of normalising stiffness.

The instructions given for plantarflexion moment production were also different for children and adults based on the difficulty children experienced in performing ramped contractions. This resulted in a marginally faster production of joint moment in children than adults which

may have influenced the mechanical properties of the tendon. Tendons display time-dependent viscoelastic properties, which can influence their mechanical properties over the duration of a contraction. For example, a greater force-relaxation, or tendon 'creep' was detected during long (~10 s) ramped contractions than for shorter (~3 s) ramped contractions, resulting in a lesser calculated tendon stiffness from the slower contraction (Pearson *et al*., 2007).To best knowledge, the effects of a ~3 s contraction on tendon elongation have not been compared to contractions performed with a maximum rate of force development (i.e. adults vs. children). However, as the time to peak force during a maximal voluntary contraction is markedly longer in children than in adults (Asai and Aoki, 1996; Falk *et al*., 2009), the resulting rate at which force was developed here was not very dissimilar between children and adults, thus the different instructions given to children and adults for MVC production are not believed to have had a significant influence on the results.

Tendon stiffness has been identified as a major factor influencing muscular force production and transmission characteristics in adults (Bojsen-Moller *et al*., 2005; Muraoka *et al*., 2005). The mechanical properties of the tendon should therefore have the same potential to influence movement performance in children. Indeed, the time lag between muscle activation and muscle force production (i.e., electromechanical delay) is greater - and the maximum rate of force development slower - in children than in adults (Asai and Aoki, 1996; Grosset *et al*., 2005), and have previously been shown to be correlated with tendon stiffness in adults (Muraoka *et al.*, 2005). The significance of tendon stiffness on these characteristics of force production has yet to be established in children and thus warrants further investigation.

4.4.5 Conclusion

This is the first study to document changes in the mechanical properties of the Achilles tendon as a function of age in prepubertal children, and the first to describe tendon properties in children younger than 9 years. Achilles tendon stiffness increased relatively linearly throughout childhood as a result of linear increases in Young's modulus and tendon hypertrophy with development. In fact, body mass, in conjunction with peak force producing ability, were suggested as the primary influences underlying both the dimensional and maturation aspects of tendon stiffness. Peak stress increased from childhood to adulthood due to greater increases in strength than tendon hypertrophy with age whilst peak strain remained constant as a result of parallel increases in tendon length and peak elongation. The differences identified in Achilles tendon properties between adults and children are most likely due to chronic increases in tendon loading with development. Determining the impact of changing tendon properties on movement performance is an important area for future research.

CHAPTER 5: The Influence of Tendon Stiffness and Muscle Activation Rate on Muscle Force Production in Children and Adults

5.0 Abstract

Differences in force production capacity between children and adults are often attributed to neuromuscular immaturity. However, tendon stiffness, which influences both electro-mechanical delay (EMD) and the contractile rate of force development (RFD) in adults, is less in children and thus has the potential to influence their muscular force production characteristics. The purpose of this study was to determine the relative neural and mechanical contributions to age-related changes in muscular force production by examining the effects of both tendon stiffness and rate of muscle activation on EMD and contractile RFD. Achilles tendon stiffness, EMD (time between the onset of muscle activity and moment), RFD (slope of the force-time relation) and rate of EMG rise (RER; slope of the EMG-time relation) were determined for plantarflexion contractions performed with a maximum RFD in forty-seven prepubertal children (age = 8.3 ± 1.6 yrs) and nineteen adults (age = 25.7 ± 2.8 yrs). Age-related differences in tendon stiffness, EMD, RFD and RER were examined between children, grouped according to chronological age (5 – 6, 7 – 8 and 9 – 10 years), and adults. Relationships were determined between 1) tendon stiffness and EMD, 2) tendon stiffness and RFD, and 3) RER and RFD. Furthermore, regression analyses were used to determine the relative influence of tendon stiffness, RER and age on RFD. Achilles tendon stiffness increased significantly (~300%) from age group 5 – 6 through to adulthood. EMD decreased significantly with increasing age and was negatively correlated with tendon stiffness in children, adults and for all ages combined (r = -0.66, -0.74 and -0.82 respectively, $p < 0.05$). RFD calculated to 50, 200 and 400 ms was significantly lower in children than adults and was related to tendon stiffness in children and all ages combined ($p< 0.05$). The relationship between normalised (to peak force) RFD and tendon stiffness was weaker in children, but was still significant across all age groups ($p< 0.05$). The regression analysis

indicated that both RER and tendon stiffness influenced RFD cumulatively accounted for 61% of the variance in RFD in children. These results demonstrate the importance of both neural and mechanical factors for rapid force development in prepubertal children. The longer EMD and slower RFD found in children indicate a less effective development and transfer of muscular forces. These findings significantly add to our understanding of the mechanisms underpinning age-related improvements in muscular force production and may have further implications with respect to the interpretation of age-related differences in complex movement performance.

5.1 Introduction

Children often adopt different inter-muscular coordination patterns than adults when performing complex motor tasks(Shiavi *et al.*, 1987; Sutherland, 1988; Frost *et al.*, 1997; Chao *et al.*, 2002; Korff *et al.*, 2009a; Lazaridis *et al.*, 2010). Age-related differences in inter-muscular coordination are often attributed to an 'immature' nervous system in children (McGraw, 1943; Forssberg, 1985). While maturation of the nervous system is undoubtedly a major factor contributing to age-related differences in inter-muscular coordination and mature movement kinematics(Sutherland *et al.*, 1980a; Forssberg, 1985; Shumway-Cook and Woollacott, 1985; Forssberg, 1999; Sundermier *et al.*, 2001; Chao *et al.*, 2002), researchers have recently started to consider the possibility that changes in non-neuromotor factors during childhood, such as changing anthropometry (Brown and Jensen, 2006; Korff and Jensen, 2008) and mechanical properties of the musculo-skeletal system (Wang *et al.*, 2004; Korff *et al.*, 2009a), may influence movement production during development.

The mechanical properties of the muscle-tendon complex are important factors influencing muscular force production and movement performance in adults, (Reeves *et al.*, 2003a; Bojsen-Moller *et al.*, 2005; Kongsgaard *et al.*, 2007). Within this context, tendons play an integral role in movement by transferring muscular forces across joints to the bones, the rate of which is strongly influenced by tendon stiffness (Fukunaga *et al.*, 1996; Muraoka *et al.*, 2004b; Bojsen-Moller *et al.*, 2005). Tendon stiffness influences the ability to generate explosive forces by affecting both the time lag between muscle activation and muscle force production (termed electromechanical delay; EMD), and the maximum rate of force development (RFD). These factors are regulated by the time taken to stretch the tendon by

an actively shortening muscle (Cavanagh and Komi, 1979; Muraoka *et al.*, 2004b). Indeed, significant relationships between muscle-tendon stiffness and both RFD (positive relationship, Reeves *et al.*, 2003a; Bojsen-Moller *et al.*, 2005) and EMD (negative relationship, Cavanagh and Komi, 1979; Muraoka *et al.*, 2004b; Grosset *et al.*, 2009)have been previously shown in adults. EMD and RFD are therefore important descriptors of rapid force development performance.

It is well documented that EMD is longer, and RFD lower in children than in adults (Asai and Aoki, 1996; Grosset *et al*., 2005; Falk *et al*., 2009; Grosset *et al*., 2009). Tendon stiffness is also lower in children (Kubo *et al.*, 2001b; O'Brien *et al.*, 2010), and in Chapter 4 was shown to vary with age-related parameters such as body mass and peak force capacity. Given the relationships identified between tendon stiffness and force production characteristics in adults, it can be hypothesised that previously observed differences in the rate of force transfer between children and adults (Asai and Aoki, 1996; Grosset *et al*., 2005; Falk *et al*., 2009) are partly dependent upon tendon stiffness. In addition to the influence of tendon stiffness, RFD depends on the rate of muscle activation (Komi, 1986; Corcos *et al.*, 1989; Nelson, 1996; Aagaard *et al.*, 2002). Neuromuscular capacity is important in the context of force development in children (Ramsay *et al*., 1990; Paasuke *et al*., 2000; Halin *et al*., 2003; Grosset *et al*., 2008) and a lower RFD found in children has been partly attributed to a lower rate of muscle activation (Falk *et al*., 2009).In an effort to explain the differences observed in movement performance between children and adults (Asai and Aoki, 1996; Chao *et al.*, 2002; Ganley and Powers, 2005; Grosset *et al.*, 2005; Korff and Jensen, 2007; Falk *et al.*, 2009), the overall purpose of this study was to tease out neural and mechanical contributions to developmental changes in force production capacity.

The specific aims of this study were to document age-related changes in Achilles tendon stiffness and EMD and RFD for the gastrocnemius medialis (GM),and describe how these variables are interrelated in typically developing prepubescent children and adults. Here, the hypothesis that EMD and RFD are related to tendon stiffness was tested. Additionally, the relative contributions of tendon stiffness and rate of muscle activation (as non-neuromuscular and neuromuscular factors) on plantarflexor RFD in children compared to adults was determined. It was hypothesised that the rate of muscle activation would have a separate and additive effect to tendon stiffness on RFD in children. By examining the hypotheses set out here, the present study has the potential to shed light on the possible mechanisms that underpin previously observed age-related differences in inter-muscular coordination during complex motor tasks.

5.2 Methods

5.2.1 Ethics and Participant Information

Forty-seven prepubertal children between the ages of 5 and 12 years (25 boys, 24 girls; mean age 8.3 ± 1.6 yrs), 10 men (27.0 ± 1.9 yrs) and 9 women (25.3 ± 3.4 yrs) volunteered to participate in this study. Prepubertal children were chosen in order to control for musculoskeletal changes that may occur as a result of increased circulating androgens with the onset of puberty (Blimkie, 1989). Prepubertal status was determined for children over 8 years of age in terms of offset from peak height velocity (Mirwald *et al*., 2002). All children within this age subgroup were determined as prepubertal. The minimum age from peak height velocity was calculated to be -1.9 years. All participants were free from known neuromuscular and musculoskeletal disorders and did not partake in competitive sports. Children provided written assent and parents/guardians provided written consent to allow their child's participation in the study. All study procedures were explained in a child-appropriate manner, and the children were made aware of their right to withdraw from the study at any time without penalty prior to commencing the study. The study was approved by the Human Research Ethics Committee at Brunel University and conformed to the guidelines of the Declaration of Helsinki.

5.2.2 Overview

The dynamometer was adjusted to each participant's anthropometry and their skin prepared for electromyography (EMG) prior to data collection as per the methods described in Chapter 4 (pages 85-87). Tendon stiffness was calculated as per the methods described in Chapter 4 (page 88). Briefly, plantarflexor moment was obtained from dynamometry during maximal voluntary isometric contractions (MVC). Antagonist coactivity was accounted for by

quantifying the EMG-moment relationship for the TA muscle and adding the associated moment to the plantarflexor moment. Plantarflexor muscle force was obtained by dividing the coactivation-adjusted plantarflexor moment by the Achilles tendon moment arm (MA_{AT}). MA_{AT} was obtained by measuring the tendon's excursion using ultrasonography as the ankle was passively rotated through a range of motion, and calculated as per the recommendations of Fath *et al.* (2010). Elongation of the tendon resulting from muscular loading was measured using ultrasound imaging of the GM MTJ during a maximum voluntary contraction (MVC). The measured displacement of the GM MTJ was then corrected for distal tendon movement (quantified by motion analysis), resulting from small heel movements during the MVC. Tendon stiffness was calculated as the slope of the plantarflexor force-tendon elongation relationship between 10 – 90% of the maximum plantarflexor force. EMD, RFD and RER were measured during three MVC trials performed with a maximum RFD. Relationships between age, tendon stiffness, EMD, RFD and RER were then determined.

5.2.3 Measurement of Electromechanical Delay

Electromechanical delay (EMD) in the GM muscle was calculated as the time lag between the onset of GM EMG activity and onset of plantarflexor moment (Figure 5-1). EMD of the GM was assumed to be most influenced by Achilles tendon stiffness because of the substantially greater tendon length (as opposed to the soleus), through which forces are transferred, and the fact that tendon stiffness was calculated from movement of the GM MTJ (encompassing a long tendon length). Despite the soleus muscle, as the largest contributor of plantarflexor force, having a shorter EMD than the GM (Komi *et al*., 1987), the GM contributes substantially to plantarflexor force production at a fully extended knee

angle (Cresswell *et al*., 1995). GM EMD is therefore of substantial functional importance. According to Muraoka *et al.* (2004), tendon slack, which influences EMD, is fully taken up at a $+10^{o}$ (plantarflexion) ankle angle and fully extended knee. They demonstrated that the difference in EMD at this position compared to more dorsiflexed ankle positions was not significantly different, therefore the use of a neutral ankle angle (foot at 90^{o} to the tibia) here would have minimised the influence of tendon slack on EMD (Muraoka *et al*., 2004). MVCs were performed with the instruction to rotate the foot away "as hard and as fast as possible". Participants were reminded not to dorsiflex the foot immediately prior to plantarflexion as this masked the onset of plantarflexor moment. Trials displaying decreases in moment immediately prior to plantarflexion at the time of collection were discarded and re-performed. Furthermore, decreases in moment greater than 0.5 Nm from baseline, as identified during data analysis, were also discarded. The procedures relating to both the skin preparation for EMG, and GM EMG recording were identical to those presented for the TA muscle in Chapter 4 (pages 85-87). GM EMG signals were digitally filtered using a 10-500 Hz band pass filter (Spike2 v5.12a software, Cambridge Electronic Design, UK). Mean (± SD) baseline GM EMG activity and moment were calculated at rest over a 200 ms window and the threshold for onset of both signals was set at ±2 SD from the baseline mean (Linford *et al*., 2006) with the signal remaining above the threshold for a minimum of 10 ms (De Luca, 1997). The mean EMD of the three MVCs for each individual was used for statistical analyses.

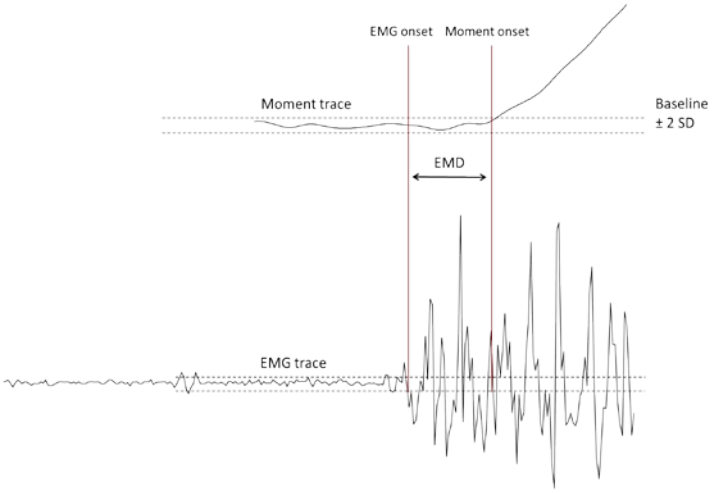


Figure 5-1. Example electromyogram and moment traces used to calculate electromechanical delay (EMD).

5.2.4 Measurement of Rate of Force Development (RFD)

RFD was calculated using a custom written program (Matlab v7.14, MathWorks, Cambridge, UK) with data obtained from three MVCs. Muscle force was calculated as the ratio of joint moment to moment arm length, and RFD to 50, 200 and 400 ms was calculated as the change in force (from force onset) divided by the time interval (Δforce/Δtime). The calculation of RFD to different time intervals is of functional importance as the capacity of a muscle to develop forces within a minimal time frame may be an important determinant of successful movement. In addition, RFD measured to these intervals provides an insight into the underlying physiological mechanisms that influence the RFD at different stages of a muscular contraction (Andersen *et al*., 2005; Andersen and Aagaard, 2006). Peak strength is known to influence the slope of the force-time curve (Hakkinen *et al.*, 1985b; Aagaard *et al.*, 2002), thus normalising RFD to peak force removes peak muscle strength as a factor influencing RFD and allows force production rate to be directly compared between

individuals (Blazevich *et al*., 2008). Normalised RFD (RFD_{norm}) was calculated to 30%, 50% and 70% of each individual's peak force (subsequently referred to as $RFD_{30\%}$, $RFD_{50\%}$ and $RFD_{70\%}$) to approximate early, mid- and late force development respectively (Aagaard *et al*., 2002). In addition, RFD to 90% ($RFD_{90\%}$) of peak force was calculated for the purpose of testing the hypothesis that RFD would best correlate with tendon stiffness when measured using the same force intervals. RFD and RFD_{norm} are reported in $kN \cdot s^{-1}$ and $\%MVC \cdot s^{-1}$, respectively.

5.2.5 Measurement of Rate of Rise of EMG Amplitude (RER)

Absolute RER was calculated from GM EMG onset to 25, 75 and 150 ms. Normalised RER (RER_{norm}) was calculated to 30%, 50% and 70% of peak EMG amplitude (subsequently referred to as $RER_{30\%}$, $RER_{50\%}$ and $RER_{70\%}$) to represent early, mid- and late EMG rise respectively. Therefore RER is reported in $\%peak\ EMG \cdot s^{-1}$. For the determination of RER, the root-mean-square of the band-pass filtered GM EMG data was calculated over a 50 ms time window (Aagaard *et al*., 2002; Blazevich *et al*., 2008).

5.2.6 Statistical Analysis

All data were analysed using SPSS statistical software (v16.0, SPSS Inc, Chicago, USA). Children were grouped according to age and are subsequently referred to as CG_{5-6},CG_{7-8} and CG_{9-10} (ages 5 – 6, 7 – 8 and 9 – 10 years, respectively). Table 5-1 provides a description of each group's statistics. A multivariate analysis of variance (MANOVA) was used to test all dependent variables (stiffness, EMD, RFD and RER) for a main effect of age. In case of significance, a one-way ANOVA was performed for each dependent variable, and Tukey-Kramer post-hoc tests were performed to identify the location of the significant differences

between age-groups (as group sizes were unequal). Statistical significance was accepted at $p < 0.05$.

Table 5-1. Descriptive statistics for participant cohorts (mean ± SD).

Group	N	Age (years)	Height (m)	Mass (kg)
CG_{5-6}	13	6.2 ± 0.6	1.20 ± 0.04	21.3 ± 2.5
CG_{7-8}	17	8.4 ± 0.6	1.35 ± 0.05	29.5 ± 6.7
CG_{9-10}	15	9.6 ± 0.7	1.39 ± 0.06	32.7 ± 5.6
Adult Men	10	26.8 ± 1.8	1.80 ± 0.06	78.6 ± 11.7
Adult Women	9	24.8 ± 3.2	1.68 ± 0.06	64.3 ± 7.8

For determining the relationship between (1) tendon stiffness and EMD, (2) tendon stiffness and RFD, and (3) RER and RFD, a polynomial was fitted to the data for children (all ages), adults and all ages combined (i.e. children and adults). Furthermore, the coefficients of determination (R^2) were derived to quantify the strength of these relationships. The order of the polynomial chosen to best represent the relationship between each pair of variables was based on the criterion that adding an additional term did not improve the R^2 value by more than 0.02 (2%). The polynomials representing the relationship between tendon stiffness and RFD for children, adults and all ages combined can be found in Table 5-2.

Finally, separate stepwise multiple regression analyses were performed to determine the contributions of tendon stiffness and RER_{norm} on RFD_{norm} for children, adults and all ages combined. The significance level for inclusion of an independent variable into the model was set at $p < 0.05$. For variables fitted with a non-linear polynomial (using the criteria set

above), higher order terms of the polynomial were included as separate independent variables in the regression analysis to represent the non-linear aspect of the data and allow a linear regression to be performed. Age was also included in the regression as a separate independent variable, to assess whether age *per se* was a suitable predictor of RFD.

5.3 Results

The results of the MANOVA revealed a main effect of age on the dependent variables. Specific results from the follow-up ANOVAs and post hoc tests are presented in the section relevant to each dependent variable.

5.3.1 Achilles Tendon Stiffness

The ANOVA revealed a main effect of age on tendon stiffness ($F(3, 61) = 46.08$, $p < 0.05$). Achilles tendon stiffness was significantly lower for $CG_{5–6}$ (85.7 ± 29.8 N/mm)than for all other age groups. Furthermore, stiffness was significantly ($p< 0.05$) greater in adults (259.33 ± 41.9 N/mm) than for $CG_{7–8}$(146.1 ± 42.1 N/mm) and $CG_{9–10}$(165.4 ± 39.9 N/mm).

5.3.2 Electromechanical Delay (EMD)

The ANOVA revealed a main effect of age on EMD ($F(3, 61) = 22.22$, $p < 0.05$). EMD in $CG_{5–6}$ (96.2 ± 16.1 ms) was significantly longer ($p< 0.05$) than for $CG_{7–8}$ (80.9 ± 15.2 ms), $CG_{9–10}$ (74.6 ± 11.4 ms) and adults (48.6 ± 15.5 ms). No difference in EMD was observed between $CG_{7–8}$ and $CG_{9–10}$, but both groups had a significantly longer EMD than adults ($p< 0.05$).

According to the criteria set, the relationship between EMD and tendon stiffness was best approximated by a first order polynomial for all groups. EMD was strongly and negatively correlated with tendon stiffness in children, adults and for all ages combined (r = -0.66, -0.61 and -0.83 respectively; see Figure 5-2). Thus, EMD was found to decrease as tendon stiffness increased from childhood through to adulthood.

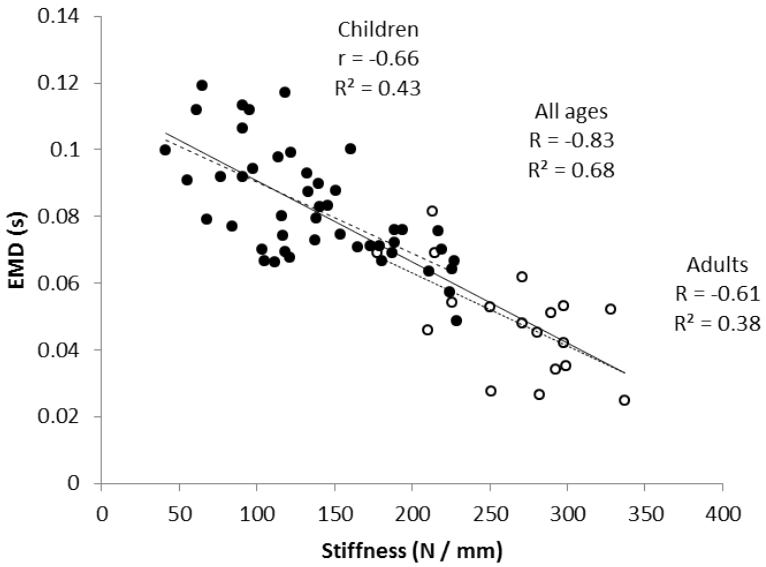


Figure 5-2. Relationship between Electromechanical delay (EMD) and Achilles tendon stiffness. Filled and open circles represent child and adult participants, respectively. Regression lines for children (dashed line), adults (dotted line) and all ages combined (solid line) are shown.

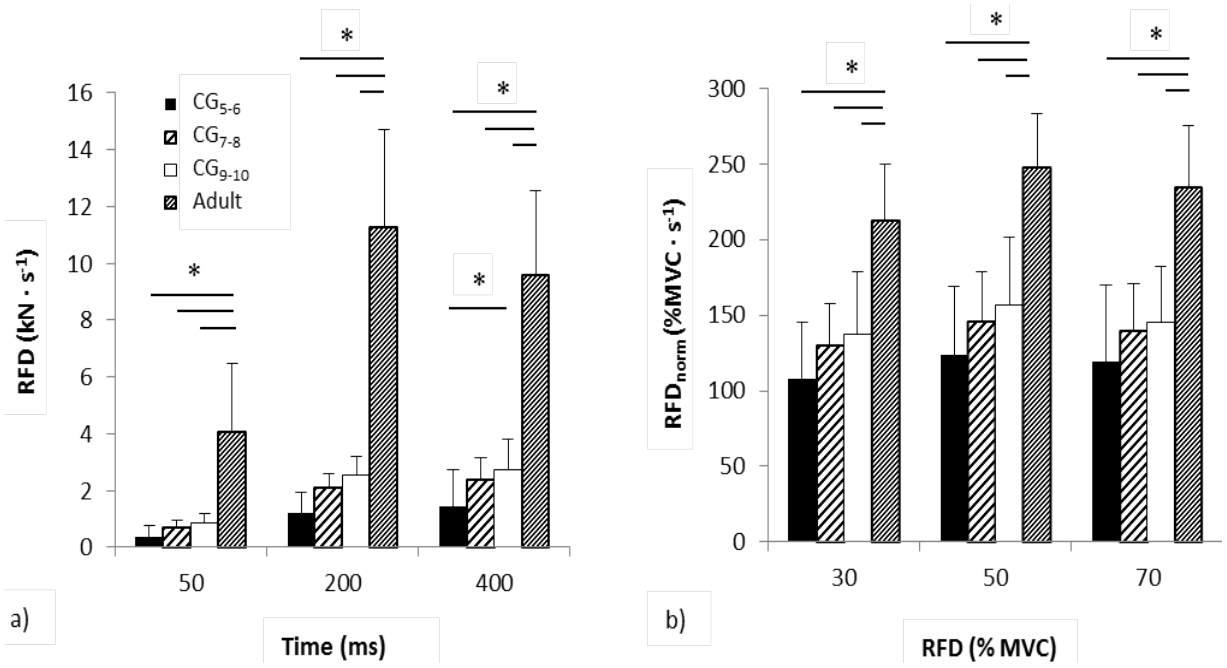


Figure 5-3. Differences in the rate of force development (RFD) from onset of force to a) 50, 200 and 400 ms and normalised RFD (RFDnorm) to b) 30%, 50% and 70% of peak force between age groups and adults. Results shown are for group means. Vertical bars represent standard deviations (SD). Asterisks indicate statistical significance between two groups ($p < 0.05$).

5.3.3 Rate of Force Development (RFD)

The ANOVA revealed a main effect of age on RFD ($F(3, 61) = 18.13 – 137.52$, $p < 0.05$). RFD (measured to 50, 200 and 400 ms) and RFD_{norm} ($RFD_{30\%}$, $RFD_{50\%}$ and $RFD_{70\%}$) were significantly lower in all children's groups than in adults ($p < 0.05$). $CG_{9\text{-}10}$ had a significantly greater RFD to 400 ms than $CG_{5\text{-}6}$. No other differences in RFD or RFD_{norm} were observed between age groups (Figure 5-3).

The relationship between RFD and Achilles tendon stiffness for children and adults was best described by a first order polynomial. A second order polynomial best approximated this relationship for all ages combined. The polynomial equations for each group can be found in Table 5-2. The coefficients of determination (R^2) quantifying the proportion of variance of RFD and RFD_{norm} explained by Achilles tendon stiffness were 0.34, 0.43 and 0.70, and 0.21, 0.29 and 0.56, respectively, for children, adults and all ages combined (Table 5-2). The relationship between stiffness and $RFD_{90\%}$ is shown graphically only (Figure 5-4) as the relationship was regarded as being too poor for a polynomial description.

5.3.4 Rate of EMG Rise (RER)

The ANOVA revealed a main effect of age on RER ($F(3, 61) = 6.63 – 49.10$, $p < 0.05$). RER to 25 ms was not significantly different between groups. RER was significantly greater in adults compared to $CG_{5\text{-}6}$ and $CG_{7\text{-}8}$ when calculated to 75 ms after EMG onset ($p < 0.05$) and also greater in adults than all child age groups when calculated to 150 ms ($p < 0.05$). $RER_{30\%}$ was significantly greater in adults than $CG_{5\text{-}6}$ and $CG_{7\text{-}8}$($p < 0.05$) and greater in adults than all child age groups for $RER_{50\%}$ and $RER_{70\%}$ ($p < 0.05$). $CG_{9\text{-}10}$ were significantly different from $CG_{7\text{-}}$

$_8$ for $RER_{30\%}$ (p< 0.05), but no other differences in RER or RER_{norm} were found between age groups (Figure 5-5).

Based on the outlined criteria, all relationships between RER and RFD were linear. The coefficients of determination quantifying the strength of the relationship between absolute RER and RFD for children, adults and all ages combined were 0.21, 0.10 and 0.50, respectively, whilst and those for RER_{norm} and RFD_{norm} were 0.33, 0.22 and 0.61 (Table 5-3).

Table 5-2. Equations describing the relationship between tendon stiffness and RFD to different time intervals and RFD normalised to peak force for children and combined ages.

Group	Interval	Equation	R^2 value
Children	RFD 50 ms	Y = 2.664 x stiffness + 287.424	0.12
	RFD 200 ms	Y = 8.807 x stiffness + 756.157	0.15
	RFD 400 ms	Y = 12.873 x stiffness + 441.912	0.34
	$RFD_{30\%}$	Y = 0.114 x stiffness + 109.530	0.03
	$RFD_{50\%}$	Y = 0.249 x stiffness + 107.620	0.10
	$RFD_{70\%}$	Y = 0.345 x stiffness + 86.884	0.21
Adults	RFD 50 ms	Y = 24.971 x stiffness – 2442.708	0.20
	RFD 200 ms	Y = 43.975 x stiffness – 597.033	0.43
	RFD 400 ms	Y = 22.933 x stiffness – 3264.286	0.37
	$RFD_{30\%}$	Y = 0.447 x stiffness + 95.834	0.29
	$RFD_{50\%}$	Y = 0.421 x stiffness + 137.904	0.28
	$RFD_{70\%}$	Y = 0.274 x stiffness + 157.831	0.10
All Ages	RFD 50 ms	Y = 0.127 x $stiffness^2$ – 28.053 x stiffness + 1952.616	0.61
	RFD 200 ms	Y = 0.229 x $stiffness^2$ – 36.171 x stiffness + 2666.007	0.69
	RFD 400 ms	Y = 0.125 x $stiffness^2$ – 6.960 x stiffness + 1014.115	0.70
	$RFD_{30\%}$	Y = 0.0021 x $stiffness^2$ – 0.295 x stiffness + 126.24	0.47
	$RFD_{50\%}$	Y = 0.0017 x $stiffness^2$ – 0.0084 x stiffness + 113.607	0.54
	$RFD_{70\%}$	Y = 0.601 x stiffness + 57.786	0.56

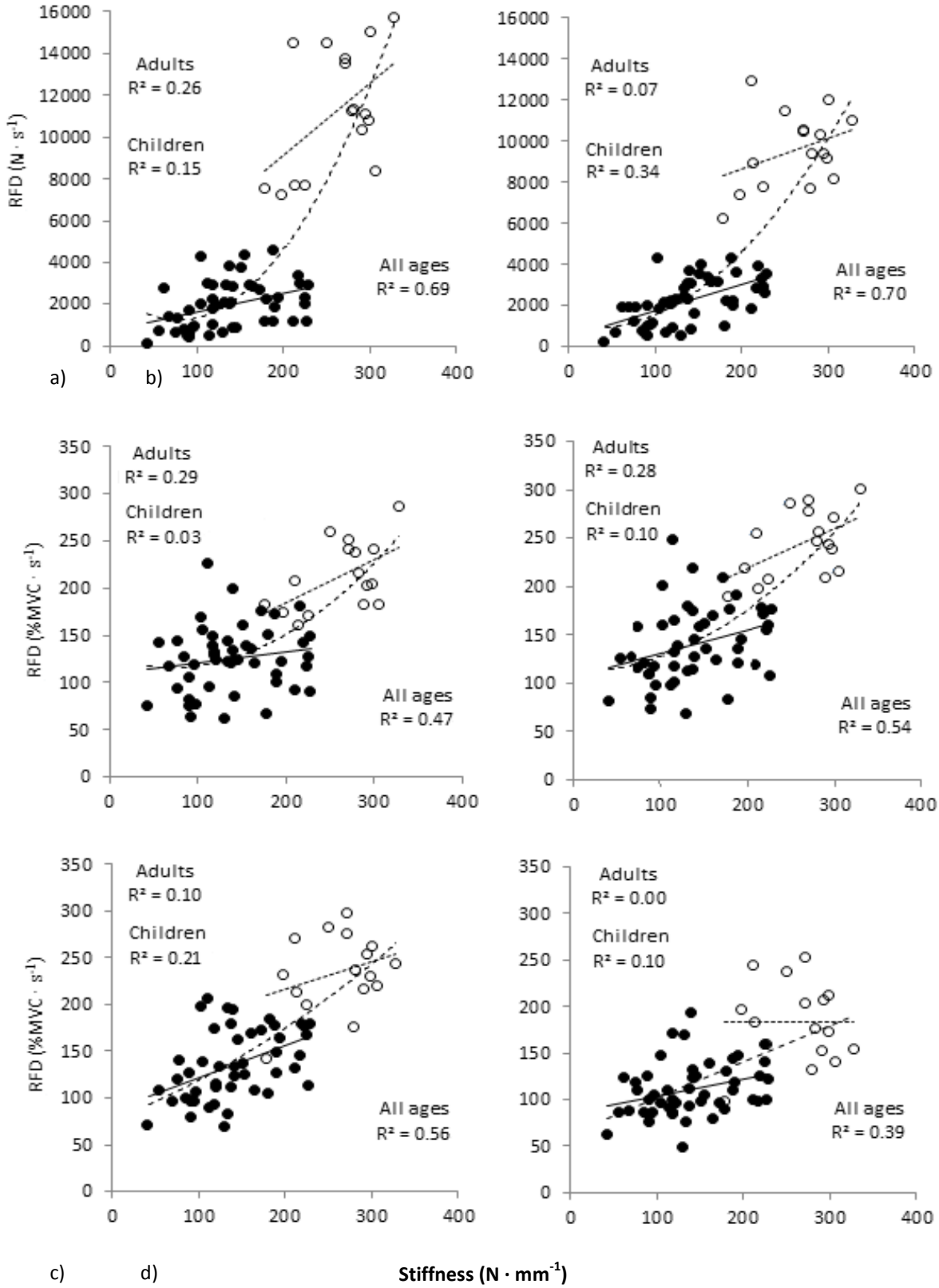


Figure 5-4. Relationship between Achilles tendon stiffness and rate of force development to a) 200 ms and b) 400 ms, and normalised rate of force development to c) 30%, d) 50%, e) 70% and f) 90% of peak force. Filled and open circles represent children and adults, respectively. Regression lines for children (solid line), adults (dotted line) and all ages combined (dashed line) are shown.

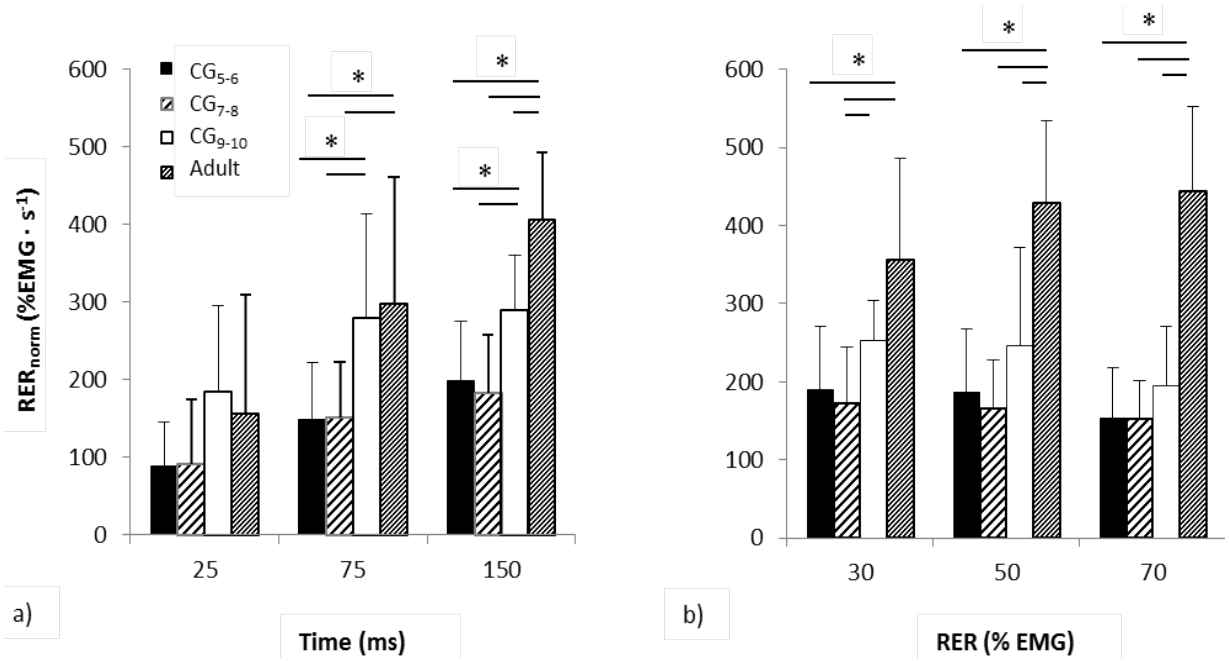


Figure 5-5. Differences in the normalised rate of EMG rise (RERnorm) from onset of EMG activity to a) 25, 75 and 150 ms, and to b) 30%, 50% and 70% of peak EMG amplitude between age groups and adults. Results shown are for group means. Vertical bars represent standard deviations (SD). Asterisks indicate statistical significance between two groups ($p < 0.05$).

Table 5-3. Coefficients of determination (R^2) for the relationships between RFD and RFDnorm and RER and RERnorm for children, adults and all ages combined. All relationships were best described using a linear polynomial according to the criteria outlined in the statistical analysis.

	RFD Children			RFD Adults			RFD All Ages		
	50 ms	200 ms	400 ms	50 ms	200 ms	400 ms	50 ms	200 ms	400 ms
RER 25 ms	0.13	0.15	0.07	0.10	0.10	0.05	0.08	0.07	0.04
RER 75 ms	0.16	0.21	0.09	0.10	0.07	0.01	0.18	0.19	0.15
RER 150 ms	0.14	0.20	0.12	0.11	0.03	0.00	0.41	0.50	0.48
	$RFD_{30\%}$	$RFD_{50\%}$	$RFD_{70\%}$	$RFD_{70\%}$	$RFD_{50\%}$	$RFD_{70\%}$	$RFD_{30\%}$	$RFD_{50\%}$	$RFD_{70\%}$
$RER_{30\%}$	0.25	0.29	0.06	0.16	0.22	0.03	0.43	0.60	0.46
$RER_{50\%}$	0.21	0.33	0.08	0.16	0.12	0.04	0.41	0.61	0.51
$RER_{70\%}$	0.11	0.28	0.11	0.08	0.00	0.01	0.33	0.51	0.53

$RFD_{30\%}$, $RFD_{50\%}$ and $RFD_{70\%}$ represent the rate of force development calculated to 30, 50 and 70% of peak force respectively. $RER_{30\%}$, $RER_{50\%}$ and $RER_{70\%}$ represent the rate of EMG rise calculated to 30, 50 and 70% of peak EMG amplitude, respectively.

5.3.5 Predictability of RFD from tendon stiffness, age and RER

The results of the stepwise multiple regression demonstrated that both tendon stiffness and RER were significant predictors of RFD. These two variables accounted for up to 61% of the variance in RFD in children ($RFD_{70\%}$) and 56% in adults ($RFD_{30\%}$). The proportion of accounted variance was 72% when both children and adults were combined. Age was not a significant predictor of RFD predictability for any group. Predictive regression equations can be found for all populations in Table 5-4.

Table 5-4. Regression equations for the prediction of RFD to 30, 50 and 70% MVC from stiffness and RER for children, adults and all ages combined.

Group	Variable	Equation	R^2
Children	$RFD_{30\%}$	$Y = 0.221 \times RER_{50\%} + 86.773$	0.29
	$RFD_{50\%}$	$Y = 0.422 \times RER_{50\%} - 0.292 \times RER_{70\%} + 0.311 \times stiffness + 69.790$	0.50
	$RFD_{70\%}$	$Y = -0.203 \times RER_{30\%} + 0.523 \times RER_{50\%} - 0.235 \times RER_{70\%} + 0.427 \times stiffness + 57.904$	0.61
Adults	$RFD_{30\%}$	$Y = 0.171 \times RER_{50\%} + 0.460 \times stiffness + 18.696$	0.45
	$RFD_{50\%}$	$Y = 0.422 \times stiffness + 137.584$	0.28
	$RFD_{70\%}$	$Y = -0.496 \times stiffness + 308.162$	0.10
All ages	$RFD_{30\%}$	$Y = -0.154 \times RER_{50\%} + 0.327 \times RER_{70\%} + 0.001 \times stiffness^2 + 73.997$	0.70
	$RFD_{50\%}$	$Y = 0.235 \times RER_{50\%} + 0.310 \times stiffness^2 + 80.260$	0.72
	$RFD_{70\%}$	$Y = 0.183 \times RER_{50\%} + 0.406 \times stiffness + 45.957$	0.70

$RFD_{30\%}$, $RFD_{50\%}$ and $RFD_{70\%}$: RFD calculated to 30, 50 and 70% of MVC, respectively. $RER_{30\%}$, $RER_{50\%}$ and $RER_{70\%}$ represent RER calculated to 30, 50 and 70% of peak EMG amplitude.

5.4 Discussion

The major findings of the present study were that (1) Achilles tendon stiffness, EMD, RFD and RER increased substantially with age to adulthood, (2) plantarflexor EMD was negatively correlated with Achilles tendon stiffness, and (3) tendon stiffness and RER have an additive effect on the prediction of RFD in children. These results extend previous findings in adults (Aagaard *et al*., 2002; Bojsen-Moller *et al*., 2005; Cavanagh and Komi, 1979; Muraoka *et al*, 2004), by demonstrating that the development of force production capabilities during childhood depend on both increased rates of muscle recruitment and changes in tendon mechanical properties. These findings significantly add to our understanding about the mechanisms underpinning age-related improvements in muscular force production, which has vast implications with respect to the interpretation of age-related differences in the execution of complex motor tasks.

5.4.1 Factors Influencing EMD

EMD is influenced by a number of factors, including mechanisms associated with the excitation-contraction coupling process (Cavanagh and Komi, 1979; Norman and Komi, 1979; Nordez *et al*., 2009) and can be modified by training (Grosset *et al*., 2009) or by inducing local muscle fatigue (Paasuke *et al*., 1999). However, the time taken to stretch the tendon is thought to account for the majority of the EMD (Cavanagh and Komi, 1979; Norman and Komi, 1979; Muraoka *et al.*, 2004b). In the present study, tendon stiffness increased with age to adulthood, which is consistent with previous findings (Kubo *et al*., 2001; O'Brien *et al*., 2010; results of Chapter 4). EMD decreased with age through to adulthood, in agreement with that previously shown for the elbow flexor (Asai and Aoki., 1996) and triceps surae muscle groups (Grosset *et al*., 2005). EMD was found to correlate

strongly and negatively with tendon stiffness in both children and adults. This study is the first to directly quantify the relationship between these variables, and supports the results of Muraoka *et al.* (2004b), who found a significant relationship between tendon slack length and EMD, and suggests that differences in tendon stiffness between children and adults underpin age-related changes in EMD. The results allow for the speculation that previously observed age-related changes in EMD (Asai and Aoki., 1996; Grosset *et al*., 2005) are at least partially due to age-related changes in tendon stiffness. In Chapter 4, it was shown that the development of tendon stiffness was largely attributable to the tendon growth and maturation associated with changes in age-related factors rather than age *per-se*. In combination with the present results, it can be concluded that tendon stiffness, and not age, is the predominant factor associated with decreases of EMD during childhood.

5.4.2 Factors Influencing RFD

RFD calculated to 50, 200 and 400 ms was significantly lower in children than in adults. This finding is in agreement with that found previously (Asai and Aoki, 1996; Grosset *et al*., 2005; Falk *et al*., 2009; Cohen *et al*., 2010). However, the single difference in RFD found presently between child groups is in relative contrast to that reported by Grosset *et al.* (2005), who found RFD increased significantly per year of age for children between 7 and 11 years. It is possible that methodological differences between the studies could account for these discrepancies in findings. Firstly, Grosset *et al.* (2005) used supramaximal electrical stimulation to find RFD, which has been shown to recruit muscle differently to voluntary contractions (Enoka, 2002; Jubeau *et al*., 2007; Zhou *et al*., 1995). Voluntary recruitment of motor units typically follows Henneman's size principle, activating motor units on an ascending size basis with increasing force requirements (Henneman *et al.*, 1965a, 1965b).

Electrical stimulation is thought to reverse this recruitment order based on the concept that large diameter axons are more excitable to imposed electrical stimulus (review: Gregory and Bickel; 2005). Action potentials generated from the depolarisation of large diameter motor neurones (from large motor units) arrive at the muscle first due to a faster conduction velocity. As these motor neurones typically innervate a large number of (fast type II) muscle fibres, a greater rise in muscle force will be initially generated. Secondly, RFD was not calculated to different time intervals, but the maximal curve gradient (i.e. maximal RFD) identified manually and RFD calculated to this point from the point of force onset. As a consequence, RFD was calculated over different intervals for each individual. These differences in methodology are likely to have a substantial effect on the outcome of RFD.

Once normalised to peak force, RFD_{norm} was still greater in adults than in children but differences between groups were reduced. This finding demonstrates that much of the difference found between absolute RFD for children and adults was associated with the adults' greater peak force capacity. Normalising RFD to peak force provides an insight into the muscle's activation rate and physiological properties as well as the mechanical properties of the tendon (Wilkie, 1950; Grimby *et al*., 1981; Harridge *et al*., 1996; Aagaard and Thorstensson, 2003). One factor that influences RFD is the muscle's maximal shortening velocity. Contractile velocity is significantly slower in children than adults (Fuchimoto and Kaneko, 1981; Belanger and McComas, 1989; Asai and Aoki, 1996). As a result of this, the time taken to reach their own peak force will be longer for children, impacting on RFD. Muscle fascicle length is a determinant of its shortening speed, as it determines the number of in-series sarcomeres. It is possible that the shorter GM muscle fibres in children (Morse *et al*., 2008) have less in-series sarcomeres, causing whole-muscle shortening velocity to be

slower, again limiting maximal RFD. Moreover, ATPase activity, which is an important determinant of contractile speed (Barany, 1967; Reiser *et al*., 1985), has been shown to be lower in infant rats than adult rats (Drachman and Johnston, 1973; Lowey *et al*., 1993) and is likely to be a factor regulating contractile speed in humans also (Resnicow *et al.*). These factors potentially contribute to the lower contractile velocity (and hence lower RFD_{norm}) observed in children and thus increase the time required to stretch the tendon for transferring forces to bone.

The relationship between tendon stiffness and RFD has been identified previously. Wilkie (1950) found that RFD decreased when a compliant structure was placed between a resistive load and a force-developing body. In agreement with these findings, Bojsen-Moller *et al.* (2005) found a greater knee extensor RFD in individuals with a stiffer vastus lateralis tendon. The low to moderate coefficients of determination found presently between Achilles tendon stiffness and plantarflexor RFD for adults is consistent with these previous findings and supports the idea that greater stiffness of the elastic structures enhances the effective transmission of contractile forces effectively (Leiber *et al*., 2000; Maganaris *et al*., 2004; Magnusson *et al*., 2003). The significant relationship demonstrated between RFD and tendon stiffness in children adds to the literature by extending the concept of mechanical influences of the tendon on force development in a prepubescent population. Given the importance of this relationship for the rapid production of muscular forces in adults, this finding is likely to have significant effects on muscular force production in children also, of which may have important consequences for movement performance. Further, this relationship might suggest that age-related differences in movement production may be a

functional rather than 'immature' adjustment to account for differences in tendon properties.

Age-related increases in RFD raise the question as to the influencing mechanisms that determine it. One specific aim of the present study was to gain a clearer understanding of the mechanisms underpinning age-related differences in RFD. Interestingly, tendon stiffness was best correlated with RFD at markedly different stages of force development in children and adults. This suggests the possibility of age-related differences in the dominant mechanisms that influence RFD at different stages of a contraction. It would appear from the results that tendon stiffness is relatively unimportant in the early rise of force in children. It is possible that the low stiffness associated with the uncrimping of collagen fibrils during the early stages of force development (i.e. toe region of the tendon force-elongation relationship) in combination with greater overall tendon compliance, causes slow and inefficient transfer of muscular forces in children.

5.4.3 Influence of the Rate of Muscle Activation (RER)

RER (to 75 and 150 ms) was greater in adults than in children (with the exception of $CG_{9\text{-}10}$ to 75 ms) but was also greater in $CG_{9\text{-}10}$ than $CG_{5\text{-}6}$ and $CG_{7\text{-}8}$. It was assumed that the temporal increase in EMG amplitude reflected an increase in both the firing frequency of motor units and the increasing recruitment of larger, high threshold motor units. The capacity to activate large motor units (typically innervating type II fibres) has been postulated to be lower in children than in adults (Ramsay *et al*., 1990; Stackhouse *et al*., 2005; Falk *et al*., 2009). As the depolarising potentials are greater in amplitude for larger motor units (Milner-Brown and Stein, 1975), individuals who are able to recruit these motor

units earlier during fast force production should display a steeper EMG-time relation. The greater EMG amplitudes exhibited by $CG_{9\text{-}10}$ compared to $CG_{5\text{-}6}$ and $CG_{7\text{-}8}$ therefore suggests that aspects of neural maturation may occur around this age. As adults demonstrate a significantly greater RER to 150 ms than $CG_{9\text{-}10}$, it would appear that improvements in the ability to rapidly recruit muscle continue through to adulthood.

RER_{norm} was also greater in adults than in children. This finding is consistent with that found for the elbow flexors (Falk *et al*., 2009), and suggests that the ability to recruit motor units for fast force production is influenced by the maturational status of the neuromuscular system (Belanger and McComas, 1989). Children also have a slower motorneurone conduction velocity than adults (Thomas and Lambert, 1960; Oh, 1984) due to possible differences in nerve myelination (Gutrecht and Dyck, 1970; Webster and Favilla, 1984) and nerve fibre diameter (Christ and Brand-Saberi, 2002). Moreover, the residual latency (delay in transmission at the neuromuscular junction) is greater in children (Thomas and Lambert, 1960). As a result of these differences, the frequency of neurone firing is lower (Paasuke *et al*., 2000), a factor that has previously been linked to RFD (Nelson, 1996). There is a growing body of literature postulating that under certain circumstances, large motor units may be preferentially recruited when faced with a task requiring rapid force development (Feiereisen *et al*., 1997; Linnamo *et al*., 2003; Wakeling, 2004; Hodson-Tole and Wakeling, 2009), possibly due to a reduced depolarisation threshold at faster shortening velocities (Christova and Kossev, 2000). In conjunction with the greater firing rate seen in adults (Piotrkiewicz *et al*., 2007), this would act to enhance the early rate of muscle activation. It is therefore concluded that the rate of muscle activation is slower in children caused by an inability to reach their own peak activation at the same rate as adults.

The rate of motor unit firing at the onset of a contraction is an important factor influencing RFD (Corcos *et al*., 1989; Nelson, 1996), and simultaneous increases in RER and RFD have been observed after a period of strength training (Van Cutsem *et al*., 1998; Aagaard *et al*., 2002; Del Balso and Cafarelli, 2007; Blazevich *et al*., 2008). Falk *et al.* (2009) found RFD_{norm} was moderately correlated with RER_{norm}(r = 0.40) for the elbow flexors of young boys (age 9.6 ± 1.6 years). In the present study, RER was positively related to RFD in children (best R^2 of 0.33 for $RER_{50\%}$ and $RFD_{50\%}$), suggesting that neural drive substantially influences the rate of muscular force development and, especially in the early ($RFD_{30\%}$) to mid ($RFD_{50\%}$) stages of a muscle contraction. The relationship between RER and RFD in adults was not as strong as that found in children (best R^2 of 0.22 found for $RER_{30\%}$ and $RFD_{50\%}$), indicating that the adults ability to produce explosive force was not heavily reliant on the ability to rapidly increase neural drive.

5.4.4 Influences of Tendon Stiffness and Neural Drive on Determining Rate Force Development

According to the results of the regression analysis, both muscle activation rate and tendon stiffness play an important role in determining RFD in children and adults. As mentioned previously, tendon stiffness does not appear to have a determining role in early force production in children, but is a significant factor in determining their mid- to late RFD_{norm}. Interestingly, RER is a key determinant during all stages of RFD_{norm} examined in children. Tendon stiffness was the sole determinant of mid- to late RFD_{norm} in adults, but the significant influence of RER on early RFD_{norm} caused an additive effect, increasing the amount of variability of RFD_{norm} that could be accounted for from a maximum of 29% to 45% (for $RFD_{30\%}$). The additive effects of tendon stiffness and RER_{norm} significantly improved the

prediction of RFD_{norm} from a maximum of 28% to 61% for $RFD_{70\%}$ in children, highlighting the importance of both factors for rapid force production in children. Despite their combined influence however, a significant proportion of the variance in RFD_{norm} remained unaccounted for, suggesting that factors not examined in the present study must play a relatively central role in rapid force production. For example, Edman and Josephson (2007) suggest that 60% of the force rise time can be attributable to the processes underlying muscle activation (e.g. calcium kinetics, cross-bridge cycling rate), and the remaining 40% represents the time taken in stretching the series elastic structures for force transfer. It is likely therefore that other physiological process impact on rapid force production, and that the significance of these processes differs between children and adults.

5.4.5 Conclusion

This is the first study to identify significant relationships between tendon stiffness and parameters relating to rapid force development (EMD and RFD) in prepubertal children. EMD was negatively related to tendon stiffness in both children and adults. Contrary to that found in adults, muscle activation rate was more important than tendon stiffness in the early rise of force in children, whilst tendon stiffness was found to influence the later rise. Nonetheless, together RER and tendon stiffness were able to account for a significant proportion of RFD variability, highlighting the role of both neural and mechanical factors in for rapid force production in children. Based on the relationships presented in the present study, it can be concluded that the significant differences in tendon stiffness and muscle activation rate found between children and adults partly explain differences in force development and might therefore influence complex movement performance.

CHAPTER 6: Resistance Training Increases Tendon Stiffness and Influences Rapid Force Production in Prepubertal Children

6.0 Abstract

As increases in muscular strength occur with age or training, a synchronous adaptation in the tendon must be achieved in order to transmit greater forces efficiently to the bone and to minimise the risk of tendon injury. Tendons have been shown to adapt to chronic loading in adults and, in addition to an increased rate of muscle activation, are thought to strongly influence muscular force production. It was therefore hypothesised that resistance training could alter tendon mechanical properties in children, and that such changes would impact on force development. Twenty prepubertal children (aged 8.9 ± 0.3 years) were divided into control (non-training) and experimental (training) groups. The training group completed a 10-week resistance training intervention consisting of 2 - 3 sets of 8 - 15 plantarflexion resistance efforts performed twice-weekly on a recumbent calf raise machine as part of a class-based physical education lesson. Achilles tendon stiffness (slope of the tendon force-elongation curve), electromechanical delay (EMD; time between the onset of muscle activity and force), rate of force development (RFD; slope of the force-time curve) and rate of EMG rise (RER; slope of the EMG-time curve) were measured during isometric plantarflexion contractions performed with a maximum rate of force development before and after training. No changes were found for any variable in the control group, however tendon stiffness and Young's modulus increased significantly in the experimental group (~35% and ~33%, respectively); tendon CSA was unchanged. Peak tendon stress showed a trend towards increasing after training as a consequence of increased plantarflexor force production, whereas peak tendon elongation and strain showed a trend to decrease. A decrease in EMD (~13%) was found after training for the experimental group which paralleled the increase in tendon stiffness (r = 0.59). Nonetheless, RFD and RER were unchanged after training. The present data show that the Achilles tendon adapts to

resistance training in prepubertal children. Such adaptation was associated with changes in muscular force production, and therefore has the potential to influence movement performance. The mechanisms underpinning rapid force production in children appear to be different to those found in adults as RFD was not influenced by the changes in tendon stiffness, and may help explain further differences in movement performance between these populations.

6.1 Introduction

Resistance training is becoming an increasingly popular mode of exercise amongst children (Faigenbaum *et al*., 1996; Guy and Micheli, 2001). Its benefits in this population include cardio-respiratory fitness (Blimkie, 1993), increased bone mineral density (Morris *et al*., 1997; Nichols *et al*., 2001) and enhanced psychosocial health (Hass *et al*., 2001; Falk and Eliakim, 2003). Additionally, resistance training promotes strength gains (Faigenbaum *et al*., 1993; Ozmun *et al*., 1994; Kubo *et al*., 2001b) beyond those associated with normal growth (Blimkie *et al.*, 1989a). These strength gains have been attributed to an improved coordination of the involved muscles and an increased motor unit activation (Ramsay *et al*., 1990; Ozmun *et al*., 1994). Androgenic and hypertrophic factors as muscular hypertrophy is rarely observed (Weltman *et al*., 1986; Ramsay *et al*., 1990; Ozmun *et al*., 1994; Faigenbaum *et al*., 2007).

As age- or training-related increases in muscular strength occur (Blimkie *et al.*, 1989a; Ramsay *et al.*, 1990), a synchronous tendon adaptation must be achieved so that: (1) these greater forces can be efficiently transmitted to the bone without tendon injury, and (2) there is a similar tendon elongation under increased load so that skeletal muscle fibres continue to work within their normal operating range (Wilson *et al*., 1994; Ishikawa and Komi, 2008). Previous research has shown that tendons adapt to chronic increases in loading by increasing their ultimate tensile strength (Woo *et al*., 1980; Vilarta and Vidal Bde, 1989) and/or stiffness (Woo *et al.*, 1980; Narici *et al.*, 1996; Kubo *et al.*, 2001a; Reeves *et al.*, 2003a; Wu *et al.*, 2010), often with an increase in CSA (Woo *et al*., 1980; Michna and Hartmann, 1989; Birch *et al*., 1999; Rosager *et al*., 2002; Kongsgaard *et al*., 2007). In Chapter 4, it was demonstrated that age-related increases in body mass and muscular strength,

which consequently increase tendon loading, correlate well with age-related increases in stiffness of the Achilles tendon in prepubescent children. This finding is consistent with the relationship observed between muscular strength and tendon stiffness in adults (Scott and Loeb, 1995; Muraoka *et al*., 2005; Arampatzis *et al*., 2007). Such results support the idea that tendons can adapt in response to changes in chronic loading in children, as has been indicated in other young mammals (Curwin *et al*., 1988; Kasashima *et al*., 2002). Nonetheless, the hypothesis that tendons can adapt in response to strength training in children has yet to be explicitly examined, so it is not known whether such training alters tendon mechanical properties in addition to that associated with normal growth and development. It is also not known whether changes in tendon CSA accompany any changes its mechanical properties. This is a particularly pertinent question as increases in muscle strength in response to training have been shown to occur without muscular hypertrophy; a lack of hypertrophic adaptation in the tendon could have a substantial impact on the potential for tendon adaptation. Thus, the main purpose of this study was to examine the effects of chronic plantarflexor strength training on both the mechanical properties and CSA of the Achilles tendon in prepubescent children. Given that children are able to increase their muscular strength in response to short-term resistance training, it was hypothesised that their tendons would be receptive to chronic increases in loading.

Understanding the relational change in muscle strength and tendon stiffness is important as tendon stiffness impacts on rapid muscular force production and thus may impact on movement capacity. For example, both electro-mechanical delay (EMD) and the rate of force development (RFD) are largely governed by the time taken to stretch the series elastic component of the muscle-tendon complex (Wilkie, 1950; Cavanagh and Komi, 1979; Bojsen-

Moller *et al*., 2005), of which the tendon is often a major component (Gray and Carter, 1860). A delay in transferring rapidly generated forces may impact on balance and stability (Mora *et al.*, 2003; Granacher *et al.*, 2010), risk of fall (Pijnappels *et al*., 2005; 2008) and movement reaction times (Wilkie, 1950; Aagaard *et al.*, 2002; Reeves *et al.*, 2003a; Bojsen-Moller *et al.*, 2005; Holtermann *et al.*, 2007; Blazevich *et al.*, 2008; Grosset *et al.*, 2009; Nordez *et al.*, 2009). Despite previous data indicating that RFD and EMD can be augmented with specific training in adults, and is typically attributed either to an increased tendon stiffness or rate of muscle activation (Van Cutsem *et al.*, 1998; Aagaard *et al.*, 2002; Reeves *et al.*, 2003a; Barry *et al.*, 2005; Del Balso and Cafarelli, 2007; Gruber *et al.*, 2007; Grosset *et al.*, 2009; Wu *et al.*, 2010), the same relationship has not been examined in young children. Therefore, a second purpose of the study was to measure the effects of plantarflexor strength training on EMD and RFD, and to determine whether any changes are related to changes of tendon stiffness. The rate of activation, estimated as the rate of rise in the electromyogram amplitude (RER), was also measured in order to examine its possible influence on RFD (Corcos *et al*., 1989; Nelson, 1996).

6.2 Methods

6.2.1 Ethics and Participant Information

Ten boys and 10 girls (aged 8.9 ± 0.3 years) volunteered to participate in the study after receiving a presentation at their school regarding the project (to which parents and guardians were invited and encouraged to ask questions). To participate in the study, written assent was provided by the children and written consent was provided by the guardians. Physical activity readiness questionnaires were completed for each child by the guardians to ensure all participants were free from known neuromuscular or musculoskeletal disorders, were not involved in competitive sports or had previous resistance training experience. Institutional ethical approval was granted by the Human Research Ethics Committee at Brunel University. The research was conducted in accordance with the guidelines set in the Declaration of Helsinki.

Based on which school class (out of two) they were enrolled in, the children were chosen at random and divided into control and experimental groups (each containing 5 girls and 5 boys). Prepubertal status was confirmed by estimating the age of the children from peak height velocity (an indicator of maturational offset) using body anthropometry (Mirwald *et al*., 2002). The minimum age from peak height velocity found for participants in this study was -3.7 years. Each participant visited the university on two separate occasions to allow pre- and post-training data to be collected before and after a 10-week ankle plantarflexion training period.

6.2.2 Pre- and Post-training Data Acquisition.

Comprehensive methodologies for calculating Achilles tendon stiffness, Young's modulus, tendon stress, strain and CSA can be found in Chapter 4 (pages 88-91). Data acquisition for the subsequent calculation of EMD, RFD and RER was identical to that presented in Chapter 5(pages 113-116) and thus will not be detailed here.

Briefly, GM MTJ elongation, measured using ultrasonography, and peak plantarflexor moment, measured using an isokinetic dynamometer, were obtained during maximal isometric plantarflexion contractions (MVCs). Displacement of the GM MTJ was corrected for distal tendon movement (caused by heel movement) using motion capture. Tendon force was calculated as the ratio of plantarflexor moment (corrected for antagonist coactivity) and Achilles tendon moment arm (estimated using the tendon excursion method). Tendon stiffness was calculated as the slope of the force-elongation curve between 10 – 90% peak force. Electromechanical delay (EMD), rate of force development (RFD) and rate of electromyographic (EMG) signal rise (RER) were calculated from each MVC trial. RFD was calculated to 25, 50, 75, 100, 150, 200, 250, 300 and 400 ms and to every 10% of MVC up to 90% MVC. RER was calculated to 25, 50, 75, 100, 150 and 200 ms and to every 10% of peak EMG amplitude up to 70% peak EMG amplitude. These intervals were chosen to maximise the chances of identifying changes in RFD after the training intervention, based on previous findings (Aagaard *et al*., 2002; Blazevich *et al*., 2008; results of Chapter 5). All procedures outlined above were carried out pre- and post-training.

6.2.3 Resistance Training Programme - Familiarisation

Children in the experimental group were familiarised with the recumbent (45^{o} incline) calf raise machine (Body Solid, Model GLPH1100, Forest Park, IL, USA) on two separate occasions prior to the start of the 10-week training period. During the first session, participants were taught the correct technique on an unloaded machine (foot plate mass of 30 kg). Once seated in the machine, participants placed the balls of their feet on the footplate of the machine and were instructed to extend the knees to a near-extended position (Figure 6-1). They then slowly raised and lowered the footplate through a full but comfortable range of motion using concentric then eccentric ankle plantarflexion contractions. In addition to providing a greater safety and longer muscle contractile (and thus tendon loading) period, slow movement speeds reduced the likelihood of changes in rate of muscle activation, a key factor influencing rapid force production (Corcos *et al*., 1989; Nelson, 1996). On the second occasion, participants completed a single set of 6 - 8 repetitions on an unloaded machine, and scored their perceived effort from 1 - 10 (1: easy, 10: hard). The number of repetitions prescribed for the initial training session was then based on their perceived effort during this set. Participants were given the opportunity to ask questions at any point during the sessions.

6.2.4 Resistance Training Programme - Design

For the duration of the study, training was performed twice-weekly (Sailors and Berg, 1987; Faigenbaum *et al*., 1993) as part of a class-based physical education lesson. The lesson was arranged as circuit training, where nine stations were organised to accommodate small groups of 3 - 4 children (27 - 28 children per class). For the class containing the experimental group, the plantarflexion exercise was set up as one of the stations. This station was

replaced with a resting station for the class containing the control group. The remaining stations were varied throughout the programme to help maintain participant motivation and typically included activities not specific to the lower extremity (basketball shooting, football dribbling, hockey slalom, sit-ups, etc.). Each lesson began with 2 - 3 minutes of low intensity aerobic exercise as a warm-up (e.g. marching on the spot, star jumps etc). Groups rotated between stations at the sound of the investigator's whistle, which was blown when all the children at a resistance machine station had each completed one set of repetitions. Rotations continued until all scheduled sets had been completed by the 10 experimental participants. 2 – 3 minutes of dynamic stretching was performed after the lesson.

Figure 6.1. Resistance machine setup. Children performed plantarflexion contractions by pushing the footplate with the balls of their feet.

The participant-instructor ratio at the resistance machine was always 1:1 to ensure that the appropriate technique, applied load and number of repetitions were adhered to and

recorded. Two further supervisors monitored activity at the remaining 8 stations. Participants started with a load they could only perform 8 - 10 repetitions (8 - 10 RM) at using the correct form. When 15 repetitions could be completed with this load, using the correct form, additional load was added in increments of 2.5 kg to generate a new 8 - 10 RM (Pediatrics, 2001). In the first two weeks, 2 sets of 8 – 15 repetitions were performed with an emphasis on safety and technique rather than maximal loading. In the remaining eight weeks, three sets of repetitions were performed (Faigenbaum *et al*., 2006). The frequency and intensity of the resistance training programme was based on progressive loading to generate substantial strength gains, and was in accordance with the current recommendations for strength training in primary school children (CSMF., 2001). The mean (± SD) load (kg) lifted (i.e. load added to the weight stack) during the 10-week training schedule is shown in Table 6-1.

Table 6-1. The 10-week training schedule and mean (± SD) weight lifted per session.

Week	**1/2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Day 1	30.0 ± 0	32.5 ± 0	34.5 ± 1.1	41.7 ± 3.3	49.1 ± 2.7	51.0 ± 1.8	52.2 ± 1.1	60.0 ± 0	59.4 ± 1.6
Day 2	30.0 ± 0	32.5 ± 0	37.3 ± 0.7	44.7 ± 2.9	47.3 ± 1.6	50.0 ± 0	60.0 ± 0	59.5 ± 2.0	62.5 ± 0

Two sets of 8–15 RM were completed per session during weeks 1 and 2, increasing to 3 sets during weeks 3 – 10. Pre- and post-training testing occurred during weeks 0 and 11, respectively. Loads reported are in kg.

6.2.5 Statistical Analysis

During the course of the training intervention, the family of one child from the experimental group (female) relocated and one child from the control group (female) was absent for the

post-training testing. Therefore statistical analyses were performed on data from 9 experimental and 9 control participants (each with 4 girls and 5 boys).

All data were analysed using SPSS statistical software (SPSS v16.0, Chicago, USA). Differences in anthropometry, tendon variables and force production characteristics between the control and experimental groups at baseline were tested for using independent *t*-tests. Changes in mean body mass, standing height, peak force production and EMD from pre- to post-training were examined with paired *t*-tests for both groups. For the post-training test, tendon-related dependent variables were calculated in two different ways. The first way of calculating peak Achilles tendon stress, strain, elongation, stiffness and Young's modulus was based on the peak forces measured during the post-training test. The second way of calculating these measures was based on the MVC values obtained during the pre-training test. This latter method of calculating the tendon-related variables was performed to allow for a direct comparison of these measures before and after training, independent of training-related strength gains by removing strength changes from pre- to post testing as a confounding factor influencing the results. These variables are subsequently referred to as MVC_{pre}. A2 x 2 mixed design analysis of variance (ANOVA) was used to analyse for differences in time (pre; post) and group (control; experimental) for each dependent variable measured (tendon variables for both pre and post MVC forces, RFD and RER).In the case of a significant time-by-group interaction, paired *t*-tests were performed to compare the pre to post values for each the experimental and the control groups. For these *t*-tests, Bonferroni adjustments were performed to control for type I error. Statistical significance was accepted at $p< 0.05$.

Finally, in order to further examine the relationship between tendon stiffness and EMD, a polynomial was fitted to the data showing the relative changes of EMD and stiffness of each individual after the training intervention.

6.3 Results

Training compliance was 92.6% and 96.7% for children in the control and experimental groups, respectively. Neither body mass nor standing height changed significantly for either group between pre- and post-training. There was a significant difference found between the control and experimental groups for pre-training stiffness ($p < 0.05$). No significant differences were found for any other variables measured at pre-training testing ($p > 0.05$).

F-statistics and levels of significance for time-by-group interactions from the ANOVAs can be found for each dependent variable in Table 6-2. Significant time-by-group interactions were found for stiffness ($p = 0.005$) and Young's modulus ($p = 0.004$) calculated from post-test MVC forces. Post hoc *t*-tests revealed a significant increase in tendon stiffness ($p < 0.001$) and Young's modulus ($p = 0.001$) from pre- to post-testing in the experimental group. Pre- to post-training differences in stiffness and Young's modulus were non-significant for the control group ($p = 0.378$ and $p = 0.320$, respectively). Time-by-group interactions were also significant for stiffness ($p = 0.004$) and Young's modulus ($p = 0.020$) calculated from pre-training MVC forces. Post hoc *t*-tests revealed a significant increase in tendon stiffness ($p = 0.001$) and Young's modulus ($p = 0.004$) in the experimental group. These differences were not significant for the control group ($p = 0.659$ and $p = 0.378$, respectively).No time-by-group interactions were found as a result of training for any RER or RFD interval (Table 6-2).

Group mean and individual changes in Achilles tendon stiffness and Young's modulus (based on peak force at pre-training) are presented in Figure 6-2. Mean (± SD) and % changes in tendon CSA, length, peak stress, peak strain, peak elongation, stiffness and Young's modulus

for both groups are shown in Table 6-3. In addition stress, strain, elongation, stiffness and Young's modulus calculated using pre-training MVC force can also be found in Table 6-3.

Table 6-2. ANOVA results for each tendon variable, RFD and RER.

Dependent Variable	*F*	*df1, df2*	*p*
Stress	0.317	1, 16	0.581
Strain	1.533	1, 16	0.234
Elongation	1.867	1, 16	0.191
CSA	0.417	1, 16	0.528
Force	0.670	1, 16	0.425
Stiffness	10.730	1, 16	0.005*
Young's modulus	11.419	1, 16	0.004*
Strain (MVC_{pre})	1.889	1, 16	0.188
Elongation (MVC_{pre})	2.253	1, 16	0.153
Stiffness (MVC_{pre})	11.056	1, 16	0.004*
Young's modulus (MVC_{pre})	6.675	1, 16	0.020*
RFD (ms)	1.333 – 0.010	1, 16	0.265 – 0.920
RFD (%)	0.778 – 0.020	1, 16	0.391 – 0.890
RER (ms)	3.538 – 0.037	1, 16	0.078 – 0.849
RER (%)	3.021 – 0.017	1, 16	0.101 – 0.899

F – F statistic; df1, df2 - degrees of freedom for group and n number respectively; p – level of significance

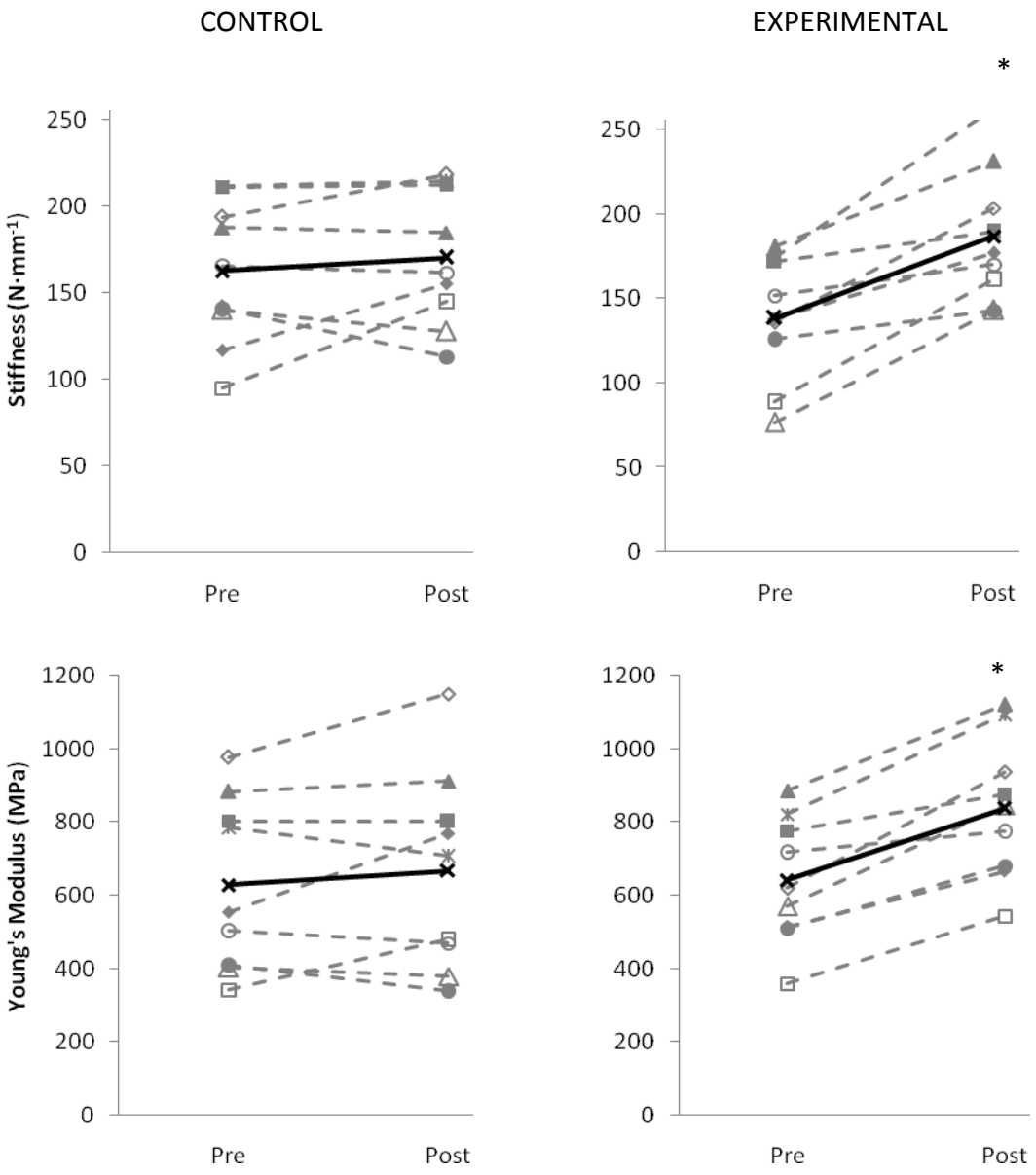


Figure 6-2. Mean (black) and individual (grey) changes in tendon stiffness and Young's modulus for control and experimental participants at pre- and post-training. * significant change relative to control, $p < 0.05$.

EMD decreased by an average 9.8 ms (~-13%) in the experimental group after training (pre: 76.3 ± 11.0 ms, post: 66.5 ± 10.2 ms; $p < 0.05$) but remained unchanged in the control group (pre: 74.5 ± 10.8 ms, post: 74.2 ± 12.3 ms). Changes in EMD were moderately, negatively correlated with tendon stiffness in the experimental group (r = 0.59) and the control and experimental participants combined (r = 0.55), as shown in Figure 6-3. Absolute and relative

(to peak EMG) RER were unchanged after training for all time intervals (Figure 6-4). RFD was also unchanged after training for all time intervals and levels of force (Figure 6-5).

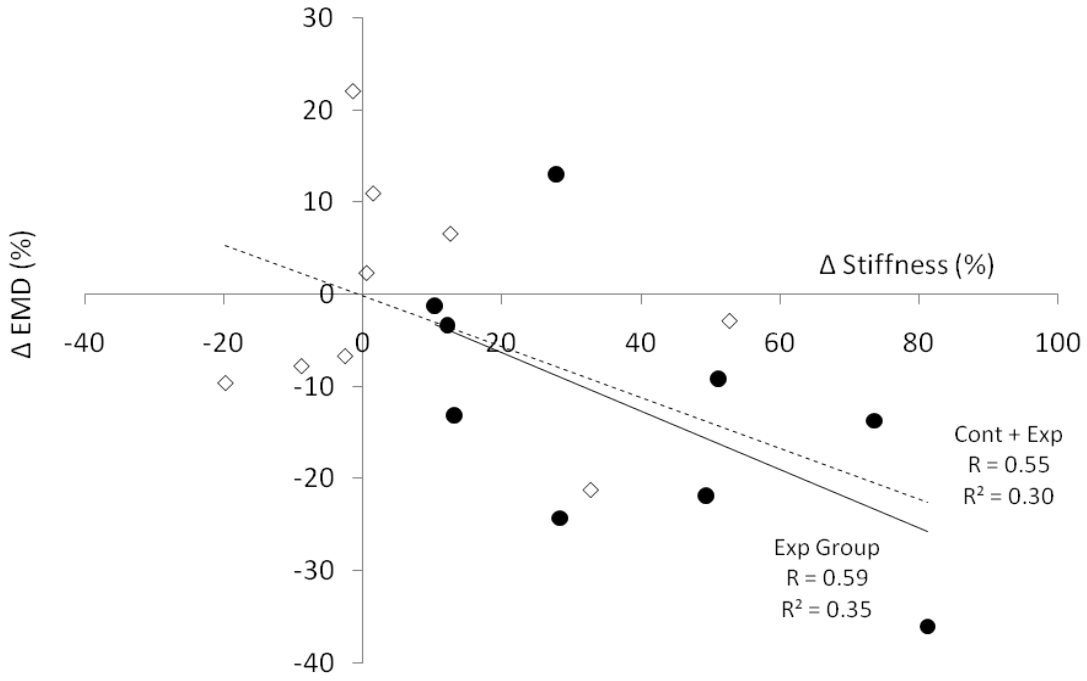


Figure 6-3. Relationship between pre- to post-training changes in tendon stiffness and electromechanical delay (EMD). Filled circles represent the experimental group and open diamonds represent the control group. Trend lines for the experimental group (solid line) and the control and experimental group combined (dashed line) are shown. A significant relationship was found between the changes in tendon stiffness and EMD for the control and experimental group combined ($p < 0.05$). Cont, control group; Exp, experimental group.

Table 6-3. Pre- and post-training training Tendon characteristics.

	Control			Experimental			
Tendon Characteristics	Pre	Post	Δ%	Pre	Post	Δ%	
Tendon CSA (mm^2)	40.7 ± 7.2	41.8 ± 7.9	2.7	35.8 ± 6.3	36.7 ± 5.9	2.5	
Tendon length (mm)	151.6 ± 32.9	153.8 ± 29.4	1.4	160.3 ± 21.3	164.5 ± 24.3	2.6	
Peak Elongation (mm)	11.8 ± 4.0	12.6 ± 2.5	7.4	11.6 ± 2.6	10.37 ± 2.4	-10.3	
Elongation MVC_{pre}(mm)	11.8 ± 4.0	11.5 ± 2.1	-2.0	11.6 ± 2.6	9.2 ± 2.1	-20.2	
Peak Strain (%)	8.1 ± 3.2	8.5 ± 2.8	5.4	7.3 ± 1.6	6.2 ± 1.4	-13.6	
Strain MVC_{pre} (%)	8.1 ± 3.1	7.8 ± 2.9	-3.2	7.3 ± 1.6	5.5 ± 0.6	-23.9	
Peak Stress (N/mm^{-2})	47.1 ± 14.1	51.7 ± 14.9	9.8	44.6 ± 9.6	50.8 ± 7.0	14.0	
Stiffness (N/mm)	162.5 ± 41.8	170.3 ± 39.1	4.8	138.4 ± 36.7	187.0 ± 44.0	35.2	*
Stiffness MVC_{pre} (N/mm)	162.5 ± 41.8	167.3 ± 37.8	2.9	138.4 ± 36.7	177.6 ± 41.3	28.4	*
Young's Modulus (MPa)	629.4 ± 235.3	673.2 ± 281.2	7.0	642.0 ± 171.2	859.4 ± 206.0	33.9	*
Young's Modulus MVC_{pre} (MPa)	629.4 ± 235.3	649.3 ± 271.7	3.2	642.0 ± 171.2	827.6 ± 187.6	28.9	*

MVC_{pre}: variable normalised to pre-training MVC force. * significant change relative to control, $p < 0.05$.

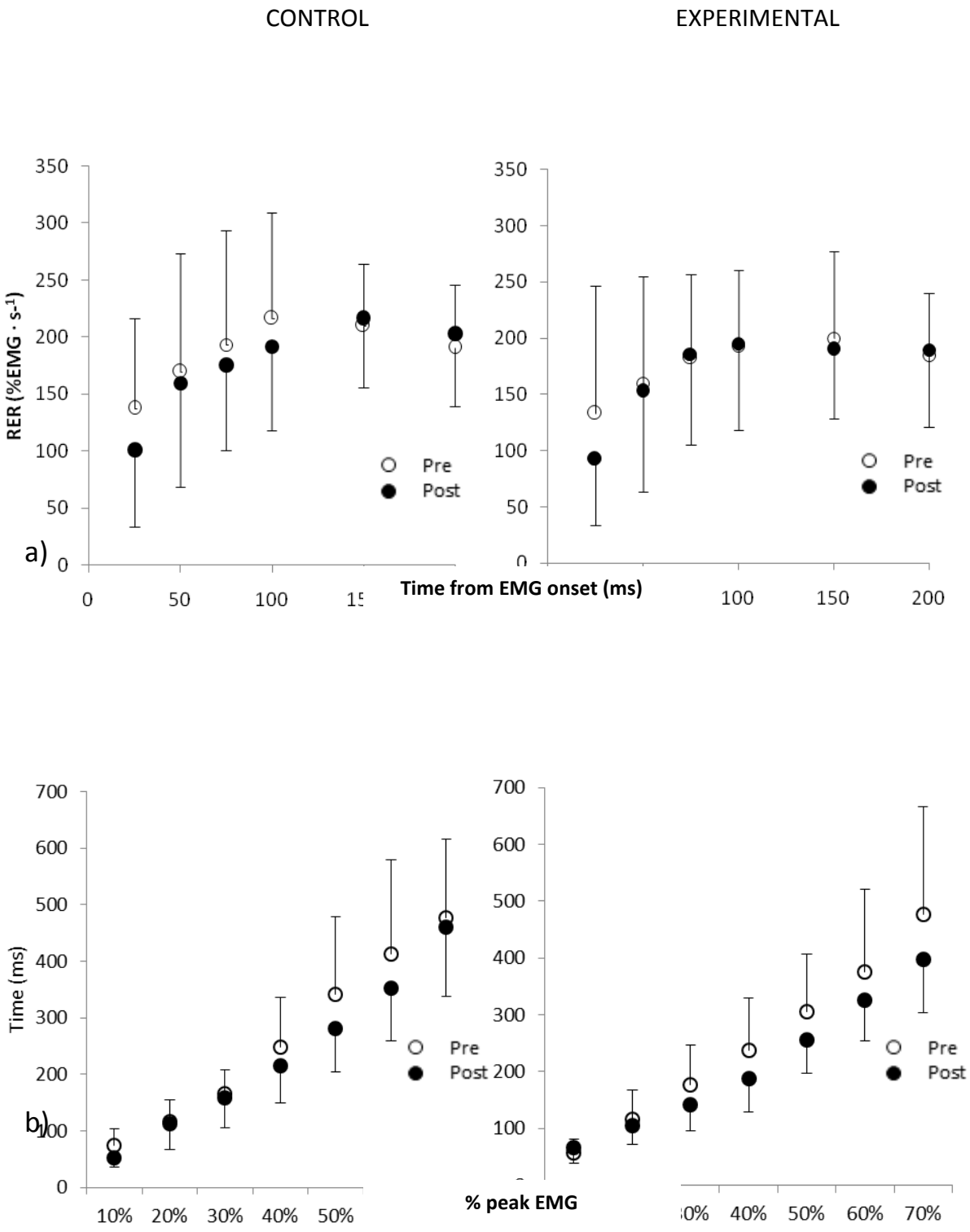


Figure 6-4. Rate of EMG rise (RER) at pre- and post-training, calculated as % peak amplitude EMG per second a) measured to 25, 50, 75, 100, 150 and 200 ms after EMG onset, b) the time taken (ms) to reach relative (to peak) EMG levels. Pre-training values are represented by open circles and post-training values are represented by filled circles. There were no statistically significant changes in RFD with the training intervention.

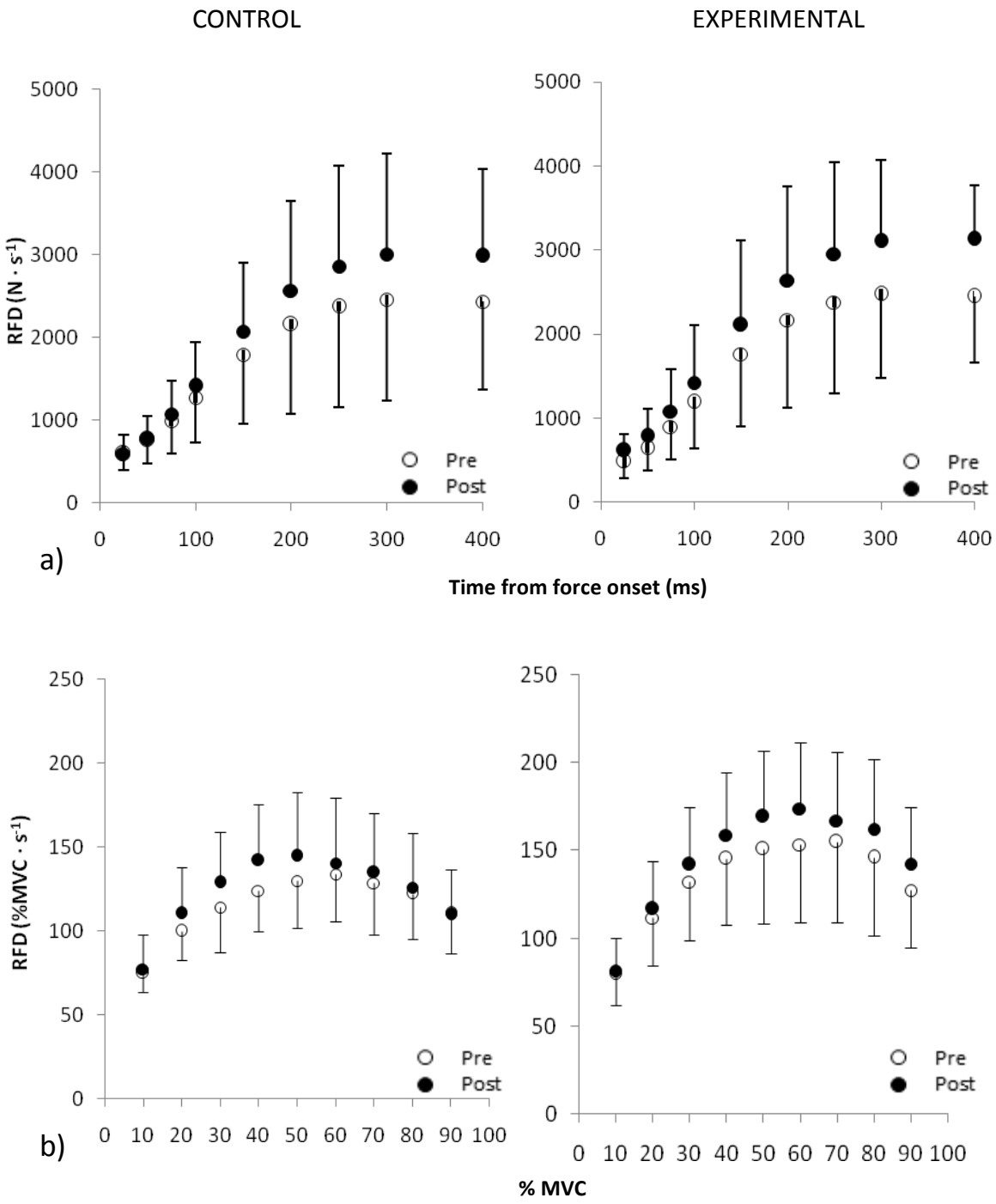


Figure 6-5. Rate of force development (RFD) at pre- and post-training, calculated as % MVC per second, measured to 25, 50, 75, 100, 150, 200, 250, 300 and 400 ms after EMG onset and b) to reach % MVC, displayed in intervals of 10%. Pre-training values are represented by open circles and post- training values are represented by filled circles. There were no statistically significant changes in RFD with the training intervention.

6.4 Discussion

6.4.1 Training-Induced Changes in Achilles Tendon Mechanical Properties

The main purpose of the present study was to examine the effects of plantarflexor strength training on the mechanical properties of the Achilles tendon in prepubescent children, with the secondary purpose to examine their relationship with muscle force production characteristics (EMD and RFD). The main finding was that the mechanical properties of the Achilles tendon were significantly altered by 10 weeks of twice-weekly resistance training in previously untrained prepubertal children. Tendon stiffness was found to increase by ~35%, which is similar to the 30% increase in Achilles tendon stiffness reported by Kubo *et al.* (2007) for adults who completed 12 weeks of resistance training, despite a far greater training intensity used by those adults. These increases are somewhat greater than previously shown for the patellar tendon. Seynnes *et al.* (2009) reported a ~24% increase in patellar tendon stiffness after 9 weeks of resistance training, whilst Kongsgaard *et al.* (2007) found a ~15% increase in patellar tendon stiffness after 12 weeks of heavy knee extension exercise in young adults. Nonetheless, it is less than the 65% increase in patellar tendon stiffness found in an elderly population after 14 weeks resistance training (Reeves *et al*., 2003). Compared to these previous findings, the present results suggest that the developing Achilles tendon is at least as, if not more, responsive to chronic increases in loading as mature tendons. These results concur with the existing literature by demonstrating an increase in tendon stiffness after short-term resistance training, and expand on previous results by confirming, for the first time, that this is also the case in children.

Importantly, no change in tendon CSA was detected after the training. As Young's modulus is a measure of normalised (to tendon dimensions) stiffness, the consequence of increased

stiffness and constant CSA was a significant increase (33%) in Young's modulus. The lack of change in tendon CSA found here after training is consistent with a number of studies in animals (Huang *et al*., 2004; Legerlotz *et al*., 2007), and both younger (Kubo *et al*., 2001a; Magnusson *et al*., 2003) and older human adults (Reeves *et al.*, 2003a) after short (< 16 weeks) periods of heavy resistance training. From these results, it is likely that chronic loading over an extended period of time may be required to induce an increase in tendon CSA (Conkrite, 1936; Rosager *et al*., 2002). Increases in Young's modulus are indicative of adaptation in the underlying microstructure and collagen arrangement. Some research has shown that microstructural changes include an increase in collagen content (Heikkinen and Vuori, 1972; Curwin *et al*., 1988), leading to an increase in mean fibril diameter (Michna and Hartmann, 1989; Enwemeka *et al.*, 1992; Patterson-Kane *et al.*, 1997b; Edwards *et al.*, 2005) and better alignment of collagen fibres with the direction of force transmission (Elliott, 1965), facilitating a denser packing of collagen material (Reed and Iozzo, 2002). These micro-adaptations improve tensile strength and stiffness (Parry *et al.*, 1978a; Bailey *et al.*, 1998; Birch *et al.*, 1999; Derwin and Soslowsky, 1999; Battaglia *et al.*, 2003; Provenzano and Vanderby, 2006; Rigozzi *et al.*, 2010). Together, these results highlight the developing tendon's preference for qualitative adaptations rather than quantitative changes in collagen in order to alter its mechanical properties under loading, much like that observed with muscle adaptation to short-term resistance training (Blazevich *et al.*, 2007b).

As a number of tendon properties are calculated from a force range based on a percentage of maximum attained force, or from peak force, training-induced increases in maximum force at post-testing causes these variables to be calculated from a new force range or peak force. For the purpose of removing strength gains as a confounding factor influencing these

variables, post-training tendon stiffness, Young's modulus, strain and elongation were also calculated over the pre-training force range. The relative changes in tendon stiffness and Young's modulus were 28% and 29% respectively. This change is only slightly less than that found using post-training forces, suggesting that absolute changes in mechanical properties as a result of a training intervention were not substantially influenced by strength improvements. Although forces are not required for the calculation of tendon strain or elongation, both variables are dependent on the force levels as this defines the tendon's deformation, depending on its stiffness. Based on peak force attained at pre-training, there were moderate, albeit non-significant, decreases in tendon elongation and strain. These small changes in tendon deformation under loading may have consequences for movement efficiency and the likelihood of injury, although further examination is required to verify this.

6.4.2 Changes in Electromechanical Delay

An important functional outcome of the training was that EMD was significantly decreased (~13%) in the experimental group. This decrease was moderately correlated with the magnitude of change in tendon stiffness (r = 0.59). We have previously shown that Achilles tendon stiffness was well correlated with EMD in children (Chapter 5), thus the moderate decrease in EMD found in the present training group is likely to be partly attributable to the increase in tendon stiffness. These results are consistent with those of Kubo *et al.* (2001a), who found a significant decrease in EMD along with a significant increase in tendon stiffness after 12 weeks of isometric training in adults. Furthermore, they are in agreement with Grosset *et al.* (2008), who found paired changes in EMD and musculo-tendinous stiffness after 10 weeks of either endurance or plyometric training in adults. These results indicate

that stiffness of the Achilles tendon is indeed a major determinant of plantarflexor EMD (Cavanagh and Komi, 1979), although the possibility of other factors influencing EMD that may have changed as a consequence of training may also be partly responsible for the decreased EMD observed. As the ability to produce movement relies on the ability to produce muscular force, a decrease in EMD of 10 ms, as found here from a short-term resistance training, would help to improve the magnitude of muscular force attainable within a time frame, thus improving the likelihood of a successful rather than unsuccessful movement outcome (Moritani, 2002; Pijnappels *et al*., 2005; Pijnappels *et al*., 2008).

6.4.3 Lack of Change in Rate of Force Development

It was hypothesised that both RFD and tendon stiffness would increase concurrently in children as a result of resistance training, based on the positive relationship found between these variables in adults (Reeves et al., 2003; Bojsen-Moller *et al*., 2005) and the simultaneous increases reported after a period of resistance training (Thorstensson *et al.*, 1976a; Narici *et al.*, 1996; Van Cutsem *et al.*, 1998; Aagaard *et al.*, 2002; Gruber *et al.*, 2007; Holtermann *et al.*, 2007; Blazevich *et al.*, 2008). However, despite the significant increase in tendon stiffness as a result of resistance training, RFD remained unchanged. A moderate relationship between tendon stiffness and RFD was shown in children (R^2 = 0.34) in Chapter 4 (page 122) for RFD calculated to 400ms. However, this relationship was demonstrated over a substantial range of Achilles tendon stiffness values (~200% difference in means between 5 – 6 and 9 – 10 year old children, ~500% difference in stiffness between the least and most stiff children). Using the equations presented in that study (Chapter 5, page122, Table 5-2) to predict the change in RFD due to change in tendon stiffness, it can be shown that RFD to 50, 200 and 400 ms, for example, would increase by 110.1, 347.3 and 562.3 $N \cdot s^{-}$

[1], respectively. These estimates are in agreement with the findings of the present study (see Figure 6-5), and show that only modest increases in RFD are likely with the magnitude of tendon stiffness changes achieved with the training performed. In fact, the range of values predicted (± 1SD) overlap considerably (e.g. 50 ms = 600.4-766.8 $N \cdot s^{-1}$ vs. 694.1-893.5 $N \cdot s^{-1}$ for pre- and post-training values, respectively), suggesting that a statistically significant changes would not have been detected. Potentially, longer or more intensive training periods might yield more favourable results with respect to RFD adaptations resulting directly from increases in tendon stiffness.

The mechanisms underpinning RFD in children were therefore speculated to involve factors other than tendon stiffness. A positive correlation has been observed previously between RFD and RER in children (Falk *et al.*, 2009; results of Chapter 5). Other studies have reported simultaneous increases in the rate of muscle activation and RFD after resistance training in adults (Aagaard *et al*., 2002; Barry *et al*., 2005; Del Balso and Cafarelli, 2007; Blazevich *et al*., 2008). Together, these results support the previous suggestion of a parallelism between these variables (Bell and Jacobs, 1986; Komi, 1986). Here, RER did not change as a result of training. Previous studies have also found a lack of change in EMG variables with training performed with a slow movement velocity (Narici *et al*., 1996; Blazevich *et al*., 2008). It is possible therefore that the lack of change in RER may possibly be due to performing the training with a slow movement velocity. The lack of change of RER may also represent a lack of improvement in the ability to recruit large motor units for force production. Motor unit recruitment has been shown to remain significantly unchanged after short-term resistance training in children (Ramsay *et al*., 1990), although other neurological adaptations have been demonstrated (Ozmun *et al*., 1994). It is also known that children do not have the

same capacity for motor unit recruitment compared to adults (Asmussen, 1973; Belanger and McComas, 1989; Paasuke *et al*., 2000). It can be concluded, therefore, that short-term training using slow movement velocities does not significantly improve neural drive in children and thus may inhibit the ability to improve RFD. Training with a fast movement velocity may be required to substantiate this conclusion.

6.4.4 Practical Implications

Tendon stiffness is thought to influence movement performance substantially and an increased tendon stiffness in a paediatric population maybe beneficial for several reasons. First, the time taken to stretch a stiff tendon to a point where forces are transmitted to the bone is shorter than that for a more compliant tendon. Thus, a stiffer tendon might improve both rapid force development and subsequent fast movement performance. Hence, the muscle may also be required to be active for a shorter time period in order to complete a task, which would reduce the metabolic cost of the activity involved (Hill, 1970; Muraoka *et al.*, 2004b). Second, a tendon's stiffness influences the proportion of a muscle-tendon unit's length change completed by the tendon and muscle, respectively (Proske and Morgan, 1987). This influence has potential implications for movement efficiency by influencing both the length of the active muscle fibres (Proske and Morgan, 1987; Lichtwark and Wilson, 2007) and the elastic energy storage capacity of the tendon (Biewener *et al*., 1998). Finally, tension-sensitive mechanoreceptors located in the muscle (golgi tendon organs and muscle spindles), which provide important proprioceptive feedback (Mora *et al.*, 2003; Granacher *et al.*, 2010), would be influenced by the tendon's stiffness and subsequent length change of the muscle fibres. This feedback may be greater when tendons are stiffer, which could influence spatial awareness of the limbs (Proske and Morgan, 1987) and impact on the

regulation of balance and motor control. Conversely, it is possible that training-induced increases in tendon stiffness without concurrent muscle adaptations may cause some functional negative effects, such as a capacity for elastic energy storage (Lichtwark and Wilson, 2007).

6.4.5 Conclusion

In summary, the present results demonstrate, for the first time, that tendons show a considerable adaptive response to strength training in prepubertal children. An increase in Achilles tendon stiffness was found without changes to the tendon's CSA, therefore microstructural changes in the tendon must have occurred to increase Young's modulus. A decrease in EMD was associated with the increase in tendon stiffness, however RFD was unchanged, suggesting that mechanisms other than tendon stiffness, such as the rate of muscle activation, might regulate RFD in children. Nonetheless, tendon adaptation in response to chronic increases in loading improves fast muscular force production in children and therefore has the potential to influence movement performance. The findings from this study may help to explain the differences in movement performance between children and adults.

CHAPTER 7: General Discussion

7.1 Introduction

Motor skill acquisition is a multifactorial process (McGraw, 1943; Zelazo *et al.*, 1972; Hopkins and Westra, 1988; Thelen, 1995). The maturational status of the nervous system (McGraw, 1943; Forssberg, 1985, 1999) and coordinative aspects of movement(i.e. neuromotor coordination: Turvey, 1990; Shumway-Cook and Woollacott, 2007),are important contributors to observed differences in movement performance between children and adults. In addition to the maturation of the neuro-motor system, the importance of non-neuromuscular factors, such as changes in segmental mass distribution (Jensen, 1989)as a contributor to motor skill acquisition in children (Brown and Jensen, 2006; Korff and Jensen, 2008) has been highlighted. Expanding on these recent findings the main purpose of this research was to examine the role of the mechanical properties of the muscle-tendon complex in the acquisition of force production capabilities in children. For this purpose, four experiments were conducted. First, we sought to gain an understanding of dimensional factors influencing age-related increases in tendon stiffness. To achieve this goal, the predictiveness of the Achilles tendon moment arm (a measure necessary to calculate tendon stiffness) was documented as a function of superficial anthropometric characteristics. Secondly, age-related increases in tendon stiffness were documented with a particular focus on differentiation between growth-dependent and maturational changes. The final two experimental chapters were then conducted with the goal of specifically creating the link between tendon stiffness and force production capabilities in children. In Chapter 5, the relationship between tendon stiffness and force production characteristics in children were examined in relation to that found in adults. This relationship was further examined in Chapter 6 by assessing the effect of a period of strength training on this relationship in children. The Achilles tendon was targeted for particular examination

because of its significance in many activities of daily living and exercise-related tasks. Children from 5 to 12 years of age were studied in order that a more complete understanding of the role of tendon stiffness could be gained throughout childhood. An overview of each experimental study and the main findings is presented in Table 7.1.

Table 7-1. Overview of thesis findings.

Chapter	Study Title	Methodology	Results	Conclusion
Chapter 3 (Study 1)	Can Achilles tendon moment arm be predicted from anthropometric measures in prepubescent children?	49 children (5 – 12 years). Achilles tendon moment arm (MA_{AT}) was estimated from tendon excursion using ultrasonography. Superficial anthropometric measures of the lower leg and foot, age, standing height and body mass were used as predictive variables.	Foot length and the distance between the calcaneus and first metatarsal were significant predictors of MA_{AT}. However, only 49% of MA_{AT} variability could be predicted using these variables.	MA_{AT} could not be accurately predicted from superficial anthropometric measurements in children.
Chapter 4 (Study 2)	Independent effects of age, body mass and muscular strength on the mechanical properties of the Achilles tendon in prepubertal children.	52 children (5 – 12 years) and 20 adults (10 men and 10 women). Tendon mechanical properties were measured and relationships with body mass, peak force and age were determined. Tendon dimensions and mechanical properties were also documented as a function of age.	Stiffness and Young's modulus increased throughout childhood and into adulthood. Body mass and tendon force could account for 66% and 39% of the variability in tendon stiffness and Young's modulus, respectively, in children.	Increases in tendon stiffness with age during childhood are largely attributable to the effects of increasing body mass and peak force production leading to both an increased tendon CSA and size-specific stiffness (Young's modulus).
Chapter 5 (Study 3)	The influence of tendon stiffness and muscle activation rate on muscle force production in children and adults.	48 children (5 – 12 years) and 20 adults (10 men and 10 women). Tendon stiffness, electro-mechanical delay (EMD), rate of force development (RFD) and rate of muscle activation (RER) were measured and relationships between the variables were documented.	EMD, RFD and RER were significantly different between children and adults. EMD and RFD were significantly correlated with tendon stiffness. Together, RER and tendon stiffness best predicted RFD in children.	Tendon stiffness impacts considerably on EMD whilst both RER and tendon stiffness appear to have separate but additive influences on force production in children.
Chapter 6 (Study 4)	Resistance training increases tendon stiffness and influences rapid force production in prepubertal children.	10 experimental and 10 controls. Achilles tendon mechanical properties were measured before and after 10 weeks of dynamic plantarflexor resistance training.	Increases in peak force, tendon stiffness and Young's modulus occurred in response to the training. EMD decreased and RFD and RER remained unchanged after training.	Resistance training causes adaptation of the developing Achilles tendon. Increases in tendon stiffness were associated with decreases in EMD, but did not influence RFD.

7.1.1 Predictability of Achilles Tendon Moment Arm with Age-Related Development in Children

Moment arm measurements on a large number of individuals can be challenging, as obtaining estimations of this measure typically requires specialist knowledge and expensive resources. Given that the distance between the centre of joint rotation and a tendon should increase with skeletal growth, it was hypothesised that there should be a relationship between the dimensions of a joints anatomic landmarks and muscle-tendon moment arm length. The purpose of the first study (Chapter 3) was to determine the predictability of the Achilles tendon moment arm from superficial anthropometric measurements. It was found that 47% of the variability in MA_{AT} could be accounted for by using a combination of foot length and the distance between the calcaneus and 1st metatarsal head. The calculation of force is sensitive to small variations in MA_{AT}, therefore it was decided that MA_{AT} could not be predicted accurately enough from the anthropometric measures that were collected in children for calculating precise tendon forces. For example, the greatest difference in predicted and actual MA_{AT} found in this study would have resulted in an error in the prediction of calculated tendon forces of ~32%, which would undoubtedly have a significant effect on tendon stiffness calculations. This finding, in conjunction with the wide range of MA_{AT} lengths found (17 – 38 mm) in the age range studied (5 – 12 years), demonstrates that simple allometric scaling should not be used to estimate MA_{AT} in children. In fact, due to the unpredictable nature of the MA_{AT}, it was considered essential that each child's own MA_{AT} be used in calculating tendon stiffness in the subsequent studies. Although the choice to measure each of the anthropometric measurements was carefully considered and based on their theoretical relationship to MA_{AT}, the possibility exists that body anthropometric

measurements that were not collected in this study may provide better predictions in children. Other researchers may choose alternative measurements in future studies.

7.1.2 Changes in Tendon Stiffness with Age-Related Development in Children

One of the primary aims of this thesis was to document Achilles tendon stiffness as a function of both age and age-related parameters such as body mass and peak muscle force (i.e. strength). This was deemed important as there is a lack of information describing the development of tendon stiffness (particularly of the Achilles) during childhood (Cornu and Goubel, 2001; Kubo *et al.*, 2001b; Lambertz *et al.*, 2003; O'Brien *et al.*, 2010). As a result, there are no normative data references for researchers or clinicians. In Chapter 4, Achilles tendon stiffness and its relationship with age-related parameters were described over a wide range of prepubertal ages (5 – 12 years). Changes in tendon stiffness of the magnitudes were found (300% from childhood to adulthood), which could be expected to have a substantial impact on the functional performance of the muscle-tendon unit in several ways. Firstly, a more compliant tendon elongates further under load compared to a stiffer tendon. This might result in a greater elastic storage ($E = ½ kx^2$, where E is the energy stored, k is the tendon stiffness and x is the elongation of the loaded tendon) and potentially an improved movement economy (Alexander and Bennet-Clark, 1977; Cavagna *et al*., 1977)in some stretch-shorten cycle activities (Bobbert *et al.*, 1986; Blazevich, 2011). The advantages associated with a stiffer tendon include a smaller overall fascicle shortening and slower shortening velocity for a given joint angle displacement or angular velocity. According to the force-length and force-velocity relationships, this would positively impact on peak force production. A stiffer tendon would also allow a greater speed of tendon recoil, based on a greater restoring force when the tendon is stretched, according to

Hooke's law (F = -kx). This relationship provides important joint power for the fast completion of a task (Ker *et al.*, 1988). In addition, a shorter time would be required to stretch the tendon for effective force transmission (Komi, 1986; Aura and Komi, 1987)due to a shorter EMD and greater RFD, thus a shorter muscle activation period would be required in order to achieve the same movement outcome as that with a compliant tendon. Importantly, this should incur a lesser metabolic cost. With the above relationships and associations in mind, it would appear that the tendon has to compromise between the conflicting requirements of appropriate force transmission and movement efficiency (Proske and Morgan, 1987). Nonetheless, the lesser Achilles tendon stiffness found in children necessitates that muscle forces must be applied with a different magnitude and timing than that in adults in order to stretch the tendon appropriately for successful movement outcomes. Further research is therefore necessary to quantify the relationships between tendon stiffness, muscle activation strategies and movement efficiency (Lichtwark and Wilson, 2007) in children. Such research would provide a real indication of whether the mechanisms influencing movement efficiency are different in children and adults.

7.1.3 Increases in Tendon Stiffness: Influence of Age and Force Transfer Requirements.

Increases in both body mass and plantarflexor force production with age were found to be more strongly related to increases in Achilles tendon stiffness and Young's modulus in children than age *per se* (Chapter 4). During daily activities and locomotor tasks, increases in muscle force requirements would likely provide a constant mechanical stimulus that would require steady tendon adaptation in order to maintain the same muscle-tendon dynamics and hence functional performance. In Chapter 6, it was also shown that peak muscle force production and body mass each likely provided an independent stimulus for tendon

adaptation, because muscle force production increased after 10 weeks of strength training without concomitant increases in body mass; the training resulted in a substantial increase in tendon stiffness (35%) and Young's modulus (33%). Importantly, these changes were found to occur without a detectable increase in tendon CSA (i.e. tendon hypertrophy), which shows that, similar to skeletal muscle adaptation, adaptations in the tendon's mechanical properties in children can occur without significant hypertrophy. The adaptation of the developing human tendon observed in response to resistance training is an important and novel finding. The practical implications of this finding are vast, and include the potential to increase a tendon's stiffness with specific resistance training in order to minimise the movement consequences of neuromuscular and musculoskeletal disorders that are associated with a decrease in MTU stiffness, such as hypermobility (e.g. Ehlers-Danlos syndrome, Marfan syndrome), or for enhancing athletic or general movement performance in children, based on the information outlined in the previous section. The potential effects of strength training in children, especially in clinical populations, should be the subject of considerable future research.

7.1.4 Separating the Effects of Tendon Stiffness and Muscle Recruitment on Force Production

To understand how humans control both simple and complex movements, knowledge of muscle force production characteristics is imperative. Until now, the importance of tendon-specific stiffness on movement production in children has received little attention. The present data show that the tendon's mechanical properties are an important determinant of muscle force production in this population, and have the potential to influence motor control strategies. Despite only a weak relationship being found between RFD and tendon

stiffness, which is consistent with that found in adults (Bojsen-Moller *et al*., 2005), the relationship between EMD and tendon stiffness was clearer: an increased tendon stiffness was associated with a shorter EMD. A shorter EMD reduces the interval between a stimulus and the onset of muscle force and thus reduces the time required to generate a specific force or successfully complete a task (for example, regaining balance after a perturbation). Thus, it might be speculated that having a stiffer tendon would be beneficial for balance recovery following a perturbation or for other tasks requiring short response times. This is an important avenue for future study.

7.1.5 Possible Limitations

The use of ultrasonography has proven an invaluable tool in determining the structure, function and mechanics of soft tissues *in vivo*. However, there are some limitations to its use in imaging tendon elongation in real time. One possible limitation is that variations in the orientation of the ultrasound probe with respect to the plane in which excursion of the MTJ occurred can induce an error. Off-plane observations would cause an underestimation of the tendon's elongation (Loram *et al*., 2006; Wilson *et al*., 2009), which has repercussions for calculating tendon stiffness. Extensive practice of imaging the GM MTJ was done during pilot testing, resulting in highly reliable data, thus this limitation is likely to be minimal. Other limitations include a requirement to manually digitise the position of the MTJ during active conditions, which is particularly time consuming and allows for measurement subjectivity. Although an automated tracking algorithm (Lee *et al*., 2008) was trialled to limit investigator-dependent subjectivity, there were several drawbacks associated with it, including the fact that it had not yet been validated for use in active muscle conditions. Thus

it was not used in the present research. Extensive practise of the manual digitisation procedure during pilot testing allowed a high reliability to be obtained (CV = 4.5%).

There are also several limitations to the tendon excursion (TE) method for determining MA_{AT}(Fath *et al.*, 2010). One such limitation central to this thesis is that MA_{AT}, used in calculating tendon stiffness, increases as a function of muscle contraction intensity (Maganaris, 2004) and is not detected using the TE method. Maganaris *et al*, (2004) found a 22 – 27% increase in the MA_{AT} during a maximal isometric contraction using MRI. As there is a strong correlation between MA_{AT} calculated using TE and COR (Fath *et al.*, 2010), consideration was given to scaling up the MA_{AT} by a similar factor to represent an active MA_{AT}. However, this was ultimately deemed unnecessary because the same method of analysis was used for all participants, thus any results or relationships found in the research conducted would not have been affected. Leaving the data unscaled also allowed direct comparison with previously published data (which were also not scaled).

7.1.6 Conclusion

The main findings from the four experimental studies (Chapters 3 – 6) include that there is an age-related increase in Achilles tendon stiffness that continues through to adulthood, but that this increase was strongly and independently associated with body mass and peak force production capacity rather than age *per se*. It is likely that the increased loading stimuli provide the tendon with a constantly increasing mechanical stimulus during standing, locomotion and other tasks. The age-related increase in tendon stiffness was associated with increases in tendon CSA and Young's modulus, although the increase in Young's modulus is suggestive of microstructural tendon adaptation from mechanical loading also

(Chapter 4). Achilles tendon stiffness was strongly linked with force production characteristics in children. A moderate proportion of EMD and RFD variability could be predicted from tendon stiffness in both children and adults, although RFD predictability was significantly better when rates of muscle activation (i.e. RER as calculated in the present research) were also included in a regression model (Chapter 5). In the final study, significant increases in Achilles tendon stiffness were found in children after 10 weeks of resistance training without a detectable increase in tendon CSA. This finding demonstrates that chronic loading from brief, high muscle forces provides sufficient stimulation for tendon adaptation in children. In support of the relationships found in Chapter 4, this finding also shows that body mass and peak muscle force have independent influences on tendon stiffness, because peak strength increased significantly as a result of the training whilst body mass remained unchanged. EMD was reduced as a consequence of both age- and training-induced increases in tendon stiffness, but RFD was unchanged after the strength training. This latter result shows that more substantial increases in tendon stiffness might be required before significant changes in RFD are identified. It is also suggestive that other factors such as the rate of muscle activation is a primary determinant of RFD in children; the training did not induce a change in the rate of muscle activation. Although movement performance was not the focus of the present research, the changes in muscle force production and tendon properties with both ageing and strength training in the children could be expected to substantially influence it. Thus, tendon stiffness should not be overlooked as a factor contributing to differences in performance between adults and children and deserves to be fully investigated in future research.

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APPENDICES

Appendix I

Ethical Approval – Studies 1, 2 and 3.

Head of School of Sport & Education
Professor Susan Capel

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Charlotte Waugh
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Brunel University

15th June 2008

Dear Charlotte

RE70-07 – Age-related Differences in Tendon Stiffness and the Effect on Force Production

I am writing to confirm the Research Ethics Committee of the School of Sport and Education received your application connected to the above project. Your application has been independently reviewed and I am pleased to confirm your application complies with the research ethics guidelines issued by the University.

On behalf of the Research Ethics Committee, I wish you every success with your study.

Yours sincerely

Dr Simon Bradford
Chair of Research Ethics Committee

Ethical Approval – Chapter 6

Head of School of Sport & Education
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Charlotte Waugh
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2nd December 2008

Dear Charlie

RE96-07 - Effects of Strength Training on Achilles Stiffness in Children

I am writing to confirm the Research Ethics Committee of the School of Sport and Education received your application connected to the above mentioned research study. Your application has been independently reviewed to ensure it complies with the University Research Ethics requirements and guidelines.

The Chair, acting under delegated authority, is satisfied with the decision reached by the independent reviewers and is pleased to confirm there is no objection on ethical grounds to the proposed study.

Any changes to the protocol contained within your application and any unforeseen ethical issues which arise during the conduct of your study must be notified to the Research Ethics Committee for further consideration.

On behalf of the Research Ethics Committee for the School of Sport and Education, I wish you every success with your study.

Yours sincerely

Dr Simon Bradford
Chair of Research Ethics Committee
School Of Sport and Education

Appendix II

Information sheets – Studies 1, 2 and 3.

Age-related differences in muscle-tendon stiffness

PARTICIPATION INFORMATION SHEET
(Child participant)

We are Charlie Waugh, and Florian Fath, two PhD students at Brunel University who would like to discover how strong children are at different ages.

You are invited to participate in a study which measures how strong you are and how your muscle and tendon move. We are trying to find out why children move differently to adults.

If you decide to take part, first you will do some star-jumps for warming up, then you will be seated on a strength testing machine and asked to push twelve and pull two times with your foot as hard and fast as possible against the machine. We will put shiny reflective stickers on your foot and leg to see how much it moves using special cameras. We will also use sticky plasters which will be connected to a computer with some wires to measure how hard your muscle is working. To put the markers on, you will have to take the sock of your right foot off and roll your trousers up to the knee. We will hold a machine against the outside of your leg which will let us see inside your leg. This is the same machine used to see babies in their mother's tummy - it uses a jelly that might be cold! You will be able to see how your muscles move when you push against the machine. We will also take some measurements of your height, weight and leg length.

If you feel uncomfortable or have any pain during the test, please let us know. You can stop at any time if you wish. We are more than happy to answer any questions you have. Please don't hesitate to ask.

As a thank you, you will get a certificate with how strong you are and a picture of your leg!

Age-related differences in muscle-tendon stiffness

PARTICIPATION INFORMATION SHEET
(Parent/Guardian)

We are Charlie Waugh, and Florian Fath, PhD students from the School of Sports Sciences and Education at Brunel University researching muscle and tendon stiffness, particularly how these change during childhood.

There is very little data on stiffness in children and even less specific to tendon stiffness. Tendon greatly influences movement, and there is a need for documenting 'normal' stiffness ranges in children of different ages. This will help us to understand why children differ to adults in the way they recruitment their muscles, and provide a valuable tool to clinicians and coaches.

Your child is invited to participate in a study which will investigate these aspects of the ankle joint during active and passive movements. The data we collect will be then be used to develop a computerised 'model' of the lower limb.

As a participant, your child will be asked to:

- Familiarise themselves with the equipment before the testing session to get used to the equipment and the required movements.
- Allow their height, weight, and some leg measurements to be taken.
- Perform a warm-up by doing 1 minute of star-jumps
- Perform 12 ankle 'pushes' and 2 'pulls' against a fixed plate which is part of a strength testing machine.

Each muscle effort will last 3-4 seconds, which is long enough for us to obtain sufficient data but short enough to prevent the muscle from getting tired. Each effort will be followed by a 1 minute rest. Including the time needed for preparing and familiarising your child with the equipment, the testing time will take no longer than 45 minutes.

During the testing, we will record your child's muscle activity using sensors placed on the leg. This will require trousers to be rolled to the knee and removal of the right shoe and sock. The sensors used are very common in this type of research and no problems have been reported following their use, although slight discomfort or minor skin irritations may be experienced when removing them, not unlike a plaster. We will also use ultrasound to collect images of the muscle and tendon complex by placing a probe and a water-based gel on the skin of the leg. This is also a common and pain-free procedure.

Filenames in data collection will be coded to protect personal details. **All personal information will remain confidential, and no personal information will be disclosed**

without permission. Unfortunately, we cannot offer monetary reimbursement for your child's participation in this study. There are no direct benefits to participating in this research study; however your assistance through participation will help us to better understand age-related changes in muscle-tendon elastic properties around the ankle joint. In addition, they will learn about how muscle and tendon work together and how they relate to strength and movement. An ultrasound image of your child's lower leg will be provided on request. The decision to allow participation or not will not affect you or your child's association with Brunel University. If you decide that you child may participate, you are free to withdraw from participation at any point without penalty.

We accept no responsibility in the unlikely event of injury as a result of your child's participation in this study. Basic first aid will be provided at the time of injury however no payment can be provided in the event of a medical problem. You will be encouraged to consult your General Practitioner under these circumstances.

Your child's participation will help us to better understand age-related changes in muscle-tendon stiffness. If you or your child have any questions, we will be more than happy for you to contact us on 01895 266500, or email Charlotte.Waugh@brunel.ac.uk and Florian.Fath@brunel.ac.uk. Alternatively, please contact Dr Thomas Korff on 01895 266477 on Thomas.korff@brunel.ac.uk.

Information sheets – Chapter 6.

Dear Parent,

Researchers at Brunel University are investigating the influence of strength training on movement efficiency and muscle force production in prepubescent children. As part of a collaboration with Ravenor Primary school, your child has been invited to participate in a project organised by the School of Sport and Education at Brunel University, looking at strength and stiffness of the lower leg with age and training.

A 10-week study will be conducted where the children complete a resistance training session during their P.E lessons. Such training has been shown to have substantial health benefits in children and is very safe to do; the programme has been specifically designed for the children in year 4. The training will be performed in circuit fashion, where the children perform several repetitions of an exercise before moving immediately to another. The exercise programme will be completed twice a week for 10 weeks during the school day. A leaflet outlining the benefits of this type of training has been included with this letter for your information.

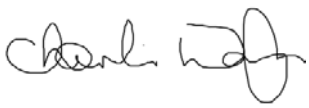
In addition to training, the project will require the pupils to come to our biomechanics department at Brunel's Uxbridge campus on two occasions – before and after the 10 weeks of training. This will be organised as part of a science morning/afternoon where children will learn about the effects of exercise on the body by participating in some simple but interesting experiments e.g. see how much oxygen they breathe whilst cycling on a stationary bike. There is also the possibility of having a lesson on our indoor climbing wall! A mini-bus will be arranged to transport the children between school and the University.

On their visits to Brunel, each student will have their strength tested. The strength test uses 3-Dimensional motion capture cameras (like the ones used in the CGI movies!) to look at movement, and an ultrasound machine to record videos of the muscle during the test - all whilst our strength testing machine tells us the strength of the muscle. The experience is a very fun, interesting & informative one! Children who complete the study will be awarded with a personalised certificate as well as an ultrasound picture of their leg muscles.

The data we collect from this project will be used for research purposes, and the data will be disseminated through published work and oral presentation. We therefore ask for you and your child's written informed consent to participate. If you agree to your child taking part after reading the attached research participation form, I would appreciate if you could fill out the attached consent and screening forms.

Thank you in advance for your time and cooperation. Please contact Mrs Hancell or myself if you have any further questions.

Sincerely,



Charlotte Waugh
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Effects of strength training on Achilles tendon stiffness in children

RESEARCH PARTICIPATION INFORMATION SHEET (Child participant)

You are invited to participate in a study which measures how strong you are and how your muscle and tendon get stronger with training as part of your class.

Your school has been given some strength training equipment, which you can use to improve your strength and even speed! If you decide to take part, you will be asked to use the equipment twice a week for 12 weeks (one school term). Your 12 week training plan will be designed from finding out how strong you are. This will be tested by me at Brunel University before the training begins. To see how well you are doing, you will also need to come to Brunel University on two other occasions, in the middle and at the end of 12 weeks.

First, you will do 5 minutes of gentle cycling and hopping, then you will be seated on a strength testing machine and we will ask you to push with your foot as hard and fast as possible against the machine. We will put shiny reflective stickers on your foot and leg to see how much it moves using special cameras. We will also use sticky plasters which will be connected to a computer with some wires to measure how hard your muscle is working. We will also use a device which enables us to see inside your leg which uses a jelly that might be cold! You will be able to see how your muscles move when you push against the machine. We will also take some measurements of your height, weight and leg length. Finally I will ask you to do some hopping exercises on a platform which can measure forces put on it. This shouldn't take more than 45 minutes.

If you feel uncomfortable or have any pain during the test, please let us know. You can stop the test at any time if you wish. We are more than happy to answer any questions you have. Please don't hesitate to ask. As a thank you, you will get a certificate with how strong you are, and a picture of your leg muscles!

Effects of Resistance training on Tendon Stiffness in Children

RESEARCH PARTICIPATION INFORMATION SHEET (Parent/Guardian)

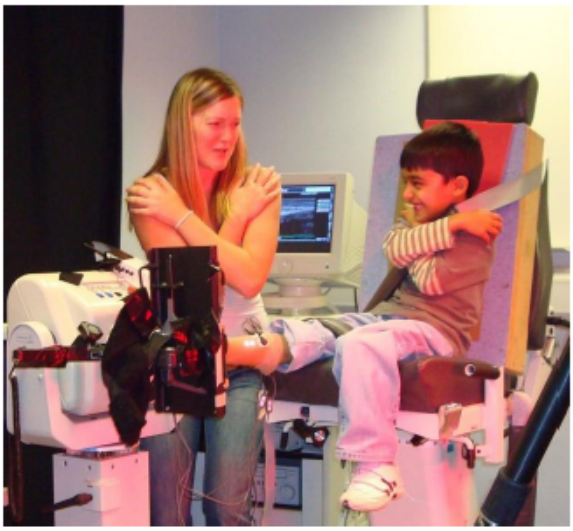
We at Brunel University, Uxbridge, are currently investigating the influence of the Achilles tendon (calf muscle's tendon) stiffness on muscle force production and movement strategies in prepubescent children. In exploring this, your child has been invited to participate in this study as part of their school class.

Increasing tendon stiffness in children may result in more adult-like movement, making it more efficient - this may benefit performance in sports requiring fast force production or simply in developing a child's ability to learn new movements.

Resistance training in children has become increasingly popular and been proven effective in promoting strength gains, improving body composition and optimising skilled performance. Because of the potential effects of resistance training on tendon stiffness, and, it is necessary to examine the influence this might have on movement, which has yet to be explored in children. A 12-week strength training study has been planned to allow us to investigate this for the first time The study requires participants to come to our biomechanics department prior to the training (week 0), and after 12 weeks of training. The set-up of this testing is shown below and will be conducted in a fun and informative manner.

Brunel University Data Collection

- Height, weight and age
- Images of the muscle and tendon will be collected by placing an ultrasound probe and a water-based gel on the skin of the leg. This is a common and pain-free procedure.
- Muscle activity will be recorded using sensors placed on the muscle 'belly' on the leg, common in this type of research. No problems have been reported following their use, although slight discomfort or skin irritation may be experienced on removal, akin to a plaster.
- Maximum calf strength will be determined by performing several maximal voluntary contractions whilst sat on a dynamometer (see picture). Each contraction will last approximately 4 seconds, followed by a 30 second rest period.



- Finally, 5 x 20 seconds of hopping will be completed on a force-measuring platform (like fancy bathroom scales), whilst infrared motion cameras detect joint movement using reflective markers positioned on the leg.

All testing will be carried out by Charlotte Waugh, whilst data collection on computer will be conducted by an additional researcher. All classroom-based training will be also supervised.

Classroom-Based Training

- Circuit training will be based at Ravenor Primary school – a 5 minute warm-up prior to exercise will be done, with a cool down and stretching to complete the session.
- Exercises included in the circuits will be a mix of cardio and core muscle training e.g. 30 second hula hoop, 1 minute of sit-ups etc. There will be 10 different stations.
- One exercise will involve resistance training completed on a leg press. To begin with, 2 sets with a light resistance will be carried out, building to 3 sets at a moderate resistance of after 2 weeks.
- After 4 weeks and for the remaining training period, 4 sets of moderate/heavy resistance will be completed. This progression is designed for safety as well as program effectiveness.

Children with orthopaedic/musculoskeletal disorders, diabetes, chronic paediatric disease or volunteers who are/have previously experienced resistance training will be unsuitable for participation.

As with all maximal exercise, there is a small chance of muscle soreness or injury, although proper warm-up, the gradual design of the training program and instruction under close supervision should further minimise these risks. Although we cannot guarantee that no injury will occur, the University is not responsible for such occurrences. A qualified First Aider will be available at all times however no payment can be provided in the event of a medical problem. You will be encouraged to consult your General Practitioner under these circumstances. All researchers involved have a valid CRB certificate.

Filenames in data collection will be coded to protect personal details. **All information will remain confidential, and no personal information will be disclosed without permission.** Unfortunately, we cannot offer monetary reimbursement for your child's participation in this study. Direct benefits from participation in this research study include improved fitness, strength, and body awareness which will aid in other sports. In addition, your child will learn about muscle structure and function and how they relate to strength and movement, amongst other information on healthy lifestyles.

Your child's participation will help us to better understand the influence of increasing strength on tendon stiffness and the implications this has for movement performance. The decision to participate or not will not affect you or your child's association with Brunel University. If you decide that your child may participate, you and your child are free to withdraw from participation at any point without penalty.

A leaflet highlighting the benefits of strength training is included with this letter. We will be more than happy for you to contact us if you have any questions. Our full contact details are listed below.

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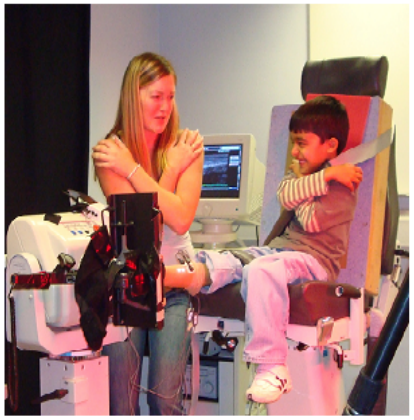
Brunel UNIVERSITY WEST LONDON

Benefits and Misconceptions of Strength Training in Children



Centre for Sports Medicine and Human Performance
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Resistance training in children has become increasingly popular and been proven effective in promoting strength gains, improving body composition and optimising skilled performance. As a result, we have seen a boom in youth gyms and child-sized training equipment, the message regarding the resistance training for health benefits in children is quite clear.

A growing body of literature into the health benefits and effects of strength training for young people have so far proved the following:

- Develops greater muscle strength & endurance without increased muscle size
- Improved motor skills, coordination & performance
- Increased lean body mass, improving body composition and helping to reducing obesity
- Increased bone mineral density to strengthen bones. Decreases risk of osteoporosis later in life.
- Improved body image & self-esteem
- Promotes favourable attitude towards lifelong physical activity
- Helps in resisting injury in other sports

Example of child-sized resistance training equipment.

Myths surrounding Strength Training...

"Resistance Training will stunt the growth ".
There are no observations indicating a decrease in stature of children who regularly participate in strength training programs in a controlled environment.

"Youth resistance training is unsafe".
The risks associated with strength training in children are no greater than with other activities given appropriate supervision

"Children cannot increase strength because they have low levels of testosterone".
Testosterone is not essential for achieving strength gains – women and elderly people are able to achieve strength gains without high levels of the hormone.

"Resistance training is only for young athletes".
Although resistance training may improve sporting performance of young athletes, regular participation in resistance training by non-athletic children may provide considerable health benefits, whilst being fun and social.

For further information on resistance training in children, please consider reading the references listed.

- Strength training by children and adults – Committee on sports medicine & fitness (by American Association of Pediatrics, available online)
- Strength training for children – Peak Performance (www.pponline.co.uk/encyc/0835.htm)

Appendix III

Consent forms – Studies 1, 2 and 3.

Age-related changes to Achilles tendon stiffness in children

PARTICIPANT CONSENT FORM
(Child participant)

STATEMENT

I agree to take part in this project.

I know that I can stop at any time.

I have been read the research information sheet and am aware of what participation will involve.

All questions that I have about the training study have been answered in a way I understand.

Name: _____

Date: _____

Age-related changes to Achilles tendon stiffness in children

INFORMED CONSENT FROM
(Parent/Guardian)

STATEMENT

1. I agree to my child participating in this project
2. I have read the research participant sheet and am aware of what my child's participation will involve.
3. I understand that my child's participation is voluntary and that I may withdraw them from the research at any time, without giving any reason. My decision not to allow participation will not alter the treatment my child would normally receive now or in the future.
4. I understand the risks involved in the participation of this study.
5. All questions that I have about the research have been satisfactorily answered.
6. I agree to these results being used for education and research on condition my child's privacy is respected.

Participant's name: _____

Parent / Legal guardian's name (print): _____

Parent / Legal guardian's signature: _____

Date: _____

Tick this box if you would like to receive a summary of the results by e-mail ☐

Tick this box if you are interested in further studies, and if it is ok for us to contact you in case ☐

Sign here if it is ok for us to take pictures of the testing. These pictures might be used for scientific communications such as posters or power point presentations:

_____

E-mail Address: _____

CONSENT FORM

Easter Sports Science Activity Day

The participant should complete the whole of this sheet him/herself with his/her guardian		
	Please tick the appropriate box	
	YES	**NO**
1. I have read the information letter about the Science Project Day at Brunel University, or has it been read to me.	☐	☐
2. I agree that, for education purposes, my child will perform simple jumping, strength and breathing tasks.	☐	☐
3. I agree that force platform (bathroom scale) measurements, or force measurements will be taken during these activities.	☐	☐
4. I agree that the measurements taken during my/my child's activities can be used for a research study.	☐	☐
5. If the answer to 4. is yes, I understand that I/my child can withdraw from the study at any time without having to give a reason for withdrawing	☐	☐
Signature of Research Participant (child) and Date:		
Name in capitals:		
Signature of Parent/Legal Guardian and Date:		
Name in capitals:		

Personal Information

Child Name_____ **Name of Parent Guardian**

_____

Date of Birth:_____ **Address:**_____

e-mail: _____ **Phone Number:** _____

	YES	**NO**
I am interested in other studies related to child development that are being conducted at Brunel University. Please contact me if there is the possibility for my child to participate in any of your studies.	☐	☐

CONSENT FORM

The influence of strength training on movement efficiency

The participant should complete the whole of this sheet him/herself with his/her guardian	*Please tick the appropriate box* YES	NO
1. I have read the information letter about the Science Project Day at Brunel University, or has it been read to me.	☐	☐
2. I agree that, for education purposes, my child will perform simple jumping and cycling tasks.	☐	☐
3. I agree that force platform (bathroom scale) measurements, Video, or force measurements will be taken during these activities.	☐	☐
4. I agree that the measurements taken during my/my child's activities can be used for a research study.	☐	☐
5. If the answer to 4. is yes, I understand that I/my child can withdraw from the study at any time without having to give a reason for withdrawing	☐	☐
Signature of Research Participant (child) and Date:		
Name in capitals:		
Signature of Parent/Legal Guardian and Date:		
Name in capitals:		

Personal Information

Child Name_____ **Name of Parent/Guardian** _____

Date of Birth:_____ **Address:**_____

e-mail: _____ **Phone Number:** _____

	YES	NO
I am interested in other studies related to child development that are being conducted at Brunel University. Please contact me if there is the possibility for my child to participate in any of your studies.	☐	☐

Appendix IV

Screening Forms

Children's Screening Form

Is your child taking any medications? If yes, please list .....

.....

Has your child been on any short term/long term medication? Yes/ No

If yes, please give details .....

.....

Has your child ever suffered from any of the following (Tick if yes)

Chest pains	( )	Asthma	( )
Joint problems	( )	Severe headaches	( )
Lower back problems	( )	Diabetes	( )
Heart problems	( )	Epilepsy	( )
Glandular fever	( )	Anemia	( )
Cystic fibrosis	( )	Muscular dystrophy	( )
Heavy nosebleeds	( )	Infectious diseases	( )

Has your doctor ever advised your child not to exercise? Yes / No

If yes, please give details .....

.....

Does your child take part in any regular physical activity now? Yes/No

If yes, please give details .....

.....

Are there any medical reasons why your child should not take part in energetic activity?

Yes / No If yes, please give details .....

.....

I, the parent/guardian of the aforementioned child acknowledge the above information to be factually correct. To the best of my knowledge I have given all relevant information on my child's health and activity. I therefore agree to my child's participation in the activities of We R Kids Gyms.

Signed .....

Print Name .....

Appendix V

Waugh, C.M., Blazevich, A.J., Fath, F. and Korff, T. Can Achilles tendon moment arm be predicted from anthropometric measures in pre-pubescent children? Journal of Biomechanics (2011), doi:10.1016/j.biomech.2011.03.023