2014

Relative importance and plasticity of anatomical and neuromuscular factors affecting joint torque production

Joanne C. Trezise

*Edith Cowan University*

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Relative importance and plasticity of anatomical and neuromuscular factors affecting joint torque production

By

Joanne C. Trezise

This thesis is presented for the award of Doctor of Philosophy (Sports Science) from the School of Exercise and Health Sciences; Faculty of Health, Engineering and Science; Edith Cowan University, Western Australia

Supervisor: Associate Professor Anthony J. Blazevich

Date of Submission: 31st March 2014
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ACKNOWLEDGEMENTS

To the people who have helped guide and support me through this journey, I finally have a chance to thank you.

To my supervisor Anthony Blazevich, without your encouragement and effort, this thesis would not have been possible. Thank you for your guidance, patience, dedication and astounding promptness in responding to my many questions and queries. Your enthusiasm and support throughout this process has been amazing.

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ABSTRACT

The present research aimed to determine (i) the relative influence of anatomical and neuromuscular variables on maximal isometric, concentric and eccentric knee extensor torque (Study 1); (ii) whether the change in strength following a 10-week strength training program is associated with changes in specific anatomical and neuromuscular variables (Study 2a); (iii) whether anatomical and neuromuscular adaptations are dependent on their pre-training magnitudes; and (iv) whether it is possible to 'predict' an individual's adaptation to strength training based on their anatomical and neuromuscular pre-training magnitudes (Study 2b).

The variables assessed throughout the studies include muscle cross-sectional area (CSA), fascicle length and angle from the proximal, middle and distal regions of the four quadriceps components; agonist (EMG:Mwave) and antagonist (EMG normalised to MVC) muscle activity, percent voluntary activation (%VA; interpolated twitch technique); maximum isometric and slow speed concentric and eccentric (60°/s), unpotentiated and potentiated twitch torques; and patella tendon moment arm distance.

Using a cross-sectional (observational) study design (Study 1; n = 56) models incorporating CSA, fascicle angle and muscle activity and activation were found to best predict both maximum isometric and eccentric torque ($R^2 = 0.72$ and 0.62). Maximum concentric torque was best predicted by a model incorporating CSA, fascicle angle and moment arm ($R^2 = 0.64$) making it suitable for predicting maximal torque in clinical/rehabilitation populations. Proximal CSA was included in the strongest models rather than the traditionally used mid-muscle CSA, indicating its potential functional importance. The strong predictive ability of models incorporating both CSA and fascicle angle indicate that the quantity of contractile tissue strongly influences inter-individual differences in strength expression.

Following 10 weeks of heavy lower-limb heavy strength training (Study 2a; n = 36), the change in isometric torque was best (although weakly; $R^2 = 0.27$) predicted by models incorporating the change in proximal-region vastus lateralis CSA and fascicle angle, and changes in concentric and eccentric torque were best predicted by average quadriceps muscle activity, proximal-region CSA (either vastus lateralis or whole quadriceps) and vastus intermedius fascicle angle ($R^2 = 0.40$ and 0.41). Changes in fascicle angle were weakly correlated with the change in strength despite its inclusion in the strongest models, highlighting the requirement to examine interactions between variables when assessing their influence on strength change. Furthermore, the weak relationships observed between the change in strength and the change in neuromuscular variables (Study 2a)
indicate that the assumption that simultaneous changes observed in strength, anatomical structure and neuromuscular function following training indicate potential causal association may need to be reconsidered.

While muscle activation measured pre-training during isometric contractions was moderately and negatively correlated with the strength change following training (Study 2b), there was no correlation for proximal-region CSA. This indicated limited scope for improvement in activation isometrically in individuals with greater levels of activation prior to training, but that all individuals had similar scope for hypertrophy. It was not possible to predict the strength change elicited by training from the measurements obtained before training \((R^2 = 0.06 \text{ to } 0.27)\).

A comparative data set presented in Study 1 provides clinicians with a tool to evaluate an individual’s maximum torque capacity, anatomical structure and neuromuscular function. While accurate prediction of strength change following training cannot be made based on pre-training testing using the current protocols (Study 2b), strength training programs targeted to improve muscle activation (Study 2a) might elicit the greatest improvements in concentric and eccentric knee extensor strength.
# Table of Contents

DECLARATION ........................................................................................................ ii
COPYRIGHT AND ACCESS STATEMENT ................................................................... iii
ACKNOWLEDGEMENTS ............................................................................................... iv
ABSTRACT .................................................................................................................. v
List of Tables ................................................................................................................. xi
List of Figures ............................................................................................................... xiii
List of Abbreviations ...................................................................................................... xiv

### CHAPTER ONE: INTRODUCTION ................................................................. 1

2.1 Introduction ........................................................................................................... 2

### CHAPTER TWO: REVIEW OF LITERATURE ............................................... 4

2.1 Overview ............................................................................................................... 5
2.2 Effect of muscle size on a muscle’s force producing capacity ......................... 6
2.3 Training-induced adaptations in muscle size ...................................................... 6
2.3.1 Factors influencing hypertrophy .................................................................. 9
2.3.2 Location of hypertrophy ............................................................................. 10
2.3.3 Relationship between changes in muscle size and changes in strength ........ 11
2.4 Effect of muscle architecture on a muscle’s force producing capacity ............. 12
2.5 Training-induced adaptations in muscle architecture ..................................... 13
2.5.1 Relationship between the change in muscle architecture and the change in strength 15
2.6 Effects of neural activation on a muscle’s force producing capacity ............... 15
2.7 Training-induced adaptations in muscle activation ......................................... 18
2.8 Influence of moment arm on torque production capacity ............................... 19
2.9 Training-induced changes in moment arm distance ......................................... 20
2.10 Considerations for determining relationships between strength and neuromuscular variables .......................................................................................... 20
2.11 Summary .......................................................................................................... 21

### CHAPTER THREE: PURPOSE OF THE RESEARCH .................................. 23

3.1 Overview ............................................................................................................. 24
3.2 Specific Aims and Hypotheses .......................................................................... 24

### CHAPTER FOUR: STUDY ONE ................................................................. 26

Anatomical and neuromuscular mechanisms influencing inter-individual variability in maximum knee extension torque ......................................................... 26

4.1 Introduction ....................................................................................................... 27
4.2 Methods ............................................................................................................ 29
Are changes in specific anatomical and neuromuscular variables associated with the changes in strength following 10 weeks of heavy strength training in previously untrained men?  

5.1 Introduction .............................................................................................................. 59  
5.2 Methods .................................................................................................................. 60  
5.2.1 Participants and Study Design ............................................................................. 60  
5.2.2 Training Program ................................................................................................. 61  
5.2.4 Measurements ...................................................................................................... 62  
5.2.5 Muscle Cross-sectional Area Analysis ................................................................. 62  
5.2.6 Data Analysis ........................................................................................................ 63  
5.3 Results ..................................................................................................................... 65  
5.3.1 Regression models ............................................................................................... 68  
5.3.2 Correlations .......................................................................................................... 73  
5.4 Discussion ................................................................................................................. 76  
5.4.1 Change in isometric torque versus changes in muscle size, architecture and activation 77
5.4.2 Changes in concentric and eccentric torque versus changes in muscle size, architecture and activation ........................................................................................................... 79
5.4.3 Are the neuromuscular variables correlated with maximum torque (cross-sectionally) also correlated with the change in torque following training (longitudinally)? ........................................ 83
5.5 Summary .......................................................................................................................................................................................... 84

CHAPTER SIX: STUDY TWO (b) ......................................................................................................................................................... 86
Can strength improvements and anatomical and neuromuscular adaptations be predicted from pre-training tests in previously non-strength-trained healthy men? .......................................................... 86
6.1 Introduction .................................................................................................................................................................................................................. 87
6.2 Methods ........................................................................................................................................................................................................ 88
6.2.1 Testing and training protocols .......................................................................................................................................................... 88
6.2.2 Data analysis ........................................................................................................................................................................................................ 88
6.3 Results ........................................................................................................................................................................................................ 89
6.3.1 Correlations between pre-training anatomical and neuromuscular variables and their change with training ............................................................................................................................................... 89
6.3.2 Correlations between pre-training torque and anatomical and neuromuscular variables and the change in torque with training ............................................................................................................. 90
6.3.3 Prediction models for the change in torque versus the combined pre-training torque and anatomical and neuromuscular variables ........................................................................................................ 94
6.4 Discussion ......................................................................................................................................................................................................... 95
6.4.1 Are changes in anatomical and neuromuscular variables dependent upon their pre-training magnitudes? ................................................................................................................................. 95
6.4.2 Are strength improvements following training dependent on pre-training anatomical structure and neuromuscular function? ............................................................................................................. 98
6.4.3 Are pre-training torque and anatomical and neuromuscular variables predictive of the change in torque following training? ........................................................................................................... 101
6.5 Summary .................................................................................................................................................................................................. 102

CHAPTER SEVEN: GENERAL DISCUSSION ........................................................................................................................................... 104
7.1 Overview ....................................................................................................................................................................................................... 105
7.2 Main findings ....................................................................................................................................................................................................... 105
7.3 Final Summary ............................................................................................................................................................................................ 108

REFERENCES .............................................................................................................................................................................................................. 110

APPENDICES ...................................................................................................................................................................................................... 121
Appendix 1. ................................................................................................................................................................................................................... 122
1A Ethical approval ..................................................................................................................................................................................................... 122
1B Information letter for participants – Study 1 ................................................................................................................................................ 123
Information letter for participants – Studies 2 and 3 ..........................................................127
Consent form – Study 1 ........................................................................................................131
Consent form – Studies 2 and 3 ..........................................................................................132
Pre-exercise medical questionnaire ..................................................................................134
Appendix 2. ..........................................................................................................................139
Calculated daily energy expenditure .................................................................................139
Calculation of knee extensor and flexor contribution to knee extension torque ..........140
Distribution of change in torque data ..............................................................................141
List of Tables

Table 2.1. Correlations ($R^2$) between muscle size measures and maximum joint torque measured during isometric, concentric and eccentric contractions ('Slow' and 'Fast' refer to ≤ 60°∙s$^{-1}$ and ≥ 180°∙s$^{-1}$, respectively) in healthy participants. 7

Table 4.1. Best-fit models for maximum isometric, and isokinetic concentric and eccentric torque (based on AIC$_C$) and the ‘best clinical model, with the equations provided to enable maximum torque predictions. 43

Table 4.2. Akaike's Information Criterion of model parameters for predicting maximal isometric (a), and isokinetic concentric (b) and eccentric (c) torque. The ‘best-fit model’ and the ‘best clinical model’ for each contraction mode are presented in bold. Models with both substantial support ($\Delta$AIC$_C$ ≤ 2) and AIC$_C$.W $\geq$ 0.10 (i.e. greater than a 10% chance that they will be the best fit model) are identified by shading. 45

Table 4.3. Correlations between individual anatomical and neuromuscular variables (predictors) and maximal isometric, and isokinetic concentric and eccentric knee joint torque. 47

Table 4.4. A comparative data set for healthy young males (aged 18-40 years) for predictors included in the ‘best-fit’, and ‘best clinical’ models, and those models with support for maximum net and total quadriceps isometric, and isokinetic concentric and eccentric torque prediction. 49

Table 5.2. Training loads, and torque, moment arm, and muscle activity and activation variables obtained before and after training during maximal isometric, and isokinetic concentric and eccentric contractions. 66

Table 5.4. The best-fit model for predicting changes in maximum isometric and isokinetic concentric and eccentric torque ($\Delta$T) from the changes in predictor variables ($\Delta$VAR). 69

Table 5.5. Akaike's Information Criterion (AIC) of model parameters which showed substantial support ($\Delta$AIC$_C$ ≤ 2) for predicting the change in isometric, and isokinetic concentric and eccentric contractions torque ($\Delta$T) based on changes in the predictor variables ($\Delta$). Models which showed moderate support ($\Delta$AIC$_C$ ≤ 4) for predicting the change in isometric torque are also included. Models with an AIC$_C$.W $\geq$ 0.10 (i.e. greater than a 10% chance that they will be the best-fit model) are identified by shading. 70

Table 5.6. Regression models using the previously-identified ‘best-fit’ model parameters for predicting torque (Study 1; Chapter 4) to determine whether adaptations in these same variables were associated with the change of strength following training. 72

Table 5.7 Correlations (r) between the change in isometric ($\Delta$T$_{ISO}$), and isokinetic concentric ($\Delta$T$_{CON}$) and eccentric ($\Delta$T$_{ECC}$) torque and changes in neuromuscular variables ($\Delta$VAR). 75

Table 6.1 Correlations between pre-training torque (maximum voluntary and electrically stimulated), muscle activity and moment arm magnitudes and the change in those magnitudes, as well as their correlations with the change in isometric, concentric and eccentric knee extension torque. 90

Table 6.2 Correlations between pre-training muscle size and architecture magnitudes and the change in those magnitudes ($\Delta$VAR), and their correlations with the change in isometric ($\Delta$T$_{ISO}$), and isokinetic concentric ($\Delta$T$_{CON}$) and eccentric ($\Delta$T$_{ECC}$) knee extension torque following training. 91
Table 6.7 Akaike’s Information Criterion of model parameters showing substantial support (ΔAIC$_C$ ≤ 2) predicting the change in maximal isometric, and isokinetic concentric and eccentric (ISO, CON and ECC, respectively) torque (ΔT) based on pre-training variables following training. Models with an AIC$_C$ weighting (AIC$_C$w$_i$) ≥ 0.10 (i.e. greater than a 10% chance they will be the best-fit model) and stronger than the null model, are identified by shading.

Table A.1. Metabolic energy equivalent calculation for two people attending university (MET/hr).
List of Figures

Figure 4.1. Outline of testings sessions. All six sessions were completed over a 2-wk period. .......... 30

Figure 4.2. Examples of raw and analysed ultrasound images of distal cross-sectional area (A and B); fascicle length at mid VL and VI (C and D) and VM fascicle length and angle (E and F). ............... 36

Figure 4.3. Illustration of the patella tendon moment arm analysis at an 80° joint angle. .............. 38

Figure 4.4. Predicted torque was modelled based on the AIC<sub>C</sub> rankings using the best-fit model (pink; y1) and the best clinical model (i.e. no maximal contraction required) (blue; y2) for maximal isometric, and isokinetic concentric and eccentric torque prediction. ........................................ 44

Figure 4.5. The effect of fascicle angle on physiological cross-sectional area (PCSA). ..................... 51

Figure 5.1. ACSA of individual quadriceps components at distal (A), middle (B) and proximal (C) regions of the thigh............................................................... 63

Figure 5.2. Predicted change in torque (ΔT) was modelled based on the AIC<sub>C</sub> rankings using the best-fit model for the change in maximal isometric, and isokinetic concentric and eccentric torque prediction......................................................... 71

Figure 5.3. Correlations between changes in maximum isometric, and isokinetic concentric and eccentric torques, versus changes in the anatomical and neuromuscular variables. ................. 74

Figure 6.1. Relationships between pre-training neuromuscular variable magnitudes and the changes in those magnitudes following training. ................................................................. 92

Figure 6.2. Relationships between pre-training neuromuscular magnitudes and the change in maximum isometric (ΔT<sub>ISO</sub>), and isokinetic concentric (ΔT<sub>CON</sub>) and eccentric (ΔT<sub>ECC</sub>) torque following training. ................................................................. 93

Figure 2C. Distribution of change in maximal isometric (a), concentric (b) and eccentric (c) torque following training................................................................. 141
**List of Abbreviations**

**Anatomical and neuromuscular measurements**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACSA</td>
<td>anatomical cross-sectional area</td>
</tr>
<tr>
<td>CSA</td>
<td>cross-sectional area</td>
</tr>
<tr>
<td>PCSA</td>
<td>physiological cross-sectional area</td>
</tr>
<tr>
<td>$\theta_f$</td>
<td>fascicle angle</td>
</tr>
<tr>
<td>$l_f$</td>
<td>fascicle length</td>
</tr>
<tr>
<td>EMG</td>
<td>electromyogram/electromyography</td>
</tr>
<tr>
<td>EMG:M</td>
<td>EMG amplitude normalised to its respective M-wave amplitude</td>
</tr>
<tr>
<td>RMS</td>
<td>root mean squared</td>
</tr>
<tr>
<td>EMD</td>
<td>electromechanical delay</td>
</tr>
<tr>
<td>%VA</td>
<td>percent voluntary activation</td>
</tr>
<tr>
<td>MA</td>
<td>moment arm distance</td>
</tr>
<tr>
<td>$\Delta$</td>
<td>change in</td>
</tr>
<tr>
<td>$\Delta$VAR</td>
<td>change in the anatomical and neuromuscular variables</td>
</tr>
</tbody>
</table>

**Suffixes**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>RF</td>
<td>rectus femoris</td>
</tr>
<tr>
<td>VL</td>
<td>vastus lateralis</td>
</tr>
<tr>
<td>VI</td>
<td>vastus intermedius</td>
</tr>
<tr>
<td>VM</td>
<td>vastus medialis</td>
</tr>
<tr>
<td>PROX</td>
<td>proximal-region</td>
</tr>
<tr>
<td>MID</td>
<td>middle-region</td>
</tr>
<tr>
<td>DIST</td>
<td>distal-region</td>
</tr>
<tr>
<td>SUM</td>
<td>sum of whole muscle or whole quadriceps</td>
</tr>
</tbody>
</table>

**Torque measurements**

<table>
<thead>
<tr>
<th>Torque Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_{ISO}$</td>
<td>isometric torque</td>
</tr>
<tr>
<td>$T_{CON}$</td>
<td>concentric torque</td>
</tr>
<tr>
<td>$T_{ECC}$</td>
<td>eccentric torque</td>
</tr>
<tr>
<td>$T_{Un-Tw}$</td>
<td>unpotentiated twitch torque</td>
</tr>
<tr>
<td>$T_{Pot-Tw}$</td>
<td>potentiated twitch torque</td>
</tr>
</tbody>
</table>

**Units**

<table>
<thead>
<tr>
<th>Unit</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ln</td>
<td>natural log</td>
</tr>
<tr>
<td>Nm</td>
<td>newton metre</td>
</tr>
<tr>
<td>RM</td>
<td>repetition maximum</td>
</tr>
</tbody>
</table>

**Data analysis**

<table>
<thead>
<tr>
<th>Measure</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>SD</td>
<td>standard deviation</td>
</tr>
<tr>
<td>SE</td>
<td>standard error</td>
</tr>
<tr>
<td>AIC</td>
<td>Akaike Information Criterion</td>
</tr>
<tr>
<td>AICc</td>
<td>Akaike Information Criterion adjusted for small sample size</td>
</tr>
<tr>
<td>$AIC_{c,d}$</td>
<td>percentage of times a models would be selected as the best model</td>
</tr>
<tr>
<td>K</td>
<td>number of parameters</td>
</tr>
<tr>
<td>LL</td>
<td>model log likelihood</td>
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CHAPTER ONE: INTRODUCTION
2.1 Introduction

The ability to apply a sufficient level of force to the ground or an object is important for the successful performance of both sporting tasks and activities of daily living [1]. Although it has been well established that the force generated by the muscle itself increases proportionally with the number of attached cross-bridges and inversely proportionally with its shortening velocity [2], joint torque, and thus external force, production is also influenced by several other anatomical and neuromuscular factors. These factors include (but are not limited to) the muscles’ sizes and the architectural arrangement of their fibres [3, 4], the level of voluntary activation of those muscles [5-8], the activation (or inhibition) level of the antagonist muscles [9, 10] and the moment arm distance across which the force is transmitted to the skeleton [11, 12]. However, although observational (cross-sectional) studies have revealed a significant inter-individual variation in these factors both between and within normal healthy [13], elderly [14] and athletic [15] populations, and the variations in some factors have been closely associated (i.e. correlated) with joint torque capacity, the extent to which each of these factors influences maximal isometric, concentric and eccentric torque has not been completely determined. Without a clear understanding of the importance of the factors influencing strength expression, it is not possible to optimise exercise training plans or target interventions specifically to an individual.

Of additional consideration is that anatomical and neuromuscular factors (with the exception of joint moment arm) show great plasticity in response to exercise training [16-19]. There is a substantial inter-individual variability in their change with training and, consequently, some individuals show greater improvements in muscular strength than others [5, 20]. If the inter-individual adaptations to these anatomical and neuromuscular variables are the cause of differences in strength improvements, then an individual’s pre-training anatomical and neuromuscular characteristics may also dictate their strength adaptations. This hypothesis is worthy of explicit testing, for if baseline characteristics are related to strength improvements following training, the optimal training program for the individual could be determined before initiation of an intervention. Understanding and interpreting an individual’s anatomical and neuromuscular status prior to training would then require a comparative data of the variables which underpin our ability to express strength. Comparing individuals to the comparative data set would allow the identification of an individual’s strengths and weaknesses before training, which would ensure the exercise prescribed was optimal for that individual.

Determining the specific influence of each anatomical and neuromuscular factor on strength expression is not easily achievable in humans. Difficulty arises because it is not possible to assess the
effect of one variable (e.g. muscle size) on strength expression without the confounding influence of other variables (e.g. muscle activation) that change in response to an exercise training intervention. Thus, the finding that an anatomical or neuromuscular variable changes simultaneously with strength after the training intervention according to an *a priori* hypothesis is often taken as evidence for causative association. However, without assessing the relationship between the changes in both variables, there is no evidence that the change in one factor (e.g. muscle size) is related to, let alone the cause of, the change in another (strength). The next-best study would thus involve a longitudinal study design focusing on the simultaneous (i.e. correlated) changes in both anatomical and neuromuscular variables and strength. Despite this possibility, relationships between the changes in variables are rarely documented in longitudinal studies, so such examinations would prove very useful in the future. Nonetheless, imposing an intervention to manipulate one or more factors is both time and financially expensive. Hence the possible influence of each factor needs to be estimated to potentially define a smaller subset of factors for study. To achieve this, large observational (cross-sectional) studies can be used preliminarily in a series of studies to determine the relative influence of various anatomical and neuromuscular variables on muscle strength.

Given the above, the purpose of the present body of work is to explore the relationships between anatomical and neuromuscular factors and strength expression using both observational (cross-sectional) and mixed-method (longitudinal) study designs. This will improve our understanding of the influence of the anatomical and neuromuscular variables on external force production, and will be the first step in the process of understanding the relative importance of training adaptations elicited by strength training interventions. From a clinical perspective, knee extension torque is a key aspect of functional lower limb rehabilitation programs [21-24] and is required for the successful completion of many activities of daily living (e.g. locomotion, chair sitting and rising, stair climbing) and athletic tasks. Thus, the knee extensors were chosen as the subject of study, and a comparative data set of the anatomical and neuromuscular variables hypothesised to be most important for strength expression were developed in young healthy men. Mathematical modelling was used in an attempt to determine the most relevant factors influencing strength expression (including the development of models using data collected without the need for maximal muscle contractions or electrical stimulation methodologies, which may not be possible in clinical populations). Finally, the potential influence of anatomical and physiological factors on strength expression was examined for isometric, concentric and eccentric contractions, since all three contraction modes are required for the successful completion of activities of daily living, and are essential for success in various sporting tasks.
CHAPTER TWO: REVIEW OF LITERATURE
2.1 Overview

In order to optimise training programs aimed to improve an individual’s strength, potential weaknesses in the chain of events leading to external force production (i.e. active joint torque) must be identified. This chapter is constructed as a narrative review focusing on the primary factors immediately influencing an individual’s strength capacity. A muscle’s size, architecture and level of activity and activation all influence its force production magnitude, and the moment arm about which that force is produced will influence the resultant torque capacity. In this chapter, the relative influence of these potentially important anatomical and neuromuscular variables will be reviewed with respect to their potential influence on muscular strength expression (see Figure 2.1).

Physical training elicits adaptations that are largely dependent upon the load, volume and velocity of the movement. Yet large individual variability is observed in the rate and magnitude of the improvements even when individuals follow the same physical training protocol. Strength training is one of the most widely practiced forms of physical training, and will be the focus of this review. Within this chapter questions related to how these anatomical and neuromuscular variables adapt to strength training, and how these adaptations may influence changes in isometric, concentric and eccentric strength will be asked. Additionally, because of their importance for successful performance in many daily living and sporting activities, a major emphasis will be placed on adaptations in the quadriceps muscles and their influence on active knee joint torque production.

![Diagram](image)

Figure 2.1: Variables influencing maximum torque production. The focus of this literature review is the primary mechanisms influencing an individual’s maximum torque production (dark blue), with training adaptations based on the three contraction modes (red). The secondary mechanisms (light blue) will not be covered within this review.
2.2 Effect of muscle size on a muscle’s force producing capacity

A muscle’s size is considered to be a key determinant of its peak force potential [11, 25-28], with moderate-to-strong correlations being reported between maximal voluntary strength (i.e. active joint torque) and muscle size (i.e. physiological cross-sectional area (PCSA) [11], anatomical cross-sectional area (ACSA) [11, 25] and muscle volume [3, 11]). These findings suggest that increases in muscle size strongly influence the increase in contractile force elicited by strength training (see Table 2.1). While there is large individual variability in muscle size across, and even within, populations (i.e. athletic [15], untrained [13], elderly [14]), the relationship between torque and muscle volume appears similar regardless of training status [3].

Nonetheless, the relationship between muscle size and strength has been observed to vary slightly when strength is measured in different contraction modes. For example, marginally stronger correlations have been shown between ACSA and joint torque developed during slow-speed concentric contractions ($r = 0.81$) compared to isometric ($r = 0.73$) or fast-speed concentric contractions ($r = 0.72$) [11], and between slow speed concentric ($r = 0.78$) compared to slow speed eccentric ($r = 0.68$) contractions [29] in the quadriceps. However, there is limited research documenting the relationship between muscle size and eccentric torque capacity, with one study completed in osteoarthritis patients ($r = 0.68$; [29]) and no studies completed in healthy adults (see Table 2.1). Considering the relative importance of eccentric muscle actions in daily living (e.g. sitting down in a chair) and athletic activities, and their use in physical training protocols for improving both muscle size [30, 31] and strength [31], the lack of data presents an important limitation in our understanding of muscle force production.

2.3 Training-induced adaptations in muscle size

Strength training stimulates increases in muscle fibre size and, in turn, whole muscle size [32]. Gross muscular hypertrophy is typically considered to be achieved slowly in the first weeks of training and the initial strength increases appear unrelated to increases in muscle size [33]. A majority of strength training studies documenting hypertrophic adaptations have utilised intervention periods of 8-12 weeks [6, 34-36], although muscular hypertrophy has been observed within 5 weeks of training initiation [37-39]. However, mechanical tension, muscle damage, and metabolic stress are considered likely requirements for hypertrophy (see review; [40]) and are all imposed during and/or after the initial sessions, so hypertrophy probably progresses from the initiation of an intervention even if not detected.
Table 2.1. Correlations ($R^2$) between muscle size measures and maximum joint torque measured during isometric, concentric and eccentric contractions ('Slow' and 'Fast' refer to ≤ 60 °·s$^{-1}$ and ≥ 180°·s$^{-1}$, respectively) in healthy participants.

<table>
<thead>
<tr>
<th>Muscle Size Measure</th>
<th>Muscle Group</th>
<th>Location</th>
<th>Participants</th>
<th>Isometric</th>
<th>Slow Concentric</th>
<th>Fast Concentric</th>
<th>Slow Eccentric</th>
<th>Fast Eccentric</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>MV</td>
<td>Elbow flexors</td>
<td>Mid</td>
<td>Sedentary to Athlete</td>
<td>0.76 (0.58)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Akagi et al. (2009) [49]</td>
</tr>
<tr>
<td>MV</td>
<td>Elbow flexors</td>
<td>Mid</td>
<td>Sedentary to Athlete (f)</td>
<td>0.93 (0.86)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Akagi et al. (2009) [49]</td>
</tr>
<tr>
<td>MV</td>
<td>Elbow flexors</td>
<td>Mid</td>
<td>Recreational</td>
<td>0.81 (0.66)</td>
<td>0.76 (0.61)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Erskine et al. (2014) [50]</td>
</tr>
<tr>
<td>MV</td>
<td>Elbow flexors</td>
<td>Mid</td>
<td>University students</td>
<td>0.94 (0.88)</td>
<td>0.65 (0.42)</td>
<td>0.86 (0.74)</td>
<td>0.72 (0.51)</td>
<td>-</td>
<td>Fukunaga et al. (2001) [3]</td>
</tr>
<tr>
<td>MV</td>
<td>Elbow extensors</td>
<td>Mid</td>
<td>University students</td>
<td>0.92 (0.85)</td>
<td>0.75 (0.61)</td>
<td>0.86 (0.61)</td>
<td>0.72 (0.51)</td>
<td>-</td>
<td>Fukunaga et al. (2001) [3]</td>
</tr>
<tr>
<td>MV</td>
<td>Knee extensors</td>
<td>Mid</td>
<td>Recreational</td>
<td>0.78 (0.60)</td>
<td>0.73 (0.53)</td>
<td>0.86 (0.74)</td>
<td>0.72 (0.51)</td>
<td>-</td>
<td>Blazevich et al. (2009) [11]</td>
</tr>
<tr>
<td>MV</td>
<td>Plantarflexors</td>
<td>Mid</td>
<td>Healthy</td>
<td>0.57 (0.32)</td>
<td>0.73 (0.53)</td>
<td>0.86 (0.74)</td>
<td>0.72 (0.51)</td>
<td>-</td>
<td>Baxter and Piazza (2014) [51]</td>
</tr>
<tr>
<td>MV</td>
<td>Plantarflexors</td>
<td>Mid</td>
<td>Endurance and untrained (f)</td>
<td>-</td>
<td>0.85 (0.61)</td>
<td>0.73 (0.53)</td>
<td>0.86 (0.74)</td>
<td>0.72 (0.51)</td>
<td>-</td>
</tr>
<tr>
<td>ACSA</td>
<td>Elbow flexors</td>
<td>Mid</td>
<td>Sedentary to Athlete</td>
<td>0.78 (0.61)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Akagi et al. (2009) [49]</td>
</tr>
<tr>
<td>ACSA</td>
<td>Elbow flexors</td>
<td>GA</td>
<td>Untrained and Bodybuilders</td>
<td>-</td>
<td>0.80 (0.64)</td>
<td>0.73 (0.53)</td>
<td>0.86 (0.74)</td>
<td>0.72 (0.51)</td>
<td>-</td>
</tr>
<tr>
<td>ACSA</td>
<td>Elbow flexors</td>
<td>Mid</td>
<td>Sedentary to Athlete (f)</td>
<td>0.91 (0.83)</td>
<td>0.74 (0.56)</td>
<td>0.86 (0.74)</td>
<td>0.72 (0.51)</td>
<td>-</td>
<td>Akagi et al. (2009) [49]</td>
</tr>
<tr>
<td>ACSA</td>
<td>Elbow flexors</td>
<td>Mid</td>
<td>University students</td>
<td>0.71 (0.50)</td>
<td>0.74 (0.56)</td>
<td>0.86 (0.74)</td>
<td>0.72 (0.51)</td>
<td>-</td>
<td>Fujimura et al. (2001) [3]</td>
</tr>
<tr>
<td>ACSA</td>
<td>Elbow extensors</td>
<td>Mid</td>
<td>University students</td>
<td>0.89 (0.79)</td>
<td>0.74 (0.56)</td>
<td>0.86 (0.74)</td>
<td>0.72 (0.51)</td>
<td>-</td>
<td>Fujimura et al. (2001) [3]</td>
</tr>
<tr>
<td>ACSA</td>
<td>Elbow extensors</td>
<td>Mid</td>
<td>Bodybuilders</td>
<td>0.58 (0.34)</td>
<td>0.74 (0.56)</td>
<td>0.86 (0.74)</td>
<td>0.72 (0.51)</td>
<td>-</td>
<td>Ikegawa et al. (2008) [54]</td>
</tr>
<tr>
<td>ACSA</td>
<td>Elbow extensors</td>
<td>Mid</td>
<td>Weightlifters</td>
<td>0.72 (0.53)</td>
<td>0.74 (0.56)</td>
<td>0.86 (0.74)</td>
<td>0.72 (0.51)</td>
<td>-</td>
<td>Ikegawa et al. (2008) [54]</td>
</tr>
<tr>
<td>ACSA</td>
<td>Knee extensors</td>
<td>Mid</td>
<td>Recreational</td>
<td>0.73 (0.53)</td>
<td>0.74 (0.56)</td>
<td>0.86 (0.74)</td>
<td>0.72 (0.51)</td>
<td>-</td>
<td>Blazevich et al. (2009) [11]</td>
</tr>
<tr>
<td>ACSA</td>
<td>Knee extensors</td>
<td>Mid</td>
<td>Sedentary and Recreational</td>
<td>0.59 (0.35)</td>
<td>0.74 (0.56)</td>
<td>0.86 (0.74)</td>
<td>0.72 (0.51)</td>
<td>-</td>
<td>Maughan et al. (1983) [25]</td>
</tr>
<tr>
<td>ACSA</td>
<td>Knee extensors</td>
<td>Mid (7)</td>
<td>Non-athletes</td>
<td>0.84 (0.71)</td>
<td>0.74 (0.56)</td>
<td>0.86 (0.74)</td>
<td>0.72 (0.51)</td>
<td>-</td>
<td>Ahtiala et al. (2003) [46]</td>
</tr>
<tr>
<td>ACSA</td>
<td>Knee extensors</td>
<td>Mid</td>
<td>Knee Osteoarthritis (f)</td>
<td>-</td>
<td>0.78 (0.61)</td>
<td>0.75 (0.56)</td>
<td>0.68 (0.46)</td>
<td>0.69 (0.48)</td>
<td>Gur and Cakun (2003) [29]</td>
</tr>
<tr>
<td>ACSA</td>
<td>VL</td>
<td>Mid</td>
<td>Healthy</td>
<td>0.75 (0.56)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Stebbings et al. (2014) [55]</td>
</tr>
<tr>
<td>ACSA</td>
<td>VL and VL (x)</td>
<td>Mid</td>
<td>Anaerobic athletes</td>
<td>0.63 (0.40)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Rutherford and Jones (1992) [56]</td>
</tr>
</tbody>
</table>

continued
<table>
<thead>
<tr>
<th>ACSA</th>
<th>Muscle Group</th>
<th>GA</th>
<th>Condition</th>
<th>MV = muscle volume; ACSA = anatomical cross-sectional area; PCSA = physiological cross-sectional area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plantarflexors</td>
<td>GA</td>
<td>Endurance and untrained (f)</td>
<td>- 0.73 (0.53)</td>
<td>Bammenn et al. (2000) [52]</td>
</tr>
<tr>
<td>PCSA</td>
<td>Elbow flexors</td>
<td>University students</td>
<td>0.95 (0.90)</td>
<td>Fukunaga et al. (2001) [3]</td>
</tr>
<tr>
<td>PCSA</td>
<td>Elbow extensors</td>
<td>University students</td>
<td>0.91 (0.83)</td>
<td>Fukunaga et al. (2001) [3]</td>
</tr>
<tr>
<td>PCSA</td>
<td>VL</td>
<td>Recreational</td>
<td>0.71 (0.50) $^1$ 0.72 (0.51) 0.62 (0.39)</td>
<td>Blazevich et al. (2009) [11]</td>
</tr>
<tr>
<td>PCSA</td>
<td>Plantarflexors</td>
<td>Endurance and untrained (f)</td>
<td>- 0.71 (0.50)</td>
<td>Bammenn et al. (2000) [52]</td>
</tr>
</tbody>
</table>

Mid = scan obtain from mid-muscle; GA = ACSA value of the region with the 'greatest area'; Mid (7) = mean ACSA of 7 mid-thigh scan locations

$^1$ is obtained at 90° flexion; $^2$ at 80°; $^3$ at 73°; $^4$ at 60°; $^5$ at 0° (neutral); $^6$ at the angle of peak torque

All participants were male unless denoted by (f) for female.
2.3.1 Factors influencing hypertrophy

The magnitude of hypertrophy following 5-12 weeks of strength training typically ranges from 5-20% [41]. The magnitude of increase appears to depend on a combination of loading intensity, previous training history, force developed during training and muscle motor unit composition. In his review, Fry [42] proposes training in the range of 80-95% of one-repetition maximum (1-RM) to optimise hypertrophy. However, the review also suggested that only 18-35% of the variance in hypertrophy is determined by intensity. This assertion is supported by Mitchell et al. [43], who observed similar increases in quadriceps muscle volume in individuals training at 30% 1-RM versus 80% 1-RM. Nonetheless, most training interventions aiming to elicit both muscular hypertrophy and strength improvements utilise 1-5 RM or 6-12 RM loads (see Kraemer and Ratamess [44]), with the moderate range (6-12 RM) considered to stimulate greater metabolic stress [42, 45] and thus potentially greater hypertrophy. Training history also appears to influence the inter-individual variance in hypertrophic adaptations, as Ahtiainen et al. [46] reported quadriceps ACSA adaptations of 5.6% in non-athletes and -1.8% in strength trained athletes following 21 weeks of lower limb heavy strength training. Also, Hakkinen et al. [47] and Alway et al. [48] have observed no further muscle growth in strength trained participants in either the quadriceps or elbow flexors following 12 and 24 weeks of strength training, respectively, suggesting a potential ceiling effect with respect to hypertrophy in trained individuals, at least when the training stimulus remains unchanged. This lack of change can be compared to hypertrophic gains of ~13-18% reported in the quadriceps in untrained individuals after 24 weeks training [13].

Another variable considered to influence hypertrophic adaptation is the muscle contraction mode, where the use of eccentric contractions allows for the development of more force per repetition compared to concentric or isometric training [57] and might be expected to promote greater hypertrophy. Indeed, Farthing and Chilibeck [57] reported greater hypertrophy in the elbow flexors after eccentric-only (~7-13%) compared to concentric-only (~2-5%) training, and Higbie et al. [35] reported a greater increase in quadriceps CSA after 10 weeks of maximal eccentric (6.6%) compared to concentric (5.0%) knee extension training in women. In contrast, Jones and Rutherford [58] reported similar increases in muscle size after 12 weeks of concentric and eccentric strength training, and Blazevich et al. (2007) observed similar increases in quadriceps muscle volume after 10 weeks of maximal isokinetic eccentric or concentric training. Thus eccentric training is not always found to elicit a greater magnitude of hypertrophy. Wernborm et al. [59] hypothesised that differences in the volume of work performed between the two contraction modes were probably the more important stimulus for the greater hypertrophy often observed following eccentric training than the shortening or lengthening of the muscle fibres, after reviewing the data from longitudinal
studies. Consideration of the total force developed for each contraction is thus necessary to truly understand the influence of different contraction modes on any of the anatomical and neuromuscular variables included in this review.

Changes in muscle size appear to at least partly result from changes in muscle fibre CSA. For example, Hortobagyi et al. [31] found a greater (x10) increase in Type II fibre area following eccentric than concentric knee extensor training, and hypertrophy has also been found to be greater in Type II than Type I muscle fibres following strength training [47, 60, 61]. Aagaard et al. [60] and Hakkinen et al. [62] both observed statistically significant increases in Type II, but not Type I, fibres in vastus lateralis (VL) following either 14 weeks of heavy (4-6 RM) or 24 weeks of explosive (60-80% 1RM) strength training, respectively. Thus, Type II fibres appear to show a greater response to strength training than Type I fibres. However, care must be taken when inferring the changes in whole muscle size from increases in muscle fibre size, as Narici et al. [13] reported that increases in fibre area (2%) where not representative in the increases in the whole muscle (~7.5%). Hypertrophic gains from training may, therefore, go unnoticed if only muscle fibre area is assessed. Additionally, part of the increase in muscle size is probably related to changes in muscle architecture. Increases in muscle fascicle angle have been shown to partly explain the increases in muscle size after strength training [60], and increases in fascicle length may influence this change. Thus, muscular hypertrophy cannot simply be considered to be reflective of hypertrophy at the muscle fibre level.

### 2.3.2 Location of hypertrophy

Hypertrophy appears to be non-uniform both between and within synergist muscles [6, 13, 38, 46, 63], although there appear to be inconsistent findings with respect to the regions expected to present hypertrophy in muscles such as the quadriceps. For example, Blazevich et al. [4] and Ahtiainen et al. [46] observed greater increases in distal and mid-quadriceps regions, Ema et al. [63] observed greater hypertrophy in VL and RF in the distal compared to the proximal region, and Narici et al. [13] noted that gains were greater in the distal and proximal regions than in the mid-region. Furthermore, some researchers have also reported greater increases in rectus femoris (RF) than vasti muscle CSA following knee extensor strength training [13, 63]. These findings of selective, or region-specific, hypertrophy make sense from the perspective that different regions within muscles can be recruited differently depending on the task goal [64, 65], and multiple innervation zones are apparent in the quadriceps muscles [66]. Nonetheless, in both cross-sectional and longitudinal studies implemented with the aim of examining the relationship between muscle size and strength, muscle size is often only measured at a single location [e.g. mid-thigh CSA; 25, 67, 68], in which case it is not known whether the muscle size response is representative of all regions within the muscle.
Alternatively, muscle volume has been measured in some studies [3, 69, 70], which provides an ideal indication of muscle size but does not allow determination of the relative changes in different muscle regions. This may be of concern considering that specific regions within a muscle may be important for force production under specific loading conditions [e.g. 71].

Furthermore, there is some debate as to whether PCSA provides the most information regarding the muscular force production capacity in vivo, compared to the more easily measured ACSA. Physiological CSA describes the magnitude of muscle fibre area perpendicular to the longitudinal axis of the muscle fibres and thus takes into account fascicle angulation, whereas ACSA is the area measured perpendicular to the longitudinal axis of the muscle [60]. Theoretically, PCSA is a more valid index of muscle size than ACSA, however their specific relationships with muscle force production measured in vivo in humans are inconsistent. For example, Bammann et al. [52] found that triceps surae PCSA and ACSA were equally strongly correlated with maximum plantar-flexor strength whereas Fukunaga et al. [3] observed stronger correlations for PCSA than ACSA for elbow flexor strength but similar correlations for the elbow extensors. By contrast Blazevich et al. [11] identified slightly stronger correlations between ACSA and knee extensor (although physiological CSA was calculated for vastus lateralis only). It is possible that methodological limitations complicating the estimation of PCSA in vivo impact on its functional use, however both CSA measurements are considered relevant and may provide somewhat similar information regarding potential muscular force capacity when measured in humans. Another drawback of PCSA measurements, however, is that they require the measurement of whole muscle volume and, therefore, cannot be used to identify region-specific differences in muscle size (or hypertrophy after training). As previous research has shown region-specific activation to be task specific [64, 65], identification of regional differences in hypertrophy is an important focus.

2.3.3 Relationship between changes in muscle size and changes in strength

The large inter-individual variability in the change in muscle size with training [e.g. 20, 36] reinforces the need for further research on the relationship between hypertrophy and strength increases following training. Previously, Hubal et al. [36] observed large individual variability in the changes in both elbow flexor muscle size (-2 to 59%) and isometric strength (-32 to 149%) following 12 weeks of dynamic training, and Erskine et al. [20] observed increases in quadriceps PCSA of -3 to 18% in the quadriceps components following 9 weeks of strength training and only a weak correlation (r = 0.48) between increases in PCSA and strength. This large range in hypertrophic responses (and strength changes) implies that other variables must be important for inducing the gains in strength following strength training. Unfortunately, there are limited data identifying whether the change in muscle
size is causative of the strength change. Higbie et al. [35] reported a correlation \( (r = 0.51) \) that was very similar to that of Erskine et al. [20] for changes in quadriceps CSA and changes in eccentric strength, and a stronger correlation \( (r = 0.70) \) between the change in quadriceps CSA and the change in concentric strength following 10 weeks eccentric or concentric training, respectively. These data suggest a possible causative link in that participants with greater increases in CSA also tended to show a greater increase in force production. Noorkoiv et al. [72] assessed the effect of the muscle length adopted during isometric strength training on the relationship between muscle size and strength and observed strong relationships \( (r = 0.85 \) and \( r = 0.80) \) between the change in proximal region VL CSA and the change in isometric force at short muscle lengths (for 30 and 40° knee angle, respectively) as well as between the change in mid-muscle VL CSA and the change in isometric force at long muscle lengths \( (r = 0.79 \) to \( 0.95, \) for 60-90° knee angles, respectively), indicating that the strength of the relationships between the change in muscle size and the change in strength maybe dependent on the training stimulus. Nonetheless, Jones and Rutherford [58], found no correlation between the change in quadriceps CSA and changes in either concentric or eccentric strength following 12 weeks training, however their measurements were obtained from muscle fibre area rather than whole muscle area, which has been observed to be unrepresentative of changes at both that site and across the whole muscle [13]. Whilst a strong correlation between changes in muscle size and strength were observed after isometric training [72], Higbie et al. [35] speculated that the typically weaker relationship between the changes in muscle size and strength is unsurprising given that whole muscle CSA does not reflect the activation of muscle fibres or the velocity-dependent nature of this activation. As training interventions affect more than one neuromuscular parameter it is probably important to assess simultaneously the relationship between the changes in various anatomical and neuromuscular mechanisms responsible for strength change to better understand the interactions between the mechanisms. Based on the large inter-individual variability in both the increases in muscle size and strength following strength training, factors other than just hypertrophy must contribute significantly to changes in strength expression, and changes in hypertrophy alone may not be expected to result in notable changes in strength.

2.4 Effect of muscle architecture on a muscle’s force producing capacity

Muscle architecture, as defined in this thesis, describes the arrangement of the muscle’s fascicles (i.e. fascicle angle and fascicle length). Fascicle angle refers to the geometric angulation of the fascicles to the longitudinal axis of the muscle, and is typically defined as the angle relative to the deep aponeurosis. Fascicle angulation is considered to improve muscular force generation capacity through three main mechanisms. First, angulation allows more contractile tissue to attach to a given area of tendon or aponeurosis [54, 73-75] and, therefore, increases the muscle’s PCSA and
consequently its peak contractile capacity. Second, the rotation of muscle fascicles (i.e. increase in fascicle angle) as muscle shortens during contraction allows the fascicles to shorten less for a given muscle shortening distance. This in turn allows fascicles (or their constituent fibres) to work nearer their optimum length (i.e. optimise force-length relationship) [2] and this phenomenon is more critical in muscles with greater fascicle angles. Third, the lesser fascicle shortening resulting from fascicle rotation also reduces the fascicle shortening speed relative to the muscle shortening speed, thus increasing muscle force in accordance with the force-velocity relationship [76]. Together with the gross size of the muscle, its architecture has been considered a key determinant of a muscle’s force producing capabilities [77, 78]; and in fact, fascicle angle tends to be moderately correlated with both muscle size [54, 63, 79] and muscle strength [80, 81].

Fascicle length, defined as the distance between the fascicle’s origin and insertion, may also affect force production. Longer fascicles typically contain a greater number of serially arranged sarcomeres and allow for faster muscle shortening speeds, as well as greater force production magnitudes over broader muscle length ranges (i.e. during dynamic muscle contractions) [15, 60, 76, 82-84]. The finding that elite sprinters have longer vastus lateralis and medial gastrocnemius fascicles than novice sprinters [83], untrained controls [85] and distance runners [15], for example, suggests a functional link between muscle shortening velocity and fascicle length when measured in vivo in humans. Theoretically, the longer fascicles should allow the faster runners to produce greater torque over a larger range of motion when muscle shortening speeds are high, and thus achieve better running times. However, exceptions are seen, with relationships between fascicle length and sprint running performance not always being apparent [86, 87]. Few observational studies, however, have examined the relationship between fascicle length and maximal force production, although Brechue and Abe [88] reported that triceps brachii and vastus lateralis fascicle length were longer in powerlifters who lifted heavier loads [88]. Therefore, while fascicle length appears to play an important role in force production during high speed movements, less is known about its influence on slow-speed high-force activities.

2.5 Training-induced adaptations in muscle architecture

Both fascicle angle and length show adaptive plasticity in response to physical (especially strength) training. In particular, increases in fascicle angle in response to strength training often occur simultaneously with increases in muscle size [4, 20, 38, 60, 63, 89] and muscular strength [6, 60, 90] following lower limb strength training interventions of 9-14 weeks. However, exceptions are seen, with some studies reporting no change (or a slight decrease) in fascicle angle with increases in muscle size and strength following both isotonic [56, 82] and eccentric [91, 92] training. Additionally,
large inter-individual variability in the changes in fascicle angle with training have been reported [60, 93], which, if fascicle angle was a key parameter influencing strength, might influence the magnitude of strength increase. Thus, while fascicle angle appears to be an important variable influencing strength improvements, the reasons for the large inter-individual variability in fascicle angle adaptations to strength training are yet to be explained.

Increases in fascicle length have been observed following both fast- [82, 93] and slow-speed dynamic [4] as well as isometric [72] training, with Seyennes et al. [38] reporting increases (9.9%) in vastus lateralis fascicle length following 10 days of high-load knee extensor training. Based on research involving rats [94-96], eccentric contractions were speculated to be more effective than concentric for stimulating fascicle length increases. However, no notable changes in fascicle length were observed after eccentric training in rabbits [97]. In humans, biceps femoris fascicle length has been observed to increase following eccentric training [92] and Reeves et al. [98] observed greater increases in VL fascicle length following eccentric (20%) compared to concentric (8%) training. Nonetheless, Blazevich et al. [4] observed similar increases following slow-speed concentric- (6.3%) and eccentric-only (3.1%) isokinetic training when fascicle length change and movement speed were kept identical between the groups. Thus, it may not be the case that eccentric contractions offer a unique stimulus for fascicle length change, although it is clear that fascicle length increases may be stimulated by it. If eccentric training is an important stimulus for fascicle length change then changes in fascicle length may be expected to be more strongly associated with the changes eccentric torque than isometric or concentric torque following training, however this has yet to be explicitly examined.

The inconsistent findings may, in some cases, be related to different measurement sites being examined between studies. For example, Erskine et al. [99] and Rutherford and Jones [56] observed no change in the lateral portion of vastus intermedius (VI) fascicle angle following strength training, whereas Ema et al. [63] observed an increase when measurements were taken from the medial portion. Another difficulty influencing the interpretation of previous results is that only one muscle within a synergist group is often assessed in isolation [15, 60]. Considering that adaptations are known to be heterogeneous both across and within synergist muscles, as well as being functionally dependent upon the task requirements, architectural changes with training might be missed when only a small section of a large muscle group is examined. In order to more clearly examine the relationship between fascicle angle and muscle strength, multiple sites should be examined in future research.
2.5.1 Relationship between the change in muscle architecture and the change in strength

While the above studies show that fascicle angle typically increases with both muscle size and strength during prolonged strength-training, there are limited data describing the relationship between the change in fascicle angle and the change in muscular strength following a period of training. Erksine et al. [20] reported a weak relationship \( r = -0.33 \) between the change in fascicle angle and the change in isometric force following 9 weeks of knee extension training, and Ema et al. [63] reported correlations ranging 0.45-0.72 between changes in size (muscle thickness) and the change in fascicle angle of the four quadriceps components after 12 weeks of strength training, however no correlations with the change in strength were calculated. Thus, whilst both fascicle angle and muscle strength may increase concurrently with training, the large individual variability in strength change may only be weakly or moderately associated with changes in fascicle angle. Clearly, more research is required to understand fully the influence of fascicle angle adaptations on muscle strength changes following training.

Additionally, it is not clear whether the change in fascicle length is related to the change in strength following training. Erskine et al. [20] reported a moderate correlation between the change in VL fascicle length and the change in isometric force \( r = -0.47 \) after 9 weeks strength training. However, Noorkoiv et al. [72] observed no correlation between the change in VL fascicle length and the change in isometric force following 6 weeks of isometric knee extensor training. Thus, when set against other parameters that might influence maximum joint torque, the importance of fascicle length change is unclear.

Muscle architecture, therefore, shows significant adaptive plasticity in response to strength training. Fascicle angle appears to be important for slow-speed high force production activities and fascicle length for improving force during higher-speed contractions. However, exceptions are seen. Additionally, while fascicle length is thought to be more important for high-speed force production, increases in fascicle length have been observed following both slow- and fast-speed forms of training. Despite the belief that muscle architecture influences peak torque and both the torque-angular and torque-angular velocity relationships, there is no clear understanding of the effect of training-related changes in muscle architecture on the change in strength following training. The relative importance of fascicle length and angle on torque production, therefore, requires further examination.

2.6 Effects of neural activation on a muscle’s force producing capacity

The contractile capacity of the muscle fibres is also determined by the magnitude of agonist activation, and resultant joint torque (i.e. strength) is influenced by antagonist activation [6, 33].
Thus the ability to activate (or deactivate) the available musculature is undeniably important for strength expression [6-8, 100, 101]. Muscle activation is a function of both the level of input from the central nervous system, at supraspinal and/or spinal levels, and muscle fibre excitability [102, 103]. As it cannot be directly measured in humans, numerous studies use electromyography (EMG) to assess muscle activity [6, 47, 100], largely because of its convenience and its ability to provide reasonable estimates of muscle activation. Electrical and magnetic cortical stimulation procedures can provide further information about corticospinal pathways, and direct nerve stimulation can be used to estimate the level of spinal input through V/H-wave measurements and assessment of voluntary activation levels through the interpolated twitch technique [103]. Since peripheral factors can strongly influence the EMG measurements [104], EMG amplitudes are often normalised to their respective M-wave amplitudes (elicited by supramaximal nerve stimulation). M-wave-normalised EMG (EMG:M_{wave}) is considered to provide a better estimate of central drive because alterations at, and distal to, the neuromuscular junction, including changes to muscle membrane excitability, should be removed by the M-wave normalisation process [105].

The large inter-individual differences observed in agonist neural activation are partly dependent on an individual's training history [106-108]. Many sites within the nervous system show adaptive potential in response to exercise training, from the supra-spinal and spinal pathways through to the neuromuscular junction [109], and adaptations to these pathways will influence an individual's force production capacity. While motor learning is associated with adaptations to the primary motor cortex enabling the production of a more efficient movement [110], adaptations to resistance training were initially considered to occur within the spinal pathways [111]. Differences in motor neurone excitability between populations have been quantified using electrical or magnetic stimulation of the peripheral nerve. For example, greater V-wave amplitudes have been observed in sprinters and weight-lifters compared to untrained controls [112, 113], indicating an enhanced neural drive, increased motor neurone excitability and/or decreased presynaptic inhibition of Type Ia afferents in the trained athletes. Increased motor neurone excitability allows the production of more powerful contractions [114] and can also be determined by the H-reflex response. Interestingly, endurance-trained athletes and untrained participants have shown larger H-reflex responses (obtained at rest) than power-trained athletes [106, 107], indicating lower Type Ia motor neurone excitability in the power-trained participants. Nonetheless, it should be noted that observational differences in H-reflex response across populations may be influenced by genetic differences, rather than just training history [8], as power-trained athletes tend to have a higher proportion of fast twitch fibres [115] and the Type Ia afferent volley of the H-reflex excites slow, rather than fast, motor units [116]. Recently, however, it has been reported using transcranial
magnetic stimulation (TMS) methodologies that strength training can elicit significant adaptations in the motor cortex [117] and that these adaptations are linked to the increase in strength [118]. Percent voluntary activation (%VA; measured using the interpolated twitch technique) of the agonist muscles also differs between populations and has been reported to be higher in weight trained participants [113] and elite sprinters [112] than untrained controls. While %VA is accepted as a good indication of activation ability it is influenced by other factors distal to the neuromuscular junction such as changes in intracellular calcium concentration [119] and the efficiency of force transmission through the series elastic components [120]. These differences in agonist activation will probably cause different force production capabilities across populations.

Antagonist muscle activation also influences the total joint torque produced [9, 10, 121]. While less is known about the relative influence of antagonist coactivation on muscular strength, it appears to differ between populations. For example, aerobically-trained athletes tend to exhibit lower levels of coactivation than anaerobic (i.e. sprint-trained) athletes [108], and anaerobic athletes showing lower levels than sedentary individuals [122]. These differences in coactivation purportedly account for ~12% of isokinetic knee extension torque in highly skilled, and up to 38% in sedentary, individuals [122]. Different methods for estimating the opposing torque created by activation of the antagonist have been developed based on the relationship between EMG amplitude and torque production [9, 122, 123]. In regards to calculating knee extension torque, although the hamstrings are a synergist group of three muscles of distinct architectural design, the opposing knee extension moment from the hamstrings appears to be reliably estimated from just one of those muscle [124]. Antagonist coactivation may thus influence maximum torque production and should be taken into account.

Muscle activation levels also differ between contraction modes. The greatest muscular force is typically produced during eccentric contractions [122, 125], although in vivo in human this phenomenon appears to be more moderate than in isolated animal muscles [126]. This difference is probably due to the non-maximal levels of voluntary activation (< 90%) produced during voluntary eccentric contractions in humans [127, 128], and it has been proposed that this activation deficit may arise from differing commands from the central nervous system during eccentric when compared to concentric and isometric contractions [129]. However, voluntary activation can improve with training [130] and the uniqueness of the maximal eccentric contraction task in some individuals may be a primary factor for differences in eccentric torque production between individuals and population groups. Differences in the level of agonist activation will clearly affect maximum force production [5] and its adaptation with training will have a strong influence on changes in functional performance.
2.7 Training-induced adaptations in muscle activation

Increases in agonist activation with training are considered vital for enhancing force production, and appear to be particularly important for strength improvements during the initial weeks of strength training [33]. Researchers have concluded that improvements in muscle activation must underpin the increase in force following training based on i) the disproportionate increases in force and muscle size [6, 58, 131, 132]; ii) the improved performance in a training task not leading to similar improvements in a dissimilar testing task [133]; and iii) the increases in EMG amplitudes observed after periods of strength training [6, 8, 33, 47]. These increases in EMG amplitude are frequently, although not always [13, 134], observed with training-related increases in strength and are considered to reflect an increased neural drive to the muscle or changes in temporal motor unit activation characteristics (e.g. changes in motor unit synchronisation) [6, 8, 33, 47, 100, 101]. While increases in motor unit synchronisation can increase EMG amplitude, a smaller reduction in amplitude cancellation of motor unit action potentials [135] has also been observed in strength-trained athletes versus untrained controls [136, 137] and following isometric training [137]. Thus, amplitude cancellation is possibly a factor leading to the observations of a greater EMG amplitude. However, these changes are often considered a lesser influence on the training-related changes in EMG amplitude and have not been demonstrated to be related to improvements in force [135]. Therefore, the increase in EMG amplitude after strength training is commonly assumed to reflect an increase in efferent neural drive to the contracting muscle.

A large individual variability in both the likelihood of increase in EMG amplitude and its magnitude of change are observed with training [138]. This observation may reflect the inherent variable features of the EMG signal or unreliability of the recording technique [104]. Conclusions based solely on EMG amplitude should, therefore, be made with caution. Also, often only a single muscle within a synergist group is assessed in research studies (i.e. VL only [13]), and thus other functionally important muscles may be excluded from the analysis. Therefore, although increases in agonist muscle activation appear to influence the improvements in joint torque following training, it has been difficult to clearly determine the relative influence of these activation changes on active joint torque production. For the knee extensors, researchers have reported both moderate [47] and weak [35] correlations between the change in EMG amplitudes and the change in strength following a period of strength training, indicating that a substantial portion of the variability in strength increase remains unaccounted for by changes in muscle activation. Higbie et al. [35] considered this result reasonable given that EMG amplitude changes are not reflective of all possible neural adaptations following training. The use of other activation measures (e.g. antagonist EMG or percent voluntary
activation) together with agonist EMG measurements may provide more information in this regard, and this possibility requires further investigation.

Strength training interventions may also stimulate changes in antagonist activation, which has been observed to decrease through an increase in volitional reciprocal inhibition [139, 140]. In general, training-induced increases in muscle strength tend to be accompanied by decreases in coactivation, and this has been clearly shown following isometric training [141]. However exceptions are seen, with researchers observing no change [142], and even an increase [143], in antagonist activation following training. de Boer et al. [143] speculated that the increase might be a safety mechanism to maintain a similar ratio of activation between the agonist and antagonist muscle groups. Further, other researchers have found no significant change in antagonist activation following concentric or eccentric training [70, 144, 145]. The effects of training on coactivation, therefore, are still unclear. Further, more research is required to clarify whether the changes in antagonist activation with training might be substantive enough to influence joint torque.

2.8 Influence of moment arm on torque production capacity

In addition to muscle force capacity, the expression of joint torque depends on the moment arm through which the force is applied. Given that joint torque \(T_{\text{JOINT}}\) is a function of muscle force \(F_M\) and the perpendicular distance (moment arm) from the rotational joint centre to the line of action of the muscle force \(d\), \(T_{\text{JOINT}} = F_M \times d\), a large moment arm can be considered theoretically ideal for high torque production, whereas small moment arms are theoretically considered to optimise joint angular excursion and velocity [146, 147]. Moment arm distance can also affect the way joint rotation influences muscle force. Individuals with larger moment arms will experience a greater change in muscle length for a given joint rotation, and the muscle will thus shorten (or lengthen) further or more rapidly for a given joint angular displacement or velocity [78]. Therefore, moment arm distance can also influence muscle force production by altering both the force-length and force-velocity properties of the contracting muscle fibres.

Despite the importance of moment arm distance for torque production, few studies have assessed moment arm as a predictor of joint torque in humans. Blazevich et al. [11] examined the relationship between patella tendon moment arm distance and maximal knee extensor isometric and concentric joint torque but found only weak correlations \((R^2 = 0.19 - 0.25)\). However, these moment arm measurements were obtained from a homogenous group of participants at rest, and were obtained in a single, extended (0°) joint position. As moment arm distance is known to vary between individuals [148] as well as with joint angle [149-151] and contraction intensity [151, 152] this relationship may be different when measured in contracted muscles at relevant joint angles. Baxter
and Piazza [51] examined the relationship between plantarflexor moment arm and isometric (both in a neutral ankle joint position) and concentric joint torque. While their participants also remained at rest during MRI image acquisition, moderate correlations between moment arm and joint torque were observed \( R^2 = 0.32 – 0.48 \). To put this into context, correlations between plantarflexor moment arm and joint torque were stronger than the correlations between muscle volume and joint torque \( R^2 = 0.22 – 0.32 \), suggesting that the leverage of the muscle is at least as important as its size in determining maximum plantarflexor strength [51]. Further research is required to determine the relative influence of patella tendon moment arm on knee extensor joint torque when compared to other factors that influence joint torque production.

2.9 Training-induced changes in moment arm distance

Moment arm distance at peak torque may be altered following training by either i) a change in the force-length properties of the muscle, which will change the angle of peak torque and hence the moment arm at peak torque, or ii) an increase in muscle size, which may alter the line of force application. However, the angle of peak torque may [153, 154] or may not [72, 155] change following training, and the effect of the minimal alterations to the line of force caused by the change in muscle size would probably be lower than the average error associated in moment arm calculations (e.g. ~1.2-1.4 mm for the patella tendon; [156]). Considering these potentially negligible changes in moment arm distance, and the small inter-individual variability in moment arm measurements [11], previous studies appear justified in measuring moment arm distance at only a single time point for use in muscle force estimation [157]. Therefore, while torque production is influenced by the joint moment arm, changes in moment arm distance are expected to have little influence on the changes in torque following training.

2.10 Considerations for determining relationships between strength and neuromuscular variables

Based on the above research as well as theoretical predictions, it is clear that muscle size, architecture, activation and joint moment arm may play an important role in active torque production changes following strength training, however it is not clear if changes in these variables are strongly associated with the changes in strength following an intervention. Researchers have generally determined this relative importance by computing correlation statistics in observational (cross-sectional) studies. These within-population correlations provide some evidence of an association between the two variables and a positive rationale for further research.
A stronger level of evidence would be to observe that a longitudinal/training intervention led to simultaneous changes in the two related variables (e.g. increases in CSA and increases in strength). In this regard, a change in two variables that occur in agreement with an *a priori* hypothesis is somewhat suggestive, but is not proof, of a causative link. With regards to strength and muscle size or activation, for example, there is considerable evidence for such a link [35, 47]. Two other study designs, however, can be considered to provide even stronger evidence. In the first, strong linear or non-linear relationships between the changes in two variables can be examined, with the supposition that a causal link should ensure that individuals with greater changes in one measured variable should also show greater changes in the other variable. Few researchers, however, have explored these relationships [35, 63, 72]. The strongest level of evidence, however, would be to manipulate a single variable and examine the change in a second (dependent) variable. This forms the basis of the randomised control trial, where participants are randomly allocated to an intervention or control group and only a single variable differs between the groups. Unfortunately, such a design cannot be implemented to examine the effects of changes in anatomical structure or neuromuscular function on the change in strength in humans as any exercise or other intervention invariably influences more than one variable, e.g. strength training affects muscle size, architecture and muscle activation simultaneously. Given this, the best level of evidence appears to be provided by the use of intervention studies in which relationships between the change scores of variables are examined. Unfortunately, such analysis is rarely done and should be more strongly considered in the future.

### 2.11 Summary

Muscle force appears to be influenced by the complex interactions between muscle size, architecture and activation, and muscle torque is further influenced by moment arm distance. Muscle size and architecture (in particular fascicle angle) appear to be key variables in influencing maximum torque production. Yet, large inter-individual variability in there adaptations to strength training interventions, and the reasons for this variability are not well understood. Inter-individual variances in muscle size, architecture and strength imply a trade-off between maximising muscle size to improve force production, and adopting specific muscle fascicle angle or fascicle length magnitudes to adjust force production for a given muscle size. However, there is currently a poor understanding of the factors influencing trade-offs between these adaptations. Muscle activation adaptations also appear vital for influencing improvements in strength, so their importance together with other variables warrants investigation. While a majority of previously published training intervention studies have reported increases in strength and changes in individual anatomical or neuromuscular variables, an optimum study design has eluded researchers since training
interventions stimulate simultaneous anatomical and neuromuscular adaptations. In future research, therefore, a more definitive examination of the relationships needs to be undertaken. Determining how the change in these variables is related to the change in strength following training appears to be the best method available for understanding the relationships between these variables. Understanding these relationships will have important implications for the future development of training programs for both sporting and rehabilitative practices.
CHAPTER THREE: PURPOSE OF THE RESEARCH
3.1 Overview

The main purpose of this research is to improve our understanding of the influence of anatomical and neuromuscular variables on maximal muscular strength (i.e. peak active joint torque). While it is known that various anatomical and neuromuscular factors influence maximum joint torque production, few studies have assessed the relative influence of these variables using either cross-sectional or longitudinal study designs. Importantly, differences between the factors influencing torque production during isometric, concentric and eccentric contractions have not been identified by examining relationships observed in a single subject cohort under the same experimental conditions. The development of a comparative data set for the most ‘influential’ characteristics would enable efficient prescription of individualised exercise programs for both training and rehabilitation, by allowing the identification and targeting of an individual’s weaknesses and needs to be a focus of future research projects. This would allow practitioners to develop individual-specific training plans to enhance muscular strength and thus improve sport performance, optimise performance in activities of daily living and shorten the time required to return to function in injured individuals. The information would also have basic scientific impact by providing important information regarding the factors influencing human muscular strength expression.

3.2 Specific Aims and Hypotheses

Study 1.

Aim: To determine which anatomical and neuromuscular factors influence inter-individual variability in maximum knee extension torque under isometric, concentric and eccentric conditions.

The hypotheses are that

i. Muscle size and fascicle angle will be most strongly correlated with maximum voluntarily slow-speed knee extension torque in a heterogeneous (healthy male) population regardless of muscle contraction mode, and thus included in all best torque prediction models.

ii. Agonist muscle activation levels will be strongly associated with maximal isometric and eccentric, but not concentric, torque production as concentric movements are more commonly performed during activities of daily living and there should, therefore, be less scope for activation deficit.
iii. Moment arm distance will influence the maximum torque production for all contraction modes.

Study 2 (a).

Aim: To investigate whether changes in maximum knee extension torque are associated with the changes in those anatomical and neuromuscular variables identified as ‘important’ in Study 1 after completion of a moderate duration (10 weeks) heavy strength training intervention.

The hypotheses are that

i. The combination of anatomical and neuromuscular variables that show the strongest relationships with the change in torque following 10 weeks of knee extensor training will be different to those variables found to best predict maximum torque in the observational study (Study 1). This is because of the smaller range of change expected with training compared to the absolute inter-individual variability observed in Study 1.

ii. Changes in agonist muscle activation will be a primary mechanism underpinning the change in torque for all contraction modes and be included in all best models for predicting torque change following training.

iii. The change in muscle size and architecture will not be strongly related to the change in torque for any contraction mode.

Study 2 (b).

Aim: To determine whether an individual’s strength and both anatomical and neuromuscular adaptations to a heavy strength training intervention can be predicted from testing performed prior to the commencement of training.

The hypotheses are that

i. Increases in muscle activation following training will be strongly related to its pre-training magnitude, with greater increases observed in individuals who displayed lesser activation potential prior to training initiation.

ii. Changes in muscle size and architecture following training will be inversely related to their pre-training magnitudes.
CHAPTER FOUR: STUDY ONE

Anatomical and neuromuscular mechanisms influencing inter-individual variability in maximum knee extension torque
4.1 Introduction

Muscle strength is an important determinant of performance in activities of daily living and sporting tasks, and has been clearly associated with decreased fall risk, morbidity and mortality [158-160]. Despite this, the importance of the various factors (e.g. anatomical and neuromuscular) underpinning strength expression have not been agreed upon, and because programs with different load, movement speed and movement pattern characteristics trigger different neuromuscular adaptations, no single exercise training program (or range of programs) has been accepted as being optimal for muscle strength development. It is well known that the maximum voluntary joint torque is dependent upon both a muscle’s size and the architectural arrangement of its fibres [3, 4] as well as on the level of voluntary activation of those muscles [5-8]. However, several other important anatomical and neurological variables also influence maximum joint torque during human movement, including the moment arm distance about which the force is produced [11, 12] and the activation (or inhibition) of the antagonist muscles by the nervous system [9, 10]. Yet cross-sectional studies have shown significant inter-individual variation in these parameters across, and within, elderly [14], athletic [15] and untrained [13] populations, so the extent to which each of these variables influences maximal torque has yet to be fully determined. Without a clear understanding of the importance of the factors influencing strength expression, targeting training and rehabilitation programs to an individual will not be possible.

Surprisingly, the majority of previous research has examined the influence of neuromuscular factors on force production during isometric and concentric contractions, despite eccentric actions being commonly performed in activities of daily living and sporting tasks. While it is accepted that muscle size and architecture appear to be strong determinants of peak joint torque during isometric and slow speed concentric contractions [3, 11, 25], the extent to which they influence eccentric torque is unknown. It is also not known whether the influence of muscle size and architecture on maximal eccentric strength is similar across individuals with a range of activity histories involving different eccentric force producing capacities; individuals who rarely perform loaded eccentric exercises may display a lower level of muscle activation during such contractions compared to the other commonly performed concentric and isometric contractions [9, 161]. Thus individuals unaccustomed to performing maximal eccentric muscle contractions may show a greater activation deficit (compared to isometric or concentric contractions), when compared to those who commonly perform eccentric actions and this variable may thus be of more significance to their eccentric torque production capacity.
Additionally, comparative data sets relating to the most important variables influencing torque production are scarce, so identification of areas of weakness within an individual is not possible and the design of individualised programs is problematic. Collectively, more data are required to define clearly the factors influencing muscle strength and to provide comparative data for these factors. To improve the design of training programs to optimise training-induced adaptations, there is thus a need to 1) understand the relative importance of variables that influence torque production, and 2) provide a comparative data set for these variables in order to determine prescription requirements.

The ideal study design for identifying the factors most important for influencing strength expression would be a longitudinal study assessing a large number of factors, in a large number of people, and clearly defining the relationship between changes in these factors and the changes in muscular strength. However, completing such a study is difficult; i.e. imposing an intervention and testing numerous variables is both time and financially expensive, so the probable influence of each factor should be determined *a priori* in the hope of defining a smaller subset of factors for future study. Large observational (cross-sectional) studies can be used for this purpose, and may thus be completed first in a series of studies to describe the strength of relationship between anatomical and neuromuscular variables and muscular strength.

Given that the knee extensor muscles are critically important for the performance of activities of daily living (e.g. stair climbing, sit-to-stand transitions, upright balance) and are, therefore, a prime target of functional lower limb rehabilitation [21-24] and athletic [89, 162] training programs, the primary purpose of this study was to examine comprehensively the relative influence of anatomical (i.e. muscle and moment arm) and neuromuscular (muscle activation and excitation-contraction coupling) variables on maximum isometric concentric and eccentric knee extension torque. A comparative data set was also established from the results to aid with identification of potential mechanisms underpinning an individual’s strength deficit (i.e. sites of weakness). As it is clinically important to track strength capacity without performing maximal contractions, a second purpose was to determine whether an individual’s maximal torque capacity could be accurately estimated from variables that may be safely measured in clinical populations (i.e. without the need for maximal contractions or nerve/muscle stimulation procedures).
4.2 Methods

4.2.1 Participants and Study Design

Fifty-six healthy men between the ages of 19 and 40 volunteered to participate in this study (29.0 ± 5.1 years; 1.78 ± 0.06 m; and 78.6 ± 14.0 kg). To provide a heterogeneous sample population, 14 were endurance trained runners, 13 were strength trained (weightlifters), 15 were recreationally active, and 14 were untrained. Participants were considered to be endurance-trained participants if they were consistently running ≥ 50 km·wk⁻¹ for 45 weeks of the year, and strength-trained if they had been weight training 3 wk⁻¹ for the past two years and were able to squat 1.5 × body mass. The recreationally active and untrained participants were classified by their response to a metabolic work rate questionnaire [163]. The recreationally active participants tended to play two games of social sport per week, while the untrained participants had not participated in regular exercise over the past three years [164] and had an average metabolic energy equivalent score (MET) < 30 day⁻¹ (Appendix 2A). The endurance and strength trained participants were recruited through local training clubs and the recreationally active and untrained participants through advertisements. Exclusion criteria included cardiovascular and inflammatory diseases, any lower-limb injury within the last three months, and any other condition that could affect performance during the testing protocols. Prior to testing this study was approved by the Edith Cowan University Human Research Ethics Committee and conformed to the Declaration of Helsinki (Appendix 1A). Participants gave their written informed consent (Appendices 1B and 1D) and confirmed they were physically capable of participating in the testing procedures (Appendix 1F).

Participants performed six testing sessions over a 2-wk period with each session separated by at least 48 h (Figure 4.1). In both of the first two sessions, participants were familiarised with the performance of maximal isometric and isokinetic muscle contractions as well as the neuromuscular assessment protocol. In the following sessions, muscle size and architecture (ultrasound imaging), isometric maximal voluntary torque and neuromuscular activation, isokinetic maximal torque and patella tendon moment arm distance (x-ray imaging) were measured on the participant’s dominant leg. Participants were required to avoid vigorous exercise 48 h prior to testing, and to avoid consumption of coffee, alcohol or other stimulants in the 6 h period prior to testing.

Prior to the commencement of isometric and isokinetic strength testing, the participants performed a standardised warm-up of 5 min stationary cycling (Monark Ergomedic 818E, Sweden) at 60 rpm at a resistance of 1.5 kp. They then completed 5-6 submaximal contractions (either isometric or
isokinetic, depending on the test) at ~40-90% of perceived maximal exertion before beginning their maximum voluntary contractions (MVC).

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<tr>
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<td>Quadriceps Ultrasound imaging</td>
<td>Isometric muscle strength and neural activation</td>
<td>Isokinetic muscle strength</td>
<td>X-ray of knee joint</td>
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**Figure 4.1** Outline of testings sessions. All six sessions were completed over a 2-wk period.

### 4.2.2 Isometric Force and Neuromuscular Measurements

To assess maximal voluntary and electrically-induced isometric knee extension strength, participants performed at least two MVCs at 5-7 knee angles. As maximum isometric torque varies with muscle length (i.e. joint angle) the angle of maximum torque production was found for each individual by performing MVCs at 5-7 sequential angles. Participants began their MVC efforts at either 45°, 55° or 65°, based on an estimated location of each participant’s potential peak torque angle (determined during familiarisation), and progressed from an extended (i.e. short muscle length) to a flexed (i.e. long muscle length) position at 5° intervals; this progression avoided the greater fatigue associated with performing a maximal efforts at longer muscle lengths [165]. The maximum torque for each participant was taken as the maximum torque ($T_{ISO}$) produced at any one of the joint angles tested.

Force was recorded through a load cell (XTran Load Cell S1W, Applied Measurement, Sydney, Australia) attached ~5 cm superior to the participants’ lateral malleoli whilst sitting in a custom-built isometric dynamometer. The participants were seated upright and tightly secured in place with shoulder and waist straps. The lever arm length, lever arm rotation axis height, and backrest position were adjusted to ensure the axis of the knee joint was aligned with the axis of rotation of the lever arm. The load cell was calibrated with known loads prior to testing and data were collected at a 1000-Hz analogue-digital conversion rate using PowerLab hardware (AD Instruments, NSW, Australia) connected to a computer running LabChart Pro software (AD Instruments, version 7.1). Based on an amplification range of ± 10 V and a resolution of 313 µV, force increments of 0.5 N could be detected. All isometric torque data were filtered in Matlab (R2010a, MathWorks Inc., USA)
using a fourth-order, zero-lag Butterworth filter with a 15-Hz cut-off frequency. Knee joint torque was calculated by converting the load cell signal (voltage) to force (newtons) and then multiplying by the external lever length (i.e. the distance from lateral epicondyle of the femur to the force transducer attachment point). Recorded torque was corrected for the gravitational influence of the shank and foot by including the weight of the lower leg and foot (5.9% bodyweight [166]), which ensured baseline torque at each angle was 0° (Eq. 1).

\[
\sin \alpha = \frac{x}{5.9\% \text{ bodyweight}} \quad \text{[Eq. 1]}
\]

The participants were required to hold each MVC for 3 s. A single [167] supramaximal electrical stimulus (140% \( M_{\text{max}} \)) was applied 2 s before contraction onset, during the torque plateau and 2-s after each MVC to allow calculation of the maximal voluntary activation of the quadriceps (see electrical stimulation protocol below). Two MVCs were performed at each angle, with a third MVC performed if the two peak torque values differed by > 5 N-m. Real-time visual feedback of the torque data was displayed on a computer screen in front of the participant and strong verbal encouragement was given throughout each MVC. A 1-min rest interval was allowed between MVCs at the same angle, and a 2-min rest was allowed between joint angles. Prior to completion of MVC testing, the test was again performed at the first joint angle to determine whether fatigue may have been induced by the protocol (fatigued was indicated by > 5% decrease in MVC from the initial trial).

Knee angle was set with a hand-held goniometer using the greater trochanter, lateral epicondyle and the lateral malleolus as landmarks. Due to tissue compression during the maximal contractions, a digital video camera (Canon MVX200I, Canon Inc.; 25-Hz) was used to record the contractions to assess possible differences between the intended and actual joint angle at the instance of peak torque. As the knee joint was hidden from view by the chair design, the centre of the knee joint was defined by two markers located at ~33 and 66% of the distance along the participants’ femur and tibia. Intra-participant differences from the intended knee joint angle at maximum voluntary contraction were 3.6° ± 0.2° (mean ± SD). The difference between the intended and measured angles was greatest at the 50° knee angle and was 4.7° ± 0.6°. Therefore, all torque angle curves were shifted by 5° so that a truer representation of joint angle was obtained and to ensure anatomical parity with joint moment arm estimates (see below).

Isometric torque measurements included maximal voluntary peak torque (\( T_{\text{ISO}} \); considered the maximum torque produced prior to stimulation), peak unpotentiated (i.e. pre-MVC) twitch torque (\( T_{\text{Un-TW}} \)) and peak potentiated (i.e. post-MVC) twitch torque (\( T_{\text{Pot-TW}} \)). While the greatest torque at each angle was used for \( T_{\text{ISO}} \) analysis, the other torque variables were defined as the mean obtained
during the two strongest contractions. Intra-session reliability testing of the electrically-induced and voluntary torques for eight participants produced coefficients of variation (CV (mean ± SD)) of 1.2 ± 0.9, 3.6 ± 1.2 and 1.5 ± 1.4, for $T_{ISO}$, $T_{Un-TW}$ and $T_{Pot-TW}$, respectively.

4.2.3 Isokinetic Torque Measurement

Maximum concentric and eccentric knee extension contractions were performed at an angular velocity of $60°\cdot s^{-1}$ on an isokinetic dynamometer (Biodex System 3, Biodex Medical Systems, Shirley, New York, USA). The participants were seated in the dynamometer with a 5° reclined back rest and firmly strapped into the seat across the hips and chest. The lower leg was attached to the dynamometer ~5 cm superior to the lateral malleolus and the centre of rotation of the dynamometer was aligned with the lateral femoral epicondyle. The knee joint moved through a range of motion (ROM) of 100° to 15° ($0°$ = full extension). Torque signals were corrected for gravity by weighing the limb at a 20° knee angle (the slightly knee flexion ensured that tension in the knee flexor muscles was minimised). One set of three repetitions of each of four knee extension conditions were completed, including both concentric and eccentric knee extensor contractions, and concentric and eccentric knee flexor contractions. A second set was completed if the two peak torque values differed by > 5 N·m. A 3-s rest was imposed between repetitions ($30°\cdot s^{-1}$ lever arm return speed) and a 2-min rest was allowed between sets. Real-time visual feedback of the torque data was provided on a computer screen in front of the participant, strong verbal encouragement was given throughout each test and the maximum torque value was used for analysis ($T_{CON}$ and $T_{ECC}$). Intra-day and inter-day reliability testing (1-wk interval) for the three maximal contractions concentric and eccentric knee extension contractions yielded CVs of 2.5 and 3.4% (intra-day) and 4.1 and 1.4% (inter-day) in 10 participants.

4.2.4 Electrical Stimulation Protocol

Electrical stimulation was used to estimate voluntary activation capacity. After a 5 min warm-up (60 rev·min⁻¹, 1.5 kg load) on a cycle ergometer (Monark Ergomedic 818E, Sweden), the femoral nerve was located using ultrasound imaging with the participant seated in the rigid chair. A self-adhesive cathode (diameter = 1 cm) was positioned 0.5 cm medial and inferior to the femoral nerve and the anode (diameter = 1 cm) was placed 2 cm lateral and superior to this position. This placement was shown to elicit the most reliable motor response in pilot testing; however the cathode location was sometimes varied in order to elicit the greatest M-wave response at a submaximal stimulation intensity. The participants were seated with a knee joint angle of 70° and instructed to remain relaxed throughout the stimulation procedure. The stimulation intensity required to elicit a maximal
motor response (maximum muscle compound action potential (M-wave)) was found through electrical femoral nerve stimulation using single 2-ms (400 V) rectangular stimuli from a high-voltage, constant-current stimulator (Digitimer, model DS7AH, Welwyn Garden City, UK). While the signal to noise ratio is increased by employing two or more stimuli, the single twitch, in this study, was able to detect incomplete activation levels similar to other recent literature [e.g. 168]. The stimulation intensity was gradually increased until the vastus lateralis (VL) M-wave plateau (maximal peak-to-peak amplitude; $M_{\text{max}}$) was reached. All further stimulations were conducted at an electrical current intensity of 140% $M_{\text{max}}$.

### 4.2.5 Muscle Activation

For both the isometric and isokinetic measurements, electromyogram signals (EMG) were obtained from rectus femoris (RF), vastus lateralis (VL) and vastus medialis (VM) using bipolar silver/silver chloride surface electrodes (10 mm diameter; Kendall Healthcare, Medi-Trace™ 200 Series, USA) arranged in a bipolar configuration with a 20-mm inter-electrode distance. To monitor hamstring involvement (coactivation) EMG signals were also recorded from the long head of biceps femoris (BF). After careful preparation of the skin (shaving, abrasion and cleaning with alcohol to obtain low inter-electrode resistance) the electrodes were placed on the muscles according to SENIAM guidelines [169] (~24, 16 and 11 cm above the patella on RF, VL and VM, respectively, and ~20 superior to the lateral epicondyle for BF), in line with the assumed longitudinal direction of the muscle fibres. A reference electrode was placed on the proximal shaft of the tibia. The exact location for each electrode was carefully measured to ensure identical placements across the testing sessions. The raw surface EMG signals were amplified (gain = 1000) and collected at an analogue-digital conversion rate of 1000 Hz (Grass Amplifier, Grass Instruments, Greenwich Ave, RI). The amplifier was interfaced with a computer via a circuit board (National Instruments Compact DAQ), and all EMG signal post-processing was conducted in Matlab (R2010a, MathWorks Inc., USA) using custom-built programs. EMG data were filtered using a fourth-order, zero-lag band pass (10-500 Hz) Butterworth filter.

To quantify EMG activity during isometric MVCs the root mean square (RMS) values were measured in the 500 ms prior to superimposed stimulation [170, 171], after accounting for an electromechanical delay (EMD) of 70 ms [16]. No statistical difference was found between the unpotentiated and potentiated M-wave amplitudes (n = 10), but greater variability was observed in potentiated M-wave amplitudes both between and within individuals, thus unpotentiated M-wave amplitudes were chosen for agonist EMG normalisation. M-wave amplitude was calculated as the unfiltered peak-to-peak amplitude from the unpotentiated stimulation and was used to normalise
agonist muscle MVC EMG (EMG:M-wave ratio), which was considered to provide a measure of central efferent drive to the quadriceps muscles [172]. For isokinetic contractions, the RMS EMG values were obtained over a 30° range (covering 500 ms) around the angle of peak torque, after again accounting for an EMD of 70 ms, and the agonist muscles’ EMG amplitudes again normalised to the average peak-to-peak M-wave amplitude. As peripheral excitability is dependent upon muscle length [170, 173], average M-wave peak-to-peak amplitudes were obtained from the unpotentiated stimulations produced at the same angle as the angle of peak torque during the eccentric and the concentric contractions. Thus, as the isometric contractions were obtained at 5° intervals, the M-wave amplitude was always obtained from a joint angle within 2.5° of the angle at isokinetic peak torque.

The isometric and isokinetic antagonist EMG amplitudes were normalised to the EMG amplitude obtained during a knee flexion MVC. As antagonist muscles typically lengthen during the inevitable joint rotation in isometric contractions [174], and the hamstrings are presumed to be active whilst lengthening during a concentric contraction [9], the antagonist EMG from both isometric and concentric contractions were normalised to eccentric knee flexion MVC. By contrast, eccentric antagonist EMG was normalised to the maximum EMG amplitude obtained during a concentric knee flexion MVC.

Maximal voluntary quadriceps activation during isometric contractions was estimated using the interpolated twitch method [171]. Percent voluntary activation (%VA) was calculated as the ratio of the amplitude of the superimposed twitch torque to the post-MVC twitch torque as per equation 2:

\[
%VA = \left(1 - \frac{\text{superimposed twitch}}{\text{final twitch}}\right) \times 100
\]

[Eq. 2]

In order to synchronise the torque and electromyogram (EMG) data for both the isometric and isokinetic contractions, torque data were collected through a custom-made cable through the Powerlab system (AD Instruments, NSW, Australia), using LabChart Pro software (AD Instruments, version 7.1) with a synchronisation stimulus appearing in both the torque data and the EMG data via a data acquisition device (Compact DAQ, National Instruments, Texas; USA).

4.2.6 Calculation of Hamstring and Quadriceps Contributions to Net Joint Torque

The net knee joint torque obtained during isometric and isokinetic contractions represents the torque generated by both the agonist and antagonist muscles. To estimate the torque produced by the quadriceps muscles alone (i.e. knee extensor torque), the antagonist torque developed by the hamstrings was estimated and then added to the total joint torque. This was done with the intent of
developing models for both total knee extension and knee extensor-only torque prediction. To do this, the linear relationship between knee flexor torque and BF EMG activity [124] was obtained from both maximal eccentric and concentric knee flexion contractions; the maximum concentric knee extensor torque was estimated after correcting for BF activity obtained from the EMG-torque relationship measured from a maximal eccentric knee flexion contraction (because the hamstrings are active whilst lengthening in a concentric knee extension), whereas the maximum eccentric knee extensor torque was calculated using the EMG-torque relation obtained during a maximal concentric knee flexion contraction. Because antagonist muscles typically lengthen during the inevitable joint rotation in ‘isometric’ contractions [174], the eccentric knee flexion contractions were also used for estimation of maximum isometric knee extensor torque. Since a quadriceps antagonist force still exists during maximal knee flexion contractions, a set of equations developed by Aagaard and colleagues [9] was used to separate joint torque into knee extensor torque ($T_Q$) and knee flexor torque ($T_H$). The full equations are shown in Appendix 2B with a slight modification. Knee extensor and knee flexor joint torques for each contraction mode were calculated separately using equations 3 and 4.

$$T_Q(\hat{\theta}) = K_1(\hat{\theta}) \cdot \text{EMG}_Q(\hat{\theta}) \quad \text{[Eq. 3]}$$

$$T_H(\hat{\theta}) = K_2(\hat{\theta}) \cdot \text{EMG}_H(\hat{\theta}) \quad \text{[Eq. 4]}$$

EMG$_Q$ was calculated as the average of RF, VL and VM EMG$_{RMS}$ amplitudes, and EMG$_H$ represents the BF EMG$_{RMS}$ amplitude. The torque and EMG$_{RMS}$ values of the strongest two trials were averaged to provide the mean EMG$_{RMS}$ for each muscle. Both EMG$_Q$ and EMG$_H$ were normalised relative to the average EMG$_{RMS}$ amplitude obtained in knee extension and flexion agonist contractions, respectively, to ensure variations in EMG readings between the two maximum trials did not influence the equations. The EMG-torque coefficients $K_1$ and $K_2$ were calculated using quadriceps- or hamstrings-only torque and EMG amplitudes obtained during each contraction at the specific angle at which maximum torque for the quadriceps contraction occurred. Using the specific angle ensured that muscle length was constant between both the quadriceps and hamstring contractions. Total knee extensor and knee flexor torque for each contraction could then be calculated.

### 4.2.7 Muscle Size and Architecture

Muscle anatomical cross-sectional area (CSA) and both muscle fascicle length ($l_f$) and fascicle angle ($\Theta_f$) were obtained using two-dimensional B-mode ultrasonography (Aloka SSD-α10, software number 6.1.0, Aloka Co., Ltd., Tokyo, Japan) using a 10 MHz 60-mm linear-array transducer. All ultrasound images were taken in extended-field-of-view mode (see Figure 4.2) [175]. Anatomical
CSA was considered representative of muscle size as it has been shown to be a good predictor of muscle force under both isometric and isokinetic conditions [11, 29, 46, 54] and allows the examination of region-specific differences between individuals. Water-soluble gel placed between the transducer and the skin aided acoustic coupling, and care was taken to minimise the pressure applied to the muscle. The images were obtained with the participants lying supine with their legs fully extended and their muscles relaxed. A rolled towel was placed underneath the knee joint to remove compression of the muscles.

Figure 4.2. Examples of raw and analysed ultrasound images of distal cross-sectional area (A and B); fascicle length at mid VL and VI (C and D) and VM fascicle length and angle (E and F).

CSA measurements were obtained at 30, 40 and 50% of thigh length (measured from the centre of the patella to the medial aspect of the anterior superior iliac spine [176]). This site placement ensured the capture of the midpoint of the muscle belly of VL and VI (i.e. 40% length) and the midpoint of the muscle belly of RF when tendon length was taken into consideration (50%). Fascicle length and angle measurements were obtained from all four quadriceps components to account for heterogeneity within and between the muscles [75]. After measuring the distance between the lateral border of the patella and greater trochanter, three sites were acquired along VL (33, 50 and
67% providing distal mid and proximal regions), and one on VI (mid-region; lateral view). VM images were obtained from the 25% ACSA site and RF from the 50% ACSA site, as mentioned above.

The measurement point was marked by 4-mm wide adhesive tape strips which caused a shadow in the ultrasound image, and fascicle length was defined as the distance between the superficial and deep aponeuroses of the fascicle that crossed the mid-point of the shadow. Fascicle angle measurements were then obtained from the same fascicle. To avoid the slightly greater fascicle curvature at the fascicle insertion onto the deep aponeurosis, fascicle angle was measured from 3-mm above the deep aponeurosis [75] to a line drawn 50% along the length of the fascicle. Because of the significant curvature of VM fascicles, their locations were slightly different; VM fascicle length was measured from the fascicle that crossed 1/3 the distance between the superficial and deep aponeuroses and VM fascicle angle was defined as that from the deep aponeurosis to 2 cm along the length of the fascicle. Three scans were acquired at each location (for both CSA and fascicle measurements) and the median of these values was used for analysis. All measurements were manually traced using ImageJ software (1.41o, National Institute of Health, USA). CVs for CSA range from 0.9 ± 0.9 to 1.5 ± 1.2 with the smallest obtained at CSA_PROX and the largest at CSA_MID; CVs for FL range from 1.7 ± 1.0 to 3.7 ± 2.1 with the smallest obtained at VI and the largest at VL_DIST; and CVs for FA range from 1.7 ± 1.0 to 3.8 ± 2.5 with the smallest obtained at VM and the largest at RF.

4.2.8 Moment Arm Distance

The patella tendon moment arm distance was obtained using seven sagittal-plane, low-radiation x-rays of the knee joint (Siemens Multi-MT 1384 model number 4803404). Participants lay in a supine position with their knees flexed and their feet against a custom-built wooden frame. The seven knee joint angles (40, 50, 60, 70, 80, 90, 100°) were set using a hand-held goniometer. As patella tendon moment arm increases during muscle contractions [151], the participants performed isometric knee extension contractions against the foot plate to approximately 60% of MVC (quantified in six participants by replicating contractions against a strain gauge in a previous session). Pilot data obtained in five participants showed a negligible difference in patella tendon moment arm in contractions ranging between 50 and 100% MVC, so the 60% effort was used in order to minimise leg movement during the scanning that occurred when maximal contractions were performed. Hand-held straps were provided to prevent possible limb extension and subsequent knee joint angle changes during the contractions.

The patella tendon moment arm distance was measured as the perpendicular distance from the line of action of the patellar tendon to the instantaneous centre of rotation (ICR) [177]. The ICR was
located in Photoshop software (Adobe Photoshop CS5) using the Reuleaux graphical analysis method [152, 177] (Figure 4.3) and the patella tendon moment arm distance then measured. One limitation of the ICR method is that the multiple steps of manual processing involved in the process increase the potential for error. Previous researchers have manually traced the outline of the bones onto transparency sheets and yielded coefficients of variation (CV) of 3.9 and 7.9% [178, 179]. In this study, the overlaying of the multiple images was performed on the computer, and inter-day analysis reliability (six participants over 3-sessions) across all five joint angles yielded a CV of 3.1 ± 2.0% (~1.4°). The method itself involved four steps: 1) the seven joint angles were overlayed along the tibia to locate an identical marker point 10 cm distal to the proximal end of the tibia (through the use of rectangular box images); 2) the femur was then assumed to be the fixed segment and the tibia the rotating limb, and all seven joint images were overlayed by superimposing the femur bones; 3) the ICR was then located on the two images surrounding the joint of interest (e.g. 70 and 90° for the MA at 80°) by finding the midpoint between the anterior and the posterior markers of the two joints (Figure 4.3 A); 4) the angle of interest was then overlayed and the MA was measured as the perpendicular distance from the ICR to the patella tendon (Figure 4.3 B). A third-order polynomial [180] ($R^2 > 0.90$) was fitted to five measured moment arm distance from 50-90° to provide an estimated moment arm distance for each individual over the entire joint angle range.

![Figure 4.3](image)

**Figure 4.3.** Illustration of the patella tendon moment arm analysis at an 80° joint angle. Perpendicular lines were drawn from the mid-point between the anterior and posterior markers on the tibia (i.e. at 70° and 90°) (A); the angle of interest (80°) was overlayed into the image (by superimposing the femoral condyles) so the ICR at this angle could be identified, and the moment arm distance (MA) was then measured as the perpendicular distance from the ICR to the patella tendon (PT) (B).

### 4.2.9 Data analysis

In order to determine which variables could be used to explain the inter-individual variation in joint torque production, the six measures of maximum torque: isometric (ISO), concentric (CON), eccentric (ECC), and quadriceps-only isometric ($ISO_{QUAD}$), concentric ($CON_{QUAD}$) and eccentric
were modelled using a set of regression models. The distribution of the data within each torque measure was visually checked for normality. Potential violations were examined using the Shapiro-Wilk test and all data were considered normally distributed.

Theoretically plausible models developed a priori were tested to determine the strength of their relationship with maximum torque (see Table 4.1 for examples). These included combinations of cross-sectional area (CSA), agonist and antagonist muscle activity magnitudes (EMG:M and EMG normalised to MVC), moment arm distance (MA), fascicle angle ($\theta_f$), fascicle length ($l_f$), percent voluntary activation (%VA) and unpotentiated twitch torque ($T_{Un-tw}$). Potentiated twitch torque was excluded from the models as it is influenced by the magnitude of the preceding MVC. The first step was to construct scatterplots of the anatomical or neuromuscular variables and maximum joint torque for each contraction mode. When the relationship between the variables and maximum joint torque appeared, on observation, to be curvilinear, these variables were included in the models (combined with the linear variables), and fitted to polynomials until the $R^2$ increased by less than 2% [181]. Correlations were then computed to assess the isolated relationships between the anatomical and neuromuscular variables and the measures of maximal torque for each contraction mode.

Because muscle size and activation (e.g. EMG activity) are commonly measured when assessing strength [6, 13, 34] a ‘CSA$_{mid}$ + EMG:M’ model was also included (using CSA obtained at mid-thigh, which is the common location of single-site CSA measurements [11, 25, 67, 68], and the best measure of ‘neural activation’ for that strength measure) to determine whether such a model would produce a better fit than the new models. Percent VA, while only obtained during the isometric contractions was also included in the eccentric models to 'infer' an individual's voluntary activation capacity in comparison to the others. Correlations between the predictor variables were also examined. When a group of variables produced high correlations ($R^2 > 0.40$ [182] (e.g. CSAPROX, CSA$_{mid}$, and CSA$_{dist}$ and CSA$_{sum}$, which all define separate CSA locations) the variable with the strongest correlation with the other predictors in that model was chosen.

The best-fitting model was then selected using an information-theoretic approach for model selection based on Akaike's Information Criterion (AIC) [183, 184]. The AIC process ranks the models and explains which model is closest to reality. It is a relative measure of the best model within the set of candidate models based on both its descriptive accuracy and minimum complexity. The information-theoretic approach was chosen over the traditionally used stepwise multiple regression as it is considered a much stronger method for model development. Strengths of the AIC model selection process are that the models are developed based on theoretic rationale, rather than multiple hypothesis testing, and also the acknowledgement that competing models may explain the
data equally as well as the best model, rather than inappropriately focusing on the single best model [185].

The models contained within the candidate model set for each contraction mode included models theoretically considered a priori to compete for the highest ranking, clinical models (models that do not incorporate maximal voluntary or electrically elicited torque measures) and comparison models (e.g. CSA_{PROX} verse CSA_{MID}). To rank the models, the AIC adjusted for small sample size (AIC_{c}) was used [183]. The model with the lowest AIC_{c} value was considered the best fit for that strength measure, and all models with a ΔAIC_{c} ≤ 2 were considered to have substantial support as prediction models [183]. Models with ΔAIC_{c} 4 – 7 have less support, and models with ΔAIC_{c} ≥ 10 are considered not to support the data [183]. For this thesis, candidate models displaying a ΔAIC_{c} ≤ 4 will be discussed as having ‘moderate’ support. The AIC_{c} weight (AIC_{c}w) identifies the probability of each model being the best-fit model amongst that combination of candidate models (i.e. an AIC_{c}w of 0.48 indicates that 48% of the time that candidate model is likely to be the best-fit model amongst that set of candidate models) [186]. Twenty-eight models were developed for each contraction mode with the combinations of variables based on both their theoretical likelihood of influencing maximum torque, and on the strength of their individual correlations with maximum torque. Models with an AIC_{c}w weighting of 0.00 were then excluded from the final results table (besides the CSA_{MID} and the best clinical model which were retained for comparison), leaving 12 to 14 models for each contraction mode (i.e. see Table 4.2).

The ‘best clinical model’ was also identified from the AIC_{c} values. The best model for clinical practice was defined as the best ranked model that did not include any maximal voluntary or electrically-elicited torque variables. The best-fit clinical model, therefore, could not include EMG and %VA predictors as they are obtained during MVCs (and are not possible in knee-injured or arthritic patients) or with electrical stimulation (i.e. not advisable in stroke patients with an intercranial haemorrhage, or individuals with a diastolic blood pressure > 120 mmHg [187]).

Model validity was checked using the variance inflation factor to ensure that no two predictors were highly correlated. Adjusted R^2 values were used in combination to show the percentage of torque that could be explained by those models. A comparative data set was then determined by taking the quartiles of each of the predictors from the best models whose ΔAIC_{c} ≤ 4 from the model of best fit. All data were analysed using R version 3.0.0 (R Development Core Team, 2013). The models were developed using the AICmodavg package [188]. Pearson’s correlation coefficients were also computed to assess the isolated relationships between the anatomical and neuromuscular variables and the measures of maximal torque for each contraction mode.
4.3 Results

Strong correlations were observed between total active knee joint torque and knee extensor torque (adjusted for the contribution of hamstring torque), which were similar for each contraction mode ($r = 0.96, 0.91$ and $0.95$ for isometric, concentric and eccentric contractions, respectively). While some individual correlations differed slightly between the two outcome variables (e.g. $r = 0.40$ vs. $0.48$ for correlations between moment arm (MA) and maximum ISO and ISO\textsubscript{QUAD} torque, and $r = 0.76$ vs. $0.68$ for correlations between proximal region CSA and maximum CON and CON\textsubscript{QUAD} torque, respectively; data not shown) very little difference was observed in the best-fit models for each contraction mode. Therefore, only total active knee joint torque was chosen for presentation and these equations can be assumed to reflect the variables that also predict knee extensor torque (i.e. adjusted for coactivation torque). Additionally, non-linear versions of the anatomical and neuromuscular variables did not improve the strength of the candidate models, so all best-fit and clinical best-fit models included only linear relationships in the final candidate model sets.

4.3.1 Prediction Models

Best-fit model:

The linear combination of ‘CSA\textsubscript{PROX} + $\theta_i$ + EMG:M + `%VA’ best predicted both maximum isometric ($R^2 = 0.72$, AIC\textsubscript{c} weight = 0.38) and eccentric ($R^2 = 0.62$, AIC\textsubscript{c} weight = 0.32) knee extension torque magnitudes, whilst maximum concentric torque was best predicted by the model ‘CSA\textsubscript{PROX} + $\theta_i$ + MA’ ($R^2 = 0.65$, AIC\textsubscript{c} weight = 0.21). However, the site from which the variables were measured (proximal, middle or distal sites) and the muscle chosen (whole quadriceps, RF, VL or VI) differed between contraction modes (Table 4.1). CSA\textsubscript{PROX} was included in the best-fit models for all contraction modes, rather than the traditionally used CSA\textsubscript{MID} variable. VL fascicle angle was included in all best-fit models, as opposed to fascicle angle measured in VI or RF, but the measurement site varied between contraction modes (i.e. VL\textsubscript{MID} for the isometric, and VL\textsubscript{PROX} for the concentric and eccentric models). EMG:M\textsubscript{AVEQ} was utilised in the isometric, but EMG:M\textsubscript{VL} in the eccentric, torque prediction models. While muscle activation was not included in the best-fit concentric torque prediction model, EMG:M\textsubscript{AVEQ} was included in other concentric prediction models ranked below the best-fit model but which had substantial support (i.e. $\Delta$AIC\textsubscript{c} $\leq$ 2, Table 4.2). MA was included in all concentric torque prediction models, and also had substantial support in eccentric torque prediction models, though it was not included in any supported model predicting isometric torque. EMG\textsubscript{BF} coactivation was included in the models that received substantial support ($\Delta$AIC\textsubscript{c} $\leq$ 2) for isometric and eccentric torque prediction. Models predicting concentric torque incorporating VI fascicle length also obtained substantial support. Comparing the best-fit models to the observed torque data, maximum
isometric, concentric and eccentric torque were predicted with a mean absolute error (± SE) of 12.2 ± 1.9%, 9.1 ± 0.9%, and 13.4 ± 1.5%, respectively (see Figure 4.4).

Best model for clinical practice:
The ‘best clinical model’ was regarded as the best model that did not include any maximal voluntary or electrically-elicited variables, which may not be possible to assess in the clinical context. All best clinical models incorporated ‘CSA_{PROX} + \theta_{f} + MA’ (Table 4.1). Fascicle angle obtained from VL_{MID} was incorporated into the best clinical model to predict isometric torque, whilst VL_{PROX} was incorporated into the concentric and eccentric torque prediction models. With regards to the AIC_{C} ranking, the best clinical model for the prediction of isometric torque had no support compared to the best-fit model (ΔAIC_{C} ≥ 10). However, if ranked against other clinical models, it still explained 62% of the inter-individual variation in maximum torque. Importantly there was strong support for the clinical model to predict concentric torque, as it was the best-fit model. The best clinical model for eccentric torque prediction had less support (ΔAIC_{C} = 5.91) and explained only 53% of the variance. Comparing the best clinical models to the observed torque data, maximum isometric, concentric and eccentric torque could be predicted with a mean absolute error (± SE) of 15.1 ± 1.7%, 9.1 ± 0.9%, and 15.0 ± 1.7%, respectively (see Figure 4.4).
Table 4.1. Best-fit models for maximum isometric, and isokinetic concentric and eccentric torque (based on $\text{AIC}_C$) and the ‘best clinical model, with the equations provided to enable maximum torque predictions.

<table>
<thead>
<tr>
<th>Maximum Torque</th>
<th>Best-fit Model</th>
<th>Equation</th>
<th>$\Delta \text{AIC}_C$</th>
<th>$R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>'Best-fit model’</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Isometric</td>
<td>$\text{CSA}<em>\text{PROX} + \text{EMG:M}</em>{\text{AVEQ}} + \theta_f \text{VL}_{\text{MID}} + %\text{VA}$</td>
<td>$Y=3.24(\text{CSA}) + 427.29(\text{EMG}) + 3.16(\theta_f) + 2.93(%\text{VA}) - 290.05$</td>
<td>-</td>
<td>0.72</td>
</tr>
<tr>
<td>Concentric</td>
<td>$\text{CSA}<em>\text{PROX} + \theta_f \text{VL}</em>\text{PROX} + \text{MA}$</td>
<td>$Y=2.34(\text{CSA}) + 2.12(\theta_f) + 2.74(\text{MA}) - 126.81$</td>
<td>-</td>
<td>0.65</td>
</tr>
<tr>
<td>Eccentric</td>
<td>$\text{CSA}<em>\text{PROX} + \text{EMG:M}</em>{\text{VL}} + \theta_f \text{VL}_\text{PROX} + %\text{VA}$</td>
<td>$Y=2.43(\text{CSA}) + 582.45(\text{EMG}) + 4.59(\theta_f) + 1.72(%\text{VA}) - 183.40$</td>
<td>-</td>
<td>0.62</td>
</tr>
<tr>
<td><strong>'Best Clinical Model’</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Isometric</td>
<td>$\text{CSA}<em>\text{PROX} + \theta_f \text{VL}</em>{\text{MID}} + \text{MA}$</td>
<td>$Y=3.17(\text{CSA}) + 3.23(\theta_f) + 1.33(\text{MA}) - 94.23$</td>
<td>15.23</td>
<td>0.62</td>
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<tr>
<td>Concentric</td>
<td>$\text{CSA}<em>\text{PROX} + \theta_f \text{VL}</em>\text{PROX} + \text{MA}$</td>
<td>$Y=2.34(\text{CSA}) + 2.12(\theta_f) + 2.74(\text{MA}) - 126.81$</td>
<td>0.00</td>
<td>0.65</td>
</tr>
<tr>
<td>Eccentric</td>
<td>$\text{CSA}<em>\text{PROX} + \theta_f \text{VL}</em>\text{PROX} + \text{MA}$</td>
<td>$Y=2.49(\text{CSA}) + 4.76(\theta_f) + 2.05(\text{MA}) - 103.44$</td>
<td>8.05</td>
<td>0.54</td>
</tr>
</tbody>
</table>

$\text{CSA}_\text{PROX} = \text{proximal cross-sectional area}; \text{EMG:M}_{\text{AVEQ}}, \text{EMG:M}_{\text{VL}} = \text{normalised average quadriceps (AVEQ) and (VL) EMG:M amplitude}; \theta_f \text{VL}_{\text{MID}}$ and $\theta_f \text{VL}_\text{PROX} = \text{fascicle angle of VL from the mid and proximal regions, respectively}; \%\text{VA} = \text{percent voluntary activation (obtained during the isometric contraction)}; \text{MA} = \text{patella tendon moment arm distance}. \text{The ‘best clinical model’ refers to the highest ranked model that did not include any maximal voluntary or electrically elicited variables. The $\Delta \text{AIC}_C$ of the best-fit model of each candidate model set = 0. The $\Delta \text{AIC}_C$ for the clinical models indicates the support for the clinical model in comparison to the best-fit model within that candidate model set ($\Delta \text{AIC}_C \geq 10$ indicates no support).} \ R^2 = \text{adjusted } R^2.$
Figure 4.4. Predicted torque was modelled based on the AICc rankings using the best-fit model (pink; y1) and the best clinical model (i.e. no maximal contraction required) (blue; y2) for maximal isometric, and isokinetic concentric and eccentric torque prediction. Figures show the mean (± SE) for each model. CSA_{PROX} = proximal cross-sectional area; EMG:M_{AVEQ} and EMG:M_{VL} = normalised average quadriceps (AVEQ) and VL EMG:M amplitudes; θ_{VL\text{MID}} and θ_{VL\text{PROX}} = VL fascicle angle obtained at mid-muscle and proximal regions, respectively; %VA = percent voluntary activation (obtained during the isometric contraction) MA = patella tendon moment arm distance. $R^2 = \text{adjusted } R^2$. 

\begin{align*}
y_1 &= \text{CSA}_{\text{PROX}} + \text{EMG:M}_{\text{AVEQ}} + \theta_{\text{VL\text{MID}}} + \%\text{VA} \quad (R^2 = 0.72) \\
y_2 &= \text{CSA}_{\text{PROX}} + \theta_{\text{VL\text{MID}}} + \text{MA} \quad (R^2 = 0.62)
\end{align*}

y represents both the best-fit and the best clinical model

\begin{align*}
y &= \text{CSA}_{\text{PROX}} + \theta_{\text{VL\text{PROX}}} + \text{MA} \quad (R^2 = 0.65) \\
y_1 &= \text{CSA}_{\text{PROX}} + \text{EMG:M}_{\text{VL}} + \theta_{\text{VL\text{PROX}}} + \%\text{VA} \quad (R^2 = 0.62) \\
y_2 &= \text{CSA}_{\text{PROX}} + \theta_{\text{VL\text{PROX}}} + \text{MA} \quad (R^2 = 0.54)
\end{align*}
Table 4.2. Akaike's Information Criterion of model parameters for predicting maximal isometric (a), and isokinetic concentric (b) and eccentric (c) torque. The 'best-fit model' and the 'best clinical model' for each contraction mode are presented in bold. Models with both substantial support (ΔAICc ≤ 2) and AICc < 0.10 (i.e. greater than a 10% chance that they will be the best fit model) are identified by shading.

(a) Isometric Torque Models

<table>
<thead>
<tr>
<th>Model</th>
<th>K</th>
<th>AICc</th>
<th>ΔAICc</th>
<th>ΔAICc</th>
<th>AICcw</th>
<th>LL</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSAprox + EMG:Maveq + θiVLm + %VA</td>
<td>6</td>
<td>559.25</td>
<td>0.00</td>
<td>0.38</td>
<td>-272.73</td>
<td>0.72</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Maveq + θiVLm + EMGBF + %VA</td>
<td>7</td>
<td>560.78</td>
<td>1.52</td>
<td>0.18</td>
<td>-272.19</td>
<td>0.72</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Maveq + θiVLm + T1un-TW</td>
<td>6</td>
<td>561.64</td>
<td>2.39</td>
<td>0.12</td>
<td>-273.93</td>
<td>0.71</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Maveq + θiVLm + VA + iVLp</td>
<td>7</td>
<td>561.67</td>
<td>2.41</td>
<td>0.11</td>
<td>-272.62</td>
<td>0.72</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Maveq + θiVLm</td>
<td>5</td>
<td>562.88</td>
<td>3.63</td>
<td>0.06</td>
<td>-275.82</td>
<td>0.70</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Maveq + θiVLm + T1un-TW + MA</td>
<td>7</td>
<td>564.00</td>
<td>4.75</td>
<td>0.04</td>
<td>-273.78</td>
<td>0.71</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Maveq + θiVLm + EMGB</td>
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<td>-275.76</td>
<td>0.70</td>
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</tr>
<tr>
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<td>564.64</td>
<td>5.29</td>
<td>0.03</td>
<td>-276.65</td>
<td>0.69</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Maveq + θiVLm + MA</td>
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<td>564.92</td>
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<td>0.02</td>
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<td>0.69</td>
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<tr>
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<td>564.99</td>
<td>5.74</td>
<td>0.02</td>
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<td>0.69</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Maveq</td>
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<td>CSAprox + θiVLm + MA</td>
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<td>-281.61</td>
<td>0.62</td>
<td></td>
</tr>
<tr>
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<td>578.20</td>
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<td>0.00</td>
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<td>0.59</td>
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</tr>
<tr>
<td>Intercept Only</td>
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<td>64.19</td>
<td>0.00</td>
<td>-309.60</td>
<td>NA</td>
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(b) Concentric Torque Models

<table>
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<tr>
<th>Model</th>
<th>K</th>
<th>AICc</th>
<th>ΔAICc</th>
<th>ΔAICc</th>
<th>AICcw</th>
<th>LL</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSAprox + θiVLm + MA</td>
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<td>573.48</td>
<td>0.00</td>
<td>0.21</td>
<td>-281.15</td>
<td>0.65</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Maveq + θiVLm + MA</td>
<td>6</td>
<td>573.69</td>
<td>0.21</td>
<td>0.19</td>
<td>-280.00</td>
<td>0.66</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Maveq + θiVLm</td>
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<td>1.75</td>
<td>0.09</td>
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<td>0.64</td>
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</tr>
<tr>
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<td>0.65</td>
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</tr>
<tr>
<td>CSAprox + EMG:Maveq + θiVLm + iVI + MA</td>
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<td>-279.57</td>
<td>0.65</td>
<td></td>
</tr>
<tr>
<td>CSAprox + MA</td>
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<td>575.43</td>
<td>1.95</td>
<td>0.08</td>
<td>-283.33</td>
<td>0.63</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Maveq + EMGBF + θiVLm + MA</td>
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<td>575.58</td>
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<td>0.65</td>
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</tr>
<tr>
<td>CSAprox + θiVLm + iVLp + MA</td>
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<td>-280.96</td>
<td>0.64</td>
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</tr>
<tr>
<td>CSAprox + EMGBF + MA</td>
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<td>575.65</td>
<td>2.17</td>
<td>0.07</td>
<td>-282.23</td>
<td>0.63</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Maveq + iVI + MA</td>
<td>6</td>
<td>577.52</td>
<td>4.05</td>
<td>0.03</td>
<td>-281.92</td>
<td>0.65</td>
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</tr>
<tr>
<td>CSAprox + EMG:Maveq</td>
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<td>579.26</td>
<td>5.78</td>
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<td>-285.25</td>
<td>0.60</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Maveq</td>
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<td>587.57</td>
<td>14.09</td>
<td>0.00</td>
<td>-289.40</td>
<td>0.54</td>
<td></td>
</tr>
<tr>
<td>Intercept Only</td>
<td>2</td>
<td>598.72</td>
<td>50.53</td>
<td>0.00</td>
<td>-297.24</td>
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</table>

(c) Eccentric Torque Models

<table>
<thead>
<tr>
<th>Model</th>
<th>K</th>
<th>AICc</th>
<th>ΔAICc</th>
<th>ΔAICc</th>
<th>AICcw</th>
<th>LL</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSAprox + EMG:Mvl + θiVLp + %VA</td>
<td>6</td>
<td>553.18</td>
<td>0.00</td>
<td>0.32</td>
<td>-269.65</td>
<td>0.62</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Mvl + θiVLp + MA + %VA</td>
<td>7</td>
<td>553.72</td>
<td>0.54</td>
<td>0.24</td>
<td>-268.59</td>
<td>0.62</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Mvl + θiVLp + EMGBF + %VA</td>
<td>7</td>
<td>554.77</td>
<td>1.59</td>
<td>0.14</td>
<td>-269.11</td>
<td>0.61</td>
<td></td>
</tr>
<tr>
<td>CSAprox + θiVLp + MA + VA</td>
<td>6</td>
<td>555.54</td>
<td>2.36</td>
<td>0.10</td>
<td>-270.84</td>
<td>0.60</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Mvl + θiVLp</td>
<td>5</td>
<td>555.59</td>
<td>2.42</td>
<td>0.10</td>
<td>-272.15</td>
<td>0.59</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Mvl + θiVLp</td>
<td>6</td>
<td>557.49</td>
<td>4.32</td>
<td>0.04</td>
<td>-271.81</td>
<td>0.59</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Mvl + θiVLp + EMGBF</td>
<td>6</td>
<td>557.57</td>
<td>4.40</td>
<td>0.04</td>
<td>-271.85</td>
<td>0.58</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Mvl</td>
<td>4</td>
<td>560.46</td>
<td>7.29</td>
<td>0.01</td>
<td>-275.81</td>
<td>0.53</td>
<td></td>
</tr>
<tr>
<td>CSAprox + EMG:Mvl + iVI + %VA</td>
<td>6</td>
<td>560.56</td>
<td>7.39</td>
<td>0.01</td>
<td>-273.35</td>
<td>0.55</td>
<td></td>
</tr>
</tbody>
</table>
4.3.2 Correlations between individual predictor variables and maximum torque

**Muscle size and architecture**

As expected, the CSA magnitudes obtained at all three sites were significantly correlated with maximum torque \(r = 0.66 - 0.77\); see Table 4.3, with CSA\textsubscript{PROX} being most strongly correlated with maximum isometric torque. For fascicle length, VL\textsubscript{PROX} was most strongly correlated (though this correlation was weak) with isometric \(r = 0.46\) and concentric \(r = 0.37\) torque, and VI was most strongly correlated with eccentric torque \(r = 0.30\). For fascicle angle, VL\textsubscript{MID} was most strongly correlated with isometric \(r = 0.39\) and VL\textsubscript{PROX} with concentric and eccentric \(r = 0.43\) and \(0.47\) torques respectively.

**Muscle activity**

EMG:M\textsubscript{AVEQ} was weakly correlated with maximum isometric torque \(r = 0.47\). EMG:M\textsubscript{VL} was the strongest muscle activation variable most strongly correlated with concentric \(r = 0.35\) and eccentric \(r = 0.31\) torque, but these correlations were weak. Antagonist (hamstrings) activation magnitude was only weakly correlated with all torque measured in each contraction mode \(r = 0.20 - 0.30\).

**Other variables**

Moment arm distance at the angle of maximum torque was correlated weakly with isometric \(r = 0.40\) and moderately with concentric \(r = 0.50\) torque, but showed no relationship with eccentric torque. Unpotentiated twitch torque was moderately correlated with isometric \(r = 0.50\), concentric \(r = 0.52\) and eccentric \(r = 0.54\) torque. The only statistically significant correlation between torque and %VA was the weak correlation observed with maximum eccentric torque \(r = 0.36\).
Table 4.3. Correlations between individual anatomical and neuromuscular variables (predictors) and maximal isometric, and isokinetic concentric and eccentric knee joint torque.

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Isometric</th>
<th>Concentric</th>
<th>Eccentric</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>R²</td>
<td>r</td>
</tr>
<tr>
<td>CSA(PROX)</td>
<td>0.77 ***</td>
<td>(0.59)</td>
<td>0.76 ***</td>
</tr>
<tr>
<td>CSA(MID)</td>
<td>0.72 ***</td>
<td>(0.52)</td>
<td>0.73 ***</td>
</tr>
<tr>
<td>CSA(DIST)</td>
<td>0.69 ***</td>
<td>(0.48)</td>
<td>0.68 ***</td>
</tr>
<tr>
<td>CSA(SUM)</td>
<td>0.74 ***</td>
<td>(0.55)</td>
<td>0.74 ***</td>
</tr>
<tr>
<td>EMG:M_RF</td>
<td>0.36 **</td>
<td>(0.13)</td>
<td>-0.02</td>
</tr>
<tr>
<td>EMG:M_VL</td>
<td>0.35 **</td>
<td>(0.12)</td>
<td>0.30 *</td>
</tr>
<tr>
<td>EMG:M_VM</td>
<td>0.39 **</td>
<td>(0.15)</td>
<td>0.26 *</td>
</tr>
<tr>
<td>EMG:M_AVEQ</td>
<td>0.47 **</td>
<td>(0.22)</td>
<td>0.25 *</td>
</tr>
<tr>
<td>EMG_BF</td>
<td>0.30 *</td>
<td>(0.09)</td>
<td>0.20</td>
</tr>
<tr>
<td>MA</td>
<td>0.40 **</td>
<td>(0.16)</td>
<td>0.50 ***</td>
</tr>
<tr>
<td>T_un-TW</td>
<td>0.50 ***</td>
<td>(0.25)</td>
<td>0.52 ***</td>
</tr>
<tr>
<td>T_pot-TW</td>
<td>0.60 ***</td>
<td>(0.36)</td>
<td>0.62 ***</td>
</tr>
<tr>
<td>VA</td>
<td>0.25 *</td>
<td>(0.07)</td>
<td>0.27 *</td>
</tr>
<tr>
<td>θi_VL_PROX</td>
<td>0.46 ***</td>
<td>(0.21)</td>
<td>0.37 **</td>
</tr>
<tr>
<td>θi_VL_MID</td>
<td>0.03</td>
<td>(0.00)</td>
<td>0.19</td>
</tr>
<tr>
<td>θi_VL_DIST</td>
<td>0.14</td>
<td>(0.02)</td>
<td>0.11</td>
</tr>
<tr>
<td>θf_RF</td>
<td>0.31 *</td>
<td>(0.09)</td>
<td>0.22</td>
</tr>
<tr>
<td>θf_VI</td>
<td>0.23</td>
<td>(0.05)</td>
<td>0.31 *</td>
</tr>
<tr>
<td>θf_VM</td>
<td>0.14</td>
<td>(0.02)</td>
<td>0.01</td>
</tr>
<tr>
<td>θf_VL_PROX</td>
<td>0.31 *</td>
<td>(0.10)</td>
<td>0.43 **</td>
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<tr>
<td>θf_VL_MID</td>
<td>0.39 **</td>
<td>(0.15)</td>
<td>0.29 *</td>
</tr>
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<td>θf_VL_DIST</td>
<td>0.21</td>
<td>(0.04)</td>
<td>0.30 *</td>
</tr>
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<td>θf_RF</td>
<td>-0.11</td>
<td>(0.01)</td>
<td>0.01</td>
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<tr>
<td>θf_VI</td>
<td>0.32 *</td>
<td>(0.10)</td>
<td>0.28</td>
</tr>
<tr>
<td>θf_VM</td>
<td>0.36 **</td>
<td>(0.13)</td>
<td>0.32</td>
</tr>
</tbody>
</table>

CSA(PROX), CSA(MID), CSA(DIST), CSA(SUM) = whole quadriceps cross-sectional area from the proximal, mid and distal regions, and the sum of all regions, respectively. EMG:M = EMG amplitude of rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM) and average quadriceps (AVERQ) normalised to their respective M wave amplitudes. EMG_MBF = biceps femoris EMG amplitude normalised to MVC; MA = patella tendon moment arm distance. T_un-TW and T_pot-TW = unpotentiated and potentiated twitch torques. %VA = percent voluntary activation. θi = fascicle length, and θf = fascicle angle for VL (proximal, mid and distal), RF, vastus intermedius (VI) and VM. R² = Adjusted R².

*p ≤ 0.05; **p ≤ 0.01; ***p ≤ 0.001

4.3.3 Comparative Data Set

A comparative data set of the predictors that were included within the models which received moderate support (ΔAIC ≤ 4) are displayed in Table 4.4. The seven predictors included are considered theoretically to predict maximum isometric concentric and eccentric torque. Comparative data for the three maximal voluntary contractions are also provided.
In cases when the angle of peak torque cannot be safely determined, as is probable in clinical practice where MVCs cannot be performed, the median angles for maximum torque for isometric, concentric and eccentric torques of 65, 71 and 76°, respectively, might be used. MA can thus be measured at these joint angles and a reasonable snapshot obtained.
Table 4.4. A comparative data set for healthy young males (aged 18-40 years) for predictors included in the ‘best-fit’, and ‘best clinical’ models, and those models with support for maximum net and total quadriceps isometric, and isokinetic concentric and eccentric torque prediction.

<table>
<thead>
<tr>
<th>Contraction Mode</th>
<th>Predictor Variables</th>
</tr>
</thead>
<tbody>
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<td></td>
<td>Torque (N·m)</td>
</tr>
<tr>
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<tr>
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<td></td>
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<td>188.8</td>
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<tr>
<td></td>
<td>449.0</td>
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<tr>
<td>Eccentric</td>
<td>321.4</td>
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<tr>
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<td>269.8</td>
</tr>
<tr>
<td>25%</td>
<td>221.7</td>
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<tr>
<td>lower</td>
<td>151.9</td>
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</tbody>
</table>

Torque = maximum voluntary torque; CSA<sub>PROX</sub> = proximal cross-sectional area; EMG: M = EMG amplitude of average quadriceps (AVEQ) and vastus lateralis (VL) normalised to the M-wave. EMG<sub>bf</sub> = biceps femoris EMG amplitude normalised to MVC; VA = percent voluntary activation obtained during isometric contractions; MA = patella tendon moment arm distance. T<sub>Un-TW</sub> = unpotentiated twitch torques; θ<sub>V</sub>L<sub>PROX</sub> and θ<sub>V</sub>L<sub>MID</sub> = fascicle angles of vastus intermedius (VI) and proximal region VL, respectively; θ<sub>V</sub>L<sub>PROX</sub> = fascicle angle of VL from the mid-muscle and proximal regions, respectively.
4.4 Discussion

The present study is the first to examine the relative importance of, and interactions between, muscle size, architecture, activation, and moment arm distance for maximum voluntary isometric, concentric and eccentric torque production. The main conclusions are that 1) muscle size, fascicle angle and the level of muscle activation imposed the greatest influence on maximal isometric and eccentric joint torque production, and their use in models predicted inter-individual differences in torque production well in a heterogeneous adult male population; 2) muscle size, fascicle angle and moment arm distance were of greater importance than muscle activation for determining maximum concentric torque; and 3) the clinical models explained a majority of the inter-individual variance in maximum torque in all contraction modes. Also of interest, the simultaneous inclusion of VL fascicle angle and CSA in the best-models may indicate that PCSA strongly influences the inter-individual differences in strength expression. Additionally, a comparative data set describing both anatomical and neuromuscular variables identified as being the most influential in producing maximum joint torque have been presented in Table 4.4. These data will aid in the detection of potential mechanisms underpinning an individual’s strength deficits (i.e. sites of weakness).

With the aim of understanding the influence of these anatomical and neuromuscular variables on maximum torque production (i.e. strength), it was considered important to determine their effect not only on maximal knee joint torque but also maximal knee extensor (quadriceps-only) torque for each contraction mode. However, due to the similarities observed between best-fit models obtained through the modelling process, data were presented for the prediction of maximum knee extension torque only.

4.4.1 The influence of muscle size, architecture, activation and joint moment arm

The best-fit model for each contraction mode was selected using Akaike’s Information Criterion (AIC) and model averaging [183, 184], as the AIC explains which model is ‘closest to reality’ and is a relative measure of the best model within the set of theoretically correct candidate models determined a priori. The models were ranked by their level of accuracy and complexity, with the more simplistic models considered to provide the highest information gain. The best-fit models explained 72%, 65% and 62% of the inter-individual variance in maximal isometric, concentric and eccentric torque, respectively (Table 4.1). This produced mean (± SE) absolute errors in the prediction of peak knee extension torque of 12.2 ± 1.9% (isometric), 9.1 ± 0.9% (concentric) and 13.4 ± 1.5% (eccentric). Thus, the anatomical and neuromuscular variables assessed in the present study appeared to be of substantial influence for maximum joint torque production (i.e. strength) in the population studied.
The three dominant predictors within the best-fit models were muscle size, fascicle angle and level of muscle activation. This was largely expected as both muscle size and activation are considered important predictors of functional performance [16, 62, 189], and increases in fascicle angle are typically associated with increases in muscle size [38, 60, 74]. The combination of CSA and fascicle angle within models that received the strongest support hints that PCSA may strongly influence the inter-individual differences in muscular strength. Greater fascicle angulation allows more contractile tissue to attach to a given area of tendon or aponeurosis [54, 73-75], which increases PCSA (see Figure 4.5) and thus contractile force. The rotation of the fascicles during muscular contraction also produces a gearing effect that maximises muscle force production according to both the force-length and force-velocity relationships [2, 76, 190], which might be of benefit for muscular force production. However, fascicle rotation (and thus the gearing effect) during dynamic contractions is thought to be minimal during high-force (slow-speed) contractions when compared to faster-speed contractions [191]. Therefore, as slow-speed maximal contractions were performed in the present study, it is probable that this mechanism of force increase was not as prominent. Regardless of the mechanism, it appears as though muscle size (CSA) and fascicle angulation exert significant and synergistic influences on knee extensor muscle force production.

Figure 4.5. The effect of fascicle angle on physiological cross-sectional area (PCSA). A muscle with a small fascicle angle (A) has a smaller PCSA than a muscle with a large fascicle angle (B) for a given muscle volume, which allows more contractile tissue to attach to the available area of tendon or aponeurosis.

Although the inclusion of both CSA and fascicle angle in the models may indicate the importance of PCSA in joint torque production, the ACSA measurements also appeared critical as they allowed the identification of the effects of region-specific differences in muscle size. The present data support this need because models including the CSA_{MID} and CSA_{PROX} variables predicted maximum torque differently; CSA measured at the proximal site was of greater benefit to model strength than CSA measured at the more commonly-used mid-muscle site. As most studies assessing CSA at a single
muscle location have previously used the mid-muscle site [25, 67, 68], a ‘CSA_{MID} + EMG:M’ model was included within the set of candidate models to allow for comparison. The $R^2$ value associated with this model was up to 0.08 lower than for the ‘CSA_{PROX} + EMG:M’ model, and was not supported as an effective model for torque prediction in any contraction mode ($\Delta$AIC$_C$ > 10; Table 4.2). Therefore, the present data indicate that the proximal quadriceps site appears to be a more functionally relevant location for obtaining single-site CSA images, particularly when the interaction between muscle size and strength is of interest.

This finding may have some practical importance as well as reflect different mechanisms of force production within skeletal muscle. Previous research has shown clear evidence that different regions within muscles may be uniquely, or at least differentially, activated and that region-specific activation can occur during unique tasks [64, 192-194]. This phenomenon may underpin the site-specific regional hypertrophy reported after periods of strength training [4, 6]. It is not yet known whether there is a specific functional importance of the proximal quadriceps muscular during knee extension, but the results of the present study suggest that inter-individual variations in knee extension torque are more associated with differences in CSA at proximal rather than middle (or distal) muscle sites. Although this finding should be explored in more detail in future research, it has an important and immediate implication in that research studies examining between-subject or time-dependent changes in CSA by measuring whole muscle volume or CSA at a mid-muscle location may not gather information regarding muscle size at the (potentially) most functionally important location.

While fascicle angle was included in all best-fit models in combination with CSA, independently it was in fact weakly correlated with maximum torque and might have otherwise been considered inconsequential (Table 4.3). The inclusion of fascicle angle in the models despite its lack of individual correlation emphasises the need to examine interactions between variables when assessing their influence on maximum joint torque rather than assessing correlations in isolation. It is important to point out that a lack of correlation can result from there being minimal inter-individual variability in a predictor variable. However, a broad sample group was included in the current study (sedentary to strength and endurance trained athletes) and thus group homogeneity was unlikely to have been an issue. The fact remains, therefore, that models that included fascicle angle as predictor variables best predicted torque, thus the inclusion of fascicle angle appears necessary even though the models include size and activation, suggesting fascicle angle is of added importance.

The inclusion of fascicle length variables did not improve the predictive capacity of the best-fit models. This finding is consistent with current theories on the importance of muscle fascicle length.
for force production, where it is typically considered more important for force production during high-speed muscle shortening [83, 84] and testing in the present study was conducted at slow speeds. However, models including vastus intermedius (VI) or proximal vastus lateralis (VLPROX) fascicle length had substantial support (ΔAICc ≤ 2) for the prediction of concentric torque (Table 4.2). The reason for this specific finding is not immediately clear, although sarcomeres within longer fascicles may shorten less and, therefore, slower, during dynamic contractions and this might have had some impact on concentric muscle force production. Given the current findings, the importance of fascicle length should not be disregarded under slow-speed contraction conditions; however, it appears not to be especially influential.

Agonist muscle activity was included in the best-fit models for the prediction of isometric (EMG:MAVEQ) and eccentric (EMG:MVL), but not concentric, torque; although it should be noted that models including EMG:MAVEQ received substantial support (i.e. AICc < 2) for concentric torque prediction; see Table 4.2. Isometric knee extension contractions are not commonly performed in activities of daily living (e.g. walking up stairs and sit-to-stand transitions involve concentric muscle contractions) so inter-individual variability in muscle activation may have become a potentially important factor for maximising isometric torque. While eccentric contractions are performed more frequently (e.g. walking down the stairs and stand-to-sit transitions), the purpose of those eccentric contractions is typically deceleration and stabilisation. The uniqueness of the maximal eccentric contractions performed under laboratory (particularly isokinetic) conditions may have also influenced the likelihood that muscle activation magnitude would be an important predictor influencing eccentric torque. This possibility is supported by the inclusion of both %VA and EMG:M variables in the best-fit models. Although %VA is often considered a useful measure of central drive and the ability to activate the available musculature is generally accepted to be greater in individuals with a higher %VA [122], the measure may also be influenced by other neuromuscular factors including the force transmission efficiency of the series elastic components [120]. Furthermore, the EMG:M ratio is considered to be a reasonable indicator of central drive to the contracting muscle [195], but is also influenced by other factors including motor unit synchronisation and amplitude cancellation [104] and alterations in action potential amplitude and velocity [196]. Therefore, both %VA and EMG:M methods may share some mechanistic similarity but may also be influenced by other non-similar and non-neural factors. The inclusion of both variables within best-fit models in the present study is suggestive that these variables provide unique and important information with regard to muscular force production. Further research is, therefore, required to determine the specific information provided by the variables’ inclusion in models.
Although %VA was obtained during isometric contractions in the present study, it was included in both the best-fit models predicting isometric and eccentric torque. This perhaps implies either that individuals who can activate their muscles more completely during maximal isometric contractions may also produce greater eccentric torque, or that information about muscle activation potential obtained during isometric contractions also has some use in understanding eccentric torque. This result is, however, supportive of previous findings where individuals unaccustomed to eccentric contractions were found to produce lower muscle activity (i.e. EMG amplitudes) than trained individuals during maximal eccentric contractions [122]. Assessing %VA during eccentric contractions [197] in future studies might further improve the predictive model for maximal eccentric torque.

While less is known about the relative influence of antagonist coactivation on joint torque production, coactivation magnitudes clearly differ between population groups [108, 122] and a number of studies have indicated a possible influence of antagonist coactivation on maximal agonist torque [9, 121, 122, 198]. In the present study, coactivation torque developed by the hamstrings resulted in opposing knee flexor torque estimated to range from 15-40 N·m. This equated to a difference between maximum knee extension joint torque and maximum knee extensor-only torque of 6.9 ± 1.6%, 8.6 ± 2.1% and 7.3 ± 1.9% for isometric, concentric and eccentric contractions, respectively (data not shown). However, as the between-subject variability was small compared to the within-subject variability, the relationship between maximum knee extension joint torque and maximum knee extensor-only torque was strong (r = 0.96, 0.91 and 0.95 for isometric, concentric and eccentric contractions, respectively) and their relationships with anatomical and neuromuscular variable magnitudes were similar. However, whilst not included in the best-fit models, there was substantial support for models including EMG_{BF} for the prediction of isometric and eccentric torque (\Delta AIC_C = 1.52 and 1.41 for isometric and eccentric, respectively). Coactivation should thus be considered as a variable that may influence maximal performance to some extent, and may be of functional significance in some individuals.

Moment arm distance appears to be an important predictor variable only in concentric torque prediction models, where it was included in the best-fit model as well as in all models for which there was substantial support. One explanation for this result is there could be a specific influence of moment arm distance on concentric but not isometric or eccentric torque production, although it is not clear why this might be the case. An alternative explanation is that as concentric contractions are commonly performed in many activities of daily living and, mechanical leverage rather than muscle activation capacity may have become a more discriminating factor between individuals. Despite its theoretical importance for maximising torque production, patella tendon moment arm
distance has previously been shown to be weakly correlated with maximum isometric \( (r = 0.50) \) and concentric \( (r = 0.43) \) torque [11], and similar results were observed in the present study \( (r = 0.40 \) and \( 0.50 \) for isometric and concentric torque, respectively; Table 4.3). The correlation between joint moment arm and eccentric torque was even weaker \( (r = 0.20) \), yet models including moment arm distance still received substantial support in the eccentric torque prediction model set \( (\Delta AIC_c \leq 2) \). One possibility is that moment arm distance at the angle of peak torque may be less of a factor during eccentric contractions than moment arm distance at the initiation of the contraction (i.e. in the extended knee position) when the muscles resist joint motion at the onset of force application. As the muscle fibre length-to-moment arm ratio (i.e. gearing) can strongly influence the functionality of a system [150, 199], a shorter fibre length and longer moment arm distance at the contraction onset may provide a more effective gearing ratio (greater leverage) for stronger than weaker individuals. Moment arm distance at the contraction start angle, rather than at the angle of peak torque, may thus prove a better predictor variable for eccentric contractions, and this hypothesis should be examined in future research.

### 4.4.2 Best clinical model for predicting maximum joint torque

A secondary purpose of the present study was to determine the best model for predicting peak torque in a clinical population (i.e. the ‘best clinical model’), as for rehabilitation purposes it is important to identify potential models for maximal torque prediction that do not involve the performance of maximal muscular contractions or the use of electrical muscle or nerve stimulation methodologies. These clinical models might allow clinicians to estimate (and track longitudinally) an individual’s strength within a rehabilitation program without the need for specific MVC testing, which may pose a significant injury risk. While developed on healthy males, these models are relevant to athletes undergoing rehabilitation for acute injuries as time has not altered the stiffness of the musculotendinous structures [200], nor intramuscular fat levels [201]. However, the accuracy of the models for athletes with chronic injuries, and clinical patients, would be dependent on the pathology and how long the condition has been present.

The results from the present analysis show that all best clinical models used the same ‘\( \text{CSA}_{\text{PROX}} + \theta_I + \text{MA} \)’ variable structure (Table 4.1). In fact, the clinical model designed to estimate concentric torque was the best fitting model and explained 65% of the inter-individual variance in maximal concentric torque. In comparison to the model of best fit, there was no support for the best clinical model to predict isometric torque \( (\Delta AIC_c \geq 10) \), clearly indicating the efficiency of the best-fit model. However, if ranked against other clinical models, the best clinical model explained 62% of the inter-individual variance in maximum isometric torque and 54% of the variance in maximum eccentric torque.
Importantly from a clinical perspective, the models allowed the prediction of maximum knee extension torque with mean (± SE) absolute errors from the observed torque values of 15.1 ± 1.7% (isometric), 9.1 ± 0.9% (concentric) and 15.0 ± 1.7% (eccentric), which differ from the best-fit model errors by < 3%. The safety with which data can be collected for use should, however, make them a suitable choice for predictive modelling in clinical conditions where maximal contractions cannot be performed.

It is important to note, however, that while the accuracy of torque prediction appears suitable for individuals with relatively average maximal torque capacities, the standard error tends to increase for individuals with the least or greatest torque production capacities (e.g. see Figure 4.4). This may be somewhat problematic in the applied context as well conditioned individuals (e.g. athletes requiring rehabilitation programs) will fall in the upper, and previously untrained clinical patients will probably fall in the lower, regions of the strength continuum. Thus, the accuracy and validity of the models developed in the present study should be tested in groups other than healthy adult men. Regardless of this requirement, both the best-fit and clinical models appear to provide reasonable estimates of an individual’s maximum torque producing capacity under isometric and slow movement-speed concentric and eccentric conditions.

### 4.4.3 Comparative data set

The present study is the first to report a comparative data set for those anatomical and neuromuscular variables found to be important for knee extension joint torque production (Table 4.4), and maximum torque data for each contraction mode has also been presented. The heterogeneous sample of 56 participants, ranging from sedentary to strength- and endurance-trained athletes, ensured the data set would be representative of the broad population of healthy males aged 18-40 years. An individual’s weaknesses (deficits) can now be identified by comparison to the normal population magnitudes for those variables and training plans then designed to target those weaknesses. If it is not plausible for an individual to perform maximal contractions or electrical stimulation techniques cannot be obtained then strength capacity can be estimated through an intervention period by use of the best clinical equations (Table 4.1).

### 4.5 Summary

The present findings add substantially to our understanding of the relative importance of, and interdependence between, anatomical and neuromuscular variables influencing maximum knee extension torque production in different contraction modes. The results indicate that the combination of predictors included in the ‘best-fit’ model varies across contraction modes, but that
CSA + θf was a constant combination in all best-fit models. The models best predicting isometric and eccentric torque also included muscle activation variables of either EMG_AVEQ or EMG_VL, and %VA, while MA was included in the concentric torque model indicating that mechanical leverage is more important than maximal activation ability in these more commonly performed muscle contractions. Interestingly, CSA_PROX was identified as a more important site for CSA measurement than CSA_MID. Speculatively, this might reflect the importance of the proximal quadriceps musculature for force production, and practically is of vital importance since many researchers compare CSA measured around mid-thigh regions between individuals (or over time in longitudinal studies). Even though fascicle angle was consistently included in the best-fit models, it was only weakly correlated with maximum torque in all contraction modes. This finding highlights the need to examine the interactions between variables when assessing their influence on maximum joint torque rather than relationships in isolation. Of practical importance, the data show that CSA_PROX + θf + MA can be used to estimate an individual’s strength change without the need to perform maximal contractions or use electrical stimulation procedures, which is an important safety benefit in some clinical populations. The best clinical models deviated only slightly in accuracy (< 3%) from the best-fit models. Finally, a comparative data set for those anatomical and neuromuscular variables found to be most influential in maximal torque production have been presented, enabling the identification of potential weaknesses in healthy adult men. An important next step to aid in the design of individual rehabilitation programs would be to determine whether the variables important for maximum torque production are also related to the magnitude of strength change elicited by a training intervention.
CHAPTER FIVE: STUDY TWO (a)

Are changes in specific anatomical and neuromuscular variables associated with the changes in strength following 10 weeks of heavy strength training in previously untrained men?
5.1 Introduction

Strength training, especially in previously untrained individuals, elicits substantial functional and structural adaptations leading to increases in muscular strength; however, these neuromuscular [20, 25, 35, 36] and strength [20, 36, 202] adaptations vary markedly between individuals. Muscle size, for example, is considered an important factor influencing strength expression and can account for ~60% of the inter-individual variability in strength in non-strength trained adults [Study 1 and 3, 11, 25] yet gains in muscle size have been found to be poorly related to training-induced strength improvements [20, 58]. Strength training also elicits adaptations in muscle architecture [4, 38, 60, 203] and activation [6, 47, 100, 101] so changes in these neuromuscular variables may confound the relationship between muscle size and strength, and potentially explain the larger inter-individual variability in strength improvements following training. To date, however, this speculation has received relatively little scientific scrutiny so the relative importance of different neuromuscular variables to the training-induced strength increase is not known. Given this, and despite a wealth of research detailing the neuromuscular responses to training, specific neuromuscular targets have not been identified in which large changes might lead to the greatest improvement in muscular strength.

Knee extensor torque production in particular is required for the successful completion of many activities of daily living (e.g. locomotion, chair sitting and rising, stair climbing) and athletic tasks, so it is an important muscle group for study. Quadriceps muscle size and activation (both the amplitude of agonist muscle EMG activity and percent voluntary activation assessed using interpolated twitch technique (ITT)) and vastus lateralis fascicle angle were identified in Study 1 (Chapter 4) as the best predictors of maximum isometric and eccentric knee extension torque, while muscle size, fascicle angle and patella tendon moment arm distance collectively were the best predictors of maximal concentric torque. However, it is possible that the variables most predictive of maximum strength within a population (i.e. in a cross-sectional analysis) have a different impact on the strength changes elicited by a training intervention (i.e. in a longitudinal analysis) since time-dependent (within-participant) changes may be considerably less than the between-subject variation. Thus, longitudinal studies are required in order to identify the neuromuscular factors most associated with longer-term strength change. The identification of the neuromuscular variables that most influence strength change would allow the targeting of these variables with specific exercise training regimes, and the provision of individualised training programs based on a person’s structural and/or functional characteristics.

Given the above, the present study was designed to determine whether changes in strength (isometric, concentric and eccentric) following moderate-duration (10 weeks) high-resistance (6-
repetition maximum [6-RM]; ~85-90% maximum load) strength training were associated with changes in specific neuromuscular variables. Determination of the strongest relationships between changes in strength and changes in the anatomical and neuromuscular variables provide evidence as to the most relevant mechanisms underpinning strength change. As anatomical and neuromuscular adaptations to strength training are known to be load, volume and velocity dependent [62, 82, 89] it is important to emphasise that the present research explored the effects of 10 weeks of heavy, and thus slow-speed, isoinertial, lower-limb strength training. Also, as many activities of daily living and athletic tasks require the performance of isometric and dynamic contractions, it was considered important to determine the relationships between the measures of anatomical structure and neuromuscular function versus isometric, concentric and eccentric strength.

5.2 Methods

5.2.1 Participants and Study Design

Thirty-six healthy untrained men between the ages of 19 and 40 volunteered to participate in the study (29.0 ± 5.1 y; 1.78 ± 0.05 m; and 78.9 ± 8.2 kg). Four additional volunteers begin the study were not included in the final analysis: two dropped out due to work commitments, and two were excluded based on personal circumstances obstructing their training during last 2 weeks. The participants were classified as untrained based on their response to a metabolic work rate questionnaire [163]. All participants had an average weekly metabolic energy equivalent score (MET) of < 30/day (Appendix 2A) and had not performed any regular lower-limb strength training in the past four years. Participants were also excluded if they suffered from cardiovascular or inflammatory disease, a lower limb injury within the last three months, or any other condition that could affect performance during the testing and training protocols. All were recruited via advertisements. Participants gave their written informed consent (Appendices 1C and 1E) and confirmed they were physically capable of participating in the testing procedures (Appendix 1F). Prior to testing this study was approved by the Edith Cowan University Human Research Ethics Committee and conformed to the Declaration of Helsinki (Appendix 1A).

Participants performed the same six testing sessions as outlined in Study 1 (Chapter Four) to measure muscle size and architecture, maximal isometric voluntary torque and muscle activation capacity, maximal voluntary isokinetic torque and activation capacity, and patella tendon moment arm distance. They then attended one pre-training gym session to determine their maximum load for 6 repetitions of each exercise (see 5.2.2) and to familiarise themselves with the training
exercises. Post-training testing began 4-5 days after their final training session (Table 5.1). Each participant completed all test sessions at the same time of day (± 2 h) [204].

Table 5.1. Outline of testing and training schedule. Pre-training testing occurred during weeks 1-3; training was completed between weeks 4-13; and post-training testing was completed in week 14 (see text for details).

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<td>6-RM test, training protocol familiarisation</td>
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<td>Week 14</td>
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5.2.2 Training Program

The participants completed two training sessions per week for 10 weeks (20 sessions). All training sessions were supervised and the participants were required to complete at least 18 training sessions. The exercise protocol consisted of incline (45°) leg press, knee extension and leg curl exercises against a heavy load on commercial fitness machines (Cybex international Inc, Medway, USA). The participants performed 3 sets of 6 repetitions per exercise (6-RM). This load and volume were chosen as they have previous been shown to stimulate the greatest strength and hypertrophic adaptations [44]. The first session was completed at 60% 6-RM to both accustom the participants to the training exercises and minimise muscle soreness, and all subsequent sessions were completed at 100% 6-RM. Two-minutes of passive rest was given between sets and 3 min was allowed between exercises; rest periods were strictly enforced by the training supervisor. To control the range of motion, participants were required to cover the range of 10 – 90° for knee extension, and 5 – 90° for the leg press. This was controlled by the used of markers taped to the exercise equipment for the participants to aim for. The participants were verbally encouraged throughout each session to give
their maximal effort. The warm-up for each session consisted of 5 min of low-intensity, self-paced stationary cycling and 2 warm-up sets of 6 repetitions of each exercise at approximately 50 and 70% of the day’s load. The warm-down consisted of 5 min of cycling and 5 min of static stretching. As post-exercise ingestion of protein will assist in eliciting an optimum training response [205] and individual variations in post-exercise nutrition might increase training adaptation variation, all participants consumed a protein shake immediately post-training (Redbak Whey Protein, International Health Investments Pty Ltd, Helensvale Queensland) containing between 20 to 40 g (0.4 g protein per kg body mass) of whey protein isolate powder comprising 86% protein and 8% of both carbohydrates and fats. Participants were also instructed as to the need to have an adequate energy intake (including proteins, carbohydrates and fats) during the 10-week training period.

5.2.4 Measurements

The maximum voluntary torque and both anatomical and neuromuscular measurements, were performed as described in Study 1 (refer to pages 29-37). Briefly, these included:

- maximum voluntary isometric, concentric and eccentric knee extension torque
- maximal voluntary concentric and eccentric knee flexion torque
- muscle cross-sectional area (CSA), fascicle length (li) and fascicle angle (θi) measured from proximal, middle and distal regions of the four quadriceps components
- agonist (EMG: M) and antagonist (normalised to MVC) muscle activation obtained during isometric, concentric and eccentric contractions;
- percent voluntary quadriceps activation (%VA; interpolated twitch technique), M-wave amplitude and unpotentiated and potentiated twitch torques obtained during the isometric contraction
- patella tendon moment arm distance (MA).

5.2.5 Muscle Cross-sectional Area Analysis

Quadriceps muscle CSA was measured using the techniques described in Study 1 (Chapter 4), however CSA was also determined for each quadriceps component (i.e. RF, VL, VI, and VM) separately at proximal, middle and distal regions; whole quadriceps CSA was also collected at those regions (Figure 5.1). This was considered necessary due to the between and within-muscle variability in hypertrophy following training interventions [13, 46, 63]. When the separation of vastii muscles was not clear in the proximal images due to a lack of observable inter-muscular septum, a line was drawn from the end of the visible septum to a landmark on the muscle’s circumference that had been observable on the mid-muscle region images [4]. Coefficient of variation (CV) for RF ranged from 2.9 ± 1.6 (distal) to 4.2 ± 3.0 (proximal), VL CVs ranged from 2.1 ± 1.5 (mid) to 2.6 ± 1.4
(proximal), VI CVs ranged from 1.3 ± 0.9 (mid) to 2.4 ± 1.4 (proximal), and VM CVs ranged from 2.1 ±1.5 (proximal) to 3.7 ± 2.8 (distal).

Figure 5.1. ACSA of individual quadriceps components at distal (A), middle (B) and proximal (C) regions of the thigh, identifying rectus femoris (RF), vastus medialis (VM) vastus lateralis (VL) and vastus intermedius (VI).

5.2.6 Data Analysis

Five separate repeated measures multivariate analyses of variance (MANOVA), time as the within-participant variable, were conducted to assess post-training changes in (1) isometric, concentric and eccentric peak knee extensor torque, and unpotentiated and potentiated twitch torques; (2) muscle activation (EMG amplitudes); (3) M-wave amplitude measured during an isometric contraction; (4) quadriceps and individual muscle CSAs; and (5) fascicle angles and fascicle lengths in each muscle. When significant time effects were observed, additional ANOVAs or univariate analyses were performed as appropriate to determine the location of the change. Changes in percent voluntary activation and moment arm (moment arm at the angle of peak torque was considered changeable with training) were analysed using paired t-tests. Normality of data distribution was confirmed using the Shapiro-Wilk test. Data that were not normally distributed (i.e. percent voluntary activation (%VA) and VM M-wave amplitude) were log transformed prior to statistical analysis. Analyses were performed using SPSS (version 20.0.0 IBM Corp., New York, USA). Descriptive data are displayed as mean ± standard deviation in the text and tables, and as mean ± standard error of the mean (SE) in the figures. Significance was accepted at $p \leq 0.05$.

Multiple regression models were developed a priori to examine the relationships between the change in maximum torque ($\Delta T$) and the changes in anatomical and neuromuscular variables ($\Delta VAR$). The predictor variables included in each model were considered to theoretically influence maximum torque production [11, 13]. Individual %VA obtained during the isometric contractions was also included in the concentric and eccentric candidate model sets to allow an inference of maximal
activation capacity. When assessing the change scores, an absolute change was considered a more important indicator of change than percentage change, as a similar relative change would require large improvements by stronger, and only small improvements by weaker, individuals. Muscle activation, however, was quantified as the percent change in order to minimise the influence of individual variability in EMG resulting from anatomical differences (e.g. adipose tissue thickness). Additionally, as a significant change in moment arm following training would only result from a change in the knee joint angle at which maximum torque is produced, and total moment arm distance is important for the amplification of muscle force production, moment arm distance measured before training was included in the models.

First, scatterplots were constructed to identify the relationships between ΔVAR and ΔT for each contraction mode. When the relationship between ΔVAR (or pre-training moment arm) and ΔT appeared to be nonlinear the nature of the relationship was identified using polynomial curve fitting, with curve order being increased until the change in $R^2$ was less than 2% [181]. These variables were added as nonlinear data in the models (combined with the linear variables). The distributions of the dependent variables were checked for normality and both the changes in isometric ($\Delta T_{ISO}$) and eccentric ($\Delta T_{ECC}$) torque were transformed using the natural log due to non-normal distributions (Appendix 2C). Correlations were then computed to assess the isolated relationships between the changes in the anatomical and neuromuscular variables and the change in maximal torque for each contraction mode.

The best model for each contraction mode was selected based on Akaike’s Information Criterion (AIC) [183, 184], as in Chapter 4. The models contained within the candidate model set for each contraction mode were all considered a priori to be theoretically influential to maximal torque production. To rank the models, the AIC adjusted for small sample size (AIC$_C$) was used [183]. The model with the lowest AIC$_C$ value was considered the best fit for that strength measure, and all models with $\Delta$AIC$_C$ ≤ 2 were considered to have substantial support [183]. Between 22 and 25 models were developed for each contraction mode combinations of variables determined by both their theoretical likelihood of influencing the change in torque, and on the strength of their individual correlations with the changes in torque. The models showing substantial support for explaining the variance in the change in torque are shown in Table 5.6. All models were assessed for collinearity using the variance inflation factor (VIF) to ensure that no two predictors within a model were strongly correlated (VIF <5). Adjusted $R^2$ values were used in combination with the AIC$_C$ rankings to identify the percentage of torque that could be explained by the models.
To determine whether the neuromuscular variables previously identified (Chapter 4; Study 1) as the best predictors from cross-sectional analysis were the same variables deemed to influence the change in torque following training, the predictors from the ‘best-fit’ model for each contraction mode from Study 1 were also correlated with ΔT. All regression models were analysed using R version 3.0.0 (R Development Core Team, 2013).

5.3 Results

All participants increased in strength following the 10-week training period (see Table 5.2). Maximal isometric, concentric, eccentric torque increased by 17.2 ± 12.6%, 12.5 ± 8.0% and 16.2 ± 14.4%, respectively (p < 0.01 for all; Table 5.2). These changes were less than the 46.6 ± 21.0% increase in 6-RM knee extension strength across the training period (data not shown). Strong correlations (r = 0.73 to 0.78) were observed between the maximum leg extension load and maximum torque for each contraction mode following training, however weak or no correlations (r = 0.07 to 0.25) were observed between the change in maximum leg extension load and the change in maximum torque for each contraction mode. Changes were observed in a number of variables relating to muscle activation but not coactivation or moment arm (at the angle of peak torque) after training (Table 5.2). Statistically significant increases were also observed for all CSA, fascicle angle and fascicle length measurements, as shown in Table 5.3. Participants who produced greater torque at pre-training were equally likely to increase absolute strength as the weaker participants, as demonstrated by strong correlations between pre- and post-training torque values (r = 0.93, 0.97 and 0.90, all p ≤ 0.001, for isometric, concentric and eccentric torque, respectively).
Table 5.2. Training loads, and torque, moment arm, and muscle activity and activation variables obtained before and after training during maximal isometric, and isokinetic concentric and eccentric contractions.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre-Training (Mean ± SD)</th>
<th>Post-Training (Mean ± SD)</th>
<th>Absolute Change (Mean ± SD)</th>
<th>Percent Change (Mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Training Load: 6-RM</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee extension (kg)</td>
<td>76.2 ± 18.5</td>
<td>109.6 ± 25.8</td>
<td>33.4 ± 15.2</td>
<td>45.8 ± 19.7</td>
</tr>
<tr>
<td>Leg press (kg)</td>
<td>134.7 ± 48.1</td>
<td>275.0 ± 91.3</td>
<td>140.2 ± 62.1</td>
<td>114.2 ± 58.6</td>
</tr>
<tr>
<td>Leg curl (kg)</td>
<td>42.9 ± 7.8</td>
<td>62.6 ± 10.6</td>
<td>19.7 ± 7.4</td>
<td>47.6 ± 19.7</td>
</tr>
<tr>
<td><strong>Isometric</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Torque_{SO} (N·m)</td>
<td>256.4 ± 69.1</td>
<td>296.8 ± 74.3</td>
<td>39.7 ± 25.6 **</td>
<td>17.2 ± 12.6 **</td>
</tr>
<tr>
<td>T_{Un-Tw} (N·m)</td>
<td>48.0 ± 13.6</td>
<td>48.6 ± 12.9</td>
<td>0.5 ± 11.4</td>
<td>6.9 ± 42.2</td>
</tr>
<tr>
<td>T_{Pot-Tw} (N·m)</td>
<td>65.8 ± 19.42</td>
<td>70.29 ± 16.8</td>
<td>4.5 ± 15.0</td>
<td>14.4 ± 48.4</td>
</tr>
<tr>
<td>MA (mm)</td>
<td>50 ± 5</td>
<td>50 ± 4</td>
<td>-0.3 ± 1.9</td>
<td>-0.6 ± 3.8</td>
</tr>
<tr>
<td>M-Wave_{BF} (amp)</td>
<td>4.31 ± 1.31</td>
<td>4.05 ± 1.33</td>
<td>-0.3 ± 1.0</td>
<td>-4.5 ± 22.9</td>
</tr>
<tr>
<td>M-Wave_{VL} (amp)</td>
<td>5.09 ± 1.58</td>
<td>4.91 ± 1.60</td>
<td>-0.2 ± 1.6</td>
<td>2.0 ± 42.0</td>
</tr>
<tr>
<td>M-Wave_{VM} (amp)</td>
<td>3.06 ± 2.13</td>
<td>2.54 ± 1.32</td>
<td>-0.5 ± 2.6</td>
<td>0.8 ± 51.4</td>
</tr>
<tr>
<td>EMG:M_{AVEQ} (mV)</td>
<td>0.075 ± 0.023</td>
<td>0.089 ± 0.022</td>
<td>-</td>
<td>23.2 ± 25.5 **</td>
</tr>
<tr>
<td>EMG:M_{BF} (mV)</td>
<td>0.066 ± 0.020</td>
<td>0.083 ± 0.025</td>
<td>-</td>
<td>33.8 ± 42.9 **</td>
</tr>
<tr>
<td>EMG:M_{VL} (mV)</td>
<td>0.072 ± 0.028</td>
<td>0.084 ± 0.027</td>
<td>-</td>
<td>24.4 ± 35.0 **</td>
</tr>
<tr>
<td>EMG:M_{VM} (mV)</td>
<td>0.087 ± 0.038</td>
<td>0.099 ± 0.040</td>
<td>-</td>
<td>23.7 ± 45.3 *</td>
</tr>
<tr>
<td>EMG_{BF} (mV)</td>
<td>0.244 ± 0.09</td>
<td>0.226 ± 0.08</td>
<td>-</td>
<td>-5.1 ± 26.7</td>
</tr>
<tr>
<td>%VA (%)</td>
<td>88.51 ± 6.71</td>
<td>92.00 ± 4.99</td>
<td>-</td>
<td>4.1 ± 3.6 **</td>
</tr>
<tr>
<td><strong>Concentric</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Torque_{CON} (N·m)</td>
<td>223.5 ± 61.5</td>
<td>248.2 ± 60.9</td>
<td>24.7 ± 13.9 **</td>
<td>12.5 ± 8.0 **</td>
</tr>
<tr>
<td>MA (mm)</td>
<td>50 ± 5</td>
<td>49 ± 5</td>
<td>0.17 ± 1.8</td>
<td>-0.4 ± 3.7</td>
</tr>
<tr>
<td>EMG:M_{AVEQ} (mV)</td>
<td>0.081 ± 0.020</td>
<td>0.094 ± 0.025</td>
<td>-</td>
<td>19.0 ± 26.4 **</td>
</tr>
<tr>
<td>EMG:M_{BF} (mV)</td>
<td>0.083 ± 0.031</td>
<td>0.092 ± 0.035</td>
<td>-</td>
<td>14.8 ± 34.9</td>
</tr>
<tr>
<td>EMG:M_{VL} (mV)</td>
<td>0.071 ± 0.022</td>
<td>0.086 ± 0.033</td>
<td>-</td>
<td>21.9 ± 38.4 **</td>
</tr>
<tr>
<td>EMG:M_{VM} (mV)</td>
<td>0.089 ± 0.037</td>
<td>0.111 ± 0.050</td>
<td>-</td>
<td>23.4 ± 36.8 **</td>
</tr>
<tr>
<td>EMG_{BF} (mV)</td>
<td>0.257 ± 0.10</td>
<td>0.272 ± 0.12</td>
<td>-</td>
<td>10.9 ± 38.5</td>
</tr>
<tr>
<td><strong>Eccentric</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Torque_{ECC} (N·m)</td>
<td>274.5 ± 73.6</td>
<td>315.8 ± 73.8</td>
<td>40.4 ± 32.0 **</td>
<td>16.2 ± 14.4 **</td>
</tr>
<tr>
<td>MA (mm)</td>
<td>48 ± 5</td>
<td>48 ± 5</td>
<td>-0.2 ± 1.8</td>
<td>-0.4 ± 3.7</td>
</tr>
<tr>
<td>EMG:M_{AVEQ} (mV)</td>
<td>0.066 ± 0.019</td>
<td>0.080 ± 0.024</td>
<td>-</td>
<td>22.7 ± 28.4 **</td>
</tr>
<tr>
<td>EMG:M_{BF} (mV)</td>
<td>0.067 ± 0.026</td>
<td>0.078 ± 0.030</td>
<td>-</td>
<td>21.6 ± 39.2 *</td>
</tr>
<tr>
<td>EMG:M_{VL} (mV)</td>
<td>0.062 ± 0.023</td>
<td>0.077 ± 0.030</td>
<td>-</td>
<td>27.0 ± 36.5 **</td>
</tr>
<tr>
<td>EMG:M_{VM} (mV)</td>
<td>0.068 ± 0.031</td>
<td>0.090 ± 0.046</td>
<td>-</td>
<td>23.3 ± 37.4 *</td>
</tr>
<tr>
<td>EMG_{BF} (mV)</td>
<td>0.226 ± 0.10</td>
<td>0.227 ± 0.10</td>
<td>-</td>
<td>5.7 ± 33.8</td>
</tr>
</tbody>
</table>

Torque variables include maximum voluntary torque (Torque_{SO/CON/ECC}) as well as unpotentiated (T_{Un-Tw}) and potentiated (T_{Pot-Tw}) twitch torques. Muscle activation variables include peak-to-peak M-Wave amplitude of rectus femoris (RF), vastus lateralis (VL) and vastus medialis (VM); EMG:M = normalised average quadriceps (EMG:M_{AVEQ}), RF (EMG:M_{BF}), VL (EMG:M_{VL}) and VM (EMG:M_{VM}) EMG amplitudes; EMG_{BF} = biceps femoris EMG amplitude normalised to EMG during MVC; MA = patella tendon moment arm measured at the angle of peak isometric, concentric or eccentric torque; %VA = percent voluntary activation. *p ≤ 0.05; **p ≤ 0.01
Table 5.3 Muscle size, fascicle angle, and fascicle length obtained before and after training.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre-training (Mean ± SD)</th>
<th>Post-training (Mean ± SD)</th>
<th>Absolute Change (Mean ± SD)</th>
<th>Percentage Change (Mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSA_{QPROX} (cm²)</td>
<td>76.3 ± 14.1</td>
<td>80.0 ± 14.3</td>
<td>3.7 ± 2.6 **</td>
<td>4.9 ± 3.4</td>
</tr>
<tr>
<td>CSA_{OMID} (cm²)</td>
<td>72.6 ± 15.3</td>
<td>77.9 ± 15.3</td>
<td>5.2 ± 2.9 **</td>
<td>7.3 ± 4.0</td>
</tr>
<tr>
<td>CSA_{QDIST} (cm²)</td>
<td>60.6 ± 11.9</td>
<td>66.0 ± 13.7</td>
<td>5.3 ± 3.8 **</td>
<td>9.0 ± 6.3</td>
</tr>
<tr>
<td>CSA_{QSUM} (cm²)</td>
<td>209.5 ± 40.9</td>
<td>223.7 ± 42.5</td>
<td>14.2 ± 8.4 **</td>
<td>7.0 ± 4.3</td>
</tr>
<tr>
<td>CSA_{RFPROX} (cm²)</td>
<td>9.6 ± 2.4</td>
<td>10.6 ± 2.6</td>
<td>0.9 ± 0.8 **</td>
<td>9.7 ± 8.2</td>
</tr>
<tr>
<td>CSA_{RFMID} (cm²)</td>
<td>5.9 ± 1.8</td>
<td>6.7 ± 2.1</td>
<td>0.7 ± 0.5 **</td>
<td>12.1 ± 8.8</td>
</tr>
<tr>
<td>CSA_{RFDIST} (cm²)</td>
<td>3.0 ± 1.1</td>
<td>3.5 ± 1.3</td>
<td>0.5 ± 0.4 **</td>
<td>16.7 ± 14.7</td>
</tr>
<tr>
<td>CSA_{RFSUM} (cm²)</td>
<td>18.6 ± 4.9</td>
<td>20.8 ± 5.6</td>
<td>2.2 ± 1.5 **</td>
<td>11.7 ± 7.1</td>
</tr>
<tr>
<td>CSA_{VPROX} (cm²)</td>
<td>24.7 ± 5.6</td>
<td>26.3 ± 5.6</td>
<td>1.6 ± 1.5 **</td>
<td>6.6 ± 5.9</td>
</tr>
<tr>
<td>CSA_{VMID} (cm²)</td>
<td>21.8 ± 5.7</td>
<td>24.2 ± 5.9</td>
<td>2.4 ± 1.8 **</td>
<td>11.2 ± 8.4</td>
</tr>
<tr>
<td>CSA_{VDIST} (cm²)</td>
<td>15.2 ± 3.8</td>
<td>17.3 ± 4.2</td>
<td>2.1 ± 1.5 **</td>
<td>13.6 ± 9.7</td>
</tr>
<tr>
<td>CSA_{VSUM} (cm²)</td>
<td>61.6 ± 14.5</td>
<td>67.6 ± 15.2</td>
<td>6.0 ± 3.7 **</td>
<td>10.2 ± 6.8</td>
</tr>
<tr>
<td>CSA_{VPROX} (cm²)</td>
<td>30.4 ± 5.9</td>
<td>31.9 ± 6.3</td>
<td>1.5 ± 1.6 **</td>
<td>5.1 ± 5.2</td>
</tr>
<tr>
<td>CSA_{VMID} (cm²)</td>
<td>26.4 ± 6.0</td>
<td>28.6 ± 6.2</td>
<td>2.2 ± 1.6 **</td>
<td>8.3 ± 5.5</td>
</tr>
<tr>
<td>CSA_{VDIST} (cm²)</td>
<td>19.8 ± 4.3</td>
<td>21.7 ± 5.0</td>
<td>1.5 ± 1.5 **</td>
<td>7.4 ± 7.5</td>
</tr>
<tr>
<td>CSA_{VSUM} (cm²)</td>
<td>76.7 ± 15.7</td>
<td>82.3 ± 16.9</td>
<td>5.6 ± 3.7 **</td>
<td>7.4 ± 4.4</td>
</tr>
<tr>
<td>θ_{VLPROX} (°)</td>
<td>19.6 ± 4.1</td>
<td>20.4 ± 4.0</td>
<td>0.9 ± 1.4 **</td>
<td>4.6 ± 7.1</td>
</tr>
<tr>
<td>θ_{VMLMID} (°)</td>
<td>17.6 ± 4.0</td>
<td>18.4 ± 3.3</td>
<td>0.9 ± 2.1 *</td>
<td>4.9 ± 12.1</td>
</tr>
<tr>
<td>θ_{VLDIST} (°)</td>
<td>17.8 ± 3.5</td>
<td>18.6 ± 3.3</td>
<td>0.80 ± 2.2 *</td>
<td>4.5 ± 12.5</td>
</tr>
<tr>
<td>θ_{VRF} (°)</td>
<td>14.1 ± 366</td>
<td>15.1 ± 3.0</td>
<td>1.0 ± 1.7 **</td>
<td>6.7 ± 12.3</td>
</tr>
<tr>
<td>θ_{VIL} (°)</td>
<td>14.1 ± 3.6</td>
<td>14.7 ± 3.5</td>
<td>0.7 ± 1.8 *</td>
<td>4.6 ± 12.7</td>
</tr>
<tr>
<td>θ_{VM} (°)</td>
<td>36.8 ± 3.7</td>
<td>38.6 ± 4.2</td>
<td>1.8 ± 2.5 **</td>
<td>5.0 ± 6.8</td>
</tr>
<tr>
<td>θ_{VLPROX} (cm)</td>
<td>7.7 ± 1.1</td>
<td>8.0 ± 1.0</td>
<td>0.3 ± 0.6 **</td>
<td>3.9 ± 7.4</td>
</tr>
<tr>
<td>θ_{VMLMID} (cm)</td>
<td>7.8 ± 137</td>
<td>8.2 ± 1.4</td>
<td>0.4 ± 0.7 **</td>
<td>4.5 ± 9.2</td>
</tr>
<tr>
<td>θ_{VLDIST} (cm)</td>
<td>7.6 ± 1.3</td>
<td>8.2 ± 1.5</td>
<td>0.6 ± 0.7 **</td>
<td>7.4 ± 8.5</td>
</tr>
<tr>
<td>θ_{RF} (cm)</td>
<td>9.0 ± 1.9</td>
<td>9.3 ± 1.8</td>
<td>0.3 ± 0.7 **</td>
<td>3.6 ± 7.4</td>
</tr>
<tr>
<td>θ_{VI} (cm)</td>
<td>7.4 ± 1.4</td>
<td>7.8 ± 1.4</td>
<td>0.5 ± 0.7 **</td>
<td>6.1 ± 9.0</td>
</tr>
<tr>
<td>θ_{VM} (cm)</td>
<td>9.0 ± 1.0</td>
<td>8.6 ± 1.0</td>
<td>0.3 ± 0.5 **</td>
<td>2.8 ± 5.9</td>
</tr>
</tbody>
</table>

CSA = cross sectional area of the quadriceps (Q), rectus femoris (RF) vastus lateralis (VL), vastus intermedius (VI), and vastus medialis (VM). θf = fascicle angle, fn = fascicle length. 

PROX, MID and DIST refer the proximal, mid-muscle and distal regions of the thigh; SUM = total of all CSA regions for that quadriceps measure.

*p ≤ 0.05, **p ≤ 0.01
5.3.1 Regression models

Change in torque versus the change in anatomical and neuromuscular variables

Moderate relationships were observed between the best-fit model and the change in torque for all contraction modes (Table 5.4). The best-fit model for the change in isometric torque was ‘ΔCSA,VL<sub>PROX</sub> + Δθ<sub>f</sub>VL<sub>PROX</sub>’ ($R^2 = 0.27$, AIC<sub>c</sub> = 0.52) while the best-fit models for the change in concentric and eccentric torques were ‘ΔEMG:M<sub>AVEQ</sub> + ΔCSA,VL<sub>PROX</sub> + Δθ<sub>i</sub>VI’ ($R^2 = 0.40$, AIC<sub>c</sub> = 0.15) and ‘ΔEMG:M<sub>RF</sub> + ΔCSA,Q<sub>PROX</sub> + Δθ<sub>i</sub>VI’ ($R^2 = 0.41$, AIC<sub>c</sub> = 0.31), respectively (Table 5.5). Models incorporating the change in mid-region vastus lateralis fascicle length ($iVL_{MID}$) or angle ($θ_{VL_{MID}}$) also had substantial support for inclusion in the concentric torque prediction models, and the change in percent voluntary activation (%VA) and pre-training moment arm distance had substantial support (AIC<sub>c</sub> < 2) for use in the eccentric models (Table 5.5). Based on the best-fit models for each contraction mode, the mean (± SE) absolute errors in the prediction of the change in torque were 16.1 ± 3.1% (isometric), 59.8 ± 12.9% (concentric) and 17.6 ± 2.3% (eccentric) (see Figure 5.2).

While fascicle angle was present in all best-fit models, Δθ<sub>f</sub>VL<sub>PROX</sub> appeared in the isometric torque prediction models whereas Δθ<sub>i</sub>VI appeared in the concentric and eccentric torque models. Similarly, the change in VL CSA was included in the best-fit isometric and concentric torque models (ΔCSA,VL<sub>PROX</sub>), while whole quadriceps CSA measured proximally (ΔCSA,Q<sub>PROX</sub>) was included in the eccentric torque prediction models. There was also substantial support for models incorporating the percent changes for both the average quadriceps (ΔEMG:M<sub>AVEQ</sub>) and rectus femoris (ΔEMG:M<sub>RF</sub>) muscle activation variables for both concentric and eccentric torque production. Models incorporating the change in antagonist EMG (EMG<sub>BF</sub>) and unpotentiated twitch torque, did not have enough support to be included in the final candidate model set for any contraction mode.

Change in torque versus the change in the ‘best-fit’ parameters from Chapter 4

In Study 1, specific variables were incorporated into the best-fit models for predicting maximum torque for each contraction mode (Table 4.1). These models were then tested in the present study to examine whether the models that could explain the greatest amount of variance in maximum torque production could also explain a significant proportion of the variance in the change in torque, however no relationship was observed for any contraction mode ($R^2 = 0.00$ to 0.07; Table 5.6).
Table 5.4. The best-fit model for predicting changes in maximum isometric and isokinetic concentric and eccentric torque (ΔT) from the changes in predictor variables (ΔVAR).

<table>
<thead>
<tr>
<th>Contraction</th>
<th>Best-fit Model</th>
<th>Equation</th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( ΔT_{ISO} )</td>
<td>( ΔCSA,VL_{PROX} + Δθ,VL_{PROX} )</td>
<td>[ Y = 0.210(ΔCSA,VL_{PROX}) + 0.199(Δθ,VL_{PROX}) + 2.924 ]</td>
<td>0.27</td>
</tr>
<tr>
<td>( ΔT_{CON} )</td>
<td>( ΔEMG:M_{AVEQ} + ΔCSA,VL_{PROX} + Δθ,VI )</td>
<td>[ Y = 0.251(ΔEMG:M_{AVEQ}) + 2.453 (ΔCSA,VL_{PROX}) + 2.537 (Δθ,VI) + 14.633 ]</td>
<td>0.40</td>
</tr>
<tr>
<td>( ΔT_{ECC} )</td>
<td>( ΔEMG:M_{AVEQ} + ΔCSA,Q_{PROX} + Δθ,VI )</td>
<td>[ Y = -0.124(ΔCSA,Q_{PROX}) + 0.016(ΔEMG:M_{AVEQ}) + 0.170(Δθ,VI) + 3.334 ]</td>
<td>0.41</td>
</tr>
</tbody>
</table>

For each candidate model: \( θ_i \) = fascicle angle of proximal vastus lateralis (VL_{PROX}) or vastus intermedius (VI); CSA = proximal-region vastus lateralis (CSA,VL_{PROX}) or whole quadriceps (CSA,Q_{PROX}) cross-sectional area of whole; ΔEMG:M_{AVEQ} = amplitude of average quadriceps normalised to the M-wave; EMG:M is represented as the percentage change while the other predictor variables are represented as an absolute change. \( R^2 \) = adjusted \( R^2 \).
Table 5.5. Akaike’s Information Criterion (AIC) of model parameters which showed substantial support (ΔAIC < 2) for predicting the change in isometric, and isokinetic concentric and eccentric contractions torque (ΔT) based on changes in the predictor variables (Δ). Models which showed moderate support (ΔAIC < 4) for predicting the change in isometric torque are also included. Models with an AIC<sub>Δw</sub> = 0.10 (i.e. greater than a 10% chance that they will be the best-fit model) are identified by shading.

<table>
<thead>
<tr>
<th>Contraction</th>
<th>Model</th>
<th>K</th>
<th>AIC&lt;sub&gt;C&lt;/sub&gt;</th>
<th>ΔAIC&lt;sub&gt;C&lt;/sub&gt;</th>
<th>AIC&lt;sub&gt;Δw&lt;/sub&gt;</th>
<th>LL</th>
<th>R&lt;sup&gt;2&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>ln (ΔT&lt;sub&gt;ISO&lt;/sub&gt;)</td>
<td>ΔCSA,&lt;sub&gt;VL&lt;/sub&gt;+Δθ&lt;sub&gt;VL&lt;/sub&gt;</td>
<td>4</td>
<td>77.48</td>
<td>0.00</td>
<td>0.52</td>
<td>-34.10</td>
<td>0.27</td>
</tr>
<tr>
<td></td>
<td>ΔCSA,&lt;sub&gt;VL&lt;/sub&gt;+Δθ&lt;sub&gt;VL&lt;/sub&gt;+Δ%VA</td>
<td>5</td>
<td>79.80</td>
<td>2.32</td>
<td>0.16</td>
<td>-33.90</td>
<td>0.25</td>
</tr>
<tr>
<td></td>
<td>ΔCSA,&lt;sub&gt;VL&lt;/sub&gt;+Δθ&lt;sub&gt;VL&lt;/sub&gt;+MA</td>
<td>5</td>
<td>80.08</td>
<td>2.60</td>
<td>0.14</td>
<td>-34.04</td>
<td>0.25</td>
</tr>
<tr>
<td></td>
<td>ΔCSA,&lt;sub&gt;Q&lt;/sub&gt;+Δθ&lt;sub&gt;VL&lt;/sub&gt;</td>
<td>4</td>
<td>81.29</td>
<td>3.81</td>
<td>0.08</td>
<td>-36.00</td>
<td>0.18</td>
</tr>
<tr>
<td>Intercept Only</td>
<td>2</td>
<td>85.82</td>
<td>8.34</td>
<td>0.01</td>
<td>-40.73</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

ΔT<sub>CON</sub>

| ΔEMG:M<sub>AVEQ</sub>+ΔCSA,<sub>VL</sub>+Δθ<sub>VI</sub> | 5 | 273.35 | 0.00 | 0.15 | -130.64 | 0.40 |
| ΔEMG:M<sub>AVEQ</sub>+ΔCSA,<sub>VL</sub>+Δθ<sub>VI</sub> | 5 | 273.91 | 0.56 | 0.11 | -130.92 | 0.40 |
| ΔEMG:M<sub>AVEQ</sub>+Δθ<sub>VI</sub> | 4 | 274.01 | 0.65 | 0.11 | -132.34 | 0.36 |
| ΔEMG:M<sub>AVEQ</sub>+ΔCSA,<sub>Q</sub>+Δθ<sub>VI</sub> | 5 | 274.55 | 1.20 | 0.08 | -131.24 | 0.38 |
| ΔEMG:M<sub>RF</sub>+ΔCSA,<sub>Q</sub>+Δθ<sub>VI</sub> | 5 | 274.55 | 1.20 | 0.08 | -131.24 | 0.38 |
| ΔEMG:M<sub>AVEQ</sub>+ΔCSA,<sub>Q</sub>+Δθ<sub>VI</sub> | 5 | 274.55 | 1.20 | 0.08 | -131.24 | 0.38 |
| ΔEMG:M<sub>AVEQ</sub>+ΔCSA,<sub>Q</sub>+Δθ<sub>VI</sub> | 5 | 274.68 | 1.33 | 0.08 | -131.31 | 0.37 |
| ΔEMG:M<sub>RF</sub>+Δθ<sub>VI</sub> | 4 | 275.01 | 1.66 | 0.07 | -132.84 | 0.36 |
| Intercept Only | 2 | 287.05 | 13.70 | 0.00 | -141.34 | - |

| ln (ΔT<sub>ECC</sub>) | ΔEMG:M<sub>AVEQ</sub>+ΔCSA,<sub>Q</sub>+Δθ<sub>VI</sub> | 5 | 75.84 | 0.00 | 0.31 | -31.77 | 0.41 |
| ΔEMG:M<sub>RF</sub>+ΔCSA,<sub>Q</sub>+Δθ<sub>VI</sub> | 6 | 77.06 | 1.22 | 0.17 | -30.85 | 0.43 |
| ΔEMG:M<sub>AVEQ</sub>+ΔCSA,<sub>Q</sub>+Δθ<sub>VI</sub> | 6 | 77.55 | 1.71 | 0.13 | -31.09 | 0.42 |
| EMG:M<sub>RF</sub>+ΔCSA,<sub>Q</sub>+Δθ<sub>VI</sub> | 6 | 77.63 | 1.79 | 0.12 | -31.14 | 0.38 |
| Intercept Only | 2 | 88.35 | 12.51 | 0.00 | -41.97 | - |

In refers to the natural log of the ΔT. For each candidate model: CSA,<sub>Q</sub> = proximal cross-sectional area of whole quadriceps, or of vastus lateralis (VL) in isolation, respectively; θ<sub>VL</sub> = fascicle angle of VL obtained at the proximal region, and vastus intermedius (VI), respectively; Δθ<sub>VI</sub> = fascicle length of VL obtained at the middle region; ΔEMG:M<sub>AVEQ</sub> and ΔEMG:M<sub>RF</sub> = amplitude of normalised average quadriceps (AVEQ) or rectus femoris (RF) EMG:M amplitude, respectively; %VA = percent voluntary activation (obtained during isometric contractions); MA = patella tendon moment arm distance. Intercept = basic control model with no predictors, and includes only the constant and residual variance (σ<sup>2</sup>).

EMG is represented as a percent change; the other predictor variables are represented as an absolute change aside from MA, which is included as the pre-training moment arm distance.

K = number of parameters tested in each model; AIC<sub>C</sub> = Akaike information criterion for a small data set; ΔAIC<sub>C</sub> = the models AIC<sub>C</sub> minus the minimum AIC<sub>C</sub> among candidate models. AIC<sub>Δw</sub> = the percentage of times that a given model would be selected as the ‘best model’ by AIC<sub>C</sub>, and serves as the weight of evidence for a given model being the best model from that set of candidate models [183]; R<sup>2</sup> = adjusted R<sup>2</sup>. 

70
Figure 5.2. Predicted change in torque ($\Delta T$) was modelled based on the AICc rankings using the best-fit model for the change in maximal isometric, and isokinetic concentric and eccentric torque prediction. Figures show the mean ($\pm$ SE) for each model. (ln) = the natural log of the change in torque. CSA,Q\textsubscript{PROX} and CSA,VL\textsubscript{PROX} = proximal cross-sectional area of whole quadriceps, or of vastus lateralis (VL) in isolation, respectively; EMG:M\textsubscript{AVEQ} = normalised average quadriceps (AVEQ) amplitude; $\theta$\textsubscript{VL\textsubscript{PROX}} and $\theta$\textsubscript{VI} = fascicle angle of VL obtained at proximal region, and vastus intermedius (VI), respectively; $R^2$ = adjusted $R^2$. 

\[
\Delta T = \Delta\text{CSA,VL\textsubscript{PROX}} + \Delta\theta\text{VL\textsubscript{PROX}} \ (R^2 = 0.27)
\]

\[
\Delta T = \Delta\text{EMG:M\textsubscript{AVEQ}} + \Delta\text{CSA,VL\textsubscript{PROX}} + \Delta\theta\text{VL} \ (R^2 = 0.40)
\]

\[
\Delta T = \Delta\text{EMG:M\textsubscript{AVEQ}} + \Delta\text{CSA,Q\textsubscript{PROX}} + \Delta\theta\text{VI} \ (R^2 = 0.41)
\]
Table 5.6. Regression models using the previously-identified ‘best-fit’ model parameters for predicting torque (Study 1; Chapter 4) to determine whether adaptations in these same variables were associated with the change of strength following training.

<table>
<thead>
<tr>
<th>Contraction</th>
<th>Model</th>
<th>( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \Delta T_{\text{ISO}} )</td>
<td>( \Delta \text{CSA}<em>{\text{PROX}} + \Delta \text{EMG}:M</em>{\text{AVEQ}} + \Delta \theta_{\text{VL}_{\text{MID}}} + \Delta %\text{VA} )</td>
<td>0.07</td>
</tr>
<tr>
<td>( \Delta T_{\text{CON}} )</td>
<td>( \Delta \text{CSA}<em>{\text{PROX}} + \Delta \theta</em>{\text{VL}_{\text{PROX}}} + \Delta \text{MA} )</td>
<td>0.00</td>
</tr>
<tr>
<td>( \Delta T_{\text{ECC}} )</td>
<td>( \Delta \text{CSA}<em>{\text{PROX}} + \Delta \text{EMG}:M</em>{\text{VL}} + \Delta \theta_{\text{VL}_{\text{PROX}}} + \Delta %\text{VA} )</td>
<td>0.07</td>
</tr>
</tbody>
</table>

\( \text{CSA}_{\text{PROX}} \) = proximal cross-sectional area; \( \Delta \text{EMG}:M \) = amplitude of average quadriceps (AVEQ) or vastus lateralis (VL) normalised to the M-wave; \( \%\text{VA} \) = percent voluntary activation; \( \text{MA} \) = patella tendon moment arm obtained pre-training. \( \text{EMG}:M \) is represented as the percentage change while the other predictor variables are represented as an absolute change. \( R^2 \) = adjusted \( R^2 \).
5.3.2 Correlations

**Correlations between the change in torque and changes in muscle activation variables**

While the percent changes in agonist muscle activation variables were not correlated with the change in isometric torque, $\Delta$EMG:M<sub>AVEQ</sub> and $\Delta$EMG:M<sub>RF</sub> were moderately correlated with the change in both concentric ($r = 0.52$, $p < 0.01$; $r = 0.56$, $p < 0.001$) and eccentric ($r = 0.56$, $p < 0.001$; $r = 0.51$, $p < 0.01$) torque, as shown in Figure 5.3 and Table 5.7. $\Delta$EMG:M<sub>VM</sub> was also weakly correlated with the change in both concentric ($r = 0.35$, $p < 0.05$) and eccentric ($r = 0.48$, $p < 0.01$) torque.

**Correlations between the change in torque and the changes in muscle size and architecture variables**

The changes in muscle size were more strongly correlated with the change in isometric, compared to the change in the concentric or eccentric, torque. The change in proximal whole quadriceps CSA ($\Delta$CSA,Q<sub>PROX</sub>) and the change in proximal ($\Delta$CSA,V<sub>PROX</sub>) and mid-region VL CSA ($\Delta$CSA,V<sub>MID</sub>) were weakly correlated with the change in isometric torque ($r = 0.36$ and $0.42$, $p < 0.05$) and ($r = 0.45$, $p <0.01$), respectively; see Table 5.7. $\Delta$CSA,V<sub>PROX</sub> was also weakly correlated with the change in concentric torque ($r = 0.35$, $p < 0.05$). The change in eccentric torque was not correlated with changes in any muscle size variable.

The change in proximal region VL FA ($\Delta$θ<sub>VL</sub>PROX) was moderately correlated with the change in isometric torque ($r = 0.41$, $p < 0.05$) and the change in VI FA ($\Delta$θ<sub>VI</sub>) was moderately correlated with the change in concentric torque ($r = 0.41$, $p < 0.05$); Table 5.7. The change in mid-region VL fascicle length was moderately correlated with the change in concentric ($r = 0.35$, $p < 0.05$), and the change in RF fascicle length was moderately correlated with the change in eccentric ($r = 0.43$, $p < 0.05$) torque. This change in fascicle length was the only muscle-based variable found to correlate with the change in eccentric torque following training.
Figure 5.3. Correlations between changes in maximum isometric, and isokinetic concentric and eccentric torques, versus changes in the anatomical and neuromuscular variables. (ln) refers to the natural log. CSA,Q\textsubscript{PROX} = whole quadriceps proximal cross-sectional area; θ\textsubscript{VL\textsubscript{PROX}} = proximal vastus lateralis fascicle angle; EMG:M\textsubscript{AVEQ} and EMG:M\textsubscript{RF} = normalised average quadriceps (AVEQ) and rectus femoris (RF) amplitude; l\textsubscript{RF} = rectus femoris fascicle length. SE ± 1.96 is shown in grey. *\(p \leq 0.05; \) **\(p \leq 0.01; \) ***\(p \leq 0.001\)
Table 5.7 Correlations (r) between the change in isometric ($\Delta T_{ISO}$), and isokinetic concentric ($\Delta T_{CON}$) and eccentric ($\Delta T_{ECC}$) torque and changes in neuromuscular variables ($\Delta VAR$).

<table>
<thead>
<tr>
<th>$\Delta VAR$</th>
<th>$\Delta T_{ISO}$</th>
<th>$\Delta T_{CON}$</th>
<th>$\Delta T_{ECC}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>%Δ EMG:M$_{AVEQ}$</td>
<td>0.17</td>
<td>0.52 **</td>
<td>0.56 ***</td>
</tr>
<tr>
<td>%Δ EMG:M$_{RF}$</td>
<td>0.10</td>
<td>0.56 ***</td>
<td>0.51 **</td>
</tr>
<tr>
<td>%Δ EMG:M$_{VL}$</td>
<td>0.00</td>
<td>0.28</td>
<td>0.31</td>
</tr>
<tr>
<td>%Δ EMG:M$_{VM}$</td>
<td>0.11</td>
<td>0.35 *</td>
<td>0.48 **</td>
</tr>
<tr>
<td>%Δ EMG$_{BF}$</td>
<td>0.00</td>
<td>0.10</td>
<td>-0.20</td>
</tr>
<tr>
<td>Δ%VA</td>
<td>0.23</td>
<td>0.08</td>
<td>0.17</td>
</tr>
<tr>
<td>MA</td>
<td>0.13</td>
<td>0.11</td>
<td>-0.10</td>
</tr>
</tbody>
</table>

ΔCSA,Q$_{PROX}$ | 0.36 * | 0.23 | -0.26 |
ΔCSA,Q$_{MID}$ | 0.30 | 0.07 | -0.26 |
ΔCSA,Q$_{DIST}$ | 0.29 | 0.20 | 0.17 |
ΔCSA,RF$_{PROX}$ | 0.32 | 0.00 | 0.04 |
ΔCSA,RF$_{MID}$ | 0.20 | 0.04 | 0.06 |
ΔCSA,RF$_{DIST}$ | 0.26 | 0.23 | 0.15 |
ΔCSA,VL$_{PROX}$ | 0.42 * | 0.35 * | 0.09 |
ΔCSA,VL$_{MID}$ | 0.45 ** | 0.03 | 0.03 |
ΔCSA,VL$_{DIST}$ | 0.26 | 0.16 | 0.12 |
ΔCSA,VI$_{PROX}$ | 0.30 | 0.36 | 0.04 |
ΔCSA,VI$_{MID}$ | 0.29 | 0.17 | 0.00 |
ΔCSA,VI$_{DIST}$ | 0.36 * | 0.08 | 0.12 |
ΔCSA,VM$_{PROX}$ | -0.18 | 0.12 | -0.05 |
ΔCSA,VM$_{MID}$ | 0.12 | 0.06 | -0.26 |
ΔCSA,VM$_{DIST}$ | 0.11 | 0.07 | -0.29 |
ΔθVL$_{PROX}$ | 0.41 * | 0.00 | -0.16 |
ΔθVL$_{MID}$ | 0.16 | 0.26 | 0.06 |
ΔθVL$_{DIST}$ | 0.13 | 0.15 | 0.10 |
ΔθRF | 0.19 | -0.06 | 0.23 |
ΔθVI | 0.05 | 0.39 * | 0.26 |
ΔθVM | 0.27 | 0.28 | -0.10 |
ΔiVL$_{PROX}$ | 0.13 | -0.20 | -0.18 |
ΔiVL$_{MID}$ | -0.06 | -0.34 * | -0.15 |
ΔiVL$_{DIST}$ | -0.12 | 0.17 | 0.00 |
ΔiRF | -0.27 | -0.22 | -0.43 * |
ΔiVI | 0.00 | 0.13 | -0.03 |
ΔiVM | 0.23 | -0.05 | -0.09 |

EMG:M = normalised average quadriceps (AVEQ), rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM) amplitude; EMG$_{BF}$ = amplitude MA=patella tendon moment arm; %VA = percent voluntary activation.

CSA = cross sectional area of the whole quadriceps (Q), rectus femoris (RF) vastus lateralis (VL), vastus intermedius (VI), and vastus medialis (VM). θ$_i$ = fascicle angle, l$_i$ =fascicle length. PROX, MID and DIST refer the proximal mid-muscle and distal regions of the thigh.

Δ = absolute change; %Δ = percent change. *p ≤ 0.05; **p ≤ 0.01; ***p ≤ 0.001
5.4 Discussion

Whilst muscle size, activation and architecture are considered to be important variables influencing maximum muscular force production [13] there is surprisingly little information regarding the relationship between changes in these variables and changes in strength following training. The present study examines the relationship between changes in isometric, concentric and eccentric knee extension strength and changes in specific anatomical and neuromuscular variables (i.e. muscle size, activation and architecture) following chronic (10 weeks) heavy (6-RM) strength training. The main conclusions are that 1) the change in isometric strength was moderately associated with changes in muscle size and fascicle angle; 2) the change in agonist muscle activation was the strongest predictor of the changes in maximum concentric and eccentric torque production ($r = 0.51$-$0.56$), and this relationship was strengthened when combined with muscle size and fascicle angle in the predictive models; 3) pre-training moment arm distance and the change in precontraction voluntary activation ($\%VA$) also appeared to influence the change in eccentric torque as they were included in models that received substantial support; 4) the best models for predicting maximum torque within a population (Study 1) did not predict the change in torque with training; and 5) overall, 27-41% of the variance in the change in isometric, concentric and eccentric torque could be predicted by the change in the anatomical and neuromuscular variables measured in the present study.

The four best candidate models for each contraction mode were ranked using Akaike’s information criterion ($AIC_c$), an information-theoretic approach for model selection that determines the best-fit model by accounting for the goodness-of-fit of a model (i.e. the difference between the expected and the observed data) in conjunction with its simplicity (i.e. the number of variables included) [183, 184]. The models were designed to predict the absolute changes in strength rather than the relative change to ensure that the influence of stronger participants was not reduced if their relative changes were modest compared to the weaker participants. Post-hoc, the data analysis was repeated using relative change scores and, interestingly, little difference in outcome was observed (data not shown) so the information presented herein appears equally applicable to relative changes in strength. The training elicited strength increases (Table 5.2) that were similar in magnitude to those reported previously following similar-duration heavy strength training interventions [i.e. 13-20%; 46, 133, 206]. Nonetheless, although strength changes in all contraction modes were substantial and statistically significant, they were also highly variable between individuals (see large SD in Table 5.2). The changes in strength were slightly less than the $33.9 \pm 15.7$ kg ($46.6 \pm 21.0\%; p < 0.001$) increase in isoinertial 6-RM knee extension strength following training. This discrepancy may have resulted
from the different contraction modes performed in the training (isoinertial) and testing (isometric and isokinetic), as well as the participants providing maximal effort during pre-training testing during the functionally simple isometric and isokinetic extensions, but (potentially) not performing maximally in the initial weeks of the functionally more complex isoinertial leg-press and concentric-eccentric knee extension training. The neuromuscular adaptations were measured during isometric and isokinetic contractions, as isoinertial training exercises also require greater activation of the stabilising and synergist muscles and thus maximum quadriceps force production may be limited by strength and activation of the stabiliser muscles [133].

The best-fit models were found to explain 27%, 40% and 41% of the inter-individual variation in the change in maximal isometric, concentric, and eccentric torque, respectively (Table 5.4). Model use led to mean (± SE) absolute errors in the prediction of the change in torque of 16.1 ± 3.1% (isometric), 59.8.2 ± 12.9% (concentric) and 17.6 ± 2.3% (eccentric) (Figure 5.2). Therefore, while changes in the anatomical and neuromuscular variables assessed in the present study appeared to be moderately associated with the change in maximum knee extension torque production (i.e. strength) following the 10-wk strength training period the change in concentric torque in particular was poorly predicted. Thus, whilst the isometric and eccentric models can provide a reasonable estimate of maximal joint torque, care should be taken when using the concentric torque prediction model. The results also indicate that mechanisms other than those measured in the present study must have influence strength changes (see discussion below).

5.4.1 Change in isometric torque versus changes in muscle size, architecture and activation
The best-fit model for the change in isometric torque included both the change in proximal VL CSA (vastus lateralis cross-sectional area) and the change in proximal VL fascicle angle. While the model explained only 27% of the change in isometric knee extension torque, it was the strongest model in the candidate set with an AICc weight (AICc,wi) of 0.57, indicating that 57% of the time the candidate model would be the best-fit model amongst that set of candidate models. The inclusion of CSA in the models is not surprising given that muscle size is considered to be a significant variable influencing joint torque production [11, 25-28], and cross-sectional analyses show moderate-to-strong correlations between maximal voluntary strength and measures of muscle size [3, 11; and Study 1, 20, 25]. However, the relationship between the change in joint torque and the change in muscle size is not as clear. Of the few studies to assess this relationship, weak correlations (r = 0.48 and 0.51) have been observed between the change in muscle size and the changes in isometric [20] and eccentric [35] strength, and a moderate correlation (r = 0.70) was found with the change in concentric [35] strength, following dynamic training. Higbie et al. [35] speculated that the weaker
relationship between the changes in muscle size and strength is unsurprising given that whole muscle CSA does not reflect the activation of muscle fibres, or the velocity-dependent nature of this activation. Interestingly, when accounting for regional-specific hypertrophy, strong relationships have been observed between the change in proximal VL and isometric force developed at short muscle lengths ($r = 0.80$ to $0.85$) and between the change in mid-region VL CSA and isometric force at long muscle lengths ($r = 0.79$ to $0.95$) [72] following isometric training. These results indicate that the strength of the relationship between the change in isometric torque and region-specific changes in muscle size may be task-dependent, and appear to be stronger following isometric training.

In the present study, the change in joint torque was most strongly associated with the change in muscle size when torque was measured isometrically ($r = 0.36$ to $0.45$, $p < 0.05$ for proximal region whole muscle, and VL CSA, and mid-region VL CSA; Table 5.7). This finding is similar to other studies examining the influence of dynamic strength training [20]. While these data suggest a causative link between changes in CSA and changes in isometric torque production a majority of the variance in torque production following training was left unexplained. Therefore, factors other than muscular hypertrophy must contribute significantly to changes in strength, and changes in hypertrophy alone may not be expected to result in notable changes in strength.

The incorporation of fascicle angle in combination with CSA (both measured proximally) improved the predictive strength of the models, which emphasises the potential importance of physiological CSA (PCSA) for maximising changes in strength. Increases in fascicle angle allow more contractile tissue to attach to a given area of tendon or aponeurosis [54, 73-75] and should thus increase PCSA and, therefore, contractile force. An alternative explanation is that increase in fascicle angle can increase fascicle rotation during contractions, which produces a gearing effect, allowing fascicles to work at slower speeds and enhancing muscle force through the optimisation of both the force-velocity and the force-length characteristics [2, 76, 190]. However, only high-force (slow-speed) contractions were examined in this study so it is probable that fascicle rotation would be minor [207] and this mechanism may not be of substantial influence. Therefore, it is more likely that the increased ability to pack contractile tissue onto the tendon and aponeurosis was the main benefit derived from the simultaneous increases in CSA and fascicle angle in the proximal region. It is not surprising proximal VL CSA was most strongly correlated with isometric torque as VL is the largest quadriceps component [208]. Its proximal CSA is slightly larger than its mid-region CSA (Table 5.7), and proximal CSA was observed to be strongly predictive of strength differences cross-sectionally (see Study 1). It is not yet known if there is a specific functional importance of the proximal quadriceps muscle during knee extension, but the results of the present study suggest that CSA
obtained at the proximal region has more influence than the middle (or distal) region on individual variations in the change in isometric knee extension torque following training. The result emphasises the need to examine changes in proximal quadriceps musculature rather than obtaining CSA from a single mid-muscle region or measuring whole muscle volume.

When considering single variable correlations rather than the models, it was of interest that the changes in proximal VL fascicle angle were moderately correlated with the change in isometric torque ($r = 0.41, p < 0.05$) whilst the other fascicle angle measures were not significantly correlated. Significant increases in fascicle angle were elicited by the training at all measurement sites (Table 5.3), however, these changes were highly variable between individuals (see SD in Table 5.3). Considering the apparent influence of the proximal region on changes in isometric torque, the possibility exists that the functional influence of other regions was less. One other study examined the relationship between the change in VL fascicle angle and the change in isometric torque following a similar training protocol to that used in the present study, with weak and non-significant correlations reported ($r = -0.33, p = 0.21$) [20]. Given these results it may be concluded that changes in fascicle angle, when considered in isolation, are relatively unrelated to changes in isometric strength but may be important when changes in CSA occur in unison.

Changes in fascicle length should, theoretically, be associated with increases in muscle shortening speed and force production during high speed or large range of movement activities [15, 76, 84]. Given this, it was not surprising that fascicle length was not included in any of the best-fit models for the prediction of isometric torque. While Erskine et al. [20] reported a weak correlation between VL fascicle length and isometric torque ($r = -0.47, p = 0.06$), Noorkoiv et al. [72] found no relationship between the change in VL fascicle length and the change in isometric torque, and in the present study, there was also no correlation observed between VL fascicle length and isometric torque (Table 5.7). The lack of relationships observed between fascicle length and isometric torque indicate that fascicle length change has little functional influence on isometric torque, at least when measured at the angle of peak torque. In future research, the impact of fascicle length on torque production at long versus short muscle lengths might be more explicitly examined.

5.4.2 Changes in concentric and eccentric torque versus changes in muscle size, architecture and activation

The best-fit models for predicting the changes in concentric ($\text{EMG} \cdot \text{M}_{\text{AVEQ}} + \text{CSA}, \text{VL}_{\text{PROX}} + \theta_{\text{VI}}$) and eccentric ($\text{EMG} \cdot \text{M}_{\text{AVEQ}} + \text{CSA}, \text{Q}_{\text{PROX}} + \theta_{\text{VI}}$) torque displayed moderate relationships ($R^2 = 0.40$ and 0.41, for concentric and eccentric torque, respectively). While the inclusion of changes in CSA and fascicle angle may again indicate the importance of an increase in contractile tissue within the
muscles for strength change following training, the change in muscle activation was also included in all isokinetic candidate models (Table 5.6) and was also the most strongly correlated with the change in torque of any neuromuscular variable ($r = 0.51$ to $0.56$ for the change in both average quadriceps (EMG:M$_{AVEQ}$) and RF (EMG:M$_{RF}$) muscle activity and the change in concentric and eccentric torque, respectively). Muscle activity can, therefore, be considered the most important variable influencing concentric and eccentric torque production.

Whilst a greater agonist muscle activity is often considered an important factor underpinning strength expression [6-8, 100, 101], the relationship between the change in muscle activity and the change in torque has not been well studied. Researchers have commonly used EMG procedures to assess changes in muscle activity [e.g. 13, 100], however peripheral factors can strongly influence these measurements [104]. To account for the potential influence of peripheral changes on EMG amplitudes in the present study, EMG signals were normalised to their respective M-wave amplitudes (elicited by supramaximal femoral nerve stimulation). M-wave-normalised EMG amplitudes (EMG:M) were considered to provide a clearer estimate of central drive because alterations at, and distal to, the neuromuscular junction, including changes to muscle membrane excitability, should be removed by the M-wave normalisation process [105]. In fact, %VA (obtained using the interpolated twitch technique) and quadriceps EMG:M amplitudes measured during the isometric contractions were both found to increase over the training period in the present study, which is some support for the supposition.

Moderate correlations were observed between the change in both concentric and eccentric knee extension torque and the percent change in average quadriceps EMG amplitude (EMG:M$_{AVEQ}$; $r = 0.52$, $p < 0.01$ and $r = 0.56$, $p < 0.001$, respectively; Table 5.7). Therefore, those individuals who displayed a greater increase in agonist EMG:M amplitude also displayed greater improvements in torque when measured during dynamic contractions. Among the quadriceps components, the percent change in RF EMG amplitude (EMG:M$_{RF}$) was most strongly related to the changes in both concentric ($r = 0.56$, $p < 0.001$) and eccentric ($r = 0.51$, $p < 0.01$) torque, while the percent change in VM and VL showed either a weak or no relationship with the change in torque for either measure (Table 5.7). These results are similar to Higbie et al. [35] ($r = 0.48$ and $0.68$, $p < 0.05$, for eccentric and concentric contractions, respectively), who considered the strength of this correlation reasonable considering EMG is not reflective of all possible neural adaptations following training. Thus models incorporating either the change in average quadriceps or rectus femoris amplitude both had substantial support for predicting the change in both concentric and eccentric torque following training.
Other muscle activity measures (i.e. voluntary activation and antagonist) were collected simultaneously with agonist EMG:M in the present study with the intention of strengthening evidence for the change in muscle activity in the regression models. In fact, the change in %VA was included along with ΔEMG:MRF in the models with strong support for predicting the change in eccentric torque (Table 5.6). The change in %VA obtained during isometric contraction (at the relevant angle of maximum isometric or eccentric torque) showed no relationship with the change in isometric torque ($r = 0.23$) despite it being shown in Study 1 (Chapter 4) to be an important predictor of maximum isometric and eccentric torque. This differs somewhat from the results observed by Erskine et al. ($r = 0.47$) in their untrained individuals following 9 weeks of strength training. In the present study, the difference in correlations between the change in torque and the changes in EMG:M and %VA makes sense in that while %VA is accepted as a good indicator of activation capacity, it is influenced by other factors distal to the neuromuscular junction, including the efficiency of force transmission through the series elastic components [120], which may influence the correlations (Table 5.7). It was also measured during isometric contractions, and thus measurement obtained during dynamic contractions may yield different results in future studies. Therefore, while %VA and EMG:M methods may share some mechanistic similarity, %VA is also influenced by non-neural factors. Regardless, as the eccentric models in which they are both included received substantial support ($\Delta AIC_c < 2$) it is probably that both measurements must be providing unique information with regard to muscular force production.

Antagonist muscle activity may also influence maximal torque production by decreasing net joint torque [9, 13, 209]; however, no change in biceps femoris EMG amplitude (EMGBF) was observed after training in any contraction condition, which is agreement with Reeves et al. [142]. The large inter-individual variability in this change (see large SD; Table 5.2) should have made relationships more, rather than less, likely to be detected, however despite being considered to have some influence on maximum torque production (Study 1), no relationships were observed between the changes in torques and the change in antagonist activity. Therefore, changes in other functional and structural variables were more clearly associated with changes in dynamic torque production. Given the findings from EMG:M and %VA analyses in the present study, the change in agonist muscle activity following training should be considered a more important contributor to the improvements in dynamic strength, whereas the change in antagonist activity does not appear to influence strength improvements.

Based on the influence of fascicle angle on isometric, concentric and eccentric torque, it might be speculated that changes in fascicle angle, when considered in isolation, are relatively unimportant
for strength increases (in the present study, only VI fascicle angle was correlated with the change in concentric torque; Table 5.7). However, the present results show that the inclusion of fascicle angle simultaneous with CSA or muscle activity variables substantially increased model strength. The specific importance of VI fascicle angle in the models cannot be readily explained, especially given that VL fascicle appeared more important for the change in isometric torque production. Speculatively, VI may play a more functional role during dynamic contractions than other quadriceps components; although VI muscle activity was not measured in this study and thus this hypothesis cannot be examined herein (this may be done in the future; [210]). In fact, there is no information regarding potential improvements in VI activity following strength training. As the change in VI fascicle angle was smaller than in other quadriceps components, and also displayed a relatively small range of change (Table 5.3), statistical effects are unlikely to underpin its inclusions and thus may indicate a particular functional importance. Speculatively, VI may have a greater influence on strength change when increases in muscle size and/or activity (or other changes that could not be examined in the present study, such as lateral force transmission efficiency [211]) occur. Thus, while not predictive in isolation, the simultaneous changes in proximal quadriceps CSA, VI fascicle angle and quadriceps muscle activity appear to strongly influence the change in concentric and eccentric torque production following strength training.

Models incorporating the change in VL fascicle length measured mid-muscle received substantial support for inclusion in the concentric models. In isolation, this measure was also weakly correlated with the change in concentric torque ($r = -0.34, p < 0.05$), indicating that the individuals who demonstrated the least increase (or a decrease) in fascicle length showed a greater increase in torque after training. Of interest, RF fascicle length was the best correlated muscular variable with the change in eccentric torque ($r = -0.43, p < 0.05$). Thus while generally considered to be associated with force production during lower-load (higher-speed) movements, these relationships indicate that the change in fascicle length may have some influence on the change in slower-speed (higher-load) dynamic strength following heavy training.

A large moment arm is theoretically ideal for high torque production, whereas a small moment arm optimises joint angular range and velocity [146, 147] and moment arm distance appears to influence the magnitude of strength improvement after a period of strength training. In the present study, models incorporating moment arm also received substantial support ($\Delta AIC_c < 2$) for inclusion in the eccentric models, and moderate support for consideration in both the isometric and concentric (data not shown) models ($\Delta AIC_c < 4$). Therefore, while not incorporated within the best-fit models, moment arm distance does appear to be influence the change in joint torque, with greater
improvements in dynamic torque production observed in individuals with a greater moment arm distance.

5.4.3 Are the neuromuscular variables correlated with maximum torque (cross-sectionally) also correlated with the change in torque following training (longitudinally)?

In the previous chapter (Study 1), several neuromuscular variables were identified as being strongly correlated with maximum isometric, concentric and eccentric torque production. Those results suggested that the targeting of these variables might allow for increases in muscular strength. However, the best-fit models identified in Study 1 did not predict the changes in strength measured in this study (Study 2a) ($R^2 \leq 0.07$; Table 5.5). Muscle activity was found to be important for predicting maximum isometric torque cross-sectionally (Study 1), but was not a strong predictor of the change in isometric torque following training. Similarly, joint moment arm distance was an important predictor of maximum concentric torque cross-sectionally, but was not a predictor of the change in concentric torque with training. While the variables used to predict the inter-individual variability in maximum eccentric torque versus those predicting the change in eccentric torque were similar (i.e. the models incorporated muscle size, fascicle angle and muscle activity), the variables were obtained from different muscles and regions within those muscles. These results clearly indicate that the results of cross-sectional and longitudinal studies can differ substantially and conclusions must be made specifically to the study design used. Thus, the functional importance of specific anatomical and neuromuscular variables for muscular strength appears to be contextual, for while strength variation within a population may be well explained by variations in muscle size, activity, architecture and moment arm, the changes in strength elicited by strength training of the duration used in the present study cannot be clearly linked with changes in those specific neuromuscular variables. Longer training periods eliciting greater strength changes may be required before clearer indications can be seen, or factors not measured in the present study (e.g. lateral force transfer [211]) might be influential with regards to strength change. Further research using longitudinal designs is required in order to provide the information necessary to allow for the specific targeting of neuromuscular factors that most clearly influence strength change.

Another factor influencing the strength of the relationship between the change in torque and the change in muscle size (and, in fact, the change in any of the neuromuscular variables) is the magnitude of change elicited by the training, which was far less than the inter-individual variation in these variables within a population. As an example, the ranges of isometric torque and CSA$_{PROX}$ measured at pre-training were 297.1 N-m and 63.4 cm$^2$, respectively, whilst the ranges of the changes in these variables were only 92.6 N-m and 8.8 cm$^2$ after training (i.e. 31% and 14% of the
variation measured pre-training). Thus, statistically, the variables that demonstrate a greater range of change may display stronger associations with the change in strength following training. This also ensures that the chance of observing strong relationships ($R^2 > 0.50$) between the prediction models and the change in torque is substantially reduced.

5.5 Summary

In the present study, models incorporating the changes in anatomical and neuromuscular variables have been found to explain up to 40% of the variance in the change in torque following 10 weeks (20 sessions) of heavy strength training. The error for the concentric torque prediction models was high (59.8 ± 12.9%), however, so factors other than those examined in the present study clearly impact on the change in strength. The present results indicated that changes in CSA and fascicle angle, in combination, are associated with the change in torque in all contraction modes, and that the changes measured in proximal VL were more influential in the change in maximal isometric torque whereas the change in VI fascicle angle were more influential in the changes in maximal concentric and eccentric torque. While changes in the proximal quadriceps region appeared to be associated with changes in isometric torque production, more research is required to determine the specific importance of changes in VI to the change in dynamic torque. The change in agonist muscle activity (i.e. EMG:M) was the strongest predictor of the changes in concentric and eccentric torque, and these associations were strengthened when CSA and fascicle angle variables were simultaneously included in the models. Pre-training moment arm distance and %VA were also included in models that received substantial support for influencing the change in eccentric torque, suggesting that individuals with favourable characteristics for strength (i.e. longer moment arms) can show greater increases in strength following neural and muscular improvements (i.e. %VA) after being exposed to a training stimulus. Fascicle length was included in models that received substantial support for influencing the change in concentric torque, indicating it should not disregarded as an influential variable even at slow-speeds. Muscle coactivation appeared to have little influence on strength changes and was thus not included in any best-fit model. Overall, the prediction models could only explain 27% (isometric), 40% (concentric) and 41% (eccentric) of the variance in the change in torque with training. The small changes (and thus variability of the changes) observed in anatomical structure and neuromuscular function may have impacted on statistical power and thus reduced the likelihood of finding strong relationships.

Additionally, it is noteworthy that the combination of variables found to most influence the change in torque in the present study differed from the combinations that best predicted maximum torque for each contraction mode (Study 1; Chapter 4). This result implies that the variables most
influencing between-subject variations in muscular strength are not the same as those influencing the change in strength with training, and that the influential factors are specific to the individual. Based on the present data, knee extensor muscle CSA, fascicle angle and muscle activity appear to explain the majority of the inter-individual variances in the change in strength following training due to their incorporation in a majority of the best-fit models. Strength training programs targeted to improve muscle activity, might elicit the greatest improvements in concentric and eccentric knee extensor strength, and individuals who might not improve neural function (e.g. some clinical populations) may have less scope for strength improvement.

The focus of the present study was the strength and neuromuscular adaptations arising from 10 weeks (20 sessions) of heavy lower-limb strength training. Future studies assessing differing loads, volumes, movement speeds and durations will further our understanding of the interactions between all these variables. Future studies assessing different sample groups (i.e. strength trained, elderly, or clinical populations) may also identify different neuromuscular adaptations, and hence relationships, between strength change and anatomical and neuromuscular changes. Based on the finding that different combinations of variables are important for predicting the change in torque (Study 2a), compared to predicting the inter-individual variability in maximum torque (Study 1), future studies should also examine whether all variables have the same potential for change between individuals following heavy strength training.
CHAPTER SIX: STUDY TWO (b)

Can strength improvements and anatomical and neuromuscular adaptations be predicted from pre-training tests in previously non-strength-trained healthy men?
6.1 Introduction

It is well established that strength training in non-strength-trained individuals elicits increases in both muscle strength and size and often, although not always, increases in muscle activity and changes in muscle architecture. However, there is a large inter-individual variability in these adaptations [e.g. 20, 36]. The studies presented so far (Studies 1 and 2) have examined which anatomical and neuromuscular variables were most associated with maximum isometric, concentric and eccentric knee extension strength (or its change) using both cross-sectional and longitudinal study designs. The results showed that the variables most associated with strength differences between individuals (i.e. cross-sectionally) may differ from those influential for the change in strength following a training intervention (i.e. longitudinally). One explanation for this is that the anatomical and neuromuscular variables might only influence the change in strength if they have scope for change with training. Therefore, individuals who already have certain anatomical characteristics or a sufficient level of neuromuscular function may have less scope for change. An individual’s pre-training anatomical and neuromuscular characteristics may, therefore, affect their capacity to adapt following training.

The present study was therefore designed for two purposes: (i) to determine whether training-induced anatomical and neuromuscular adaptations were dependent upon their magnitudes measured before training, and (ii) to determine whether the strength improvements achieved by an individual following training were associated with, and could thus be predicted by, their pre-training anatomical and neuromuscular characteristics. If improvements in strength following an intervention can be linked to pre-training anatomical and neuromuscular magnitudes then the effectiveness of a training regime for an individual might be determined before that intervention is initiated, and a decision made as to whether to progress or to use an alternative training stimulus. This study will use the data obtained in Study 2a, and the relationships between pre-training anatomical and neuromuscular magnitudes and the changes in both those anatomical and neuromuscular variables will be established. Subsequently, models will be developed to determine the accuracy with which the changes in maximum isometric, concentric and eccentric torque following 10 weeks of heavy, lower-limb strength training can be predicted from pre-training variable magnitudes.
6.2 Methods

6.2.1 Testing and training protocols

Pre-training torque, pre-training anatomical and neuromuscular variables, and changes in all these variables following 10 weeks of training were obtained from Study 2a. Participant details, along with ethics, and all training and testing procedures, were thus described previously (Study 2a, Chapter 5).

6.2.2 Data analysis

**Relationships between pre-training anatomical and neuromuscular variables and their changes with training**

Scatterplots were constructed to identify the relationships between the pre-training magnitudes of the anatomical and neuromuscular variables and their change following training. If a relationship appeared to be nonlinear, polynomial curve fitting was performed beginning at the 2nd order and increasing until the increase in $R^2$ was less than 2% [181]. As no relationships were improved by 2% using polynomial fitting, the relationships were considered linear and correlations were computed to quantify the relationships.

**Relationship between pre-training torque and anatomical and neuromuscular variables and the change in strength**

As above, scatterplots were constructed to identify the relationship between the pre-training magnitudes of the anatomical and neuromuscular variables and their change in magnitude following training. Polynomial curve fitting was completed if a relationship appeared nonlinear. Similarly, no relationships were improved by 2% using polynomial fitting, the relationships were considered linear and correlations were computed to quantify the relationships.

**Can the change in torque following training be predicted prior to training by baseline torque and anatomical and neuromuscular measures**

Linear regression models were developed to determine the strength of their relationship with the change in torque, with the parameters included in each model considered *a priori* to theoretically influence torque production [11, 13]. These included combinations of cross-sectional area (CSA), agonist (EMG:M) and antagonist (EMG) muscle activity magnitudes, moment arm distance (MA), fascicle angle ($\theta_f$), fascicle length ($l_f$), percent voluntary activation (%VA) and unpotentiated twitch torque ($T_{Un-Tw}$). Potentiated twitch torque was excluded from the models as it is influenced by the magnitude of the preceding MVC. The distributions of the dependent variables were checked for normality and both change in isometric ($\Delta T_{ISO}$) and eccentric ($\Delta T_{ECC}$) torques were transformed using the natural log due to non-normal distributions (Appendix 2C).
To rank the models, the AIC adjusted for small sample size ($\text{AIC}_c$) was used [183]. The model with the lowest $\text{AIC}_c$ value was considered the best fit for that strength measure, and all models with $\Delta\text{AIC}_c \leq 2$ were considered to have substantial support [183]. Between 20 and 24 models were developed for each contraction mode with the combinations of variables determined by their theoretical likelihood of influencing the change in torque, the frequency of their incorporation in models which had gained substantial support in the Study 2a, and on the strength of their individual correlations with the change in torque. The models showing substantial support for explaining the variance in the change in torque are shown in Table 6.7. Each model was assessed for collinearity using the variance inflation factor (VIF) to ensure that no two predictors within each model were highly correlated (VIF <5). All data were analysed using R version 3.0.0 (R Development Core Team, 2013).

### 6.3 Results

6.3.1 Correlations between pre-training anatomical and neuromuscular variables and their change with training

**Muscle activity**

Normalised average quadriceps (EMG:M$_{\text{AVEQ}}$) and VL (EMG:M$_{\text{VL}}$) amplitudes and percent voluntary activation (%VA) measured during the isometric contractions at pre-training were moderately correlated with their change following training ($r = -0.60$ to $-0.74$; $p < 0.001$; Table 6.1), such that less change was apparent when magnitudes were greater at pre-training. Weak correlations were observed for concentric and eccentric contractions between pre-training EMG:M$_{\text{AVEQ}}$ ($r = -0.40$ and -0.38; $p < 0.05$), EMG:M$_{\text{RF}}$ ($r = -0.44$ and -0.46; $p < 0.01$), and antagonist activity ($r = -0.36$ and -0.38; $p < 0.01$) amplitudes and their change with training.

**Muscle size and architecture**

Of the muscular variables, pre-training fascicle angles were most consistently related to their change following training. RF and mid-region VL fascicle angles (Figure 6.1) were moderately, and VI and distal-region VL weakly, correlated (Table 6.2) such that lesser change was observed in individuals with greater fascicle angles at pre-training. Pre-training proximal-region VL and RF fascicle lengths were also weakly correlated with their change following training, while only mid-region RF and distal-region VI CSA measures showed significant correlations. While proximal-region whole quadriceps CSA (CSA$_{\text{Q_PROX}}$) was included in all models best predicting inter-individual variation in maximum voluntary torque (Study 1) and the change in torque following training (Study 2a), no relationship was observed between pre-training proximal-region CSA and its change with training ($r = 0.00$; Table 6.2).
6.3.2 Correlations between pre-training torque and anatomical and neuromuscular variables and the change in torque with training

Maximum voluntary torque and muscle activity

The change in torque with training was not correlated with pre-training torque for any contraction mode (r ≤ -0.15, p > 0.05; Table 6.1), so both stronger and weaker participants showed an equal capacity to improve their torque production. EMG:M amplitudes increased significantly with training (Table 5.2, Chapter 4), yet while pre-training RF EMG:M was weakly correlated with the change in concentric torque (r = -0.34, p < 0.05), indicating a trend towards greater increases in torque in individuals with lower muscle activity before training, there were no correlations between the other muscle activity variables and the change in torque (Table 6.1).

Table 6.1 Correlations between pre-training torque (maximum voluntary and electrically stimulated), muscle activity and moment arm magnitudes and the change in those magnitudes, as well as their correlations with the change in isometric, concentric and eccentric knee extension torque.

<table>
<thead>
<tr>
<th>Pre-Training Variable</th>
<th>ΔVAR (isometric)</th>
<th>ΔVAR (concentric)</th>
<th>ΔVAR (eccentric)</th>
<th>ΔT_ISO</th>
<th>ΔT_CON</th>
<th>ΔT_ECC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Torque MVC (N·m)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-0.08</td>
<td>-0.15</td>
<td>-0.14</td>
</tr>
<tr>
<td>T_U-TW (N·m)</td>
<td>-0.40 *</td>
<td>-</td>
<td>-</td>
<td>0.09</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>T_P-TW (N·m)</td>
<td>-0.47 **</td>
<td>-</td>
<td>-</td>
<td>-0.07</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>MA (mm)</td>
<td>-0.37 *</td>
<td>-0.43 **</td>
<td>-0.13 *</td>
<td>0.13</td>
<td>0.11</td>
<td>-0.09</td>
</tr>
<tr>
<td>EMG:M AveQ (mV)</td>
<td>-0.63 ***</td>
<td>-0.40 *</td>
<td>-0.38 *</td>
<td>0.11</td>
<td>-0.32</td>
<td>-0.02</td>
</tr>
<tr>
<td>EMG:M RF</td>
<td>-0.49 **</td>
<td>-0.44 **</td>
<td>-0.46 **</td>
<td>0.04</td>
<td>-0.34 *</td>
<td>-0.14</td>
</tr>
<tr>
<td>EMG:M VL</td>
<td>-0.60 ***</td>
<td>-0.22</td>
<td>-0.30</td>
<td>0.06</td>
<td>-0.04</td>
<td>0.07</td>
</tr>
<tr>
<td>EMG:M VM</td>
<td>-0.47 **</td>
<td>-0.24</td>
<td>-0.14</td>
<td>0.13</td>
<td>-0.20</td>
<td>0.02</td>
</tr>
<tr>
<td>EMG BF</td>
<td>-0.27</td>
<td>-0.36 *</td>
<td>-0.38 *</td>
<td>0.01</td>
<td>0.09</td>
<td>-0.07</td>
</tr>
<tr>
<td>%VA</td>
<td>-0.74 ***</td>
<td>-</td>
<td>-</td>
<td>0.00</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

ΔVAR = change in the anatomical and neuromuscular measurements; ΔT = change in maximum isometric (ΔT_ISO) and isokinetic concentric (ΔT_CON) and eccentric (ΔT_ECC) torque.

*p,0.05, **p<0.01, ***p<0.001.

Muscle size and architecture

Only two pre-training muscular variables were related to the change in torque following training. VI fascicle angle was weakly correlated with the change in concentric torque (r = -0.46, p < 0.01), indicating that individuals with smaller fascicle angles before training showed a greater change in torque following training, and RF fascicle length was moderately correlated (r = 0.53, p < 0.01) with the change in eccentric torque suggesting that individuals with longer RF fascicles before training displayed greater torque improvements following training. No other pre-training muscle size or architecture measures were correlated with the change in torque following training (Table 6.2).
Table 6.2 Correlations between pre-training muscle size and architecture magnitudes and the change in those magnitudes (ΔVAR), and their correlations with the change in isometric (ΔT\textsubscript{ISO}), and isokinetic concentric (ΔT\textsubscript{CON}) and eccentric (ΔT\textsubscript{ECC}) knee extension torque following training.

<table>
<thead>
<tr>
<th>Pre-training Variable</th>
<th>ΔVAR</th>
<th>ΔT\textsubscript{ISO}</th>
<th>ΔT\textsubscript{CON}</th>
<th>ΔT\textsubscript{ECC}</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSA, Q\textsubscript{PROX}</td>
<td>0.00</td>
<td>-0.01</td>
<td>-0.06</td>
<td>0.12</td>
</tr>
<tr>
<td>CSA, Q\textsubscript{MID}</td>
<td>-0.05</td>
<td>0.04</td>
<td>-0.06</td>
<td>0.11</td>
</tr>
<tr>
<td>CSA, Q\textsubscript{DIST}</td>
<td>0.17</td>
<td>0.03</td>
<td>-0.10</td>
<td>0.05</td>
</tr>
<tr>
<td>CSA, Q\textsubscript{SUM}</td>
<td>0.09</td>
<td>0.02</td>
<td>-0.07</td>
<td>0.10</td>
</tr>
<tr>
<td>CSA, RF\textsubscript{PROX}</td>
<td>0.14</td>
<td>-0.04</td>
<td>-0.09</td>
<td>0.08</td>
</tr>
<tr>
<td>CSA, RF\textsubscript{MID}</td>
<td>0.36 *</td>
<td>-0.07</td>
<td>-0.04</td>
<td>0.14</td>
</tr>
<tr>
<td>CSA, RF\textsubscript{DIST}</td>
<td>0.15</td>
<td>0.04</td>
<td>-0.05</td>
<td>0.15</td>
</tr>
<tr>
<td>CSA, RF\textsubscript{SUM}</td>
<td>0.28</td>
<td>0.01</td>
<td>-0.07</td>
<td>0.12</td>
</tr>
<tr>
<td>CSA, VL\textsubscript{PROX}</td>
<td>-0.16</td>
<td>-0.06</td>
<td>-0.11</td>
<td>0.05</td>
</tr>
<tr>
<td>CSA, VL\textsubscript{MID}</td>
<td>0.01</td>
<td>-0.02</td>
<td>-0.01</td>
<td>0.12</td>
</tr>
<tr>
<td>CSA, VL\textsubscript{DIST}</td>
<td>0.12</td>
<td>0.04</td>
<td>-0.11</td>
<td>0.03</td>
</tr>
<tr>
<td>CSA, VL\textsubscript{SUM}</td>
<td>0.06</td>
<td>-0.02</td>
<td>-0.08</td>
<td>0.08</td>
</tr>
<tr>
<td>CSA, VI\textsubscript{PROX}</td>
<td>0.12</td>
<td>0.02</td>
<td>-0.03</td>
<td>0.09</td>
</tr>
<tr>
<td>CSA, VI\textsubscript{MID}</td>
<td>0.02</td>
<td>0.10</td>
<td>-0.04</td>
<td>0.05</td>
</tr>
<tr>
<td>CSA, VI\textsubscript{DIST}</td>
<td>0.33</td>
<td>-0.04</td>
<td>-0.13</td>
<td>-0.03</td>
</tr>
<tr>
<td>CSA, VI\textsubscript{SUM}</td>
<td>0.23</td>
<td>0.03</td>
<td>-0.06</td>
<td>0.05</td>
</tr>
<tr>
<td>CSA, VM\textsubscript{PROX}</td>
<td>0.07</td>
<td>-0.03</td>
<td>0.00</td>
<td>0.11</td>
</tr>
<tr>
<td>CSA, VM\textsubscript{MID}</td>
<td>-0.15</td>
<td>-0.06</td>
<td>-0.17</td>
<td>0.15</td>
</tr>
<tr>
<td>CSA, VM\textsubscript{DIST}</td>
<td>-0.27</td>
<td>0.08</td>
<td>0.00</td>
<td>0.06</td>
</tr>
<tr>
<td>CSA, VM\textsubscript{SUM}</td>
<td>-0.11</td>
<td>0.00</td>
<td>-0.07</td>
<td>0.11</td>
</tr>
<tr>
<td>θ\textsubscript{f} VL\textsubscript{PROX}</td>
<td>-0.24</td>
<td>-0.30</td>
<td>-0.09</td>
<td>-0.24</td>
</tr>
<tr>
<td>θ\textsubscript{f} VL\textsubscript{MID}</td>
<td>-0.52 **</td>
<td>0.12</td>
<td>-0.17</td>
<td>0.03</td>
</tr>
<tr>
<td>θ\textsubscript{f} VL\textsubscript{DIST}</td>
<td>-0.40 *</td>
<td>0.00</td>
<td>-0.15</td>
<td>-0.05</td>
</tr>
<tr>
<td>θ\textsubscript{f} RF</td>
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<tr>
<td>θ\textsubscript{f} VI</td>
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<td>0.00</td>
<td>-0.46 **</td>
<td>-0.19</td>
</tr>
<tr>
<td>θ\textsubscript{f} VM</td>
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<td>0.03</td>
<td>-0.16</td>
<td>-0.07</td>
</tr>
<tr>
<td>i\textsubscript{f} VL\textsubscript{PROX}</td>
<td>-0.42 *</td>
<td>-0.10</td>
<td>-0.14</td>
<td>-0.02</td>
</tr>
<tr>
<td>i\textsubscript{f} VL\textsubscript{MID}</td>
<td>-0.03</td>
<td>-0.16</td>
<td>0.08</td>
<td>-0.27</td>
</tr>
<tr>
<td>i\textsubscript{f} VL\textsubscript{DIST}</td>
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<td>0.07</td>
<td>-0.06</td>
<td>0.24</td>
</tr>
<tr>
<td>i\textsubscript{f} RF</td>
<td>-0.38 *</td>
<td>-0.05</td>
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<td>0.53 **</td>
</tr>
<tr>
<td>i\textsubscript{f} VI</td>
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<td>-0.10</td>
<td>0.22</td>
<td>0.23</td>
</tr>
<tr>
<td>i\textsubscript{f} VM</td>
<td>-0.30</td>
<td>-0.11</td>
<td>0.06</td>
<td>0.02</td>
</tr>
</tbody>
</table>

CSA = cross sectional area of the whole quadriceps (Q), rectus femoris (RF) vastus lateralis (VL), vastus intermedius (VI), and vastus medialis (VM). θ\textsubscript{f} = fascicle angle, i\textsubscript{f} = fascicle length. PROX, MID and DIST refer the proximal mid-muscle and distal regions of the thigh. Δ = absolute change; %Δ = percent change.

*p ≤ 0.05; **p ≤ 0.01; ***p ≤ 0.001
Figure 6.1. Relationships between pre-training neuromuscular variable magnitudes and the changes in those magnitudes following training. CSA,QPROX = proximal cross-sectional area; θVL_MID = middle vastus lateralis fascicle angle; EMG = normalised average quadriceps (AVEQ) and rectus femoris (RF) EMG:M_WAVE amplitude; %VA = percent voluntary activation. CSA and θ obtained at rest, muscle activity measures were obtained during maximal isometric (ISO) concentric (CON) and eccentric (ECC) contraction. Δ = absolute change; R² represent the adjusted-R². SE ± 1.96 is shown in grey. **p ≤ 0.01; ***p ≤ 0.001
Figure 6.2. Relationships between pre-training neuromuscular magnitudes and the change in maximum isometric (ΔT_ISO), and isokinetic concentric (ΔT_CON) and eccentric (ΔT_ECC) torque following training. EMG:M = EMG:M normalised average quadriceps (AVEQ) and rectus femoris (RF) amplitude; θ_f = fascicle angle of vastus intermedius (VI); l_RF = fascicle length of rectus femoris (RF); Δ = absolute change; R² represent the adjusted-R². SE ± 1.96 is shown in grey. *p ≤ 0.05, **p ≤ 0.01
6.3.3 Prediction models for the change in torque versus the combined pre-training torque and anatomical and neuromuscular variables

The change in isometric torque could not be adequately (i.e. significantly) predicted from the pre-training measurements ($R^2 = 0.06$, Table 5.7) and was ranked only slightly above the null model ($\text{AIC}_{C, w_f} = 0.32$ and 0.30, respectively). A moderate correlation was observed between the best combination of anatomical and neuromuscular variables found to influence the change in eccentric torque ($\text{EMG}:M_{\text{RF}} + \theta \text{VI}; R^2 = 0.27$). The prediction of the change in eccentric torque was not improved by the inclusion of further predictor variables additional to RF fascicle length ($R^2 = 0.26$).

Table 6.7 Akaike’s Information Criterion of model parameters showing substantial support ($\Delta\text{AIC}_C \leq 2$) predicting the change in maximal isometric, and isokinetic concentric and eccentric (ISO, CON and ECC, respectively) torque ($\Delta T$) based on pre-training variables following training. Models with an $\text{AIC}_C$ weighting ($\text{AIC}_C, w_f$) $\geq 0.10$ (i.e. greater than a 10% chance they will be the best-fit model) and stronger than the null model, are identified by shading.

<table>
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<tr>
<th>Contraction</th>
<th>Model</th>
<th>K</th>
<th>$\text{AIC}_C$</th>
<th>$\Delta\text{AIC}_C$</th>
<th>$\text{AIC}_C, w_f$</th>
<th>LL</th>
<th>$R^2$</th>
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<tr>
<td>$\Delta T_{\text{ISO}} (\ln)$</td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td></td>
<td>$\theta_1 \text{VL}_{\text{PROX}}$</td>
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<td>0.06</td>
</tr>
<tr>
<td></td>
<td>Intercept Only</td>
<td>2</td>
<td>85.82</td>
<td>0.98</td>
<td>0.18</td>
<td>-40.73</td>
<td>-</td>
</tr>
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<td>$\text{CSA, Q}<em>{\text{PROX}} + \theta_1 \text{VL}</em>{\text{PROX}}$</td>
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<td>86.01</td>
<td>1.17</td>
<td>0.16</td>
<td>-38.36</td>
<td>0.07</td>
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<tr>
<td></td>
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<td>1.64</td>
<td>0.13</td>
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<td>$\theta_1 \text{VL}<em>{\text{PROX}} + \theta_1 \text{VL}</em>{\text{MID}}$</td>
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<tr>
<td>$\Delta T_{\text{CON}}$</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>$\text{EMG: M}_{\text{RF}} + \theta_1 \text{VI}$</td>
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<td>0.27</td>
<td>-134.83</td>
<td>0.27</td>
</tr>
<tr>
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<td>$\text{EMG: M}_{\text{AVEQ}} + \theta_1 \text{VI}$</td>
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<td>0.14</td>
<td>-135.86</td>
<td>0.24</td>
</tr>
<tr>
<td></td>
<td>$\text{EMG: M}<em>{\text{AVEQ}} + \theta_1 \text{VI} + T</em>{\text{MVC}}$</td>
<td>5</td>
<td>280.28</td>
<td>1.29</td>
<td>0.14</td>
<td>-134.11</td>
<td>0.27</td>
</tr>
<tr>
<td></td>
<td>$\text{EMG: M}<em>{\text{RF}} + \theta_1 \text{VI} + \text{CSA, Q}</em>{\text{PROX}}$</td>
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<td>1.71</td>
<td>0.11</td>
<td>-134.32</td>
<td>0.27</td>
</tr>
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<td>0.10</td>
<td>-134.44</td>
<td>0.26</td>
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<td>287.05</td>
<td>8.05</td>
<td>0.01</td>
<td>-141.34</td>
<td>-</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>$\theta_1 \text{RF}$</td>
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<td>-36.70</td>
<td>0.26</td>
</tr>
<tr>
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<td>0.28</td>
</tr>
<tr>
<td></td>
<td>$\theta_1 \text{RF} + T_{\text{MVC}}$</td>
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<td>1.00</td>
<td>0.14</td>
<td>-35.89</td>
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<td>-36.05</td>
<td>0.24</td>
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<tr>
<td></td>
<td>Intercept</td>
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<td>88.35</td>
<td>8.09</td>
<td>0.01</td>
<td>-41.97</td>
<td>-</td>
</tr>
</tbody>
</table>

($\ln$) refers to the natural log of the $\Delta T$. For each candidate model: $\theta_i =$ fascicle angle of proximal vastus lateralis ($\text{VL}_{\text{PROX}}$) vastus intermedius (VI) or rectus femoris (RF); $\text{CSA, Q}_{\text{PROX}} =$ proximal cross-sectional area; $\text{EMG} =$ amplitude of average quadriceps (AVEQ) or rectus femoris (RF); $\theta_i =$ fascicle length of rectus femoris (RF); Intercept = basic control model with no predictors, and includes only the constant and residual variance ($\sigma^2$). $K =$ number of parameters tested in each model; $\text{AIC}_C =$ Akaike information criterion for a small data set; $\Delta\text{AIC}_C =$ the models $\text{AIC}_C$ minus the minimum $\text{AIC}_C$ among candidate models. $\text{AIC}_C, w_f =$ the percentage of times that a given model would be selected as the ‘best model’ by $\text{AIC}_C$, and serves as the weight of evidence for a given model being the best model from that set of candidate models [183]; $R^2 =$ adjusted $R^2$. 

94
6.4 Discussion

While muscle size, activity and architecture are considered important variables influencing maximal force production [13], there is surprisingly little information regarding how these variables are influenced by strength training. In view of the large inter-individual variability in strength improvements following training interventions [13, 20, 36], the adaptations in mechanisms underpinning strength changes need to be more completely understood. Additionally, if improvements in strength following an intervention can be linked to pre-training anatomical and neuromuscular magnitudes, training programs could be designed to target an individual’s specific weaknesses. In the present study, two main questions were therefore asked: (i) are training-induced anatomical and neuromuscular adaptations following moderate-duration (10 weeks) heavy-load (6-repetition maximum; 6-RM) strength training dependent upon their magnitudes measured before training; and (ii) are strength improvements achieved by an individual following training associated with their pre-training anatomical and neuromuscular magnitudes? Based on these results it was aimed to determine whether the change in torque following training could be predicted prior to training using models including specific combinations of pre-training anatomical and neuromuscular variables.

6.4.1 Are changes in anatomical and neuromuscular variables dependent upon their pre-training magnitudes?

The first purpose was to determine whether pre-training muscle size, activity and architecture were associated with their change following training. For this purpose, relationships between the pre-training magnitudes and changes after training were examined. The main conclusions were that (i) the changes in muscle size were not influenced by pre-training size; (ii) the changes in fascicle angle were moderately related to pre-training angle; and (iii) the changes in isometric muscle activity were moderately related to pre-training EMG:M amplitudes.

Effect of pre-training muscle size on hypertrophy

In line with previous findings [20, 36], the changes in muscle size in the present study were highly variable between individuals. This variability was observed despite ensuring similarity in both the participants’ previous physical activity (via their average daily energy expenditure – Appendix 2A) and strength training experience (none in the past 4 years) as well as providing appropriate post-exercise nutrition (i.e. 20-40 g dose of whey protein, dependent on body size) and guidelines to healthy eating. Nonetheless, the magnitude of change in muscle size was not related to pre-training muscle size. Perhaps importantly, proximal-region CSA, either whole quadrieps (CSA,QPROX) or vastus lateralis (CSA,QPROX), was consistently included in the best-fit models for predicting both inter-
individual variability in maximum voluntary torque (Study 1) and the change in maximum torque following training (Study 2a), and the present results indicate that individuals were equally likely to increase it with training (Table 6.2 and Figure 6.1). The strength of the relationship between pre-training CSA and the change in CSA increased only slightly when individual quadriceps components were examined separately (e.g. mid-thigh rectus femoris: $r = 0.36, p < 0.05$; no other significant correlations). Thus, the hypertrophic response appeared independent of pre-training muscle size. This makes sense in the context that all participants were non-strength-trained prior to study commencement and should therefore have had equal scope for increase.

**Effect of pre-training muscle architecture on its change with training**

Correlations between pre-training fascicle angle and its change with training varied depending on the muscle and the region (Table 6.2). While no correlation was observed between pre-training proximal-region VL fascicle angle and its change with training ($r = -0.24$, $p > 0.05$) there were moderate and weak correlations at mid-muscle and distal sites ($r = -0.52$, $p < 0.001$ and $r = -0.42$, $p < 0.05$, respectively), indicating variable influences of pre-training fascicle angle on its change with training. Moderate and weak correlations were also observed in both RF and VI ($r = -0.56$, $p < 0.001$ and $r = -0.42$, $p < 0.05$, respectively), providing evidence that changes in fascicle angle were somewhat related to pre-training fascicle angle magnitudes, where individuals with smaller angles had a greater potential for change.

With regards to fascicle length, weak correlations were observed between pre-training RF and proximal-region VL fascicle length and their change with training ($r = -0.38$ and $-0.42$; $p < 0.05$), indicating a tendency for individuals with longer fascicles to show less change with training. These data are in agreement Noorkoiv et al. [72], who observed that only participants with shorter VL fascicles showed substantial lengthening after 6 weeks of isometric training at either short or long quadriceps muscle lengths ($r = -0.50$). Based on these results there appears to be a small effect of pre-training fascicle length on the change in fascicle length, where individuals with shorter fascicles before training have more scope for length increase. Collectively, the present data suggest that muscle architectural adaptations with training are moderately influenced by pre-training fascicle angle and length. Given the potential influence of this effect on statistical outcomes in research studies, relationships between pre-training muscle architecture and its change across an intervention period should be examined in case adjustments to statistical methods (e.g. use of covariates [72]) are required, and also to estimate the likelihood of observing a statistical change given the possible ‘ceiling effect’ (i.e. type II error inflation).
Effect of pre-training muscle activity on the change in muscle activity with training

Increases in agonist muscle activity (e.g. EMG:M amplitude) commonly [6, 47, 100, 101], although not always [13, 134, 202], occur after periods of strength training. Maximum muscle activity measured prior to training may potentially influence the changes in muscle activity with training. It could be speculated that improvements would be minimal if an individual’s agonist muscle activity was exceptional prior to training. Given the relationship observed between changes in muscle activity and changes in strength in Study 2a, strength gains might thus be attenuated. In the present study, significant increases in M-wave-normalised EMG amplitudes were found in all quadriceps components (except RF EMG:M in concentric contractions) under all three contraction modes (Chapter 5; Table 5.2). Under isometric conditions, moderate-to-weak correlations were observed between pre-training EMG:M amplitudes and their change with training (e.g. r = -0.63, -0.49, -0.60 and -0.47 for average quadriceps, RF, VL and VM EMG:M, respectively; Table 6.1), such that smaller improvements were observed in individuals with larger EMG:M ratios before training. Furthermore, a strong negative correlation was found between %VA measured before training and the change in %VA with training (r = -0.74, p < 0.001: Table 6.1). This relationship for %VA contrasts that observed in clinical patients with knee joint osteoarthritis in which no relationship was found [212], suggesting that such a finding might be population specific. It is also of interest that the change in %VA was more strongly correlated with pre-training magnitude than the normalised EMG:M amplitudes. While %VA is accepted as a good indication of activation ability, its calculation using the interpolated twitch method (ITT) is influenced by the potentiated twitch torque magnitude. Therefore changes in muscle CSA, intracellular calcium ion concentration [119] and the efficiency of force transmission through the series elastic components [120] may influence this measure. Regardless, the present data indicate that participants who displayed the lowest levels of agonist activity during isometric contractions before training also displayed the greatest improvements in agonist activity with training (Figures 6.1). This may indicate a limited scope for improvement in maximal isometric muscle activity in individuals who display greater levels of activity prior to training. This was supported by muscle activity not being incorporated into the best-fit models predicting the change in torque (Study 2a), despite being influential for predicting inter-individual variations in maximum torque production (Study 1). This effect may have been enhanced by the use of different contraction modes in training and testing. However, this was not observed isokinetically.

When measured during the concentric and eccentric contractions, however, the correlations between pre-training muscle activity and change in muscle activity were weak (r = -0.14 to -0.46; Table 6.1). This is suggestive of only a small trend toward greater improvements in muscle activity following training in those displaying lower muscle activity pre-training. This indicates all
individuals had similar ability to improve concentric and eccentric muscle activity with training regardless of their pre-training capacity, and is consistent with the moderate relationships observed between the changes in muscle activity and the changes in concentric and eccentric torque in the previous chapter (Study 2a; Table 5.4). That pre-training muscle activity had a strong influence on the change in muscle activity in isometric but not concentric and eccentric contractions may be traced to the use of dynamic (i.e. concentric + eccentric) training in the present research. Whilst dynamic training may have provided a strong stimulus for improvements in muscle activity in these contraction modes the lack of isometric training ensured that there was no clear stimulus for improvement in this contraction mode. Thus perhaps only individuals with the least pre-training isometric muscle activity had sufficient stimulus for improvement.

Antagonist muscle activity measured before training was weakly correlated with the change following training during both concentric and eccentric contractions ($r = -0.36$ and $-0.38$, respectively, $p < 0.05$; Table 6.1). Thus, individuals who used less antagonist activity during dynamic movements prior to training tended to produce a greater increase (or less of a decrease) following training. Clinically, it is important to determine if this increase in antagonist activity is large enough to influence functional performance. The results of Study 2a strongly suggest that changes in antagonist muscle activity have little influence on the change in strength with training so it is unlikely that the small effect of pre-training antagonist activity on the change in antagonist activity is of functional significance.

6.4.2 Are strength improvements following training dependent on pre-training anatomical structure and neuromuscular function?

After determining whether pre-training anatomical structure of neuromuscular function influences the ability to adapt following a training intervention, a second question was to determine if these magnitudes are associated with the change in torque following training. Understanding these relationships is the first step in being able to predict an individual’s likely strength improvement prior to the initiation of a training intervention. For this purpose, relationships between the magnitudes of anatomical and neuromuscular variables measured before training and the changes in absolute torque were examined. To the author’s knowledge, few studies have examined these relationships, yet such predictions would allow for an indication of whether heavy strength training might elicit a significant strength improvement in an individual before training commencement.

Relationship between pre-training strength and the change in strength

In the present study, there was substantial inter-individual variability in the change in joint torque production under isometric ($5.9 - 131.6$ N·m), concentric ($2.1 – 51.6$ N·m) and eccentric ($6.5 – 102.6$ N·m).
N·m) conditions. However, these changes were not correlated with pre-training torque magnitudes ($r < -0.15$; Table 6.1 and Figure 6.2), indicating that strong and weak individuals are equally likely to improve maximum torque production following 10 weeks of heavy strength training. While it might be speculated that stronger participants should have less scope for improvements following a training intervention [45], the participants in the present study were non-strength-trained and had similar exercise histories (MET levels classifying their habitual activity and lack of experience with strength training.). Thus, all participants were treated to a new physical conditioning stimulus and had an equal chance for strength improvement. This finding is similar to lack of correlation observed in the elbow flexors following 12 weeks training ($r < 0.15$) [50]. While the change in absolute torque was considered the most functionally important measure of strength it is of interest that there were also no correlations between pre-training strength and the change in relative (i.e. percent) change in strength (data not shown).

Relationship between pre-training muscle size and the change in strength

Muscle size is typically considered an important factor influencing muscle strength [11, 25-28] and it could therefore be hypothesised that individuals with a greater pre-training muscle mass might have less scope for strength improvements when increases in muscle activation are elicited through chronic training. However, in the present study pre-training muscle size provided no indication of the magnitude of strength improvement expected following training for either isometric concentric or eccentric ($r \leq 0.15, p > 0.05$; see Table 6.2). Thus, it appears that strength improvements following training are equally likely in those with larger and smaller muscles before the commencement of training.

Relationship between pre-training muscle architecture and the change in strength

Fascicle angle is considered to be an important determinant of force production characteristics, and a moderate correlation was observed between pre-training fascicle angle and its change with training in the present study. It could therefore be hypothesised that pre-training fascicle angle might also be correlated with the change in strength, and there was some evidence for this. For example, individuals with smaller VI fascicle angles before training tended to increase concentric torque production more, although the correlation was weak ($r = -0.46, p < 0.01$). In fact, of all neuromuscular measurements made at pre-training, VI fascicle angle was most strongly correlated with the change in maximal concentric torque; however, there was no relationship between pre-training fascicle angle measured at other locations and the changes in torque in any contraction mode.
Pre-training fascicle length measured in RF was moderately correlated ($r = 0.53$, $p < 0.01$) with the change in eccentric torque after training, but no other significant correlations between pre-training fascicle length and the change in torque were found. Speculatively, longer fascicles may be ideal for eccentric force production since muscle elongation would be completed with less sarcomere elongation, allowing force to be generated in a smaller region of the force-length relationship and potentially minimising strain-related fibre damage [213]. As RF fascicles were the longest within the quadriceps (see Table 5.3, Chapter 5) it is possible the improvement in RF muscle activity following training may have allowed the muscle to contribute more, providing a mechanism for improved eccentric force production. The fact that 26% of the variance in the change in eccentric torque was accounted for by the variability in pre-training RF fascicle length suggests that RF fascicle length may be functionally important variable [13], although variability in the change in RF fascicle length only explained 16% of the change in eccentric torque. The smaller correlation between the change in fascicle length and the change in torque may also imply that the stronger correlation with pre-training RF reflected its large inter-individual variability (larger range), which allowed for a stronger correlation to be observed, i.e. this relationship may indicate a statistical rather than functional effect. However, this might be considered unlikely given the importance of RF during eccentric contractions [13] and the greater increase in RF CSA and fascicle angle compared to the other quadriceps components noted in Study 2a (Table 5.3), although further research is necessary to exclude this possibility.

**Relationship between pre-training muscle activity and the change in strength**

As individuals with lower muscle activity prior to training showed a slightly greater improvement in activity following training (e.g. Figure 6.1), and given that muscle activity was associated with changes in strength in Study 2a, it could be hypothesised that these individuals may show more improvement in torque production following training. The present data partly confirm this hypothesis as the change in concentric torque was weakly correlated with pre-training RF EMG:M magnitudes ($r = -0.34$, $p < 0.05$). However, the changes in torque measured under isometric and eccentric conditions were not associated with pre-training muscle activity, and are similar to the results observed in the elbow flexor muscle group ($r = 0.19$) [50]. This lack of relationship with changes in maximum isometric and eccentric torque indicates that while increases in muscle activity may be associated with improvements in torque production following training (Study 2a), some individuals were able to increase torque production through other mechanisms. Thus, individuals with greater activity before training were in fact equally likely to increase peak strength as those with a lesser pre-training activity.
While a strong correlation was observed between pre-training %VA and its change with training \((r = -0.74)\), the change in %VA had no relationship with the change in strength \((r = 0.00)\). While this finding is consistent with the relationship between pre-training %VA and the change in torque following training in clinical studies \((r < -0.06 [212, 214])\), it contrasts with one study incorporating electrical stimulation training [215] where a strong relationship was observed \((r = -0.72)\). The cause of this discrepancy is not immediately clear, although it might indicate that broader adaptations are elicited by voluntary strength training, or that important changes in the neuromuscular system are elicited by electrical simulation training. In the present study there were also no relationships found between the changes in knee extension torque and antagonist activity measured before training. Thus, neither percent voluntary activation, nor pre-training agonist or antagonist muscle activity appear to be predictive of the magnitude of strength increase elicited by heavy strength training.

**Relationship between pre-training patella tendon moment arm distance and the change in strength**

In addition to muscular force capacity, joint torque is also dependent upon the moment arm across which the force is produced. While a large moment arm is theoretically ideal for high torque production, a smaller moment arm may optimise joint excursion and angular velocity [146, 147]. As moment arm distance is known to vary with both contraction intensity and joint angle [149-151] the moment arm-joint angle relationship was determined for each individual during muscular contraction and the moment arm at the angle of peak torque in each contraction condition was found. It was hypothesised that changes in strength might be related to moment arm distance, since the effects of increases in muscle size and activity (i.e. muscle force) should be amplified in individuals with larger moment arms. Nonetheless, no relationship was found between moment arm distance and changes in isometric, concentric or eccentric torque \((r < 0.13, p > 0.05; \text{Table } 6.1)\). Based on the present data, it can be concluded that knee extension strength increases following training are not strongly influenced by an individual’s joint moment arm. As no comparative data could be found describing the influence of moment arm distance in the changes in strength, this appears to be a novel finding which should be further examined in future research.

### 6.4.3 Are pre-training torque and anatomical and neuromuscular variables predictive of the change in torque following training?

In Studies 1 and 2 (Chapters 4 and 5) variables that were not correlated with strength (i.e. cross-sectionally) or its change with training (i.e. longitudinally) were often incorporated into best-fit models. This suggests that some variables provide an influence only when considered in the context of other variables (or changes in them) and thus modelling approaches are essential for determining the true influence of a variable. Regression models were developed to determine whether a
combination of variables measured pre-training could be used to predict the increase in strength with training. However, no combination of variables was found to be related to the changes in isometric torque following training ($R^2 < 0.06$; Table 5.7). With respect to concentric torque production, the combination of pre-training average quadriceps EMG:M and VI fascicle angle showed a moderate relationship ($R^2 < 0.27$), while RF fascicle length in isolation best predicted the change in eccentric torque ($R^2 < 0.26$). Therefore, while no model was found to predict changes in isometric torque, pre-training measurements could be used to predict ~25% of the variance in the improvements in maximal concentric and eccentric torque. Nonetheless, ~75% of the variation in the change in concentric and eccentric torque remained unexplained and thus strength changes do not appear to be well predicted from pre-training measurements. It appears that continual monitoring is required in order to determine the influence of a strength training program on muscular strength and neuromuscular adaptation.

6.5 Summary

The moderate negative correlations between the change in muscle activation measured during an isometric contractions and its pre-training magnitude indicate that individuals who already display greater activation pre-training have less scope for improvements following training. Muscle architectural adaptations with training are also moderately influenced by pre-training fascicle angle and length. This ‘ceiling effect’ has the potential to explain the lack of support for any model containing agonist EMG:M measurements to predict the change in isometric torque (Chapter 5), and the substantial support for any models incorporating agonist EMG:M to predict maximum isometric torque (Chapter 4). No relationship was observed between pre-training CSA and its change with training, indicating that all individuals had similar scope for hypertrophic gains with training. Thus the scope for change of a variable following 10 weeks heavy strength training needs to be considered. This emphasises the need to infer training adaptations from longitudinal, and not cross-sectional, study designs.

With regards to predicting an individual’s change in torque capacity from pre-training measurements, model predictions were poor. The change in isometric torque could not be explain by any combination of the pre-training variables ($R^2 = 0.06$). Models incorporating rectus femoris muscle activity and vastus intermedius fascicle angle variables explained only 27% of the variance in the change in concentric torque, and rectus femoris fascicle length in isolation explained 26% of the variance in eccentric torque change. Thus, nearly 75% of the change in torque cannot be explained from pre-training measurements of strength, anatomical structure or neuromuscular function in untrained healthy males following 10 weeks heavy strength training, at least using the present tests.
At present, the change in strength following training cannot, therefore, be predicted from pre-training anatomical and neuromuscular variables and it appears that continual monitoring is still clearly necessary when developing an individually-optimised strength training program. Future studies examining different aspect of anatomical structure and neuromuscular function may further our understanding of the interactions between all these variables.
CHAPTER SEVEN:  GENERAL DISCUSSION
7.1 Overview

The present body of work aimed to explore the relationships between anatomical and neuromuscular variables and strength expression. Although it has been established that a muscle’s size, architecture and level of activity and activation influence its force production, and that moment arm distance across which that force is transmitted impacts on resultant joint torque, there are significant inter-individual variations in these variables both between and within populations. Of additional consideration is that these variables also show great plasticity in response to exercise training. If the inter-individual adaptations in these variables explain the differences in strength improvements following training, then an individual’s baseline characteristics may also dictate their strength adaptations. The research presented in this thesis, therefore, had three main aims: (1) to determine the relative influence of anatomical (i.e. muscle and moment arm) and neuromuscular (i.e. activity and activation) variables on maximum isometric, concentric and eccentric knee extension torque production; (2) to determine whether the changes in specific combinations of anatomical and neuromuscular variables were related to the change in strength following a moderate-duration (10-week) heavy strength training program; and (3) to determine whether an individual’s strength and both anatomical and neuromuscular adaptations to heavy training could be predicted from testing performed prior to the commencement of training. To answer these questions, observational (cross-sectional) and mixed-method (longitudinal) study designs were utilised.

7.2 Main findings

In Study 1, strong relationships \( R^2 = 0.62 \) to 0.72 were observed between the best-fit anatomical and neuromuscular models and maximum isometric, concentric and eccentric torques. While muscle cross-sectional area (CSA) was included in all the best-fit models, it was observed that all models including CSA also included fascicle angle measurements, as hypothesised. This combination of CSA and fascicle angle hints that a greater physiological CSA (i.e. a greater quantity of contractile tissue) may strongly influence the inter-individual differences in muscular strength. It is worth noting that models including proximal-region CSA measurements explained a greater variance in the maximum torque than measurements obtained mid-muscle. This has important implications for studies assessing CSA as most research has presented data obtained at a single muscle location, which is typically at the muscle mid-region. These data obtained in Study 1 indicate that the proximal quadriceps site appears to be a more functionally relevant location for obtaining single-site CSA images, particularly when the interaction between muscle size and strength is of interest. Additionally, even though fascicle angle was consistently included in the best-fit models, it was only weakly correlated with maximum torque in all contraction modes. This finding highlights the need to
examine the interactions between variables when assessing their influence on maximum joint torque rather than relationships (e.g. correlations) in isolation.

The best-fit models for predicting maximal isometric and eccentric torque included both CSA and fascicle angle along with muscle activity and activation measures (EMG:M and %VA). This may be due to the uniqueness of these contractions compared to the normal movements performed during daily living. Instead of muscle activity, moment arm distance was included in the best concentric torque prediction model, indicating that mechanical leverage maybe more important than maximal activation ability for more commonly-performed concentric muscle contractions. Models including antagonist muscle activity also received substantial support for explaining maximal isometric and eccentric torque. Thus, while the best-performing models developed to predict maximal knee extensor (i.e. quadriceps only) torque were similar to those that best explained maximal knee extension torque (i.e. total torque), the inclusion of an antagonist muscle activity measure in supported models indicates that coactivation may influence maximal performance to some extent, and may be of functional significance in some individuals.

Of practical importance, models were also developed for use in clinical populations and athletes undergoing rehabilitation programs where maximal muscular contractions and electrical stimulations procedures may be contraindicated. It was also found that the combination of CSA, fascicle angle and moment arm distance can be used to estimate an individual’s knee extension strength without the need to perform maximal contractions or use electrical stimulation procedures. Notably, these best clinical models deviated only slightly in accuracy (< 3%) from the best-fit models. For the first time, a comparative data set for those anatomical and neuromuscular variables found to be most influential in maximal torque production have also been presented. An individual’s weaknesses (deficits) can thus be identified by comparison to the normal healthy male (18-40 y) population and training plans then designed to target those weaknesses.

It was identified in Study 2a that the adaptations in the mechanisms underpinning the change in strength appear to be highly individual. The linear regression models developed only yielded moderate relationships ($R^2 = 0.27$ to 0.41) between the change in the anatomical and neuromuscular variables and the changes in isometric, concentric and eccentric torque. The variables within these models also differed from the variables considered to explain the greatest variance in inter-individual differences in maximal torque (Study 1). While CSA and fascicle angle variables (potentially reflecting contractile tissue content) were included in each best-fit model, muscle activity variables were not included in the best isometric torque prediction models, which contrasts the model outcomes of Study 1. Also in opposition to Study 1, models incorporating muscle activity with CSA
and fascicle angle, rather moment arm, received substantial support for the prediction of the change in concentric torque. Interestingly fascicle length was also incorporated in models receiving substantial support for predicting the change in concentric torque. The best prediction models for changes in eccentric torque were similar to those obtained in Study 1 to predict inter-individual variations in torque, although the specific muscles or sites within muscles from which the variables were measured differed somewhat. Nonetheless, the changes in muscle CSA and fascicle angle in combination clearly, but moderately, predicted the change in isometric, concentric and eccentric strength and may thus be influential mechanisms. The change in quadriceps muscle activity (EMG:M) displayed the strongest relationships with the change in concentric and eccentric torque and their inclusion improved those prediction models. Based on the present data, strength training programs targeted to improve agonist muscle activity might elicit the greatest improvements in concentric and eccentric knee extension strength; whereas proximal muscle size and fascicle angle appear important for improvements in all contraction modes. While the weaker model predictions in Study 2a may be partly explained by statistical factors (i.e. the small range of change in the variables in Study 2a compared to the large inter-individual variation in Study 1), it also suggest that the factors influencing strength change with training may be somewhat different to the factors explaining individual variability in maximum strength.

Study 2b was conducted to assess whether the changes in the anatomical and neuromuscular variables were influenced by their pre-training magnitudes, and also whether these pre-training magnitudes were related to the change in strength following training. Moderate and negative correlations were observed between the change in muscle activation measured during an isometric contraction and its pre-training magnitude, suggesting that individuals with greater activation capacity prior to training may have less scope for improvement following training. This might partly explain why muscle activation variables were included in the models to predict maximum torque cross-sectionally (Study 1), but not the change in torque with training (Study 2); individuals with better pre-training activation capacity may still have shown improvements in torque because of changes in other anatomical and neuromuscular variables. This supposition is supported by the weak correlations between both pre-training muscle size and concentric and eccentric muscle activity capacities and their changes with training, indicating their pre-training magnitudes had little influence on their potential to change with training. In fact, concentric and eccentric torque prediction models incorporating both the change in muscle activity and muscle size received the strongest support for the prediction of changes in torque following training. There was also evidence to suggest that muscle architectural changes are moderately influenced by pre-training fascicle
angles and lengths. Nonetheless, there was little evidence that stronger individuals or those with greater CSA might show less adaptation in these variables.

Regarding the ability to determine an individual’s change in torque capacity from pre-training measurements, model predictions were poor. While the inclusion of rectus femoris muscle activity and vastus intermedius fascicle angle variables, or rectus femoris fascicle length alone, could be used to explain over 25% of the variance in concentric and eccentric torque change, respectively, too much of the variance in torque change remains unexplained for these models to be relied on in a functional setting. At present, the change in strength following training cannot be accurately predicted from pre-training anatomical and neuromuscular variables and it appears that continual monitoring is thus necessary when designing individually-optimised strength training programs. However, it appears that increases in muscle size, fascicle angle and muscle activity explain the majority of the inter-individual variances in the change in strength following training based on their inclusion in a majority of the best-fit models, and thus could be targeted. Strength training programs designed to improve muscle activity might elicit the greatest improvements in concentric and eccentric knee extensor strength. Individuals who may struggle to improve neural function (e.g. some clinical populations) might thus have less scope for strength improvement.

7.3 Final Summary
To better understand the factors influencing active joint torque production, anatomical structure and neuromuscular function were comprehensively examined and mathematical modelling techniques used to quantify their relationship with torque production. Maximum knee extension torque was measured through the range of motion to ensure the maximum torque was obtained regardless of variation between individuals or changes with training. Whilst isometric and concentric torque production have been regularly assessed in previous studies, eccentric torque production was also included in the present study given its importance in activities of daily living and sporting movements. Muscle CSA and architectural heterogeneity both between and within the quadriceps muscles was considered to potentially influence joint torque production, thus anatomical CSA and fascicle angle and length measurements were obtained at proximal, mid-muscle and distal regions. Extend-field-of-view ultrasonography was used so that muscle CSA and fascicle length could be measured without the need to overlay single images, or use extrapolation procedures or estimation. EMG amplitudes were normalised to their respective M-wave amplitudes to provide a robust measure of agonist muscle activity. Moment arm distance was obtained during muscular contraction, and at the angle of maximum torque rather than a single pre-determined angle. Additionally, an information-theoretic approach to model selection was used to estimate the
proportion of variance in combinations of anatomical and neuromuscular variables that could explain inter-individual variations, and training-related change, in maximum joint torque production.

The research conducted presently was considered the first step in understanding how the changes in specific anatomical and neuromuscular variables influence the change in torque following training. While previous studies have often been conducted under the assumption that parallel increases in neuromuscular variables and strength are indicative of a potentially causative relationship, the results of the present research suggest that this may not be the case when relationships between the changes in these variables are examined. Additionally, the strength of correlations between anatomical and neuromuscular variables (or their changes) and joint torque may provide some information as to their influence, and it was notable that these correlations did not consistently reflect the level of support for models incorporating those variables. These data emphasise the need to examine interactions between variables when assessing their influence on maximum joint torque, rather than assessing correlations in isolation. Thus, the research presented in the current thesis has demonstrated the importance of identifying the interaction between the anatomical and neuromuscular variables when assessing torque. While studies 2 and 3 assessed the effects of 10 weeks of heavy strength training, future studies assessing the effects of different training programs (i.e. training loads, volumes and durations) are required to further our understanding of the interactions between these variables.
REFERENCES


Appendix 1.
1A Ethical approval

Dear Joanne

Project Number: 4668 TREZISE
Project Name: Anatomical and physiological factors influencing force production capabilities

Supervisors: - Anthony Blazevich

Ethics approval for your research project was granted from 23 February 2010 to 30 November 2013.

The National Statement on Ethical Conduct in Human Research requires that all approved projects are subject to monitoring conditions. This includes completion of an annual report (for projects longer than one year) and completion of a final report at the completion of the project.

A FINAL REPORT was due on 30 November 2013.

A copy of the ethics report form can be found on the Ethics Website

Please complete the ethics report form and return the signed form to the Research Ethics Office.

Note that ethics approval is required for both the collection and use (analysis) of data. If the project is still continuing, please complete the form and apply for an extension of ethics approval.

Hi Joanne,

Project 4668 TRESIZE
Program Name - Anatomical and physiological factors influencing force production capabilities

Thank you for your Annual Ethics Report and advise our records have been updated to reflect the information provided. Your request for an extension of ethics approval has been granted until 30 May 2014.

Regards

Faye

Faye Walmsley
Ethics Support Officer,
Office of Research & Innovation,
Edith Cowan University,
270 Joondalup Drive,
Joondalup, WA 6027
Tel: +61 08 6304 5032 | Fax: +61 08 6304 5044 | CRICOS IPC 00279B
INFORMATION LETTER FOR PARTICIPANTS

Anatomical and physiological factors influencing force production capabilities

Study 1: Factors influencing force production capabilities.

Thank you for showing an interest in this research project. Please read this information letter carefully before deciding whether or not to participate. If you decide not to participate there will be no disadvantage to you of any kind and we thank you for considering participating.

Purpose of this research project

This project is being undertaken as part of the requirements of a Doctor of Philosophy in Sport and Exercise Science at Edith Cowan University. While the ability to perform high force and high speed movements is important for the successful performance of both sporting and daily activities (e.g. stair climbing, standing, and fall prevention), the relative importance of certain anatomical and physiological variables (e.g. muscle structure, nervous system, tendon properties) in producing these movements has not yet been determined. We know that these variables adapt in response to exercise loading, but we do not know why people show different improvements in strength and speed following training. Understanding why some people display greater (and different) adaptations than others is important for the optimum prescription of training plans for both sport and rehabilitative practices. This project, therefore, aims to establish which neural, muscular and tendinous factors are most clearly associated with an individual’s peak torque and power producing ability.

Why were you selected?

You have been selected as a potential participant for the study as you have indicated that you are a healthy male aged between 19-40 years. You also fit into one of the following five criteria:

You are either a trained participant who is:

1) An endurance trained runner who runs ≥40 km / week and has been doing so for over three years.

2) A sprint trained athlete (either team sport or track athlete) who can run 40 m in < 5.3 s. You have also been competing / playing for over three years.

3) A strength trained participant who has regularly performed resistance training for the past year for 3+ sessions / week, and you can squat 1.5× your body weight.

Or, you are an untrained participant who has either:

1) Been completing some exercise over the past three years, but nothing consistently.

2) Not participated in exercise for the past three years.
If you are a sprint-strength- or endurance-trained participant, you will complete a performance test prior to beginning this project. The sprint trained participants will complete the 40 m distance on a grass track with the use of timing gates, the strength-trained participant will perform full squat exercises and the endurance-trained participants will complete a modified Bruce Treadmill protocol to determine their VO2 max, all to be completed on Joondalup campus. If you are in either of the untrained participant groups, you will complete an average weekly activities form prior to beginning this study.

**What will be asked of you?**

All participants will be involved in six separate sessions over a 10 day period. All testing sessions will involve the quadriceps (knee extensor) muscles. The first two sessions will be ‘familiarisation’ sessions, where you will practice maximal contractions and be introduced to the electrical stimulation protocol. The next four sessions will involve data collection for this project - they will be explained below. Five of the six sessions will be held on ECU Joondalup campus. The sixth session will be conducted at a Radiology centre in Subiaco. You will be responsible for transporting yourself to all locations. You will be asked to refrain from any vigorous exercise 48 hours prior to testing, and from consuming caffeine, alcohol or any other stimulant or depressant for at least 6 hours prior to testing.

**Outline of the Testing Sessions:**

<table>
<thead>
<tr>
<th>First familiarisation session</th>
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</table>

*Familiarisation sessions:* As testing session 3 involves electrical stimulation of your femoral nerve (located in the groin area) and performing maximal contractions (testing sessions 3 and 4), the two familiarisation sessions are included so you can become accustomed to the protocol. During pilot studies for this protocol, the participants all agreed that while they knew the only negative aspect of the electrical stimulation was slight discomfort, they still felt apprehensive for the first two sessions. After that they all showed no hesitation about the stimulation. The familiarisation period is designed to make you apprehension free by the beginning of testing session 3. The familiarisation sessions will also involve the performance of both static and dynamic maximal contractions to allow you to become familiar with performing these movements. Each familiarisation session will take approximately 40 min.

*Testing session 1:* During session one, you will lie in a relaxed position on your back while an ultrasound probe is moved across your leg to take images of your muscles. This session will take 60 min.

*Testing session 2:* Seven x-ray images will be taken of your knee joint to allow the calculation of your patella tendon moment arm (the distance between the centre of your knee joint to your patellar tendon). This session will take place at Envision Medical Imaging in Subiaco. You will be required to find your own transport there. The time in the clinic itself will be 15 - 30 min.
Testing session 3: This session will determine your peak muscular strength during static muscle contractions and your quadriceps muscle activation. Eleven non-invasive surface electromyography electrodes (EMG) (each 2 cm²) will be placed on your right thigh muscles to measure the muscle activity during muscle contraction. To determine muscle activation your femoral nerve will be electrically stimulated approximately 10 times to determine your level of maximum tolerable intensity. The stimulation electrode will be positioned in the fold between your upper thigh and your pelvis (i.e. groin area). To determine the maximal strength of your quadriceps muscles you will perform 15 static maximal voluntary contractions (holding for 3 s). During these 15 contractions your femoral nerve will also be electrically stimulated (to elicit a further increase in contraction force) to enable analysis of voluntary activation verses your potential maximal activation. Maximal hamstring strength will also be determined by three static maximal contractions. This session will take 60 - 70 min.

Testing session 4: This session will determine your peak muscular strength during dynamic muscle contractions, and the stiffness of your patellar tendon. EMG electrodes will again be placed on your right thigh muscles to measure muscle activity. Twenty-eight dynamic maximal knee extension contractions (ranging over 7 different speeds) will then be performed to determine your velocity-specific peak strength. Three final static maximal contractions will be completed with the ultrasound device held above your patellar tendon to allow the calculation of tendon stiffness. This session will take 50 min.

Will you experience any discomfort or inconvenience? What are the potential risks?

1. To improve the detection of muscle activity by EMG, the skin under the electrodes will be gently shaved, abraded and cleaned with alcohol. This may sting a little, and you will be left with small 3 cm bald patches on your leg.

2. To maximally stimulate the quadriceps muscle group electrical stimulation lasting 2 milliseconds will be applied to your femoral nerve. The stimulation will begin at a low level and be slowly increased with your consent. This is likely to feel strange and slightly uncomfortable to begin with but becomes easily tolerable.

3. As the strength testing component of this study involves maximal muscle contractions there is a risk of delayed onset muscle soreness (1 to 3 days after exercise) and /or injury. Participants not accustomed to exercise are likely to experience this muscle soreness - this is very normal when loading a muscle more than it is used to. We will minimise this effect through adequate warm-up and warm-down procedures.

4. The radiation dose from the low-dose x-ray session is equivalent to 3 months of radiation exposure from natural daily background radiation. The time at the clinic will be 20 min.

What are the benefits to you?

You will learn how anatomical and physiological variables interact to improve your maximum force producing capabilities. You can be provided with measures of your maximal knee strength (both flexion and extension), neural activation, muscle architecture values, and cross-sectional images of your quadriceps muscles. The individual profile obtained from this study will enable you to identify which areas (neural, muscular or tendinous) you may be able to focus on to enhance your performance. If you
are one of the untrained participants you may decide to also take part in study 2 of this research program which involves a 10-week fully supervised resistance training program.

Confidentiality of information

All information provided and collected by the investigator will be used in a strictly professional and confidential manner. During the course of the study information will be stored either in a locked drawer or on a password-protected computer. Your data shall be given an identification code so that only the people directly involved in collecting the information will know exactly which person it belonged to. When the results are published in a scientific journal your identity will not be revealed. After a study is completed the data must be retained for a minimum of five years. It will be stored in a locked filing cabinet with restricted access.

Results of the research study

The results of this study will comprise my PhD Thesis. The data will also be published in academic journals and be presented conferences. Feel free to ask if you wish to know any of your individual results, and, following analysis, they can be relayed to you.

Withdrawing from the study

If you decide to participate, you are free to withdraw from the study at any time. Whether you decide to participate or not, your decision will not prejudice you in any way. If you do withdraw from the study any information or data pertaining to you will be excluded from the final results.

If you would like to participate, or have any questions and would like further information regarding this study, please contact me. If you would like to speak to the research supervisor, please contact Assoc Prof. Anthony Blazevich (08) 6304 5472. If you have any concerns of complaints about the research project and wish to talk to an independent person, please contact the ECU Research Ethics Officer on (08) 6304 2170.

Thank you for your time
Kind Regards,

Joanne Trezise MPhEd (PhD Candidate)
Faculty of Computing, Health and Science
School of Exercise, Biomedical and Health Science
Edith Cowan University
100 Joondalup Drive, Joondalup WA 6027
Office phone: (08) 6304 5819
Email: j.trezise@ecu.edu.au
INFORMATION LETTER FOR PARTICIPANTS

Anatomical and physiological factors influencing force production capabilities

Study 2: Investigation of the relationships between pre-training neural, muscular and tendon properties, and their changes with heavy strength training.

Thank you for showing an interest in this research project. Please read this information letter carefully before deciding whether or not to participate. If you decide not to participate there will be no disadvantage to you of any kind and we thank you for considering participating.

Purpose of this research project
This project is being undertaken as part of the requirements of a Doctor of Philosophy in Sport and Exercise Science at Edith Cowan University. While the ability to perform high force and high speed movements is important for the successful performance of both sporting and daily activities (e.g. stair climbing, standing, and fall prevention), the relative importance of certain anatomical and physiological variables (e.g. muscle structure, nervous system, tendon properties) in producing these movements has not yet been determined. We know that these variables adapt in response to exercise loading, but we do not know why people show different improvements in strength and speed following training. Understanding why some people display greater (and different) adaptations than others is important for the optimum prescription of training plans for both sport and rehabilitative practices. This project, therefore, aims to establish which neural, muscular and tendinous factors are most clearly associated with an individual’s peak torque and power producing ability.

Why were you selected?
You have been selected as a potential participant for this study as you have indicated that you are a healthy untrained male aged between 19-40 years. To qualify as ‘untrained’, you have not participated in regular exercise for the past two years.

What will be asked of you?
All participants will be involved in six separate sessions over a 10 day period. All testing sessions will involve the quadriceps (knee extensor) muscles. The first two sessions will be ‘familiarisation’ sessions, where you will practice maximal contractions and be introduced to the electrical stimulation protocol. The next four sessions will involve data collection for this project - they will be explained below. Five of the six sessions will be held on ECU Joondalup campus. The sixth session will be conducted at a Radiology centre in Subiaco. You will be responsible for transporting yourself to all locations. You will be asked to refrain from any vigorous exercise 48 hours prior to testing, and from consuming caffeine, alcohol or any other stimulant or depressant for at least 6 hours prior to testing.
Outline of the Testing Sessions:

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**Familiarisation sessions:** As testing session 3 involves electrical stimulation of your femoral nerve (located in the groin area) and testing sessions 3 and 4 require the performance of maximal contractions, the familiarisation sessions are included so you can become accustomed to the protocol. During pilot studies for this protocol, the participants all agreed that while they knew the only negative aspect of the electrical stimulation was slight discomfort, they still felt apprehensive for the first two sessions. After that they all showed no hesitation about the stimulation. The familiarisation period is designed to make you apprehension free by the beginning of testing session 3. The familiarisation sessions will also involve the performance of both static and dynamic maximal contractions to allow you to become familiar with performing these movements. Each familiarisation session will take approximately 1 hr 30 min.

**Testing session 1:** During session one, you will lie in a relaxed position on your back while an ultrasound probe is moved across your leg to take images of your muscles. This session will take 70 - 80 min.

**Testing session 2:** Seven x-ray images will be taken of your knee joint to allow the calculation of your patella tendon moment arm (the distance between the centre of your knee joint to your patellar tendon). This session will take place at Envision Medical Imaging in Subiaco. You will be required to find your own transport there. The time in the clinic itself will be 30 min.

**Testing session 3:** This session will determine your peak muscular strength during static muscle contractions and your quadriceps muscle activation. Eleven non-invasive surface electromyography electrodes (EMG) (each 2 cm²) will be placed on your right thigh muscles to measure the muscle activity during muscle contraction. To determine muscle activation your femoral nerve will be electrically stimulated approximately 10 times to determine your level of maximum tolerable intensity. The stimulation electrode will be positioned, by yourself, in the fold between your upper thigh and your pelvis (i.e. groin area). To determine the maximal strength of your quadriceps muscles you will perform 18 static maximal voluntary contractions (holding for 3 s). During these 15 contractions your femoral nerve will also be electrically stimulated (to elicit a further increase in contraction force) to enable analysis of voluntary activation verses your potential maximal activation. Maximal hamstring strength will also be determined by three static maximal contractions. This session will take 2 hr.

**Testing session 4:** This session will determine your peak muscular strength during dynamic muscle contractions, and the stiffness of your patellar tendon. EMG electrodes will again be placed on your right thigh muscles to measure muscle activity. Twenty-eight dynamic maximal knee extension contractions (ranging over 7 different speeds) will then be performed to determine your velocity-specific peak strength. Three final static maximal contractions will be completed with the ultrasound
device held above your patellar tendon to allow the calculation of tendon stiffness. This session will take 1 hr 30 min.

**Training Intervention:**
The training sessions will focus on your thigh muscles. You will be required to complete two training sessions per week for 10 weeks (20 sessions). The exercise protocol for each session will consist of 3 sets of 6 repetitions of three exercises: leg press, leg extension and leg curl. You will have 3 min rest between each set. Each session will be fully supervised. To limit muscle soreness (as you have not recently been performing this type of exercise), the loads you shall lift during the first week of the intervention have been minimised to 60 and 80% of your intended load, to ensure that you begin gradually. You will then perform three hard sessions (100% 6RM load) followed by one easy session (50% of your normal load) in order to prevent overtraining (see diagram).

*Training intensity for each session over the 10 weeks—three hard sessions followed by one easy session.*

![Diagram showing training intensity over 10 weeks]

Allowing for the rest periods, a warm-up, and some stretching on completion, each training session will take 45 min.

**Post-Training Testing Sessions**
In the week following completion of the training intervention you will be required to repeat Testing sessions 1 3 and 4 only.

**Will you experience any discomfort or inconvenience? What are the potential risks?**
1. To improve the detection of muscle activity by EMG during the testing sessions, the skin under the electrodes will be gently shaved, abraded and cleaned with alcohol. This may sting a little, and you will be left with small 3 cm bald patches on your thigh.

2. To maximally stimulate the quadriceps muscle group electrical stimulation lasting 2 milliseconds will be applied to your femoral nerve. The stimulation will begin at a low level and be slowly increased with your consent. This is likely to feel strange and slightly uncomfortable to begin with but becomes easily tolerable.

3. As the strength testing component of this study involves maximal muscle contractions there is a risk of delayed onset muscle soreness (1 to 3 days after exercise) and /or injury. Participants not accustomed to exercise are likely to experience this muscle soreness - this is very normal when loading a muscle more than it is used to. Through the testing session we will minimise this effect through adequate warm-up and warm-down procedures, and
during the training intervention we have taken the added precautions of building up the load slowly over the first week.

4. The radiation dose from the low-dose x-ray session is equivalent to 3 months of radiation exposure from natural daily background radiation. The time at the clinic will be 20 min.

What are the benefits to you?

This study aims to determine how anatomical and physiological variables interact to improve our maximum force producing capabilities. As an athlete the individual profile obtained from this study will enable you to identify which areas (neural, muscular or tendinous) you may be able to focus on to enhance your performance. You will also receive 20 fully supervised resistance training sessions and have the opportunity to ask questions about training recovery and exercise safety. Furthermore, you will be provided with a 250 ml protein shake following each training session to enhance your training and aid in recovery.

Confidentiality of information

All information provided and collected by the investigator will be used in a strictly professional and confidential manner. During the course of the study information will be stored either in a locked drawer or on a password-protected computer. Your data shall be given an identification code so that only the people directly involved in collecting the information will know exactly which person it belonged to. When the results are published in a scientific journal your identity will not be revealed. After a study is completed the data must be retained for a minimum of five years. It will be stored in a locked filling cabinet with restricted access.

Results of the research study

The results of this study will comprise my PhD Thesis. The data will also be published in academic journals and be presented conferences. If you wish to know any of your individual results, feel free to ask and following analysis they can be relayed to you.

Withdrawal from the study

If you decide to participate, you are free to withdraw from the study at any time. Whether you decide to participate or not, your decision will not prejudice you in any way. If you do withdraw from the study any information or data pertaining to you will be excluded from the final results.

If you would like to participate, or have any questions and would like further information regarding this study, please contact me. If you would like to speak to the research supervisor, please contact Assoc Prof. Anthony Blazevich (08) 6304 5472. If you have any concerns of complaints about the research project and wish to talk to an independent person, please contact the ECU Research Ethics Officer on (08) 6304 2170.

Kind Regards,

Joanne Trezise MPhEd (PhD Candidate)
School of Exercise, Biomedical and Health Science
Edith Cowan University
100 Joondalup Drive, Joondalup WA 6027
Office phone: (08) 6304 5819
Email: j.trezise@ecu.edu.au
CONSENT TO PARTICIPATE IN RESEARCH
Anatomical and physiological factors influencing force production capabilities

Study 1: Factors influencing force production capabilities.

This is to certify that I _______________________________________________ hereby agree to participate as a volunteer in a scientific investigation performed at Edith Cowan University.

The investigation and my part in the investigation have been defined and fully explained to me and I understand the explanation. A copy of the procedures of this investigation and a description of any risks and discomforts has been provided to me and has been discussed in detail with me.

- I have read and understood the information sheet about this research project and the testing protocols have been explained to me.
- I have been given an opportunity to ask any questions and all such questions and inquiries have been answered to my satisfaction.
- I understand that I am free to ask any questions and that they will be answered to my satisfaction.
- I understand that as part of the testing I will be required to undergo maximal voluntary contractions, have my skin prepared for electromyography (measure of muscle activity), have electrical stimulation on my femoral nerve, have ultrasound imaging of my knee extensor muscles and patella tendon, and eight x-ray images of my knee joint.
- I understand that the maximal voluntary contractions may lead to muscle soreness if I am unaccustomed to the technique and that the electrical stimulation will initially feel uncomfortable.
- I understand that I am free to withdraw my consent and to discontinue participation in the project or activity at any time.
- I understand that my data will remain confidential with regard to my identity.
- I certify that to the best of my knowledge and belief, I have no physical condition that would increase the risk to me participating in this investigation.
- I agree that the research data obtained from this study may be published, provided I am not identifiable in any way.

Participant _______________________________ Date __________________________

I, the undersigned, was present when the study was explained to the participant in detail and to the best of my knowledge and belief it was understood.

Investigator _______________________________ Date __________________________

Joanne Trezise MPhEd (PhD candidate)
School of Exercise, Biomedical and Health Science
Edith Cowan University
100 Joondalup Drive, Joondalup WA 6027
(W) 08 6304 5819 Email: j.trezise@ecu.edu.au
CONSENT TO PARTICIPATE IN RESEARCH

Anatomical and physiological factors influencing force production capabilities

*Study 2: Investigation of the relationships between pre-training neural, muscular and tendon properties, and their changes with heavy strength training.*

This is to certify that I ____________________________________________________________________ hereby agree to participate as a volunteer in a scientific investigation performed at Edith Cowan University.

The investigation and my part in the investigation have been defined and fully explained to me and I understand the explanation. A copy of the procedures of this investigation and a description of any risks and discomforts has been provided to me and has been discussed in detail with me.

- I have read and understood the information sheet about this research project.
- I have been given an opportunity to ask any questions and all such questions and inquiries have been answered to my satisfaction.
- I understand that I am free to ask any questions and that they will be answered to my satisfaction.
- I understand that as part of the testing I will be required to undergo maximal voluntary contractions, have my skin prepared for electromyography (measure of muscle activity), have electrical stimulation on my femoral nerve, have ultrasound imaging of my knee extensor muscles and patella tendon, and an x-ray image of my knee joint.
- I understand that the maximal voluntary contractions may lead to muscle soreness if I am unaccustomed to the technique and that the electrical stimulation will initially feel uncomfortable.
- I understand that I shall be undertaking a 10-week heavy resistance training program, that I must attend 2 sessions/wk, and that I may feel some muscle soreness in my thigh muscles from the training.
- I understand that I am free to withdraw my consent and to discontinue participation in the project or activity at any time.
- I understand that my data will remain confidential with regard to my identity.
- I certify that to the best of my knowledge and belief, I have no physical condition that would increase the risk to me participating in this investigation.
- I agree that the research data obtained from this study may be published, provided I am not identifiable in any way.

Participant __________________________________ Date ______________________

I, the undersigned, was present when the study was explained to the participant in detail and to the best of my knowledge and belief it was understood.
Joanne Trezise MPhEd (PhD candidate)
School of Exercise, Biomedical and Health Science
Edith Cowan University,
100 Joondalup Drive, Joondalup WA 6027
(W) 08 6304 5097,
Email: j.trezise@ecu.edu.au
Pre-exercise Medical Questionnaire

The following questionnaire is designed to establish a background of your medical history, and identify any injury and/ or illness that may influence your testing and performance.

Please answer all questions as accurately as possible, and if you are unsure about anything please ask for clarification. All information provided is strictly confidential.

Personal Details

ID Code:______________________________________________

Date of Birth (DD/MM/YYYY):__________________

PART A

1. Are you a regular smoker or have you quit in the last 6 months?  Y  N  _______________

2. Did a close family member have heart disease or surgery, or stroke before the age of 60 years?  Y  N  Unsure _______________

3. Do you have, or have you ever been told you have blood pressure above 140/90 mmHg, or do you current take blood pressure medication?  Y  N  Unsure _______________

4. Do you have, or have you ever been told you have, a total cholesterol level above 5.2 mmol/L (200 mg/dL)?  Y  N  Unsure _______________
5. Is your BMI (weight/height²) greater than 30 kg/m²?  
   Y  N  Unsure ________________

**PART B**

1. Have you ever had a serious asthma attack during exercise?  
   Y  N  ______________________

2. Do you have asthma that requires medication?  
   Y  N  ______________________

3. Have you had an epileptic seizure in the last 5 years?  
   Y  N  ______________________

4. Do you have any moderate or severe allergies?  
   Y  N  ______________________

5. Do you, or could you reasonably, have an infectious disease?  
   Y  N  ______________________

6. Do you, or could you reasonably, have an infection or disease that might be aggravated by exercise?  
   Y  N  ______________________

**PART C**

1. Are you currently taking any prescribed or non-prescribed medications?  
   Y  N  ______________________
2. Have you had, or do you currently have, any of the following?

If YES, please provide details

<table>
<thead>
<tr>
<th>Condition</th>
<th>Y</th>
<th>N</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rheumatic fever</td>
<td>Y</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>Heart abnormalities</td>
<td>Y</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>Y</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>Epilepsy</td>
<td>Y</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>Recurring back pain that would make exercise problematic, or where exercise may aggravate the pain</td>
<td>Y</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>Recurring neck pain that would make exercise problematic, or where exercise may aggravate the pain</td>
<td>Y</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>Any neurological disorders that would make exercise problematic, or where exercise may aggravate the condition</td>
<td>Y</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>Any neuromuscular disorders that would make exercise problematic, or where exercise may aggravate the condition</td>
<td>Y</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>Recurring muscle or joint injuries that would make exercise problematic, or where exercise may aggravate the condition</td>
<td>Y</td>
<td>N</td>
<td></td>
</tr>
</tbody>
</table>
A burning or cramping sensation in your legs when walking short distances

Y  N  ____________________________

Chest discomfort, unreasonable breathlessness, dizziness or fainting, or blackouts during exercise

Y  N  ____________________________

PART D

Have you had flu in the last week?  Y  N  ____________________________

Do you currently have an injury that might affect, or be affected by, exercise?

Y  N  ____________________________

*Is there any other condition not previously mentioned that may affect your ability to participate in this study?

Y  N  __________________________________________________________

PART E

Have you ever been told by a medical practitioner or health care professional that you have a nerve or muscle disorder?

Y  N  ____________________________

Do you have a heart pacemaker?

Y  N  ____________________________

Do you have any metallic implants (e.g. bone pins)?

Y  N  ____________________________
Declaration (to be signed in the presence of the researcher)

I acknowledge that the information provided on this form, is to the best of my knowledge, a true and accurate indication of my current state of health.

Participant

Name:________________________ Date (DD/MM/YYYY):_______________

Signature:____________________________

Researcher:

Signature:____________________________

Date (DD/MM/YYYY):_________________

Practitioner (only if applicable)

I, Dr _______________________________________ have read the medical questionnaire and information/ consent form provided to my patient Mr ____________________________________, and clear him medically for involvement in exercise testing.

Signature:____________________________

Date (DD/MM/YYYY):___________________
Appendix 2.

2A Calculated daily energy expenditure

Calculated daily energy expenditure during work and recreational time using METs (Metabolic equivalents)

One MET is the energy expended at rest, so the METs/hr score is a measure of the physical intensity of daily activities. To qualify as an untrained participant, the participant must not be participating in regular exercise. Table A.1 displays the daily energy expended by two people calculated using MET values per hour. The only difference between the two people is there mode of transport to university. While a 30-min cycle to school for this person may be considered a mode of transport and not regular exercise, they would not be classified as untrained due to their energy expenditure levels being > 30 METs/day.

Table A.1. Metabolic energy equivalent calculation for two people attending university (MET/hr).

<table>
<thead>
<tr>
<th>Activity (Person One)</th>
<th>MET</th>
<th>Activity (Person Two)</th>
<th>MET</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dressing, bathing, breakfast</td>
<td>2.0</td>
<td>Dressing, bathing, breakfast</td>
<td>2.0</td>
</tr>
<tr>
<td>Riding to university on the train</td>
<td>0.5</td>
<td>Cycle to university</td>
<td>5.0</td>
</tr>
<tr>
<td>Walk to university</td>
<td>0.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sitting at computer from 9am-5pm</td>
<td>14.4</td>
<td>Sitting at computer from 9am-5pm</td>
<td>14.4</td>
</tr>
<tr>
<td>Incidental walking on the job</td>
<td>1.2</td>
<td>Incidental walking on the job</td>
<td>1.2</td>
</tr>
<tr>
<td>Walk during lunch break</td>
<td>0.6</td>
<td>Walk during lunch break</td>
<td>0.6</td>
</tr>
<tr>
<td>Walk to the train</td>
<td>0.6</td>
<td>Cycle home from university</td>
<td>5.0</td>
</tr>
<tr>
<td>Riding home on the train</td>
<td>0.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Household chores</td>
<td>1.3</td>
<td>Household chores</td>
<td>1.3</td>
</tr>
<tr>
<td>Cooking and eating dinner</td>
<td>1.0</td>
<td>Cooking and eating dinner</td>
<td>1.0</td>
</tr>
<tr>
<td>Leisure: reading, TV, coffee date</td>
<td>3.0</td>
<td>Leisure: reading, TV, coffee date</td>
<td>3.0</td>
</tr>
<tr>
<td>Leisurely walk after dinner</td>
<td>1.7</td>
<td>Leisurely walk after dinner</td>
<td>1.7</td>
</tr>
<tr>
<td>Preparing for bed</td>
<td>1.5</td>
<td>Preparing for bed</td>
<td>1.5</td>
</tr>
<tr>
<td>Total METs/hr</td>
<td>28.9</td>
<td>Total METs/hr</td>
<td>36.7</td>
</tr>
</tbody>
</table>

MET values adapted from Ainsworth et al (2000) [163]
The equation to determine contribution of knee extensor and knee flexor torque to total knee extension was developed by Aagaard et al. (2000) “Antagonist muscle coactivation during isokinetic knee extension” [9].

A change has been made on line [6] where ‘\(K_2 = A_1 + K_2 \cdot B_1\)’ becomes ‘\(K_2 = A_1 + K_1 \cdot B_1\)’. 

**Appendix**

The extension moment \(M_1\) measured during concentric quadriceps action (quadriceps Q: agonist; hamstring H: antagonist) is determined by the difference between the agonist extension moment \(M_{Q,ext}\) and antagonist flexion moment \(M_{H,flex}\) (\(K_1\) and \(K_2\) denoting EMG-to-force constants):

\[
M_1 = K_1 \cdot \text{EMG}_{Q,agon} - K_2 \cdot \text{EMG}_{H,antag} \quad [1]
\]

Correspondingly, the force moment \(M_2\) measured in knee extension movements involving eccentric hamstring action (H agonist, Q antagonist) is determined by

\[
M_2 = K_2 \cdot \text{EMG}_{H,agon} - K_1 \cdot \text{EMG}_{Q,antag} \quad [2]
\]

The pair of equations [1] and [2] can be solved for any knee angle \(\Theta\) as it consists of two equations with two unknown variables: the EMG-to-force constants \(K_1\) and \(K_2\). Dividing equation [2] with \(\text{EMG}_{H,agon}\) yields

\[
\frac{M_2}{\text{EMG}_{H,agon}} = \frac{K_2 - K_1 \cdot \text{EMG}_{Q,antag}}{\text{EMG}_{H,agon}} \quad [3]
\]

Correspondingly, dividing equation [1] with \(\text{EMG}_{H,antag}\) and rearranging gives

\[
\frac{M_1}{\text{EMG}_{H,antag}} = -K_2 + K_1 \cdot \text{EMG}_{Q,agon} \quad [4]
\]

Subsequently, adding left and right sides of eqs. [3] and [4], respectively, and isolating \(K_1\) results in the following solution for [1] and [2]:

\[
K_1 = \frac{A_1 + A_2}{B_2 - B_1} \quad [5]
\]

\[
K_2 = A_1 + K_1 \cdot B_1 \quad [6]
\]

Where

\[
A_1 = M_2/\text{EMG}_{H,agon} \quad B_1 = \text{EMG}_{Q,antag}/\text{EMG}_{H,agon}
\]

\[
A_2 = M_1/\text{EMG}_{H,antag} \quad B_2 = \text{EMG}_{Q,agon}/\text{EMG}_{H,antag}
\]

At any given knee angle \(\Theta\), calculating \(K_1\) and \(K_2\) according to [5] and [6] yields quadriceps and hamstring muscle moments \(M_{Q,ext}\) and \(M_{H,flex}\):

\[
M_{Q,ext}(\Theta) = K_1(\Theta) \cdot \text{EMG}_{Q}(\Theta)
\]

\[
M_{H,flex}(\Theta) = K_2(\Theta) \cdot \text{EMG}_{H}(\Theta)
\]

The fact that \(K_1\) and \(K_2\) were determined at identical joint angular velocity, at specific contraction modes (\(K_1\): concentric Q, \(K_2\): eccentric H) and separately for every 0.05° knee joint angle between 10° and 90°, ensures that the specificity of 1) muscle length, 2) muscle contraction velocity, 3) internal muscle lever arm length and 4) contraction mode is inherent in the EMG-to-force relationships depicted. Prior to being fed to the set of equations the EMG signal of each muscle was normalized relative to its average EMG amplitude obtained during agonist contraction, thereby avoiding that the various EMG signals should contribute to eqs. [1] and [2] in proportion to their numeric EMG amplitude size (in mV).
Figure 2C. Distribution of change in maximal isometric (a), concentric (b) and eccentric (c) torque following training (left). Based on a non-normal distribution, isometric and eccentric torques were log transformed (right).