PREEXERCISE STRATEGIES: THE EFFECTS OF WARM-UP, STRETCHING, AND MASSAGE ON SYMPTOMS OF ECCENTRIC EXERCISE-INDUCED MUSCLE DAMAGE AND PERFORMANCE

Pornratshanee Weerapong

BSc (Physical Therapy), MSc (Physiology of Exercise)

A thesis submitted to Auckland University of Technology

in fulfillment of the degree of

Doctor of Philosophy

February 2005

TABLE OF CONTENTS

Chapter 3 Stretching: mechanisms and benefits on performance and injury prevention 47

ATTESTATION OF AUTHORSHIP

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the award of any other degree or diploma of a university or other institution of higher learning, except where due acknowledgement is made in the acknowledgements.

Chapters 2-6 of this thesis represent five separate papers that have been submitted to peer-reviewed journals for consideration for publication. In each of these submitted papers, 85% of work is my own work, 10% is that of Associate Professor Patria Hume, and 5% is that of Professor Gregory Kolt. Both of these coauthors have approved the inclusion of the joint work in this doctoral thesis.

……………………………………

Pornratshanee Weerapong

Date……………..

LIST OF TABLES

LIST OF FIGURES

ACKNOWLEDGMENTS

This thesis is the result of three years of work whereby I have been accompanied and supported by many people. It is a pleasant aspect that I have now the opportunity to express my gratitude to all of them.

I gratefully acknowledge the excellent support of my supervisors Associate Professor Patria Hume and Professor Gregory Kolt. Being in the first group of PhD students at Auckland University of Technology, and being the only international PhD student in the Faculty of Health, was not easy for my supervisors and I. Patria and Greg helped me pass through difficulties and were always with me when I needed assistance. Their enthusiastic and integral view on research, and their mission for providing 'only high-quality work and not less', has made a deep impression on me. I owe them lots of gratitude for showing me this way of research, and providing me with valuable comments on several versions of this thesis.

Special thanks to Professor Will Hopkins who tirelessly assisted me in shaping my research methodology and helped me with statistical analyses. His guidance had a direct impact on improving the quality of this thesis. Special thanks also to Professor Peter McNair, Dr Elizabeth Bradshaw, and Dr John Cronin for valuable advice on experimental protocol and stiffness calculation.

This research has been supported and funded by Huachiew Chalermprakiet University, Thailand (for my PhD study) and the American Massage Therapy Association Foundation through a research grant. I am also grateful to the Physical Rehabilitation Research Centre, School of Physiotherapy, Auckland University of Technology, and the New Zealand Institute of Sport and Recreation Research, Auckland University of Technology, for equipment and technical advice, and for providing me an excellent work environment during the past years. The Auckland University of Technology staff is also thanked for their assistance.

Special thanks go to all the participants who contributed their time and energy to my studies, Simon Pearson who helped me collect my data, and Karoline Cheung for her technical advice. Special thanks also go to my fellow postgraduate students who

were very supportive, especially Clara Soper and Erica Hinckson who started their PhD journey at the same time as me and were kind and very helpful friends.

I feel a deep sense of gratitude for the Hume, the Soper, and the Nicolson families for being my families when I was alone in New Zealand. Their cheerfulness helped me get through several difficult times. Special thanks to my best friends, Craig Nicolson and Pei Leong Kong, for their unconditional support.

I am very grateful to my sisters, Patcharin, Waleephan, and Patchara, and many close friends in Thailand, who were always with me even though physically we were very far apart. The happy memories of my parents still provide a persistent inspiration for my life journey. Special thanks to staff at the Faculty of Physical Therapy, Huachiew Chalermprakiet University (Thailand) for their confidence and encouragement throughout my PhD study period.

LIST OF PUBLICATIONS, CONFERENCE PRESENTATIONS, AND TECHNICAL REPORTS ARISING FROM THIS PHD THESIS

Peer-reviewed Journal Publications

Chapters 2-6 of this thesis represent individual papers that have been submitted to peer-reviewed journals for consideration for publication. These five papers are listed below.

1. Hume, P. A., Cheung, K., Maxwell, L., & Weerapong, P. (2004). DOMS: An overview of treatment strategies. *International SportMed Journal, 5,* 98-118.

2. Weerapong, P., Hume, P. A., & Kolt, G. S. The effects of active dynamic warm-up, passive dynamic stretching, and massage on stiffness, range of motion, maximum voluntary isometric strength, and soreness in hamstring muscles before and over fours days after eccentric exercise. *Manuscript submitted for publication.*

3. Weerapong, P., Hume, P. A., & Kolt, G. S. (2004). A comparison of preexercise interventions (dynamic stretching and massage) on leg stiffness and jumping and sprinting performance. *Manuscript submitted for publication*.

4. Weerapong, P., Hume, P. A., & Kolt, G. S. (2004). Preventative strategies for exercise-induced muscle damage. *Critical Reviews in Physical and Rehabilitation Medicine, 16*, 133-150.

5. Weerapong, P., Hume, P. A., & Kolt, G. S. (2004). Stretching: mechanisms and benefits on performance and injury prevention. *Physical Therapy Reviews, 9,* 189-206.

6. Weerapong, P., Hume, P. A., & Kolt, G. S. (in press). The mechanisms of massage and benefits for performance, muscle recovery, and injury prevention. *Sports Medicine*.

Conference Presentations and Associated Publications

1. Weerapong, P., Hume, P. A., Kolt, G. S. (2004, October). Warm-up, stretching, and massage before exercise: Effects on passive stiffness and delayed-onset muscle soreness. Poster session presented at the annual meeting of the American Massage Therapy Association, Nashville, TN, USA.

2. Weerapong, P., Hume, P. A., Kolt, G. S. (2004, June). Warm-up, stretching, and massage before exercise: Effects on passive stiffness and delayed-onset muscle soreness. Poster session presented at the 51st annual meeting of the American College of Sport Medicine, Indianapolis, IN, USA.

3. Weerapong, P., Hume, P. A., Kolt, G. S. (2003, November). Preexercise strategies: The effects of warm-up, stretching, and massage on muscle properties. Preceding of the New Zealand Sports Medicine Conference (p. 138). Nelson, New Zealand: Sports Medicine New Zealand.

Technical reports

1. Weerapong, P., Hume, P. A., Kolt, G. S. (2004). Preexercise strategies: The effects of warm-up, stretching, and massage on the severity of muscle soreness. Progress report on the American Massage Therapy Association Foundation Research Grant. 1st January – 31st March 2004. Auckland, New Zealand: Auckland University of Technology, Division of Sport and Recreation.

2. Weerapong, P., Hume, P. A., Kolt, G. S. (2003). Preexercise strategies: The effects of warm-up, stretching, and massage on the severity of muscle soreness. Progress report on the American Massage Therapy Association Foundation Research Grant. 1st October – 31st December 2003. Auckland, New Zealand: Auckland University of Technology, Division of Sport and Recreation.

3. Weerapong, P., Hume, P. A., Kolt, G. S. (2003). Preexercise strategies: The effects of warm-up, stretching, and massage on the severity of muscle soreness. Progress report on the American Massage Therapy Association Foundation Research Grant. 1st July - 30th September 2003. Auckland, New Zealand: Auckland University of Technology, Division of Sport and Recreation.

4. Weerapong, P., Hume, P. A., Kolt, G. S. (2003). Preexercise strategies: The effects of warm-up, stretching, and massage on the severity of muscle soreness. Progress report on the American Massage Therapy Association Foundation Research Grant. 1st April - 30th June 2003. Auckland, New Zealand: Auckland University of Technology, Division of Sport and Recreation.

5. Weerapong, P., Hume, P. A., Kolt, G. S. (2003). Preexercise strategies: The effects of warm-up, stretching, and massage on the severity of muscle soreness. Progress report on the American Massage Therapy Association Foundation Research Grant. 1st January - 31st March 2003. Auckland, New Zealand: Auckland University of Technology, Division of Sport and Recreation.

6. Weerapong, P., Hume, P. A., Kolt, G. S. (2002). Preexercise strategies: The effects of warm-up, stretching, and massage on the severity of muscle soreness. Progress report on the American Massage Therapy Association Foundation Research Grant. 1st September - 31st December 2002. Auckland, New Zealand: Auckland University of Technology, Division of Sport and Recreation.

ABSTRACT

Preexercise activity is performed with an expectation to reduce the risk of eccentric exercise-induced muscle damage and enhance performance. There is a lack of information on what type of preexercise activity can benefit both eccentric exercise-induced muscle damage prevention and performance enhancement. Therefore, the aim of the thesis studies was to examine the effects of preexercise activities (warm-up, stretching, and massage) on passive stiffness and the symptoms of eccentric exercise-induced muscle damage, and leg stiffness and jumping and sprinting performance. To investigate the effects of preexercise activities on passive stiffness and the symptoms of muscle damage (Study A), thirty healthy adult males were randomly assigned into one of three groups: warm-up, stretching, or massage intervention. On Day 1, measurements (stiffness, range of motion, maximum voluntary isometric strength, and soreness) were taken before and after each intervention. On Day 2 the same procedure was repeated but with the addition of a bout of eccentric exercise immediately after the interventions. On each of Days 3-5 all measures were assessed once. Mean changes in the measures from the baseline were expressed as effect sizes (ES, fractions of baseline between-subject standard deviations). The threshold for a substantial ES was 0.20. Warm-up showed an immediate small effect in reducing stiffness (ES -0.49, 90% confidence limits ±0.26) compared to the baseline. Strength and flexion range of motion decreased immediately after eccentric exercise with all three interventions, but the decrease was least with warm-up (-0.38 ±0.32 and -0.88 ±0.67 for strength and range of motion, respectively). By Day 5, all measures returned to baseline in the warm-up group, whereas the stretching and massage groups still showed substantial negative effects. Therefore, warm-up was the best intervention for reducing passive stiffness and functional loss from eccentric exercise.

To investigate the effects of preexercise activities on leg stiffness and jumping and sprinting performance (Study B), twelve healthy adult males with no musculoskeletal problems were randomised into one of the three interventions on Day 1 and performed each intervention on each day over three weeks. The warm-up group ran at a self-selected speed for five minutes. The stretching group performed dynamic stretching exercises on their legs as demonstrated to them by a qualified sports scientist. The massage group received a 10-minute effleurage, petrissage, and tapotement intervention on their legs administered by a physiotherapist. Jumping height and sprinting time were measured three times: immediately after intervention, at 10 minutes after intervention, and 20 minutes after intervention. Leg stiffness was calculated from the force over the displacement during contact with the ground during jumping. Data were analysed by using paired-t tests and effect size statistics. The dynamic stretching group showed a small positive effect on jumping height when compared with the warm-up group. For this reason, dynamic stretching was shown to be a preferable preexercise activity when compared with massage and warm-up only.

The main finding from this PhD thesis was that active dynamic movement (warm-up in Study A and dynamic stretching in Study B) resulted in the least symptoms of muscle damage and demonstrated a potential to enhance jumping performance. Therefore, athletes and sports participants should perform active dynamic movement on muscles predominantly used in sports and exercises at the beginning of training or exercise sessions to reduce functional loss from exercise-induced muscle damage and maintain their muscle power to exercise.

CHAPTER 1

INTRODUCTION

Many coaches and athletes hold the belief, based on observations, experiences, and anecdotal evidence, that warm-up, stretching, and massage can provide several benefits to the body prior to participating in sports activities and exercise. More specifically, warm-up, stretching, and massage are used as preexercise activities to enhance performance and reduce the risk of eccentric exercise-induced muscle damage through biomechanical, neurological, and psychological mechanisms. In this PhD thesis, however, only the biomechanical mechanisms of warm-up, stretching, and massage, and their effectiveness in minimising muscle damage (caused from eccentric exercise) and improving performance (specifically jumping height and sprinting time) have been investigated.

A warm-up is believed to enhance performance by overcoming viscous resistance to movement of the joint and muscle (McNair & Stanley, 1996), to increase muscle blood flow (Tiidus & Shoemaker, 1995), and to increase neurological excitability (Shellock & Prentice, 1985). Stretching is believed to enhance performance and to prevent injuries by increasing flexibility (Smith, 1994). Massage is believed to increase muscle blood flow and increase muscle temperature (Drust, Atkinson, Gregson, French, & Binningsley, 2003), thereby enhancing performance (Cafarelli & Flint, 1992). Massage is also believed to reduce tissue adhesion and to increase muscle flexibility (Braverman & Schulman, 1999; Nordschow & Bierman, 1962), which could help to reduce injury risk factors (Gleim & McHugh, 1997). These three preparation strategies, therefore, are usually advised for general exercise, training, and competition to improve athletic performance and to reduce the risk of injuries (Cafarelli & Flint, 1992; Callagan, 1993; Shellock & Prentice, 1985; Smith, 1994).

However, recent research does not support the notion that these preparatory exercises (warm-up, stretching, and massage) benefit performance and minimise the symptoms of eccentric exercise-induced muscle damage. Warm-up is the most common practice that is generally used prior to sport events or exercises. The majority of studies have found that a warm-up has no effect on performance (Arnett, 2002; Bishop, Bonetti, & Dawson, 2001; Gray & Nimmo, 2001; Hawley, Williams, Hamling, & Walsh, 1989). It is essential to allow participants to warm-up before exercise because of ethical issues.

The literature did not show any detrimental effects of warm-up therefore warm-up was used as a control treatment.

There are three basic categories of stretching techniques commonly performed as preexercise activity: (a) the static technique – which stretches the muscle to the point of slight muscle discomfort and is held for an extended period; (b) the ballistic technique – which makes use of repetitive bouncing movements; (c) the proprioceptive neuromuscular facilitation (PNF) technique – which uses alternating contractions and stretching of muscles (Shellock & Prentice, 1985). Unfortunately, recent studies have provided evidence that these three types of stretching result in decreased force production (Fowles, Sale, & MacDougall, 2000; Kokkonen, Nelson, & Cornwell, 1998; Nelson, Guillory, Cornwell, & Kokkonen, 2001; Nelson & Kokkonen, 2001) and jumping performance (Church, Wiggins, Moode, & Crist, 2001; Cornwell, Nelson, & Sidaway, 2002). Dynamic stretching – which uses functional movements for stretching - is another type of stretching that has been used before exercise and training (Shellock & Prentice, 1985). There are no data on the effects of dynamic stretching on sports performance. Therefore, one of the aims of this PhD thesis was to elucidate whether dynamic stretching was better than static stretching for enhancing performance in sport type activities of running and jumping. Moreover, there are no data on the effects of passive dynamic stretching on the symptoms of eccentric exercise-induced muscle damage. Passive dynamic movement has been found to reduce muscle passive stiffness (McNair, Dombroski, Hewson, & Stanley, 2000), which is related to the severity of muscle damage (McHugh, Connolly, Eston, Kremenic et al., 1999).

Even though the number of athletes requiring massage is increasing (Blood, 2000), the benefits of massage on athletic performance and eccentric exercise-induced muscle damage are still questioned. There were only two studies on the effects of preexercise massage on performance. Wiktorsson-Moller, Oberg, Ekstrand, & Gillquist (1983) found that between six to 15 minutes of petrissage (a massage technique that is applied with firm pressure on the tissues) reduced muscle strength. However, the tests of muscular function were not suitable for monitoring performance as there was no relationship between the training-induced changes in muscle function and the training-induced change in performance (Murphy & Wilson, 1997). Another study on the effects of preexercise massage investigated sprinters (Harmer, 1991) and reported that massage had no effects on mean stride frequencies. However, the outcome measure can be considered inappropriate as the stride frequency needs to be combined with stride length to determine performance. Therefore, there are limited studies on the effects of preexercise massage on performance. In addition, there are no data on the effects of preexercise massage on eccentric exercise-induced muscle damage prevention.

The reviews of the limited published research on the effects of warm-up, stretching, and massage on both reducing severity of muscle damage and enhancing performance are presented in Chapters 2, 3, and 4. The methodological limitations of previous research contribute to inconclusive results and difficulties in clinical application. Their limitations can be summarised as:

Previous published research in preexercise activity investigated only the effects of single preexercise activities (e.g., warm-up, stretching, massage) on severity of muscle damage or performance. Ideally, preexercise activity should be beneficial to both aspects of reducing symptoms of muscle damage and enhancing performance.

- The variation of methodologies of warm-up, stretching, and massage interventions (i.e., definitions, types, techniques, frequency, duration) need to be selected carefully to achieve the aim of research. Such variations of intervention lead to difficulty when comparing the results from different published research, and for practical application. A comparison between several preexercise activities within the same research project might help to differentiate the effects of each intervention.
- The majority of previous research on preexercise activity has been limited by not incorporating a control group, or by not controlling the intensity of eccentric exercise, or using appropriate outcome measures.
- Previous research on preexercise activity showed that some stretching and massage techniques were detrimental to performance. The mechanisms responsible for the detrimental effects on performance need to be researched as the understanding of the mechanisms will help coaches, sport and health professionals, and researchers to develop appropriate preexercise activities.
- There is limited research in various techniques of preexercise activities (e.g., specific warm-up, dynamic stretching, and petrissage and friction massage

techniques) on the biomechanical, physiological, neurological, and psychological mechanisms and effectiveness on severity of muscle damage and performance.

Research on these preexercise activities may help coaches, sport and health professionals, and researchers to select to an appropriate preexercise activity.

Based on the limitations of previous research, the immediate effects of warm-up, dynamic stretching, and massage interventions on muscle stiffness were compared. Passive stiffness was investigated as a responsible mechanism for the severity for muscle damage in Study A because previous research showed a positive relationship between passive stiffness and severity of muscle damage (McHugh, Connolly, Eston, Kremenic et al., 1999). Leg stiffness was examined as a responsible mechanism in Study B. Leg stiffness is described as a linear movement that occurs in the vertical direction during jumping (Butler, Crowell, & Davis, 2003) when the legs are modeled like spring to support the mass of the body. To increase practical application knowledge, the effectiveness of three preexercise interventions (warm-up, dynamic stretching, and massage) on the severity of muscle damage from eccentric exercise (Study A) and jumping and sprinting performance (Study B) were investigated further. The study aims and questions are summarized in Figure 1.1 for Study A and Figure 1.2 for Study B.

STUDY A (reported in Chapter 5)

The effects of active dynamic warm-up, passive dynamic stretching, and massage on stiffness range of motion, maximum voluntary isometric strength, and soreness in hamstring muscles before and over four days after eccentric exercise.

Aims:

1. To examine the possible mechanisms of three preexercise interventions (warm-up, stretching, and massage) in terms of biomechanics (evaluated by range of motion, passive muscle stiffness, and muscle strength) on the hamstring muscles in healthy adult male participants.

2. To compare the effectiveness of three preexercise interventions (warm-up, stretching and massage) in reducing the severity of muscle damage (as determined by soreness sensation, range of motion, muscle strength, and muscle stiffness) caused by eccentric exercise.

Questions:

Does preexercise intervention (warm-up, stretching, and massage):

1. Increase hamstrings range of motion as measured by a goniometer?
2. Increase muscle strength as measured by isometric contraction?
3. Reduce hamstrings passive muscle stiffness as measured by the slope of the torque-angle curve?
4. Reduce the severity of hamstrings muscle damage as measured by soreness sensation, range of motion, muscle strength, and muscle stiffness if preexercise intervention is applied prior to eccentric exercise?

Figure 1.1. Summary of the aim and questions of Study A (Chapter 5).

STUDY B (reported in Chapter 6)

A comparison of preexercise interventions (dynamic stretching, and massage) on leg stiffness and jumping and sprinting performance.

Aim:

To compare the effectiveness of three preexercise interventions (warm-up, warm-up and stretching, and warm-up and massage) on jumping and sprinting performance in healthy adult male participants.

Questions:

Does preexercise intervention (warm-up, stretching, and massage):

1. Change leg stiffness?
2. Enhance jumping height?
3. Enhance sprinting time?

Figure 1.2. Summary of the aims and questions of Study B (Chapter 6).

The results in Study A and Study B provide benefits for physical therapists, athletic trainers, athletes, and coaches for the selection of proper preexercise activities that help to minimize the severity of eccentric exercise induced muscle damage and enhance performance. The information from this thesis adds to the body of knowledge in providing research-based evidence on the effects of preexercise activities (warm-up, dynamic stretching, and massage) on passive stiffness and severity of eccentric exercise- induced muscle damage, and leg stiffness and performance of jumping and sprinting performance.

There are seven chapters in this PhD thesis. Chapters 2 to 6 have been submitted to peer-reviewed journals as separate papers for publication. The three literature reviews (chapters 2-4) were accepted for publication in Sports Medicine, Physical Therapy Reviews, and Critical Review in Physical and Rehabilitation Medicine, respectively. Chapter 2 is a review of the literature on the mechanisms and benefits of massage on performance and the risk of injury. Chapter 3 is a review of the literature on the mechanisms and benefits of stretching on performance and the risk of injury. Chapter 4 is a review of the literature on the preventative strategies for muscle damage. Chapter 5 is an experimental study on the effectiveness of preexercise strategies (warm-up, stretching, and massage) on the severity of muscle damage. Chapter 6 is an experimental study on the effectiveness of preexercise strategies (warm-up, stretching, and massage) on performance. Passive stiffness and leg stiffness have been investigated as the responsible mechanisms for the severity of muscle damage and jumping and sprinting performance. Chapter 7 consists of general discussion, limitations, recommendations, and conclusions for the whole thesis.

In this PhD thesis, the term “eccentric exercise” is used to indicate the type of exercise that induces muscle damage. The term “severity of muscle damage” indicates the magnitude of the symptoms after eccentric exercise, including soreness sensation, strength and range of motion loss, swelling, and tenderness. In terms of muscle damage from eccentric exercise, soreness sensation has been the major concern in earlier research (High, Howley, & Franks, 1989; Hilbert, Sforzo, & Swensen, 2003; Johansson, Lindstrom, Sundelin, & Lindstrom, 1999) because the symptoms after eccentric exercise are usually termed “delayed-onset muscle soreness”. However, other symptoms, such as strength and range of motion loss, need to be considered as they might have more impact on athletes or patients than soreness sensation, and they may take longer to recover. The other symptoms of muscle damage such as strength and range of motion loss, and stiffness should be considered along with soreness sensation after exercise-induced muscle damage.

There have been no previous publications on the effects of preexercise massage and dynamic stretching exercise on the severity of muscle damage and performance. Therefore it was difficult to calculate the sample size from power analysis. There are two reasons ten participants per group were used in Study A and 12 participants in Study B. Pilot work showed high reliability (intraclass correlation coefficients (ICC > 0.90) for all parameters in Study A (Appendix H) and Study B (Appendix Q). Study A was designed to compare three interventions. The minimal number of participants for the controlled trial was ten participants per group or per treatment. Participants were asked to come to the laboratory at the same time each day for five consecutive days. The measurements took two hours for the first day and one hour for the following days. The exercise protocol aimed to damage muscle and caused soreness, strength and range of motion loss on the following days. Therefore, thirty participants were recruited because of these ethical and practical limitations. Study B used a crossover design where participants received each intervention each day. Within subject comparisons were made. The crossover design helped to minimise the individual response effects on treatment. Participants were asked to come to the stadium for four consecutive weeks. The measurement took one hour each day. The major problem for data collection of Study B was access to, and cost for, the stadium. The three interventions were arranged in order resulting in six groups (warm-up-stretching-massage, warm-up-massage-stretching, stretching-warm-up-massage, stretching-massage-warm-up, massage-warm-up-stretching, massage-stretching-warm-up) to minimise learning effects. Therefore, 12 participants were recruited because of these practical limitations.

The results from this PhD thesis were analysed using effect size statistics to present the magnitude of the effects of the treatment (warm-up, stretching, and massage). The precision of the estimate on the effect of the treatment was expressed using 90% confidence limits (Sterne & Smith, 2001) derived from paired t-tests. The confidence intervals (CI) were used for qualifying the uncertainty in the results (Wolfe & Cumming, 2004). The CIs were chosen for statistical analysis because they show the extent to which study results are applicable in general, beyond the participants involved in the study (Wolfe & Cumming, 2004). Furthermore, 90% confidence limits represent adequate precision for making probabilistic inferences, because the probability that the true value is less than the lower confidence limit and more than the upper confidence limit are both only 0.05, which could be interpreted as *very unlikely* (Peterson, Wilson, & Hopkins, 2004). *P*-values were calculated and presented in the precise number so they could be used for CI calculation and allow the readers to have a better indication of the uncertainty of the results. *P*-values were not used to interpret data of this study because it is not clearly separate the information of the precision of the study (data were presented by CI) and the magnitude of the treatment effects (data were presented by the magnitude of effect sizes).

Limitations

There are many aspects that this PhD study has not investigated:

- There are several types of muscle stiffness (e.g., active stiffness, passive stiffness, leg stiffness, vertical stiffness). Each type of muscle stiffness depends on structures of muscle that resist the application of external force. In this PhD thesis, passive stiffness was investigated in Study A and leg stiffness was investigated in Study B. The selection of muscle stiffness for each experiment was based on a significant contribution on the outcome measures from previous research.
- Participants in this PhD study were healthy adult males. Therefore, the results from this study may not be directly applicable to professional or elite athletes or aspects of sport performance other than jumping and sprinting.

Delimitations

This PhD study was delimited by:

- Participants were allowed to become familiar with equipment and the experimental protocol to minimise learning effects.
- The severity of muscle damage depends on individual responses. The variation of individual responses was minimised by controlling the intensity of exercise at 80% of maximum voluntary isometric contraction for each participant and randomising participants into each group.
- There are several types of performance. Jumping and sprinting were selected because they are generally performed in the majority of sports and they demonstrate functional movement.

CHAPTER 2

THE MECHANISMS OF MASSAGE AND BENEFITS FOR PERFORMANCE, MUSCLE RECOVERY, AND INJURY PREVENTION

Weerapong, P., Hume, P. A., & Kolt, G. S. (in press). The mechanisms of massage and benefits for performance, muscle recovery, and injury prevention. *Sports Medicine*.

2.1 Abstract

Many coaches, athletes, and sports medicine personnel hold the belief, based on observations and experiences, that massage can provide several benefits to the body such as increased blood flow, reduced muscle tension and neurological excitability, and an increased sense of well-being. Massage can produce mechanical pressure which is expected to increase muscle compliance resulting in increased range of joint motion, decreased passive stiffness, and decreased active stiffness (biomechanical mechanisms). Mechanical pressure might help to increase blood flow by increasing the arteriolar pressure, as well as increasing muscle temperature from rubbing. Depending on the massage technique, mechanical pressure on the muscle is expected to increase or decrease neural excitability as measured by the H-reflex (neurological mechanisms). Changes in parasympathetic activity (as measured by heart rate, blood pressure, and heart rate variability) and hormonal levels (as measured by cortisol levels) following massage result in a relaxation response (physiological mechanisms). A reduction in anxiety and an improvement in mood state also cause relaxation (psychological mechanisms) after massage. Therefore, these benefits of massage are expected to help athletes by enhancing performance and reducing injury-risk. However, limited research has investigated the effects of preexercise massage on performance and injury prevention. Massage between events is widely investigated because it is believed that massage might help to enhance recovery and prepare athletes for the next event. Unfortunately, very little scientific data has supported this claim. The majority of research on psychological effects of massage has concluded that massage produces positive effects on recovery (psychological mechanisms). Postexercise massage has

been shown to reduce the severity of muscle soreness but massage has no effects on muscle functional loss. Notwithstanding the belief that massage has benefits for athletes, the effects of different types of massage (e.g., petrissage, effleurage, friction) or the appropriate timing of massage (preexercise versus postexercise) on performance, recovery from injury, or as an injury prevention method are not clear. Explanations are lacking, as the mechanisms of each massage technique have not been widely investigated. Therefore, this article discusses the possible mechanisms of massage and provides a discussion of the limited evidence of massage on performance, recovery, and muscle injury prevention. The limitations of previous research are described and further research is recommended.

2.2 Introduction

Massage has been used for rehabilitation and relaxation for thousands of years around the world. Recent research from the United Kingdom showed that in the past 11 years, massage treatment was administered for approximately 45% of the total time in physiotherapy treatment (Galloway, Watt, & Sharp, 2004). Massage is used in general approaches, such as preparation for competition, between competitions, and in assisting recovery from competition, rather than treatment for specific problems (Galloway et al., 2004). The large proportion of massage application in sports events is due to many coaches and athletes holding the belief, based on observations and experiences, that massage can provide several benefits to the body such as increased blood flow, reduced muscle tension and neurological excitability, and an increased sense of well-being. There is limited scientific evidence, however, to support the use of massage for enhancing performance, enhancing recovery from injury, or for preventing muscular injury. There is a relative lack of good studies or information on massage and its potential to influence muscle recovery, injury prevention and physical performance. Many claims are made about massage, but few have any empirical data to back them up and what little data there is tends to point more to the limitations of massage than to any significant effects. Rather the possible mechanisms and the effects of massage usually result from authors’ speculations based on general biomechanical, physiological, or psychological knowledge. More scientific data on the benefits of massage is required.

This literature review outlines possible mechanisms of massage (biomechanical, physiological, neurological, and psychophysiological), and evaluates the evidence for the benefits of massage in improving performance, enhancing recovery, or preventing muscular injury.

Literature was located using three computer databases (PubMed, SPORT Discus, and ProQuest 5000 International) in addition to manual journal searches. The computer databases provided access to biomedical and sport-oriented journals, serial publications, books, theses, conference papers, and related published since 1964. The key search phases used included: sport massage, massage, performance, sport injury, delayed onset muscle soreness, injury prevention, range of motion, and muscle stiffness. Articles not published in English nor in scientific journals, nor articles that focused on a specific type of massage (such as connective tissue massage, acupressure), nor articles that focused on the effects of massage in special populations, were not included in this review. The criteria for inclusion were:

- The article must have used normal, healthy participants. Age, gender, and fitness differences were not excluding factors.
- The article must have used Swedish-type massage as an intervention. The massage technique such as effleurage and petrissage were not excluding factors.
- The article may have discussed the possible mechanisms of massage in relation to biomechanical and/or neuromuscular properties of muscle, sport performance, rate of injury, and muscle soreness.
- The article may have been a review of previous research

2.3 Types of massage

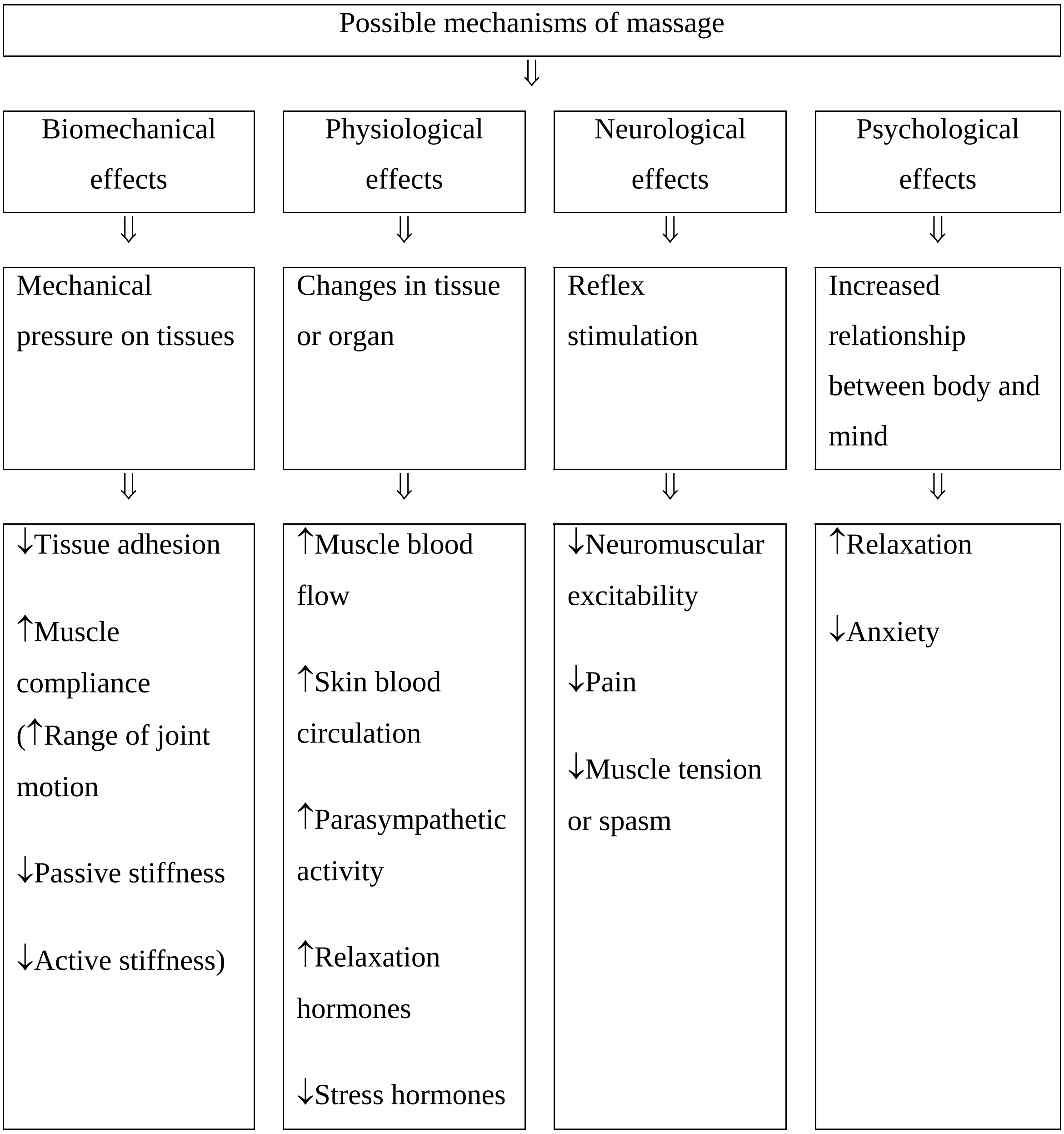
Massage has been defined as “a mechanical manipulation of body tissues with rhythmical pressure and stroking for the purpose of promoting health and well-being”(Cafarelli & Flint, 1992) (pp. 1). Classic Western massage, or Swedish massage, is the most common form of massage currently used around the world for athletes with purported clinical advantages (see Table 2.1). There are a number of techniques in existence, and their use depends on the experience of the therapist and the intended clinical advantage desired. The majority of research has used a combination of Western techniques to investigate the effects of massage, with a few studies having used other techniques such as myofascial trigger point massage.

Table 2.1 Summary of classic Western massage techniques.

Techniques	Definition	Application	Suggested clinical advantage
Effleurage	Gliding or sliding movement over the skin with a smooth continuous motion (Tappan & Benjamin, 1998).	• Beginning of a session. • During a break after applying a specific technique. • End of each session.	• Stimulate the parasympathetic nervous system and evoke the relaxation response. • Enhance venous return.
Petrissage	Lifting, wringing, or squeezing of soft tissues in a kneading motion, or pressing or rolling of the tissues under or between the hands (Tappan & Benjamin, 1998).	• Following effleurage.	• Mobilize deep muscle tissue or the skin and subcutaneous tissue. • Increase local circulation. • Assist venous return.
Friction	An accurately delivered penetrating pressure applied through the fingertips (Goats, 1994).	• Used for a specific purpose such as to reduce muscle spasm.	• Treat muscle spasm or break up adhesions from old injuries.
Tapotement	Various parts of the hand striking the tissues at a fairly rapid rate (De Domenico & Wood, 1997).	• Finishing a section of the body. • Before and during a competition.	• Stimulate the tissues either by direct mechanical force or by reflex action.

2.4 The possible mechanisms of massage

The effects of massage are most likely produced by more than one mechanism (Braverman & Schulman, 1999; De Domenico & Wood, 1997; Goats, 1994; Tappan & Benjamin, 1998). A theoretical model of how biomechanical, physiological, neurological, and psychological mechanisms (Bell, 1964; Braverman & Schulman, 1999) may be affected by massage is presented in Figure 2.1. As stated earlier the majority of these mechanisms are speculated by the authors with little empirical data to support the statement. For example, it has been speculated that the possible increase in muscle blood flow, as well as the possible decrease in neuromuscular excitability resulting from mechanical pressure may be factors in any potential effectiveness of massage on muscle compliance. Speculations on the possible mechanisms are needed so further research can be developed to establish the true mechanisms and effects of massage.



↓ = decrease; ↑ = increase.

Figure 2.1 Theoretical model of the expected mechanisms of massage.

2.4.1 Biomechanical mechanisms

Massage involves the application of mechanical pressure on the muscle tissue in order to decrease tissue adhesion. Increased muscle-tendon compliance is believed to be achieved by mobilising and elongating shortened or adhered connective tissue. Improved muscle compliance results in a less stiff muscle-tendon unit (Magnusson, 1998). Biomechanically three main measures are used to assess muscle-tendon unit compliance: dynamic passive stiffness; dynamic active stiffness; and static joint end range of motion (Gleim & McHugh, 1997). A summary of the evidence for the effects of massage on muscle-tendon unit compliance as measured by passive stiffness, active stiffness, and joint range of motion is presented in Table 2.2.

2.4.1.1 Passive stiffness

Only one study (Stanley et al., 2001) examined the effects of massage on passive stiffness. A 10-minute effleurage had no significant effects on passive gastrocnemius stiffness properties when compared with a 10-minute rest. The pressure of effleurage might not have been enough to produce the mechanical effects of massage, or if effleurage could produce a reflexive response the change in muscle properties might present in the contractile elements (active muscle stiffness) rather than passive elements of muscle. Further research is needed to investigate the effects of other massage techniques (e.g., petrissage, friction), which provide more mechanical pressure on muscle, on passive properties of muscles.

2.4.1.2 Active stiffness

A search of the literature identified no research on the effects of massage on active stiffness. The level of active muscle stiffness depends on passive joint properties, the intrinsic muscle and joint properties, and the effects of stretch reflex (McNair & Stanley, 1996). Massage might be able to alter active muscle stiffness by changing the level of neurological activation. However, the optimal level of muscle stiffness that benefits both performance and injury prevention is still unknown.

Table 2.2 The effects of massage on muscle-tendon unit compliance as measured by passive stiffness, active stiffness and joint range of motion.

Reference	Trial design	Sample	Intervention	Outcome measures	Main results
Passive stiffness					
Stanley et al (2001)	RCT	19 healthy subjects	10-min effleurage on gastrocnemius.	a) Passive muscle stiffness. b) Maximum tension.	NS
Active stiffness					
No published studies to date.					
Range of motion					
Nordschow and Bierman (1962)	CCT	25 normal subjects	Swedish and Hoffa massage on the back and at the back of lower limbs for 30 min.	Finger to floor test.	S: ↑ lumbar range of motion
Wiktorsson -Moller et al. (1983)	CCT	8 healthy males	a) Warm-up. b) Legs massage (kneading for 6-15 min). c) Warm-up & massage. d) Warm-up & stretching.	a) ROM of lower extremities. b) Hamstrings and quadriceps strength.	S: ↑ ankle dorsiflexion. S: ↓ quadriceps and hamstrings strength.
Leivadi et al. (1999)	RCT	30 dance students	a) Massage (whole body –effleurage, petrissage, friction). b) Progressive muscle relaxation exercise (30 min session, twice a week, 5 consecutive weeks).	Short term: a) STAI. b) POMS. c) Salivary cortisol Long term: a) Neck, shoulder ROM.	Short term: a) Both groups S : ↓ STAI, POMS b) Massage group S: ↓ cortisol Long term: a) Massage group S: ↑ ROM.

CCT = controlled clinical trial; RCT = randomised controlled trial; CT = counterbalance trial; S = significant; NS = non-significant; ROM = range of motion; STAI = State-Trait Anxiety Inventory; POMS = Profile of Mood States; ↑ = increase; ↓ = decrease.

2.4.1.3 Joint range of motion

Static flexibility is defined as the range of motion available to a joint or series of joints (Gleim & McHugh, 1997), and is usually measured with a goniometer (Clarkson, 2000). The majority of studies that have evaluated the effects of massage on muscle and connective tissue have been based on range of motion measurement (Leivadi et al., 1999; Nordschow & Bierman, 1962; Wiktorsson-Moller et al., 1983). For example, Leivadi et al. (1999) investigated the effects of neck and back massage on neck extension and shoulder abduction after massage was applied to the posterior region of the neck. The range of neck extension motion was limited by anterior muscles and ligaments and bony contact between spinous processes (Clarkson, 2000), therefore, neck extension was not a good outcome measure for the effectiveness of massage in this study. In another study (Nordschow & Bierman, 1962), finger to floor distances increased significantly after massage of the back and lower extremities, however, this study did not provide an appropriate control group and did not blind the examiner to whether the subject had massage or control. The massage therapist measured the distance between the fingers and the floor which may have caused bias during measurement. When the effects of massage on lower extremity range of motion were compared with the other preexercise activities such as warm-up and stretching (Wiktorsson-Moller et al., 1983), massage increased only ankle dorsiflexion range of motion while stretching significantly increased all lower extremity range of motion measurements. Thus, the effectiveness of massage on range of motion is still questioned especially when compared with more economical techniques like stretching.

2.4.2 Physiological mechanisms

2.4.2.1 Increased skin and muscle temperature

Superficial skin friction increases local heating, and consequently, causes hyperemia within the massaged area. Local heating increases local blood circulation (Black, Vickerson, & McCully, 2003). There is published evidence that skin and muscle temperature increased after massage application (effleurage technique).

Longworth (1982) reported an increase in skin temperature during a six-minute back massage but skin temperature returned to baseline level after ten minutes. Recently, Drust et al. (2003) reported an increase in skin and intramuscular temperature (at 1.5 and 2.5 cm) of the vastus lateralis muscle irrespective of massage duration (5, 10 and 15 minute of effleurage). Even though massage was shown to increase skin (1982) and intramuscular temperature (Drust et al., 2003), such effects on skin and intramuscular temperature might not be relevant to muscle blood flow.

It is still questionable whether increased skin and intramuscular temperature (Drust et al., 2003) without increasing muscle blood flow (Shoemaker, Tiidus, & Mader, 1997; Tiidus & Shoemaker, 1995) and muscle compliance (Stanley et al., 2001) would be beneficial to enhance performance or prevent injuries. The other limitations evident from effleurage technique administration were that skin temperature quickly returned to baseline level (1982) and muscle temperature did not increase in deep muscle temperature (deeper than 2.5 cm) (Drust et al., 2003). This leads to the implication that massage (effleurage technique) may not be suitable as a preparation and/or preventative strategy for exercise.

2.4.2.2 Increased blood flow

One expected benefit of massage with respect to enhancement of athletic performance is the increase in blood circulation. A variety of controlled clinical trials have used venous occlusion pethysmography, the ^{133}Xenon wash out technique, or pulsed Doppler ultrasound velocimetry to examine the effects of massage on blood flow (see Table 2.3). Although several authors have agreed that massage could increase blood flow (Bell, 1964; Dubrosky, 1982, 1983; Hansen & Kristensen, 1973; Hovind & Nielsen, 1974), study results have been inconclusive largely due to their design limitations. Besides small sample size (Bell, 1964; Hansen & Kristensen, 1973; Hovind & Nielsen, 1974) most of these studies had no reported statistical analysis (Bell, 1964; Dubrosky, 1982, 1983; Hansen & Kristensen, 1973; Hovind & Nielsen, 1974), nor did they use a control group (Bell, 1964; Dubrosky, 1982, 1983; Hansen & Kristensen, 1973; Hovind & Nielsen, 1974), These limitations make it hard to differentiate the changes from normal variations. More importantly, the venous occlusion pethysmograph and ^{133}Xenon wash-out techniques used in these studies had their own limitations. The venous occlusion pethysmograph demonstrated underestimation of blood flow due to the inflation of the cuff and was very sensitive to movement artifacts (Shoemaker et al., 1997; Tiidus & Shoemaker, 1995). The changes of blood flow could not be expressed quantitatively (Hansen & Kristensen, 1973). Moreover, the venous occlusion pethysmograph technique could not be used to measure blood flow during actual massage (Tiidus, 1999). The ^{133}Xenon wash-out technique overestimated blood flow because of the local trauma from injection of the tracer (Hovind & Nielsen, 1974; Shoemaker et al., 1997; Tiidus, 1999; Tiidus & Shoemaker, 1995). Pulsed Doppler ultrasound has been used to investigate muscle blood flow and has indicated that manual massage did not affect blood flow in the muscle after treatment of the muscle (1997; 1995). However, the ultrasound used in these studies detected changes in the large artery and vein but did not detect microcirculation in muscle that could be affected by massage. In summary there is a lack of confirming evidence that massage does anything significant (with a few exceptions) for the blood flow physiological response. The only studies to look at blood flow with limited technique problems (Shoemaker et al., 1997; Tiidus & Shoemaker, 1995) have shown no change in total muscle blood flow. This indicates that there is likely little real effect of massage on muscle blood flow (microcirculatory changes excepted).

Table 2.3.. The effects of massage on blood flow.

Reference	Trial design	Samples	Intervention	Outcome measures	Main results
Venous occlusion pethysmograph					
Bell (1964)	CCT	Unknown	10-minute effleurage and kneading on calf muscle.	Venous blood flow.	↑ blood flow 2 times for 40 min.*
^{133}Xenon wash out technique					
Hansen and Kristensen (1973)	CCT	12 healthy volunteers (*n*=4 for each intervention)	a) Effleurage on calf muscle. b) Ultrasound. c) Short wave diathermy (all for 5 min).	a) Muscle blood flow on right calf. b) Subcutanous blood flow on left calf.	↑ muscle blood flow immediately and returned to baseline 2 min after massage.*

Table 2.3 (continued) The effects of massage on blood flow.

Reference	Trial design	Samples	Intervention	Outcome measures	Main results
Hovind and Nielsen (1974)	CCT	9 healthy volunteers	a) Petrissage on one calf and forearm. b) Tapotement on another calf and forearm.	Muscle blood flow.	↑ muscle blood flow and lasted for 10 min after tapotement on both forearm and calf.*
Dubrovsky (1982)	CCT	12 class I and II athletes	Not known.	a) Muscle blood flow. b) Venous blood flow. c) Muscle tone. d) Arterial blood saturation.	↑ muscle blood flow.* ↑ venous blood flow.* ↓ muscle tone.* ↑ arterial blood saturation.*
Dubrosky (1983)	CCT	28 class II and III athletes	Classical whole body massage (stroking, rubbing, kneading, vibration) for 15-35 min.	a) Lung ventilation. b) Venous pressure. c) Microcirculation. d) Circulating blood volume.	Only stroking, rubbing, kneading: ↑muscle blood flow for more than 3 hr.*
Pulsed Doppler ultrasound velocimetry					
Tiidus and Shoemaker (1995)	CCT	9 healthy volunteers (one leg for massage, one leg for control)	10-minute effleurage on leg on 3 days before, immediately after, and repeated on day 2 and 3 after quadriceps eccentric exercise.	a) Muscle strength. b) Arterial blood velocity. c) Venous blood velocity. d) DOMS perception.	NS
Shoemaker et al. (1997)	CCT	10 healthy volunteers	5-minute effleurage, petrissage, and tapotement (5 min rest between each treatment) on right forearm and quadriceps.	a) Brachial artery. b) Femoral artery.	NS

CCT = controlled clinical trial; * = No statistical analysis; ↑ = increase; ↓ = decrease.

2.4.2.3 Hormones

Mechanical pressure of massage might stimulate parasympathetic activity as shown by reducing saliva cortisol levels (an indirect measure of parasympathetic activity). Changes in hormonal levels (serotonin and cortisol) after massage have been reported mostly in specific conditions such as patients with low back pain (Hernandez-Reif, Field, Krasnegor, & Theakston, 2001), HIV positive patients (Ironson et al., 1996), and depressed adolescent mothers (Field, Grizzle, Scafidi, & Schanberg, 1997), which are beyond the scope of this literature review. A reduction of saliva cortisol (stress hormones) in dance students has also been reported (Leivadi et al., 1999). The dancers, who were randomly assigned to massage or relaxation therapy groups, received treatment for five consecutive weeks. Saliva cortisol levels were investigated after the session on the first and last days of the study. Both groups reported less anxiety and depressed mood after their sessions, but only the massage group showed a decrease in cortisol levels. A reduced pre-participation anxiety (nervousness) after massage may have large implications for performance. The mechanisms responsible for reducing cortisol levels after massage application are still unknown as the evidence for cortisol changes is weak.

2.4.2.4 Parasympathetic activity

Massage has shown some evidence of increasing parasympathetic activity by reducing heart rate, reducing blood pressure (Corley, Ferriter, Zeh, & Gifford, 1995; Fraser & Kerr, 1993; Groer et al., 1994; Labyak & Metzger, 1997; Longworth, 1982), increasing relaxation substances such as endorphins (Kaada & Torsteinbo, 1989), and increasing heart rate variability (Delaney, Leong, Watkins, & Brodie, 2002). The majority of research in this area has been conducted in nursing using a specific sequence of massage called “back rub”, and has been performed in older people (Corley et al., 1995; Fraser & Kerr, 1993; Groer et al., 1994). One study investigated people with chronic pain (Kaada & Torsteinbo, 1989), while other studies used connective tissue massage (Kaada & Torsteinbo, 1989) and myofascial trigger point massage (Delaney et al., 2002). Therefore, only two studies, which met the criteria (participants were healthy people and Swedish massage was used as the intervention) were reviewed. The effects of back massage on several psychophysiological indices of arousal such as heart rate, blood pressure, galvanic skin response, electromyography, skin temperature, and psycho-emotional response (using the State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970)) were examined in 32 female staff and students in a nursing school (Longworth, 1982). The participants were massaged on the back for six minutes using a slow stroke technique (effleurage). Heart rate, blood pressure, and skin temperature increased after the massage, indicating an increase in autonomic arousal level. The galvanic skin response increased, indicating a lower level of sympathetic stimulation. The inconsistency in responses of the psychophysiological parameters might be due to individuals having a unique response pattern. The participants in the study were healthy females and were not in a stressful situation, so it may have been difficult to show a great relaxation response. In another study (Zeitlin, Keller, Shiflett, Schleifer, & Bartlett, 2000), there were no significant changes in blood pressure, pulse, and temperature after a 30-minute Swedish back massage in nine female medical students one day before an academic examination. Only respiratory rate was significantly reduced. These results might support the unique response pattern or simply mean that massage could activate only some parameters indicating parasympathetic responses in healthy female participants.

2.4.3 Neurological mechanisms

2.4.3.1 Neuromuscular excitability and the Hoffman reflex

Massage is believed to stimulate sensory receptors and decrease muscle tension by reducing neuromuscular excitability as measured by changes in the Hoffman reflex (H-reflex) amplitude (Morelli, Chapman, & Sullivan, 1999; Morelli, Seaborne, & Sullivan, 1990, 1991; Sullivan, Williams, Seaborne, & Morelli, 1991). H-reflex is considered to be the electrical analogue of the stretch reflex (Zehr, 2002). In a study by Morelli et al. (1990), one-hand petrissage for three to six minutes decreased H-reflex amplitude but the amplitude returned to baseline levels immediately after the termination of massage. The reduction of H-reflex amplitude was considered to be due to a decrease in spinal reflex excitability (Morelli et al., 1991) and showed specificity to the muscle group that had received the massage (Sullivan et al., 1991). The inhibitory effects of massage on the soleus H-reflex amplitude did not originate from mechanical stimulation of cutaneous mechanoreceptors (Morelli et al., 1999). Therefore, the inhibitory effects of massage might originate from muscle or other deep tissue mechanoreceptors.

The potent inhibitory effects of massage on neuromuscular excitability might be one of the explanations for the reduction of muscle tension or spasm after massage application. However, the reduction of H-reflex after massage (petrissage technique) might not be the reason for reduction of muscle strength as reported by Wiktorsson-Moller et al. (1983) (see the effects of massage on performance section) because the H-reflex amplitude returned to baseline levels immediately after massage termination (Morelli et al., 1990). Therefore, further research is needed to investigate the relationship between the neurological effects of massage and performance, and the effects of other types of massage application on neuromuscular excitability.

2.4.3.2 Pain and muscle spasm

Massage has been applied in order to relieve pain (Gam et al., 1998; Hernandez-Reif et al., 2001; Leivadi et al., 1999; Pope et al., 1994; Puustjarvi, Airaksinen, & Pontinen, 1990). The possible responsible mechanisms are neurological (gate-control theory), physiological (biochemical substances), and mechanical (realignment of muscle fibres). Massage may reduce pain by activating the neural-gating mechanism in the spinal cord. Tactile information from massage might stimulate large fast nerve fibres and then, block the smaller, slower nerve fibres that detect pain. This effect presumably results from local lateral inhibition in the spinal cord (Guyton & Hall, 2000) and explains why touching the painful area is an effective strategy for relieving pain. However, there are no objective data to support this idea. Massage can increase biochemical substances such as serotonin (Leivadi et al., 1999), which is a neurotransmitter that plays a role in reducing pain (Guyton & Hall, 2000).

Physiotherapists usually use massage to break the vicious cycle that causes muscle spasm, and consequently, muscle pain. Muscle spasm causes muscle pain directly by stimulating mechanosensitive pain receptors or indirectly by compressing the blood vessels resulting in ischemia (Guyton & Hall, 2000). Massage might help to rearrange muscle fibres and increase microcirculation. The realignment of fibres helps to reduce muscle spasm that stimulates pain receptors and helps to reduce the pressure on blood vessels. The increase in blood microcirculation helps to increase nutrition to the damaged area. However, there is no scientific evidence to support these ideas because massage is unlikely to increase muscle blood flow and there is no published study on the effects of massage on the realignment of fibres.

2.4.4 Psychophysiological mechanisms

Various mechanisms for the cause of the relaxation response resulting from massage have been proposed. These include an increase of plasma endorphins (Kaada & Torsteinbo, 1989), decreased arousal level (Longworth, 1982), decreased stress hormone levels (Field et al., 1997; Hernandez-Reif et al., 2001; Ironson et al., 1996; Leivadi et al., 1999), or an activation of the parasympathetic response (Delaney et al., 2002; Labyak & Metzger, 1997; Lund, Lundberg, Kurosawa, & Uvnas-Moberg, 1999). This section will focus on the effects of Swedish massage on psycho-emotional responses in normal populations (see Table 2.4 for a summary of studies).

2.4.4.1 Anxiety

The majority of research in psychological area has reported that massage provided positive effects on anxiety. Most studies have used the State-Trait Anxiety Inventory (STAI) (Spielberger et al., 1970) to measure anxiety. Studies of the effects of massage on anxiety, however, had several limitations such as no control group (Zeitlin et al., 2000), inappropriate control group (Leivadi et al., 1999), and small sample size (Zeitlin et al., 2000). For example, the anxiety levels after being massaged on the day before an academic examination were assessed for nine medical students (Zeitlin et al., 2000). The mean STAI score decreased significantly after the massage intervention and the respiratory rate was also significantly decreased between pre- and post-massage measures. The small sample size and lack of a control group make the effectiveness of acute single massage treatments unclear.

Table 2.4 The effects of massage on psychological variables.

Reference	Trial design	Samples	Intervention	Outcome measures	Main results
Anxiety					
Zeitlin et al. (2000)	CCT	9 female medical students	Whole body Swedish massage (effleurage, petrissage, friction) for 1 hr.	a)Vital signs. b) STAI. c) VAS. d) WBC. e) T-cell. f) NKCA.	S: ↓ RR, WBC, T-cell. S: ↑ NKCA.
Leivadi et al. (1999)	RCT	30 dance students	a) Massage (whole body –effleurage, petrissage, friction) b) Progressive muscle relaxation exercise (30 min session, twice a week, 5 consecutive weeks).	Short term: a)STAI. b) POMS. c) VITAS. d) Salivary cortisol. Long term: Neck, shoulder ROM.	Short term: Both groups S: ↓ STAI, POMS b) Massage group S : ↓ cortisol Long term: Massage group S: ↑ROM.
Relaxation					
Weinberg et al. (1988)	RCT	279 university students	a) Massage (full body Swedish massage) (*n*=40). b) Swimming (*n*=39). c) Jogging (*n*=47). d) Racquet ball (*n*=52). e) Tennis (*n*=45). f) Rest (*n*=56).	a) POMS. b) STAI. c) Thayer’s adjective checklist.	S: ↑ POMS, STAI, Thayer’s adjective checklist.

Table 2.4 (continued) The effects of massage on psychological variables.

Reference	Trial design	Samples	Intervention	Outcome measures	Main results
Perceived recovery					
Hemmings (2000a)	CBD	9 boxers	a) Whole body massage (effleurage, petrissage). b) Touching control. c) Rest control (all for 20 min).	a) Perceived recovery. b) Saliva flow rate.	S: ↑ perceived recovery.
Hemmings et al. (2000)	CBD	8 amateur boxers	a) Massage (20 min effleurage & petrissage whole body). b) Passive rest.	a) Heart rate. b) Blood lactate. c) Blood glucose. d) Perceived recovery scale. e) Boxing performance.	NS: heart rate blood glucose, blood lactate, boxing performance. S: ↑ perceived recovery.

CCT = controlled clinical trial; RCT = randomised controlled trial; CT = counterbalance trial
S = significant; NS = nonsignificant; RR = respiratory rate; WBC = white blood cell; NKCA = natural killer cell activity; STAI = State-Trait Anxiety Inventory; VAS = Visual Analogue Scale; POMS = Profile of Mood States;↑ = increase; ↓ = decrease.

The long term effects of a 30-minute massage, twice a week for four consecutive weeks were examined in 30 dance students (Leivadi et al., 1999). A progressive relaxation therapy group was used for comparison. Both groups reported lower state anxiety levels and depressed mood subscale of the Profile of Mood States (POMS) (McNair, Lorr, & Droppleman, 1971), however, only the massage group revealed a significantly lower saliva cortisol after the massage session. The study did not state the time of blood collection, therefore the cortisol level should be interpreted carefully because of the effects of circadian rhythm (Guyton & Hall, 2000). The progressive relaxation therapy was not an appropriate control group as it was an active relaxation technique which the participants had to carry out by themselves while massage therapy was a passive relaxation technique applied by the massage therapist. The relaxation group completed their exercises at home by following a recorded tape which raises the issue of compliance to the intervention. Therefore, further studies on the effects of massage on anxiety need to provide more appropriate control groups.

2.4.4.2 Relaxation

The effects of massage on relaxation have been investigated by using valid questionnaires such as the POMS (McNair et al., 1971), however, the use of the POMS for indicating the level of relaxation is questionable as the questionnaire is composed of six scales: tension, depression, anger, vigor, fatigue, and confusion (McNair et al., 1971). Only the tension, vigor, and fatigue subscales are appropriate for relaxation measurement. POMS is also considered too long to complete (Terry, Lane, Lane, & Keohane, 1999) as there are 65-items in the original version and 72-items in the bipolar version. Therefore, the POMS is not an appropriate questionnaire from which to measure relaxation. Interestingly, there are no questionnaires available to allow a direct investigation of the level of relaxation.

Weinberg et al. (1988) reported a preliminary study on the effects of massage on mood enhancement in 183 physical education students. Massage intervention was compared with several physical activities such as swimming, jogging, tennis, and racquetball. The students completed a battery of psychological questionnaires before and after each intervention including the POMS, the State Anxiety Inventory (SAI) (Spielberger et al., 1970), and the high and general activation subscales from Thayer's adjective checklist (Thayer, 1967). Interestingly, only the massage and running groups reported a significant positive mood enhancement with significant decreases in tension, confusion, fatigue, anxiety, anger, and depression. Only the massage group showed a significant decrease in the Thayer's high activation subscale and the SAI scores. In a similar study, Hemmings (2000b) compared the psychological effects of massage, lying resting, or touching control using the POMS during boxing training. Massage application during training improved the tension and fatigue subscales of the POMS, which are applicable to measurement of relaxation. Both Weinberg et al.'s (1988) and Hemmings (2000b) studies showed significant positive psychological effects attributable to massage despite the mood state of the participants.

2.4.4.3 Recovery from fatigue

Positive perceived psychological benefits of massage using the Perceived Recovery linear scale have been shown during the recovery phase of boxing performances and after a training session (Hemmings, 2000b; Hemmings et al., 2000). Despite no changes in physiological fatigue indicators such as blood lactate and heart rate, nine boxers reported that massage positively affected the perception of recovery following boxing performance and seemed to be a useful recovery strategy. The Perceived Recovery scale is a useful questionnaire to indicate recovery because it is short and easy to understand. However, the Perceived Recovery scale has not been widely used in research studies. To date, there is no published article reporting the correlation between the Perceived Recovery scale and physiological markers of fatigue.

2.4.5 Summary of the mechanisms of massage

Massage is believed to benefit athletes by enhancing performance and recovery, as well as promoting relaxation through biomechanical, physiological, neurological, and psychological mechanisms. Despite the general belief of the benefits of massage, there are limited empirical data on possible mechanisms of massage. Mechanical pressure from massage is believed to increase muscle compliance. Several studies reported an increase in static flexibility, as measured by joint range of motion, but these studies were methodologically flawed. One study reported poor effects of massage (effleurage technique) on dynamic flexibility as measured by passive stiffness. Studies on physiological mechanisms such as the changes of blood circulation, hormonal levels, and psychophysiological parameters such as blood pressure and heart rate are still inconclusive. The explanations might be due to the unique response pattern of individuals and the variations of massage interventions (i.e., massage technique, duration of massage, and pressure of massage) used in each study. The effects of massage on neurological mechanisms have been reported to reduce the amplitude of the Hoffman reflex, however, results were limited to the petrissage technique. Many studies have reported that massage can promote relaxation by improving psychophysiological response. Therefore, further studies are needed to investigate the biomechanical, physiological, neurological, and psychological mechanisms for each massage technique. The results will help to provide appropriate massage applications for specific sports purposes.

2.5 The evidence for massage on performance, recovery, or muscular injury prevention

Sport massage has been used for centuries in an attempt to prevent and cure injuries (Braverman & Schulman, 1999; Callagan, 1993; Goats, 1994; Tiidus, 1997). Massage is considered to enhance muscle relaxation (Nordschow & Bierman, 1962; Wiktorsson-Moller et al., 1983), reduce muscle tension (Dubrosky, 1982), and soreness (Smith, Keating et al., 1994; Tiidus & Shoemaker, 1995), promote the healing process (Starkey, 1976), and consequently, improve athletic performance (Rinder & Sutherland, 1995; Viitasalo et al., 1995; Zelikovski, Kaye, Fink, Spitzer, & Shapiro, 1993). Massage is also thought to provide a soothing, sedative, invigorating feeling and can give the athlete confidence by the positive reaction that takes place within the body (Hemmings, 2000a, 2000b; Hemmings et al., 2000; Weinberg et al., 1988). Massage might be an effective way to prevent acute injuries resulting from abnormal tissue conditions (e.g., muscle tears in tight muscle) and chronic injuries caused by wear and tear (e.g., tendinosis) (Benjamin & Lamp, 1996) by rearranging the muscle fibres (Cinque, 1989). As a result of these suggested benefits, manual massage may be a useful modality to enhance performance and prevent injury for athletes who use their muscles vigorously.

Sport massage may help to optimise positive-performance factors such as healthy muscle and connective tissues and normal range of motion (Benjamin & Lamp, 1996). Massage is used to minimize negative-performance factors such as dysfunctional muscle and connective tissue, restricted range of motion, and pain and anxiety (Benjamin & Lamp, 1996). Therefore, preventive massage is commonly recommended to help athletes prepare both physically and mentally for a forthcoming event (Tappan & Benjamin, 1998). In addition, sports massage is believed to decrease injury-potential factors. Even though massage has benefited several injury-risk factors such as increased range of motion (Leivadi et al., 1999; Nordschow & Bierman, 1962; Wiktorsson-Moller et al., 1983), reduced pain (Danneskiold-Samsoe, Christiansen, Lund, & Anderson, 1982; Gam et al., 1998), and anxiety (Leivadi et al., 1999; Weinberg et al., 1988), there have been no intervention studies to assess the effects of these possible injury prevention strategies. There is no clear evidence that massage can actually improve performance, enhance recovery, or prevent muscular injury. In addition the cost to benefit ratio of massage compared with other methods such as jogging or stretching has not yet been investigated.

2.5.1 The effects of massage on performance

Sport massage is used both pre- and post-event in an attempt to increase athletes' performance, overcome fatigue, and help recovery (Callagan, 1993). An increase in muscle blood flow would hasten the delivery of oxygen, increase muscle temperature, and buffer blood pH which would then aid in the performance of exercise (Cafarelli & Flint, 1992). Increased muscle blood flow, theoretically, should help to remove waste products after exercise and should enhance delivery of protein and other nutrients needed for muscle repair (Tiidus, 1999). Increased lymph flow, could, in theory, reduce post-exercise swelling and stiffness by reducing muscle interstitial content and thereby reduce muscle discomfort (Tiidus, 1997). However, there are no data to support these ideas, and the few studies on massage and blood flow has shown no increases in blood flow.

A search of the literature identified only two studies on the effects of preexercise massage on performance. Wiktorsson-Moller et al. (1983) found that 6-15 minutes of petrissage, with the aim of promoting relaxation and comfort, reduced muscle strength. However, Wiktorsson-Moller et al. (1983) used isokinetic movement to test muscle strength. Research has shown that the tests of muscular function were not suitable to monitor performance (Murphy & Wilson, 1997). There were no relationships between the percentage changes in the tests of muscular function (concentric and eccentric contraction of isoinertial and isokinetic tests) and the changes in performance (sprinting and cycling) after an 8-week weight-training programme (Murphy & Wilson, 1997). Another study on the effects of 30 minutes of preexercise whole-body Swedish massage (including effleurage, petrissage and tapotement) in 14 sprinters (Harmer, 1991) showed that mean stride frequencies were not significantly different between massage and control groups. However, it should be noted that the highest absolute stride frequency was obtained in the trial immediately following massage. Stride frequency needs to be combined with stride length to determine performance. Therefore, the effects of preexercise massage on performance are still inconclusive due to the lack of well-controlled studies.

2.5.2 The effects of massage on recovery

2.5.2.1 Performance recovery

It is believed that one of the greatest advantages of sport massage is to overcome fatigue and reduce recovery time, especially during periods of competition, and consequently, enhance performance at the next event. Even though many elite athletes believe that massage is an important part of their success (Cinque, 1989; Samples, 1987), the effects of massage itself are still questioned. Massage can improve some physiological markers (Balke, Anthony, & Wyatt, 1989) but some studies have shown no effect on any recovery parameters (Hemmings et al., 2000). A summary of the effects of massage on performance recovery is presented in Table 2.5.

Table 2.5 The effects of massage on performance recovery.

Reference	Trial design	Samples	Intervention	Outcome measures	Main results
Balke et al (1989)	CT	7 healthy subjects	15-20 min a) Manual massage. b) Mechanopercussive massage.	a) Max MET. b) Max HR. c) Max BP. d) Leg muscle endurance. e) Leg muscle strength.	Both types of massage can reduce both physiological and muscular fatigue.
Rinder and Sutherland (1995)	CT	20 healthy subjects	a) Massage (effleurage, petrissage on both legs). b) Rest (for 6 min).	Maximal number of leg extensions against half maximum lift leg extension.	S: ↑ number of leg extensions more than control group.

Table 2.5 (continued) The effects of massage on performance recovery.

Reference	Trial design	Samples	Intervention	Outcome measures	Main results
Monedero and Donne (2000)	CT	18 male cyclists	a) Passive recovery. b) Active recovery. c) Massage (effleurage, stroking, tapotement on legs). d) Combined treatment (all for 15 min).	a) Performance time. b) Blood lactate. c) Heart rate.	S: a) Combined treatment in ↑ performance time b) Combined treatment & active recovery in ↑ blood lactate removal.
Boone and Cooper (1995)	CT	10 healthy subjects	a) Massage of lower extremities. b) Rest (all for 30 min).	a) $\dot{V}O_{2.}$ b) HR. c) SV. d) Q. e) $(a\text{-}v)O_{2.}$	NS
Hemmings et al. (2000)	CT	8 amateur boxers	a) Massage (20 min effleurage & petrissage whole body). b) Passive rest.	a) HR. b) Blood lactate. c) Blood glucose. d) Perceived recovery scale. e) Boxing performance.	NS: heart rate, blood glucose, blood lactate, boxing performance. S: ↑ perceived recovery.

CCT = controlled clinical trial; RCT = randomised controlled trial; CT = counterbalance trial; S = significant; NS = non-significant; MET = metabolic equivalent; HR = heart rate; BP = blood pressure; $\dot{V}O_2$ = maximum oxygen consumption; SV = stroke volume; Q = cardiac output; $(a\text{-}v)O_2$ = arterial-venous oxygen difference; ↑ = increase; ↓ = decrease.

To investigate the effects of massage on recovery, several studies provided massage between sport sessions. However, there were some limitations in these studies leading to inconclusive data. For example, Monedero and Donne (2000) administered combination treatments (active exercise and massage) so the true benefits of individual massage treatments are still unclear. Some studies had problems with credible data including small sample size (Boone & Cooper, 1995; Hemmings et al., 2000) and lack of statistical analysis (Balke et al., 1989). An appropriate design (such as cross-over design), use of a control group (placebo treatment), and maximisation of motivation of participants in both control and massage groups are factors which need to be considered in massage studies in order to minimise psychological effects.

Curative massage can facilitate soft tissue healing in a number of ways. Massage may help reduce both primary oedema and the possibility of secondary oedema caused by the pressure of increased fluid in the area of trauma (Braverman & Schulman, 1999; Starkey, 1976). Starkey (1976) found that combination treatments including cold, exercise, and mechanical massage could reduce total time lost from practice by approximately two days when compared to the normal ice, compression, and elevation treatment. Unfortunately, the published report showed neither the results section nor the statistical analysis. Therapists apply massage to the injured area because they expect massage to improve blood circulation to the injured area and, consequently, to help enhance healing. The mechanical pressure from massage is generally used to treat adherent or contracted connective tissue in order to restore fibres to a more normal alignment. Nevertheless, there are no data to support these suggested mechanisms.

2.5.2.2 Blood lactate removal

Blood lactate has been used as a marker for fatigue and recovery (Bale & James, 1991; Dolgener & Morien, 1993; Gupta, Goswami, Sadhukhan, & Mathur, 1996; Hemmings et al., 2000; Monedero & Donne, 2000). A summary of the effects of massage on blood lactate removal is presented in Table 2.6.

Table 2.6 The effects of massage on blood lactate removal.

Reference	Trial design	Samples	Intervention	Outcome measures	Main results
Bale and James (1991)	CT	9 male athletes.	a) Massage on leg. b) Warm down. c) Passive rest (all for 17 min).	a) Blood lactate. c) Flexibility. d) Stiffness.	S: ↓ blood lactate & stiffness at 12 hr after massage treatment.
Dolgener and Morien (1993)	RCT	22 runners.	a) Passive recovery (*n*=7). b) Bicycle recovery (*n*=7). c) Massage recovery (*n*=8)-effleurage & petrissage on legs (all for 20 min).	Blood lactate before, 3, 5, 9, and 15, 20 min after treatment.	NS
Gupta et al. (1996)	CT	10 male athletes (all subjects performed all interventions, interval period 48 hr).	a) Passive recovery (40 min). b) Active recovery (40 min). c) Massage recovery (10 min of kneading & stroking).	a) Blood lactate. b) Gas exchange ($\dot{V}O_2$ & $\dot{V}CO_2$). c) Heart rate.	NS
Hemmings et al. (2000)	CT	8 amateur boxers.	a) Massage (20 min effleurage & petrissage whole body). b) Passive rest.	a) Heart rate. b) Blood lactate. c) Blood glucose. d) Perceived recovery scale. e) Boxing performance.	NS: heart rate, blood glucose, blood lactate, boxing performance. S: ↑ perceived recovery.

Table 2.6 (continued) The effects of massage on blood lactate removal.

Reference	Trial design	Samples	Intervention	Outcome measures	Main results
Monedero and Donne (2000)	CT	18 male cyclists.	a) Passive recovery. b) Active recovery. c) Massage (effleurage, stroking, tapotement at legs). d) Combined treatment (all for 15 min).	a) Performance time. b) Blood lactate. c) Heart rate.	S: a) Combined treatment in ↑ performance time b) Combined treatment & active recovery in ↑ blood lactate removal.

RCT = randomised controlled trial; CT = counterbalance trial; S = significant; NS = non-significant; $\dot{V}O_2$ = maximum oxygen consumption; ↑ = increase; ↓ = decrease.

Only one study has reported that massage treatment could increase blood lactate removal after strenuous exercise (Bale & James, 1991). Cool-down, however, produced a superior blood lactate removal rate than massage therapy. Other studies showed no benefit from massage (Dolgener & Morien, 1993; Gupta et al., 1996; Hemmings et al., 2000; Monedero & Donne, 2000). Therefore, there is little empirical evidence to support the effectiveness of massage for blood lactate removal despite participants reporting less fatigue after massage application (Dolgener & Morien, 1993; Gupta et al., 1996; Hemmings, 2000a, 2000b; Hemmings et al., 2000; Monedero & Donne, 2000). If an elevated muscle blood flow is the aim of treatment then light exercise would be more beneficial than massage (Shoemaker et al., 1997; Tiidus, 1997, 1999). If psychological effects of fatigue are to be considered then massage might provide some benefit. However, no studies have compared the psychological effects of massage and cool-down.

2.5.3 The effects of massage in preventing muscular injury

2.5.3.1 Delayed onset muscle soreness

Delayed onset muscle soreness (DOMS) is a very important problem for coaches and athletes because it causes chronic pain and diminishes muscle function and ability to participate in sport (Ernst, 1998). DOMS commonly occurs between 24 and 72 hours after unaccustomed eccentric exercise (Appell, Soares, & Durate, 1992; Clarkson & Sayers, 1999; Ebbeling & Clarkson, 1989; Howell, Chleboun, & Conaaster, 1993). The consequences of damage to muscle function include prolonged loss of muscle strength (Chleboun et al., 1995; Clarkson, Nosaka, & Braun, 1992; Clarkson & Sayers, 1999; McHugh, Connolly, Eston, & Gleim, 2000; McHugh, Connolly, Eston, Gartman, & Gleim, 2001), soreness sensation (Howell et al., 1993; McHugh et al., 2000; McHugh et al., 2001), decreased range of motion (Clarkson et al., 1992), increased muscle stiffness (Chleboun et al., 1995; Howell et al., 1993), increased resting metabolic rate (Dolezal, Potteiger, Jacobsen, & Benedict, 2000), and perturbed athletic performance (Harris, Wilcox, Smith, Quinn, & Lawson, 1990; Hone, Siler, & Schwane, 1990; Smith, 1992). These changes might increase the risk of sports injury.

The sequence of DOMS events consists of the mechanical stress of exercise on muscle fibres (Appell et al., 1992; Armstrong, 1984; Cheung, Hume, & Maxwell, 2003; Ebbeling & Clarkson, 1989; W. J. Evans & J. G. Cannon, 1991; Faulkner, Brooks, & Opiteck, 1993), causing sarcomeres to rupture (Friden & Lieber, 1992) followed by calcium homeostasis disturbance. The damage of sarcoplasmic reticulum or muscle membrane can increase intracellular calcium and trigger calcium-sensitive pathways (Armstrong, 1990; Armstrong, Warren, & Warren, 1991). Calpain, the calcium-activated neutral protease, plays a role in the ultrastructural muscle damage (Clarkson & Sayers, 1999). The inflammatory response to damaged muscle fibres causes a transfer of fluid and cells to the damaged tissue (Smith, 1991). The increased fluid produces swelling after injury. Neutrophils and macrophages migrate to the inflammatory sites and play a role in both the damage and repair processes (Clarkson & Sayers, 1999). The exact mechanisms to explain how soreness develops and why there is a delay in pain sensation is not fully understood (Cheung et al., 2003; Clarkson, 2000; Clarkson & Hubal, 2002; deVries & Housh, 1996).

Several treatments which aim to prevent and/or reduce the severity of muscle damage have been investigated including acupuncture (Barles, Robinson, Allen, & Baxter, 2000), ultrasound (Ciccone, Leggin, & Callamaro, 1991; Craig, Bradley, Walsh, Baxter, & Allen, 1999), cryotherapy (Eston & Peters, 1999), compression (Chleboun et al., 1995; Kraemer et al., 2001), antiinflammatory drugs (Bourgeois, MacDougall, MacDonald, & Tarnopolsky, 1999), hyperbaric oxygen therapy (Mekjavic, Exner, Tesch, & Eiken, 2000), warm-up (High et al., 1989; Nosaka & Clarkson, 1997), stretching (High et al., 1989; Johansson et al., 1999; Lund, Vestergaard-Poulsen, Kanstrup, & Sejrsen, 1998a), and massage (Hasson et al., 1992; Lightfoot, Char, McDermont, & Goya, 1997; Rodenburg, Steenbeek, Schiereck, & Bar, 1994; Smith, Keating et al., 1994; Tiidus & Shoemaker, 1995; Weber, Servedio, & Woodall, 1994; Wenos, Brilla, & Morrison, 1990). These treatments have been applied as a prophylactic and/or a therapeutic intervention. However, the benefits of these treatments are still inconclusive. From a clinical point of view, the treatment given prophylactically is more desirable for reducing or preventing injury, and consequently, for producing a reduction in further injuries, chronic pain, cost of injury treatment, and time lost from training activities.

Massage is one of the treatments commonly used to alleviate DOMS because it is thought to increase local blood and lymph flow, decrease oedema, and reduce pain. Significant reductions in soreness perception of DOMS after massage have been reported (Bale & James, 1991; Rodenburg et al., 1994; Smith, Keating et al., 1994; Tiidus & Shoemaker, 1995). Some studies explained the mechanism of DOMS reduction by the increase of neutrophils (Smith, Keating et al., 1994), and the reduction of blood creatinine kinase (Rodenburg et al., 1994; Smith, Keating et al., 1994), while some researchers failed to explain the mechanism at all (Bale & James, 1991; Tiidus & Shoemaker, 1995). Many researchers, however, reported that massage was not beneficial in reducing DOMS (Hasson et al., 1992; Lightfoot et al., 1997; Weber et al., 1994; Wenos et al., 1990; Yackzan, Adams, & Francis, 1984). A summary of studies that have investigated the effects of massage on muscle soreness is presented in Table 2.7.

Table 2.7 The effects of massage on muscle soreness.

Reference	Trial design	Samples	Intervention	Outcome measures	Main results
Smith et al. (1994)	RCT	14 untrained males (*n*=7 for each intervention)	a) 30-min of effleurage, shaking, petrissage, cross-fiber. b) Control. Intervention performed 2 hr after biceps and triceps eccentric exercise.	a) Muscle soreness rating. b) Blood CK, neutrophils, cortisol.	S: ↓ DOMS & CK. S: ↑ neutrophils.
Rodenburg et al. (1994)	RCT	50 untrained males (*n*=27 for experimental group)	a) Warm-up, stretching, massage (15 min of effleurage, tapotement, petrissage). b) Control.	a) DOMS scale. b) Maximal isotonic force. c) Flexion angle of elbow. d) Extension angle of elbow. e) CK level. f) Mb concentration.	S: ↓ DOMS (extensor muscles), CK, flexed arm angle. S: ↑ maximal isotonic force.
Tiidus and Shoemaker (1995)	CCT	9 healthy volunteers (5F, 4M) (one leg massage, one leg control)	10-minute effleurage on leg 3 days before, immediately after, and repeated on day 2 and 3 after quadriceps eccentric exercise.	a) Muscle strength. b) Arterial blood velocity. c) Venous blood velocity. d) DOMS perception (follow up 5 consecutive days).	S: ↓ soreness at 48 hr posteccentric exercise.

Table 2.7 (continued) The effects of massage on muscle soreness.

Reference	Trial design	Samples	Intervention	Outcome measures	Main results
Farr et al. (2002)	CT	8 healthy males (one leg massage, one leg control).	30 min leg massage (effleurage & petrissage) 2 hr after 40 min treadmill walk.	a) Muscle strength. b) Plasma CK. c) Vertical jump. d) Soreness. e) Tenderness.	S: ↓ soreness and tenderness at 48 hr post-eccentric exercise.
Hilbert et al. (2003)	RCT	18 healthy volunteers	a) Swedish massage (7 min of effleurage, 1 min of tapotement, 12 min of petrissage). b) control (placebo lotion applied and rest for 20 min). Intervention performed 2 hr after hamstrings eccentric exercise.	1) POMS. 2) ROM. 3) Peak torque (eccentric contraction). 4) DDS. 5) Neutrophils.	S: ↓ soreness at 48 hr posteccentric exercise.
Wenos et al. (1990)	CCT	9 untrained participants (not specific gender) (one quadriceps as control, the other was massaged).	Unknown.	a) Torque. b) ROM-hip flexion, extension, abduction, adduction. c) Soreness perceptions.	NS
Lightfoot et al. (1997)	RCT	31 healthy volunteers (12M, 19F) (*n*=10 for each group).	a) Massage (10 min of petrissage) immediately & 24 hr postexercise on the left calf muscle. b) Stretching. c) Control.	a) DOMS scale. b) Lower leg volume. c) CK level.	NS

Table 2.7 (continued) The effects of massage on muscle soreness.

Reference	Trial design	Samples	Intervention	Outcome measures	Main results
Hasson et al. (1992)	RCT	16 healthy subjects (no specific gender).	a) Retrograde massage (*n*=6). b) Placebo massage (*n*=5). c) Control (*n*=5) (treatment 24 hr postexercise).	a) Maximum isotonic KE. b) Maximum concentric KE. c) Maximum eccentric KE. d) Maximum 1 leg jump. e) Maximum 2 leg jump. f) Soreness perception.	NS
Weber et al. (1994)	RCT	40 untrained F (*n*=10 for each group).	a) Massage (effleurage, petrissage) b) Microcurrent electrical stimulation. c) Upper body ergometry. d) Control (all for 8 min).	a) Soreness scale. b) Maximal isometric contraction. c) Peak torque.	NS

CCT = controlled clinical trial; RCT = randomised controlled trial; CT = counterbalance trial; S = significant; NS = non-significant; DOMS = delayed onset muscle soreness; F = female; M = male; CK = creatine kinase; DDS = Differential Descriptor Scale; POMS = Profile of Mood States; ↑ = increase; ↓ = decrease.

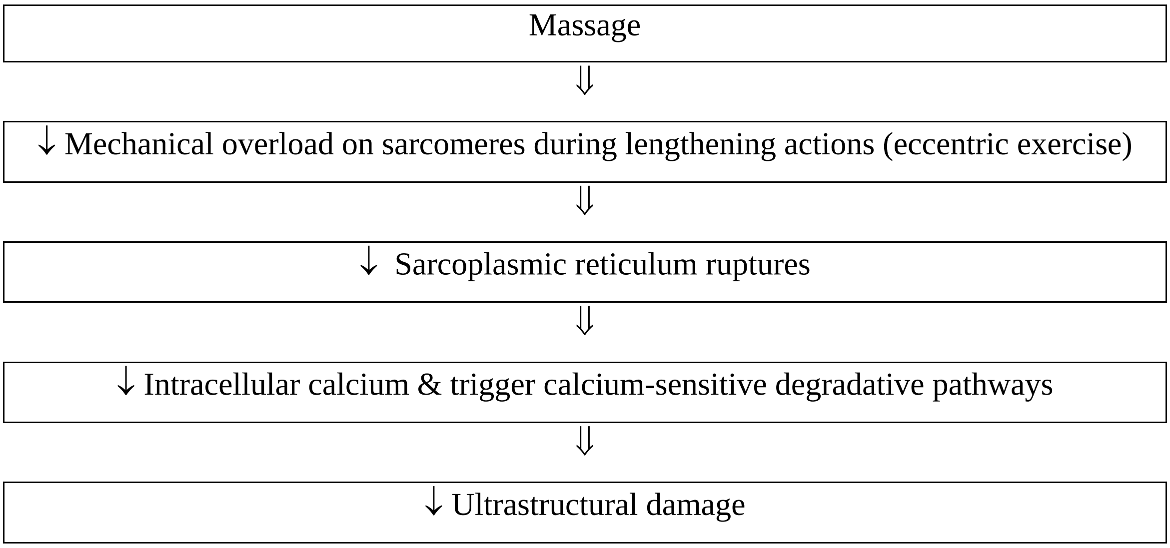
The inconclusive data on the effects of massage on DOMS may be due to the limitations of previous research. The majority of studies used small sample sizes, which limited the statistical power of the studies (Bale & James, 1991; Hasson et al., 1992; Lightfoot et al., 1997; Smith, Keating et al., 1994; Tiidus & Shoemaker, 1995; Weber et al., 1994; Wenos et al., 1990). Some studies used another limb as a control group that could have introduced intrasubject bias (Tiidus & Shoemaker, 1995; Wenos et al., 1990). One study used a combination of treatments making it difficult to establish the effectiveness of each of the treatments alone (Rodenburg et al., 1994). Two studies were reported only in abstract form (Hasson et al., 1992; Wenos et al., 1990). The different genders of participants might affect the results (Lightfoot et al., 1997; Tiidus & Shoemaker, 1995) because of the different patterns of DOMS between males and females as reported in the literature (MacIntyre, Reid, Lyster, & McKenzie, 2000). The wide variation of massage techniques, duration of massage application, area of the body massaged, and outcome measures also affect the conclusions that can be drawn from the studies.

The unclear mechanisms of massage may also lead to inappropriate massage application. In practice, massage is often applied immediately after exercise in order to enhance blood circulation. The effects of massage on blood circulation are still questionable as described previously. The eccentric exercise, which induced muscle damage, does not produce waste products that require extra blood flow. Research which investigated the effects of massage immediately after exercise found a decrease of DOMS 48 hours after exercise but did not find any change in blood circulation (Tiidus & Shoemaker, 1995). Some studies have not found any effects of massage immediately after exercise (Weber et al., 1994; Wenos et al., 1990).

Some researchers have speculated that massage may reduce DOMS sensation by decreasing muscle oedema. However, in studies by Hasson et al. (1992) and Lightfoot et al. (1997), leg volume and soreness sensation did not change after massage immediately after exercise and/or 24 hours after exercise. Massage performed two hours post-exercise was reported to benefit DOMS by reducing an inflammatory process (Smith, Keating et al., 1994). The neutrophil values in the massage group were significantly higher than in the control group at eight and 24 hours. The authors speculated that the elevation of the neutrophil counts was the result of the mechanical action of massage by the shearing of the neutrophils from the vessel walls. The increased blood flow from the proposed physiological mechanism of massage might prevent the migration of the neutrophils from the circulation into the injury sites. Thus, the neutrophil values would be elevated in the blood count. Two studies used the protocol of Smith et al. (1994) to examine the effects of massage application two hours after eccentric exercise (Farr et al., 2002; Hilbert et al., 2003). Farr et al. (2002) and Hilbert et al. (2003) reported that massage performed two hours post-exercise was effective in reducing soreness sensation. It is important to note that Farr et al.'s study investigated massage on one leg and used the other leg as the control group. Therefore, it is likely that massage might provide a psychological advantage as only soreness sensation - the subjective measure reported by the participants - was reduced after massage application. However, there was no benefit of massage for preventing muscle strength and function loss (as determined by isometric and isokinetic tests and jumping height, respectively) (Farr et al., 2002; Hilbert et al., 2003). Interestingly, both research studies did not find any change in neutrophil count.

It is hypothesized, that if the mechanical effect of massage can increase muscle flexibility and reduce muscle stiffness, enhance local microcirculation and lymph flow, and increase muscle compliance, massage should be applied before eccentric exercise in order to lower the initial mechanical overload of eccentric exercise. A theoretical model of the expected mechanism of massage on the severity of DOMS is presented in Figure 2.2.

Figure 2.2 Theoretical model of the expected mechanisms of massage on the severity of muscle soreness.



2.5.4 Summary of the evidence for massage improving performance, enhancing recovery, or preventing muscular injury

There are relatively few, well controlled studies that have examined the potential for massage to influence performance, recovery or injury-risks. Limited research has investigated the effects of preexercise massage on performance. The results are inconclusive due to the inappropriate massage techniques and outcome measures used. There is no research that has looked at the effects of preexercise massage on injury-prevention. Massage is widely administered between events because it is believed that massage might help to enhance recovery and prepare athletes for the next coming event. Unfortunately, very little scientific data has supported this claim. A large number of studies of massage have reported the psychological benefits of massage between events. Several research studies have reported that post-eccentric exercise massage could help to reduce muscle soreness sensation but could not affect muscle functional loss.

2.6 Summary and conclusions

Massage is believed to benefit athletes through its biomechanical, physiological, neurological, and psychological mechanisms. Research has reported the effects of massage on physiological (investigated by blood flow and blood-borne substance), neurological (investigated by H-reflex), and psychological (investigated by questionnaire and psychophysiological parameters such as heart rate, blood pressure) mechanisms. There are limited data on the possible mechanisms of massage, especially mechanical mechanisms of pressure and motion of massage on muscle properties such as passive or active muscle stiffness.

There are several limitations of previous research on the effects of massage on performance and injury prevention which have lead to inconclusive results. Further research is clearly needed to establish the possible benefits of massage. The unclear effects of massage on muscle blood flow lead to uncertain benefits for performance and recovery from fatigue. Only petrissage has been studied and shown to reduce the H-reflex. Other massage techniques have not been examined in terms of their neurological effects. Therefore, there is no evidence to support the claim that some massage techniques (e.g., tapotement, vibration) can increase neuromuscular excitability. The lack of studies on the mechanical effects of massage on muscle properties such as active and passive stiffness provides unclear information on the biomechanical mechanisms of massage. The understanding of the mechanism of exercise-induced muscle soreness, as well as the mechanisms of massage, will help to select the appropriate massage technique, duration of massage application, and time to apply massage. Therefore, more research on the effects of massage is needed to clarify whether massage is beneficial for enhancing performance, enhancing recovery from injury, or reducing the risk of muscular injury.

The effects of different types of massage (e.g., petrissage, effleurage) or the appropriate timing of massage (preexercise versus postexercise) on performance, recovery from injury, or as an injury prevention method also needs to be examined.

2.7 Recommendations

Research has not shown any clear benefits of massage on sport performance or injury prevention. The use of massage before training and competition to enhance performance is, therefore, questionable. Future research should address the following questions:

- Can massage increase muscle blood flow, muscle temperature, neuromuscular excitability, or muscle flexibility?
- Can massage increase performance such as sprinting, jumping, or endurance athletic events?
- What type of massage can produce benefits? How long should massage be applied? When should athletes receive massage?
- Are the effects of massage universal or are they specific to each massage therapist?
- Is the cost and time for massage appropriate when a warm-up or cool-down may be as, or more, effective?

In order to overcome limitations of previous research, massage studies should consider these points:

- An appropriate control group should be provided. The ideal control group for a massage study should be passive therapy where the participants receive the same attention in terms of time from the therapists as the massage group. However, the therapists should not apply any pressure on the muscle. Some physiotherapy equipment might be appropriate such as a sham shortwave diathermy.

- Studies should be designed as a counterbalance design in order to minimise the different responses of individual participants.
- Appropriate outcome measures and massage techniques should be used in the study.

CHAPTER 3

STRETCHING: MECHANISMS AND BENEFITS ON PERFORMANCE AND INJURY PREVENTION

Weerapong, P., Hume, P. A., & Kolt, G. S. (2004). Stretching: mechanisms and benefits on performance and injury prevention. *Physical Therapy Reviews, 9,* 189-206.

3.1 Summary

Stretching is usually performed before exercise in an attempt to enhance performance and reduce the risk of injury. Most stretching techniques (static, ballistic, and proprioceptive neuromuscular facilitation) are effective in increasing static flexibility as measured by joint range of motion, but the results for dynamic flexibility as measured by active and passive stiffness, are inconclusive. The mechanisms of various stretching techniques in terms of biomechanics and neurology, the effectiveness of the combination of stretching with other therapies such as heat and cold, and the effectiveness of stretching for performance and injury prevention are discussed. The possible mechanisms responsible for the detrimental effects of stretching on performance and the minimal effects on injury prevention are conferred, with the emphasis on muscle dynamic flexibility. Further research is recommended to explore the mechanisms and effects of other stretching techniques besides static stretching on dynamic flexibility, muscle soreness, sport performance, and rate of injury.

3.2 Introduction

Common clinical practices suggest that preexercise stretching can enhance performance and prevent injuries by increasing flexibility. However, current scientific research does not support this notion (Cornwell et al., 2002; Fowles et al., 2000; Johansson et al., 1999). Rather the acute effects of stretching can have detrimental effects on performance parameters such as muscle strength ((Fowles et al., 2000; Kokkonen et al., 1998; Nelson, Guillory et al., 2001), and jumping performance (Knudson, Bennett, Corn, Leick, & Smith, 2001; Young & Behm, 2003). In this paper, the possible mechanisms of stretching are reviewed in order to provide guidelines regarding use of stretching as an appropriate strategy to enhance performance and reduce the risk of injury. The effects of stretching on performance and injury prevention are presented. Further areas for research are also recommended.

Several reviews of stretching have been published recently, so the reader is encouraged to refer to these for more information; e.g. the effects of stretching on muscle properties (Magnusson, 1998), the effects of stretching on injury prevention (Weldon & Hill, 2003; Witvrouw, Mahieu, Danneels, & McNair, 2004), and the effects of stretching on performance (Thacker, Gilchrist, Stroup, & Kimsey, 2004). The current review focuses on the possible mechanisms of stretching on biomechanical and neurological changes of muscles, and consequently, how these mechanisms affect the performance and the risk of injury from exercise. This paper also focuses on dynamic stretching techniques and dynamic flexibility which may be more beneficial to athletes than the more traditional stretching in term of performance and injury prevention.

Literature for this review was located using three electronic databases (PubMed, SPORT Discus, and ProQuest 5000 International) in addition to manual journal searches. The computer databases provided access to biomedical and sport-oriented journals, serial publications, books, theses, conference papers, and related research published since 1965. The key search terms included: sport stretching, static stretching, dynamic stretching, ballistic stretching, proprioceptive neuromuscular facilitation, performance, sport injury, delayed onset muscle soreness, injury prevention, and muscle stiffness. There were a limited number of published randomised controlled trials, therefore, other types of research such as clinical controlled trials and literature reviews were included in this review. Articles not published in English and/or in scientific journals, that focused on the psychological effects of stretching, or the effects of stretching in special populations were not included in this review. The criteria for inclusion were that the article must have:

- focused on normal, healthy participants. Age, gender, and fitness differences were not excluding factors.
- investigated the acute effects of stretching. Immediate and long-term effects of flexibility training were not excluding factors.
- discussed the possible mechanisms of stretching in relation to biomechanical and/or neuromuscular properties of muscle, sport performance, rate of injury, or muscle soreness.

3.3 Definition of stretching

Several literature reviews have considered flexibility (Gleim & McHugh, 1997; Shellock & Prentice, 1985) as the outcome of stretching exercise. However, the definition of stretching itself has not been well defined yet. Magnusson et al. (Magnusson et al., 1996) stated that “stretching has been characterised in biomechanical terms in which the muscle-tendon unit is considered to respond viscoelasticity during the stretching manoeuvre” (pp. 77). However this definition of stretching is more a biomechanical result of stretching rather than a definition of the action of stretching. In our review stretching is defined as movement applied by an external and/or internal force in order to increase muscle flexibility and/or joint range of motion. The aim of stretching before exercise is to increase muscle-tendon unit length (Taylor, Dalton, Seaber, & Garrett, 1990) and flexibility. The increase in flexibility may help to enhance athletic performance and decrease the risk of injury from exercise (Gleim & McHugh, 1997).

3.4 Types of stretching technique

There are various types of stretching techniques that are used, often depending on athlete choice, training programme, and the type of sport. An earlier review of stretching (Shellock & Prentice, 1985) indicated that four different methods are commonly used for sport activities: static, ballistic, proprioceptive neuromuscular facilitation (PNF), and dynamic (see Table 3.1).

Table 3.1 Summary of the advantages and disadvantages of stretching techniques.

Techniques	Definition	Advantages	Disadvantages
Ballistic stretching.	Repetitive bouncing movements at the end of joint range of motion (Shellock & Prentice, 1985).	Increased range of motion (Shellock & Prentice, 1985).	Reduced muscle strength (Nelson & Kokkonen, 2001). May cause injury (Smith et al., 1993).
Proprioceptive neuromuscular facilitation (PNF) stretching.	Reflex activation and inhibition of agonist and antagonist muscles (Burke, Culligan, & Holt, 2000).	Increased range of motion (Spernoga, Uhl, Arnold, & Gansneder, 2001).	Reduced jump height (Church et al., 2001). Need experience and practice (Smith, 1994).
Static stretching.	Passive movement of a muscle to maximum range of motion and then holding it for an extended period (Shellock & Prentice, 1985).	Increased range of motion (Halbertsma, van Bolhuis, & Goeken, 1996). Simple technique.	Reduced muscle strength (Cornwell et al., 2002; Fowles et al., 2000). May cause injury (Smith et al., 1993).
Dynamic stretching.	Slow movement of a joint as a result of antagonist muscle contraction throughout the range of movement (Shellock & Prentice, 1985).	Unknown.	Unknown.

3.5 Mechanisms of stretching

Stretching results in elongation of muscles and soft tissues through mechanical and neurological mechanisms. Stretching activities may benefit athletes mentally through psychological mechanisms, however, there have been no studies on the psychological effects of stretching.

3.5.1 Biomechanical mechanisms

Muscle-tendon units can be lengthened in two ways: muscle contraction and passive stretching. When muscle contracts, the contractile elements are shortened, and a compensatory lengthening occurs at the passive elements of tissues (tendon, perimysium, epimysium, and endomysium) (Taylor, Brooks, & Ryan, 1997). When muscle is lengthening, the muscle fibres and connective tissues are elongated because of the application of external force (Taylor et al., 1997). Stretching increases muscle-tendon unit length by affecting the biomechanical properties of muscle (range of motion and viscoelastic properties of the muscle-tendon unit).

3.5.1.1 Range of motion

The majority of previous research on the effects of stretching on flexibility used range of motion as an indicator (Halbertsma et al., 1996; Henricson, Fredriksson, Persson, Pereira, & Westlin, 1984; Spernoga et al., 2001; Wiktorsson-Moller et al., 1983; Zito, Driver, Parker, & Bohannon, 1997). The exact physiological mechanism of stretching resulting in increased range of motion still remains unclear (Muir, Chesworth, & Vandervoort, 1999) as most research has failed to show changes in muscle properties such as passive (Klinge et al., 1997; Magnusson et al., 1996; Magnusson, Simonsen, Aagaard, Sorensen, & Kjaer, 1996; Magnusson, Simonson et al., 1996) or active (Cornwell & Nelson, 1997; McNair & Stanley, 1996) stiffness. The increase in range of motion is thought to be the influence of increasing stretch tolerance (Gajdosik, Giuliani, & Bohannon, 1990; Halbertsma et al., 1996; Magnusson, 1998; Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996) and pain threshold, or subject bias following an intervention (Magnusson, Simonsen, Aagaard, Gleim, & McHugh, 1995). Therefore, the increase in static flexibility, as indicated by range of motion, does not provide clear information on musculotendinous behaviour (Magnusson, 1998; Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996).

3.5.1.2 Viscoelastic properties of the muscle-tendon unit

The viscoelastic properties of muscle exhibit several phenomena when external load is applied. When tissues are held at a constant length, the force at that length gradually declines and is described as the “stress relaxation” response (Magnusson, 1998; Magnusson et al., 1995; Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996; McHugh, Magnusson, Gliem, & Nicholas, 1992; McNair et al., 2000). When tissues are held at a constant force, the tissue deformation continues until approaching a new length and is termed “creep” (Taylor et al., 1990). Creep might be another explanation for the immediate increased range of motion after static stretching (Gajdosik et al., 1990). The musculotendinous unit also produces a variation in the load-deformation relationship between loading and unloading curves (Taylor et al., 1990). The area between the loading and unloading curves is termed “hysteresis” and represents the energy loss as heat due to internal damping (Kubo, Kanehisa, & Fukunaga, 2001; Taylor et al., 1990). Several researchers have studied the effects of stretching on stress-relaxation, creep, and hysteresis (Cavagna & Citterio, 1974; Halbertsma et al., 1996; Kubo et al., 2001; Kubo, Kanehisa, & Fukunaga, 2002; Magnusson, 1998; Magnusson, Aagaard, Larsson, & Kjaer, 2000; Magnusson et al., 1995; Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996; McNair et al., 2000; Taylor et al., 1997; Taylor et al., 1990), however, none of the previous research clearly showed the relationship of these phenomena to the rate of muscle injury or performance.

Tendon, which is the major resistance to the final range of motion of the musculotendinous unit, shows similar characteristics. The mechanics of tendon have been recently reviewed (Kjaer, 2004; Silver, Freeman, & Seehra, 2003; Vanderby & Provenzano, 2003). Passive stiffness refers to the passive resistance of the muscle-tendon unit in a relaxed state when external forces are applied. The slope of the force and deformation curve at any range of motion is defined as passive stiffness (Gleim & McHugh, 1997; Magnusson, 1998). Passive torque, which occurs during passive movement, is resistance from stable cross-links between actin and myosin, non-contractile proteins of the endosarcomeric (titin) and exosarcomeric cytoskeletons (desmin), and connective tissues surrounding muscles (endomysium, perimysium, and epimysium) (Gajdosik, 2001). Perimysium is considered to produce major resistance (Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996). Active stiffness is defined as the resistance of the contracted muscle to transiently deform when the external forces were applied briefly, and can be measured by the damped oscillation technique (McNair & Stanley, 1996; Wilson, Elliott, & Wood, 1992; Wilson, Murphy, & Pryor, 1994). The oscillation of the contracted muscle after the application of external force results from the viscoelasticity of muscle and the level of muscle activation (McNair & Stanley, 1996). Passive and active stiffness provide more information on muscle-tendon unit behavior during movement than range of motion only.

3.5.2 Neurological mechanisms

Biomechanical responses of muscle–tendon units during stretching are independent of reflex activity (Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; McHugh, Kremenic, Fox, & Gleim, 1998; McHugh et al., 1992; Mohr, Pink, Elsner, & Kvitne, 1998; Taylor et al., 1990) as indicated by the lack of muscle activity (EMG) responses during stretching. However, a decrease in the Hoffman reflex response (H-reflex) during (Vujnovich & Dawson, 1994) and after stretching (Avela, Kyrolainen, & Komi, 1999; Gollhofer, Schopp, Rapp, & Stroinik, 1998; Moore & Kukulka, 1991; Rosenbaum & Hennig, 1995) has been reported.

Some research reports have stated that all stretching techniques affect neural responses by reducing neural sensitivity (Avela et al., 1999; Gollhofer et al., 1998; Moore & Kukulka, 1991; Rosenbaum & Hennig, 1995; Thigpen, Moritani, Ythiebaud, & Hargis, 1985; Vujnovich & Dawson, 1994). The majority of research on the effects of stretching on neurological mechanisms have investigated the changes of the H-reflex - the electrical analogue of the stretch reflex but without the effects of gamma motoneurons and muscle spindle discharge (Zehr, 2002). Electrical stimulation of a mixed peripheral nerve (both sensory and motor axons) (Zehr, 2002) will evoke the H-reflex. The activation of the motor axons directly induces the M-wave (from the point of stimulation to the neuromuscular junction) prior to evoking the H-reflex (from Ia afferents arising from annulospiral endings on the muscle spindle) via a monosynaptic connection to the alpha motoneurons (Zehr, 2002). H-reflex is widely used to study changes in the reflex excitability of a group of muscle fibres (Gollhofer et al., 1998; Moore & Kukulka, 1991; Morelli et al., 1999; Morelli et al., 1990, 1991; Vujnovich & Dawson, 1994; Zehr, 2002). The depressed amplitude of H-reflex after stretching might be due to several possibilities relating to presynaptic and/or postsynaptic change (Guissard, Duchateau, & Hainaut, 2001). The presynaptic changes might be due to a presynaptic inhibition inducing an autogenic decrease in Ia afferents and/or an altered capacity for synaptic transmission during repetitive activation (Guissard et al., 2001). The postsynaptic changes might be due to an autogenic inhibition from the Golgi tendon organ (GTO), recurrent inhibition from the Renshaw loop, or postsynaptic inhibition of afferents from joint and cutaneous receptors (Guissard et al., 2001).

3.6 Mechanisms of each stretching technique

Even though each technique of stretching is expected to increase muscle and joint flexibility, different stretching techniques produce increases in flexibility by different mechanisms.

3.6.1 Static stretching

Static stretching is the most widely used technique by athletes due to its simplicity. Static stretching was found to affect both mechanical (Cornwell et al., 2002; Kubo et al., 2001, 2002; Magnusson et al., 1995; Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996; McHugh et al., 1998; McHugh et al., 1992; McNair et al., 2000) and neurological (Avela et al., 1999; Guissard et al., 2001; Ribot-Ciscar, Tardy-Gervet, Vedel, & Roll, 1991; Rosenbaum & Hennig, 1995; Vujnovich & Dawson, 1994) properties of the muscle-tendon unit resulting in increased musculoskeletal flexibility (see Table 3.2).

Table 3.2 The effects of static stretching on muscle properties.

References	Trial design	Sample	Interventions	Outcome measures	Main results
ROM and passive stiffness					
McHugh et al. (1992)	PPT	9 men and 6 women (hamstrings).	Static stretch (hold 45s) 1.At onset of EMG. 2. Five degrees below the onset of EMG (negligible EMG activity).	1.Peak torque. 2. ROM. 3. EMG.	S: ↓ torque. ↑ ROM.
Magnusson et al. (1995)	PPT	10 men (hamstrings).	1.Static stretch (hold 90s, rest 30s) 5 times (stretch 1-5). 2. Repeated static stretch one time (stretch 6).	1.Peak torque. 2. ROM. 3. EMG.	S: ↓ stress relaxation. ↑ ROM.
Magnusson et al. (1996)	PPT	7 women (one leg stretch one leg control) (hamstrings).	Static stretch (45s hold x 15-30s rest x 5times), twice daily, 20 consecutive days.	1.Stress relaxation. 2. Energy. 3.EMG. 4.ROM.	S : ↑ ROM.
Halbertsma et al. (1996)	RCT	10 men and 6 women with short hamstrings.	1.Static stretching (30 s hold x 30 s rest) for 10 min (*n*=10) 2.Control-rest (*n*=6)	1.Peak torque. 2. ROM. 3.Passive stiffness.	S: ↑ ROM.
Magnusson et al. (1996)	CCT	8 neurological intact and 6 spinal cord injury volunteer (hamstring).	Static stretch (hold 90s).	1.Stress relaxation. 2. Passive torque. 3.EMG.	NS

Table 3.2 (continued) The effects of static stretching on muscle properties.

References	Trial design	Sample	Interventions	Outcome measures	Main results
Magnusson et al. (1996)	PPT	13 men (hamstrings).	5 static stretches (hold 90s, rest 30s) and repeated 1 hr later.	1.Stiffness. 2. Energy. 3. Passive torque.	S: ↓ energy, stiffness, and peak torque.
Klinge et al. (1997)	CCT	12 men in experimental group, 10 men in control group.	4 x 45 s static stretch.	1. ROM. 2. Passive stiffness.	NS
McHugh et al. (1998)	CCT	8 men and 8 women (hamstrings).	SLR stretch.	1. Peak torque. 2. ROM. 3. EMG.	S: ↑ ROM.
Magnusson et al. (1998)	CCT	12 men (hamstrings)	1. 90 s static stretches. 2. Continuous movements 10 times at 20°.s^{-1}.	1. ROM. 2. Passive stiffness.	S: ↑ ROM.
Muir et al. (1999)	RCT	10 men (one leg-stretching, one leg-control) (hamstrings).	1. Static stretching (30sx10s) for 4 times. 2. Control-rest.	1.Peak torque 2. Centre range (of hysteric loop).	NS
McNair et al. (2000)	CBT	15 men and 8 women (plantarflexors).	Static stretching 1. 1x60s hold. 2. 2x30s hold. 3. 4x15s hold. 4.Continuous passive movement for 60s.	1. Passive stiffness. 2. Peak torque.	Continuous movement S: ↓ passive stiffness. Hold condition S: ↓ peak tension.

Table 3.2 (continued) The effects of static stretching on muscle properties.

References	Trial design	Sample	Interventions	Outcome measures	Main results
Magnusson et al. (2000)	PPT	20 men	3 static stretches (hold 45s, rest 30s) and repeated 1 hr later.	1. Stiffness. 2. Energy. 3. Passive torque.	S: ↓ stress relaxation.
Kubo et al. (2001)		7 men (plantarflexors).	Passive stretching to 35° dorsiflexion at 5°.s^{-1} for 10 min.	1. Tendon stiffness. 2. Tendon hysteresis. 3. MVC.	S: ↓ tendon stiffness (10%), tendon hysteresis (34%).
Kubo et al. (2002)	CBT	8 men (plantarflexors).	Passive stretching to 35° dorsiflexion at 5°.s^{-1} for 5 min.	1. Tendon stiffness. 2. Tendon hysteresis.	S: ↓ tendon stiffness (8%), tendon hysteresis (29%).
ROM and active stiffness					
Wilson et al. (1992)	CCT	16 male weightlifters (*n* = 9 in experiment, *n* = 7 in control group).	Flexibility training (6-9 rep) of upper extremities, 10-15 min per session, twice a week for 8 weeks.	1.Rebound bench press (RBP). 2.Purely concentric bench press (PCBP).	S: ↑ ROM (13%). S: ↑ RBP (5.4%). S: ↓ SEC stiffness (7.2%).
Cornwell et al. (1997)	PPT	10 men (plantarflexors).	Passive stretching.	1. ROM. 2. Active stiffness.	S : ↑ ROM.

Table 3.2 (continued) The effects of static stretching on muscle properties.

References	Trial design	Sample	Interventions	Outcome measures	Main results
McNair and Stanley (1996)	CCT	12 men and 12 women (plantarflexors).	1. Static stretch (30sx30s). 2. Jogging (60%MHR). 3. Combined 2+1. Randomly order, each intervention for 10 min.	1. ROM. 2. Active stiffness.	Jogging group S: ↓ active stiffness. All groups S: ↑ ROM.
Hunter and Marshall (2001)	CCT	15 men, 15 women (*n* =15 in experiment and control groups) (plantarflexors).	10 x 30 s static stretches.	Active stiffness.	NS
Cornwell et al. (2002)	PPT	10 men (plantarflexors).	Passive stretching (30 s x 6 times).	1. Active muscle stiffness. 2. EMG. 3. Jump height.	S: ↓ jump height (7.4%). ↓ active stiffness (2.8%).

CCT = controlled clinical trial; RCT = randomised controlled trial; PPT = pre- & post-test trial; CBT, counterbalance trial; ROM = range of motion; EMG = electromyography; SEC = series elastic components; S = significant; NS = non-significant.

Despite static stretching being effective in increasing static flexibility as measured by range of motion (Halbertsma et al., 1996; Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996; McNair et al., 2000; McNair & Stanley, 1996), static stretching does not affect dynamic flexibility as measured by passive (Halbertsma et al., 1996; Magnusson, 1998; McNair et al., 2000) or active (Cornwell & Nelson, 1997; McNair & Stanley, 1996) stiffness, but affects viscoelastic properties by reducing stress relaxation (Halbertsma et al., 1996; Magnusson et al., 1995; Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996; McNair et al., 2000; Muir et al., 1999). The reduction of stress relaxation is an acute adaptation of the parallel elastic component to lower the imposed load across the myotendinous junction where injury usually occurs (Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996). However, there is no clear evidence that static stretching can reduce the rate of injury. Static stretching has produced similar muscle-tendon unit property responses between neurologically intact participants and spinal cord-injured participants with complete motor loss (Magnusson, Simonson et al., 1996). As well, there were reports of no EMG activity during passive stretching (Magnusson et al., 1995; Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996; McHugh et al., 1998; McHugh et al., 1992). Therefore, the effects of static stretching on muscle properties do not involve the neurological mechanism.

The effects of stretching on muscle properties depend on various factors including the stretching techniques used, time to stretch, holding duration, time to rest, and the time gap between intervention and measurement. The majority of research has examined the acute effects of static stretching on passive properties of the muscle-tendon unit (Magnusson, 1998; Magnusson et al., 2000; Magnusson et al., 1995; Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996; McNair et al., 2000). In a series of studies by Magnusson et al. (Magnusson, 1998; Magnusson et al., 2000; Magnusson et al., 1996; Magnusson et al., 1995; Magnusson, Simonsen, Aagaard, & Kjaer, 1996; Magnusson, Simonson et al., 1996) static stretching at 90 s for five repetitions reduced muscle resistance measured by passive stiffness, peak torque, and stress relaxation. The decline of muscle-tendon unit resistance returned to baseline within one hour (Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996) except for stress relaxation (Magnusson et al., 1995). Unfortunately, shorter stretch holding times (less than 60 s), and lower stretching repetitions (less than four times) did not provide such effects (Halbertsma et al., 1996; Magnusson et al., 2000; McNair et al., 2000). Interestingly, long term training using ten stretches for 45 s per day (three weeks) (Magnusson, Simonsen, Aagaard, Sorensen et al., 1996) and four stretches for 45 s, two sessions per day, seven days per week for 13 weeks (Klinge et al., 1997) did not change the mechanical or viscoelastic properties of muscle (Magnusson, Simonsen, Aagaard, Sorensen et al., 1996). Therefore, the changes of viscoelasticity of muscle-tendon units depend more on the duration of stretch rather than the number of stretches (Kubo et al.,

2001) or the length of the stretching training period. Indeed, if a decrease in muscle-tendon unit resistance is required long stretch duration (up to 90 s) might be most beneficial.

The protocol used in Magnusson et al.' studies (1998) showed an acceptable reproducibility (correlation coefficient; r = 0.91-0.99, 0.92-0.95 and coefficient of variation; 5.8%-14.5%, 9.5%-17.6%, for test and retest within one hour and between days, respectively). The key to the high reproducibility was the participants' trunk being fixed perpendicular to the seat in order to fix the origin of the hamstring muscles at the pelvic. The rate of stretch was controlled at an angular velocity of 5°/s which does not stimulate muscle activity. This rate of stretch might be an appropriate protocol to study passive properties of muscle at rest. During sports activities or injury, however, muscle-tendon units are stretched at 25-50 times more than the velocity used in these studies (e.g., 120°/s) (Kubo et al., 2001). It was found that the tensile strength and energy absorption changed with different rates in vitro (Taylor et al., 1990). Therefore, the high velocity of stretch as used during sport performance is needed to be researched to investigate the change of viscoelastic properties of muscle.

Prolonged static stretch (five to ten minutes) has been shown to decrease tendon and aponeurosis stiffness (elasticity) and hysteresis (viscosity) as measured passively by ultrasonography (Kubo et al., 2001, 2002). The decrease in stiffness from stretching may be due to an acute change in the arrangement of collagen fibres in tendon (Kubo et al., 2001). However, the holding time in the Kubo et al. (2001, 2002) studies is considered very long when compared with practical stretching (30-60 s hold). Unfortunately, no research on the effects of static stretching on the tendon for shorter durations has been identified in the published literature. The same group of researchers (Kubo et al., 2002) also reported that the combination of long term resistance (70% of one repetition maximum, 10 repetitions per set, five sets per day, four days per week for eight weeks) and stretching (ten minutes per day, seven days per week for eight weeks) training did not change tendon elasticity (determined by stiffness) but reduced hysteresis (17%). Unfortunately, the mechanisms responsible for the decrease in stiffness and hysteresis of the tendon are still unknown. The acute response of stretching on tendon and aponeurosis stiffness might be partly responsible for the increase in range of motion. The effects of stretching on series elastic components are also unclear. The findings of several researchers are in agreement that static stretching does not affect active stiffness. McNair and Stanley (1996) and Hunter et al. (2001) suggested that soleus stretching (five stretches of 30 s hold and 30 s rest, and 10 stretches of 30 s hold, respectively) did not reduce active stiffness. Similarly, Cornwell and Nelson (1997) reported that stretching did not affect the active stiffness of musculotendinous units of triceps surae. Unfortunately, Cornwell and Nelson (1997) did not report the details of the research process such as the stretching technique nor active stiffness measurement. Recently, Cornwell et al. (2002) found a slight but significant reduction of active muscle stiffness of the soleus muscle (2.8%) after stretching (six stretches of 30 s hold and 30 s rest). McNair and Stanley (1996) and Hunter et al. (2001) only investigated the soleus muscle while Cornwell et al. investigated the triceps surae. Stretching in Cornwell et al.'s study was slightly longer than in McNair and Stanley's study (180 s and 150 s, respectively) but not longer than Hunter et al.'s study (300 s). In the McNair and Stanley and Hunter et al. studies, the participants stretched the plantar flexors by adopting a step-standing position and stretching by flexing both knees. The participants in the Cornwell et al. study stretched in two ways. The participant placed the foot on the inclined board and maximally dorsiflexed the ankle joint whilst keeping the sole of the foot flush with the board surface and the knee joint fully extended. The second stretching technique employed the same protocol but the knee was flexed in order to increase the stretching force on the soleus. These stretching protocols might provide more strain on series elastic components of muscle than the method used in the McNair and Stanley study.

In evaluating the effects of long-term stretching, Wilson et al. (1992) reported that flexibility training of pectoralis and deltoid muscles (10 to 15 minutes per session, twice per week for eight weeks) reduced active muscle stiffness by 7.2%. The decrease in active stiffness might be from the long-term adaptation of connective tissue, sarcomere, contractile tissue, and/or reflex responses. These effects may not occur with acute stretching.

Despite the fact that the neurological mechanism is not responsible for the change of muscle-tendon unit properties during passive stretching, static stretching has been reported to decrease neuromuscular sensitivity as indicated by H-reflex responses (Avela et al., 1999; Guissard et al., 2001; Vujnovich & Dawson, 1994). Rosenbaum and Hennig (1995) reported that a static stretch of triceps surae (30 s each for three times) reduced the peak force of reflex force production, force rise rate, and EMG activity. Stretching might improve muscle compliance (reduced peak force and force rise rate), reduce muscle spindle sensitivity (reduced peak-to-peak amplitude), and reduce excitation-contraction coupling (increased force-to-EMG ratios). Thigpen et al. (1985) proposed that the decrease of evoked H-wave amplitude might be due to inhibitory effects of the Ib afferent from the GTO. Avela et al. (1999) also reported a reduction of H-reflex (46%) after prolonged stretching for one hour. The reduction of stretch reflex activity (reduced peak-to-peak amplitude) and α-motoneuron pool excitability (reduced H-wave /M-wave ratio) were suggested to occur from reduced sensitivity of the large-diameter afferents. Vujnovich and Dawson (1994) compared two stretching techniques (static and ballistic stretching) on the excitability of the α-motoneurons as indicated by amplitude changes in the H-reflex. H-reflex was reduced by up to 55% during static stretching of the soleus muscle (maintained for 160 s) but returned to baseline immediately following the termination of stretching. The amplitude of stretching (mid- and full-range of motion) provided a similarity in the mean depression of the H-reflex amplitude. Therefore, the receptor that is likely to mediate the inhibitory effect during static muscle stretch is the muscle spindle type II afferent. The results of this study were questioned because there were only two participants in the mid-range of motion stretching group. In contrast, Guissard et al. (2001) reported that greater stretching amplitude produced a greater reduction of H-reflex. When the ankle was moved passively for 10°, the H-reflex reduced by 25% but when the ankle was moved up to 20°, the H-reflex was reduced by 54%. Each stretch was held for 20 s to 30 s. The results from Guissard et al.'s study showed that a reduction of motoneuron excitation during stretching resulted from both pre-and post-synaptic mechanisms. The pre-synaptic mechanism was responsible for the small stretching amplitude while postsynaptic mechanisms played a dominant role in larger stretching amplitudes.

Table 3.3 The effects of static stretching on neuromuscular activity.

Reference	Trial design	Samples	Interventions	Outcome measures	Main results
Thigpen et al. (1985)	CCT	6 men, 2 women.	3 x 20 s toe touch stretching.	H-reflex.	S: ↓ H/M ratio 21.49%.
Vujnovich and Dawson (1994)	CCT	*Group A* (*n*=14): static stretching. *Group B* (*n*=2): Similar as A but followed up every 2 min for 10 min. *Group C* (*n*=5): Similar to B but followed by ballistic stretch (1 rad.s^{-1} for 160 s). *Group D* (*n*=2): Stretching at midway between neutral and fully dorsiflexion.	Maximally dorsiflexion for 160 s.	H-reflex.	Group A: S: ↓ H-reflex (45%). Group B: S: ↓ H-reflex during stretching but NS afterward. Group C: S: ↓ H-reflex during ballistic stretching (84%) >static stretching (40%). Group D: S: ↓ H-reflex 40%.
Rosenbaum et al. (1995)	CCT	50 male athletes.	Three min static stretch: hold 30s.	H-reflex of triceps surae: 1. Peak force. 2. Force rise rate. 3. Half relaxation rate. 4. EMG amplitude & integral. 5. EMG latencies.	S: ↓ peak force, force rise rate, half relaxation rate, EMG amplitude and integral. S: ↑ EMG latencies.

Table 3.3 The effects of static stretching on neuromuscular activity.

Reference	Trial design	Samples	Interventions	Outcome measures	Main results
Avela et al. (1999)	CCT	6 men (plantar flexors).	Repeated passive stretching (1 hr).	1. MVC. 2. 50%MVC. 4. H_{max} 5. Motor unit firing rate (ZCR).	S: ↓ MVC (23.2±19.7%). S: ↓ H reflex (46.1±38.3%). S: ↓ ZCR (12.2±11.4%).
Guissard et al. (2001)	PPT	7 men, 4 women.	Static stretch at 10° and 20°.	H/M ratio.	Dorsiflexion (10°) S: ↓ H/M ratio (25%). Dorsiflexion (20°) S: ↓ H/M ratio (55%).

CCT = controlled clinical trial; RCT = randomised controlled trial; PPT = pre- & post-test trial; CBT, counterbalance trial; ROM = range of motion; EMG = electromyography; SEC = series elastic components; S = significant; NS = non-significant; MVC = maximum voluntary contraction; H/M ratio = H-reflex/M-wave ratio.

A reduction of neuromuscular sensitivity during stretching, such as the amplitude of the H-reflex, might be due to the tension on stretched muscle applied by an external force being higher than the resistance from a protective mechanism of muscle from a stretch reflex. When muscle is stretched further, neuromuscular excitability from the stretch reflex still works but cannot resist the external force, which might cause the reduced sensitivity of the muscle spindles and, consequently, H-reflex during stretching. A decreased neuromuscular sensitivity, as indicated by the amplitude of the H-reflex, however, reverted to the control levels immediately after termination of static stretching in a study by Vujnovich and Dawson. (1994). Therefore, the neuromuscular mechanism is not likely as the mechanism to increase muscle flexibility after static stretching.

3.6.2 Ballistic stretching

Ballistic stretching is likely to increase flexibility through a neurological mechanism. The stretched muscle is moved passively to the end range by an external force or agonist muscle. Holding a muscle at the end range of joint motion might reduce muscle spindle sensitivity, with repeated stretch applied at the end range inhibiting the GTO. Research has reported an increase in range of motion (Vujnovich & Dawson, 1994; Worrell, Smith, & Winegardner, 1994), decrease in EMG (Wiemann & Hahn, 1997) and decrease H-reflex (Vujnovich & Dawson, 1994) with ballistic stretching.

Only one study in the published literature (Vujnovich & Dawson, 1994) examined the effects of ballistic stretching on neuromuscular excitability. Ballistic stretch applied following static stretch demonstrated lowered H-reflex mean amplitude than that obtained during static stretching (Vujnovich & Dawson, 1994). The lower H-reflex might be due to the inhibition of GTO or presynaptic inhibition from type Ia afferents. The results should be interpreted with caution as the number of subjects in the static stretching and ballistic stretching groups were different (n = 14 and 5 respectively) and there was no control group. Ballistic stretching was performed immediately after static stretching, therefore, the effects of ballistic stretching alone on H-reflex are still unknown.

It has been suggested that ballistic stretching may be more harmful than other stretching techniques. During ballistic stretching, muscle is stretched at a fast rate and rebounded back repetitively, resulting in greater tension and more absorbed energy within the muscle-tendon unit (Taylor et al., 1990). Muscle, which is released immediately after applying a high force, does not allow enough time for muscle to reduce tension (stress relaxation) or increase length (creep) (Taylor et al., 1990). Surprisingly, scientific evidence did not support this suggestion that ballistic stretching is more harmful than static stretching. Ballistic stretching (60 bounces per minute, 17 stretches per set for three sets) resulted in less severity of muscle soreness than static stretching (the same intensity and duration, but static stretching was held for 60 s) in college-age male volunteers (Smith et al., 1993). Despite ballistic stretching being less harmful than static stretching, according to Smith et al. (1993), most researchers still recommend slow static stretch before exercise (Shellock & Prentice, 1985; Smith, 1994).

3.6.3 Proprioceptive Neuromuscular Facilitation (PNF)

Several PNF techniques have been used to increase flexibility including slow-reversal-hold, contract-relax, and hold-relax techniques (Shellock & Prentice, 1985). These techniques include the combination of alternating contraction and relaxation of both agonist and antagonist muscles (Shellock & Prentice, 1985). The theory of PNF has been discussed and reviewed recently (Burke et al., 2000). The contractility property of muscles provides flexibility in the PNF technique on the basis of the viscoelastic characteristics of muscle and neuromuscular facilitation. The contracted muscle results in lengthening non-contractile elements (perimysium, endomysium, tendon) of muscle, and consequently, causes a relaxation of the muscle–tendon unit and decreased passive tension in a muscle (Taylor et al., 1997). The contracted muscle also stimulates the muscle sensory receptors within the muscle-muscle spindle (negative stretch reflex) and GTO which help to relax the tensed muscle. Therefore, the muscle-tendon-unit becomes more relaxed after the contraction.

Some PNF techniques, such as slow-reversal-hold, require agonist muscle to contract in order to relax antagonist muscle (Shellock & Prentice, 1985). “Reciprocal inhibition” occurs when the excitability signal from agonist muscle is transmitted by one set of neurons in the spinal cord to elicit muscle contraction, and then an inhibitory signal is transmitted through a separate set of neurons to inhibit the antagonist muscle (Burke et al., 2000). Reciprocal inhibition helps all antagonistic pairs of muscle to make smooth movement. When antagonist muscle is inhibited, muscle will be stretched to the opposite direction more easily.

Isometric contraction is commonly performed prior to passive stretching in the PNF technique. Post-isometric contraction exhibited a brief decrease in H-reflex response (83% by one second and 10% by ten seconds, respectively) (Moore & Kukulka, 1991). The depressed H-reflex was regardless of the intensity of isometric contraction (Enoka, Hutton, & Eldred, 1980), velocities, and amplitude of stretch (Gollhofer et al., 1998). Researchers have proposed that the decrease in H-reflex after isometric contraction could be a result of pre-synaptic inhibition (Gollhofer et al., 1998; Moore & Kukulka, 1991). The suppression of reflex activity was short-lasting (less than 10 s), indicating that passive stretching should be performed immediately after pre-isometric contraction in order to gain the maximal efficiency of stretching.

PNF stretching has been reported to result in a greater improvement of range of motion compared with static stretching (Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996; Sady, Wortman, & Blanke, 1982). Post-isometric contraction reduced neuromuscular sensitivity (Gollhofer et al., 1998; Moore & Kukulka, 1991) and might help to enhance the effectiveness of stretching (see Table 3.4). Toft et al. (1989) compared short-term (90 minutes after stretching) and long-term (three weeks) effects of contract-relax stretching (maximal contraction of plantarflexors for eight seconds, relaxation for two seconds, and passive stretch for eight seconds) on stress relaxation of ankle plantarflexors. There was no difference between short-term and long-term effects of PNF stretching. In the studies by Toft et al. (1989) and McNair et al. (McNair et al., 2000), peak torque of the plantarflexors sixty seconds after the start of the stress-relaxation phase was reduced by approximately 15% by PNF stretching (Toft et al., 1989), and 20% by static stretching (McNair et al., 2000). Similarly, peak torque of the hamstrings declined 18% after PNF stretching and 21% after static stretching in other studies by Magnusson et al. (Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996) If dynamic flexibility is required, the claim that PNF stretching provides a better flexibility technique than static stretching is still questioned.

PNF stretching is a complicated stretching technique with a combination of shortening contraction and passive stretching. Therefore, PNF stretching might be harmful as it has been found to increase blood pressure (Cornelius, Jensen, & Odell, 1995) and EMG activity (Osternig, Robertson, Troxel, & Hansen, 1987) during the contraction phases. Moreover, PNF technique needs some experience to be performed, and a partner is needed to help with stretching. When compared with other stretching techniques, the selection of PNF stretching prior to exercise is still being questioned. Further study is required to investigate the effects of PNF stretching on dynamic muscle properties (e.g., active and passive stiffness), performance, and muscle soreness

Table 3.4 The effects of PNF stretching on biomechanics and neuromuscular activity.

Reference	Trial design	Samples	Interventions	Outcome measures	Main results
Sady et al. (1982)	CCT	1. Control (*n* = 10). 2. Static (*n* = 10). 3. Ballistic (*n* = 11). 4. PNF (*n* = 12).	1. Static: 3 x 6 s. 2. Ballistic: repeated movements for 20 times. 3. PNF: 3 x 6 s. 3 days/week for 6 weeks.	ROM.	PNF and control group. S: ↑ ROM.
Toft et al. (1989)	PPT	10 men	Contract-relax (8 s maximum contraction, 2 s relax, 8 s static stretch) 6 times.	Stress relaxation.	NS
Magnusson et al. (1996)	CCT	7 women (one leg stretch one leg control) (hamstrings).	Static stretch (45s hold x 15-30s rest x 5times), twice daily, 20 consecutive days.	1. Stress relaxation. 2. Energy. 3. EMG. 4. ROM.	S: ↑ ROM.
Magnusson et al. (1996)	CCT	8 neurological intact and 6 spinal cord injury volunteers (hamstring).	Static stretch (hold 90s).	1.Stress relaxation. 2.Passive torque. 3.EMG.	NS

CCT = controlled clinical trial; RCT; PPT = pre- & post-test trial; ROM = range of motion; EMG = electromyography; SEC = series elastic components; S = significant; NS = non-significant.

3.6.4 Dynamic stretching

In an extensive review of warm-up and stretching (Shellock & Prentice, 1985), it was stated that “dynamic stretching is important in athletic performance because it is essential for an extremity to be capable of moving through a non-restricted range of motion" (pp.272). Unfortunately, there was no published research cited for any aspects of dynamic stretching.

Cyclic stretching, or passive continuous motion, has been demonstrated to be effective for decreasing passive muscle stiffness (McNair et al., 2000). A less stiff muscle is believed to absorb greater energy when forces are applied to it (McNair et al., 2000). As well, less muscle stiffness might be beneficial in reducing the severity of muscle soreness as research has shown the positive relation between passive stiffness and the severity of muscle soreness (McHugh, Connolly, Eston, Kremenic et al., 1999). However, there is no evidence that dynamic movement of stretching can reduce the severity of muscle soreness.

Dynamic stretching might be a useful protocol for increasing flexibility without decreasing athletic performance. Dynamic contraction of muscle throughout the range of motion is expected to decrease dynamic flexibility as indicated by passive muscle stiffness (McNair et al., 2000). Movement, without holding at end range of motion, may not reduce neuromuscular sensitivity. If the effect of decreasing passive stiffness, however, is more pronounced than the effects of neuromuscular sensitivity on performance, a reduction in performance might still occur. Further research is needed to elucidate the benefits of dynamic stretching on flexibility, muscle properties, neuromuscular sensitivity, performance, and injury prevention.

3.7 Combination of stretching with other therapies

3.7.1 Warm-up

Stretching is generally performed after warm-up (Shellock & Prentice, 1985). The theory is that warm-up will increase muscle temperature to help enhance tissue flexibility (Magnusson et al., 2000). Warm-up has been shown to increase tissue temperature but not to affect muscle properties (passive energy absorption) during stretching (Magnusson et al., 2000). In a study by Magnusson et al. (2000), warm-up (jogging) was performed at 70% of maximum O_2 uptake for 10 minutes and resulted in elevation of muscle temperature by 3°C. After warm-up, four static stretch manoeuvres of 90 s reduced passive energy absorption by 25% while five static stretching exercises (held for 90 s) at resting temperature (no warm-up) reduced passive energy absorption by 30% (Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996). However, there were no data on other parameters of passive muscle properties such as passive muscle stiffness and peak torque collected in this study. In the same way, warm-up (running (Stewart & Sleivert, 1998) and heel raising (Knight, Rutledge, Cox, Acosta, & Hall, 2001)) did not help to improve range of motion of lower limbs regardless of the warm-up intensity (60, 70, and 80% of VO_2 max) (Stewart & Sleivert, 1998) or training period (two, four, and six weeks) (Knight et al., 2001). Therefore, warm-up might not be an effective way to enhance flexibility of passive properties of muscle.

McNair and Stanley (1996) reported the effects of warm-up (treadmill jogging for 10 minutes at 60% of maximum age predicted heart rate) on series elastic muscle stiffness. Surprisingly, warm-up reduced active (series elastic component) stiffness (6%) more than the combination treatments of warm-up and stretching (3%) and stretching alone (-1%). Warm-up might be more effective in reducing resistance from various properties associated with active stiffness (passive joint properties, level of muscle activation, tendon properties, and the effect of stretch reflex) than stretching.

Warm-up might help to increase muscle relaxation by reducing EMG activity. Mohr et al. (1998) reported that post-warm-up EMG activity was significantly less than pre-warm-up EMG activity in gastrocnemius and soleus muscle. The authors proposed that muscle architecture and arrangement of connective tissue might influence the effects of warm-up (cycling) on reducing EMG activity of the gastrosoleus complex. EMG during stretching in the Mohr et al. (1998) study was slightly higher than previous reports (McHugh, Connolly, Eston, Kremenic et al., 1999; McHugh, Connolly, Eston, & Gleim, 1999; McNair et al., 2000) due to this study using needle electrodes and participants stretching by themselves (weight-bearing position). Participants in previous research were investigated for EMG activity by using surface electrodes and were passively stretched in a relaxed position. Therefore, an application of warm-up prior to stretching in order to reduce EMG activity might be important in some muscles (i.e., gastrocnemius and soleus) which are dense in connective tissue, and with some challenging stretch positions that need more control and balance of the body such as a standing bent knee stretch or a standing straight knee stretch with heel overhanging a step.

3.7.2 Heat and cold

Temperature has effects on muscle (Noonan & Best, 1993) and connective tissue (Warren, Lehmann, & Koblanski, 1976) in vivo. In a study of skeletal muscle tensile behaviour, warm muscle (40 °C, measured using an intramuscular probe) showed less stiffness and more load-to-failure than cold (35 °C) muscle (Noonan & Best, 1993). A study of rat tail tendon indicated that an elevated tissue temperature (45 °C), before the application of low force (one-quarter of full load to failure), produced greater residual length and reduced tissue damage (indicated by tissue rupture which was defined as the point at which elongation continued with no increase in load) than tissues with a normal temperature (Warren et al., 1976). The results of these animal studies (Noonan & Best, 1993; Warren et al., 1976) support the general practice by therapists of applying superficial heat before stretching in order to maximise the effectiveness of treatment.

In human, results of research on the combined treatment of stretching with either heat or cold on flexibility as measured by range of motion were inconclusive (Burke et al., 2001; Henricson et al., 1984; Taylor, Waring, & Brashear, 1995). In the study by Henricson et al. (1984), application of an electric heating pad (43°C) for 20 minutes before PNF stretching significantly increased range of motion of hip flexion and abduction. The stretching only group increased range of motion of hip flexion and external rotation, while the heat only group did not show any effect on range of motion. The stretching in this study was performed in only one direction (hip flexion) while the participants were investigated for hip flexion, abduction, and external rotation. Taylor et al. (1995) compared the effects of static stretch alone (held for one minute), heat (77°C) and static stretch, and cold (-18°C) and static stretch. There was a significant increase in hamstring length regardless of treatment but no significant difference between treatments. Similarly, the comparison among PNF stretching alone (isometric contraction for 10 s, five s rest for four times), PNF stretching and cold (immersed in a cold-water bath (8°C) for 10 minutes before performing the same PNF protocol), and PNF and heat (immersed in a hot-water bath (44°C) for 10 minutes before performing the same PNF protocol) for five consecutive days did not lead to any difference in hip range of motion (Burke et al., 2001). Knight et al. (2001) studied the effects of four treatments (static stretching alone, warm-up prior to static stretching, superficial moist heat for 15 minutes prior to static stretching, and seven minutes of continuous ultrasound prior to static stretching) on plantar flexor flexibility over six consecutive weeks. The use of ultrasound for seven minutes prior to stretching was the most effective treatment for increasing ankle dorsiflexion range of motion. Ultrasound may have provided a deeper heat at the muscular level (Draper & Prentice, 2002) compared to the hot pack or hot bath that might only increase skin temperature.

3.7.3 Massage

Stretching, warm-up, and massage are often performed in sport practice as treatment to prevent muscle injury. Rodenburg et al. (1994) reported that the combination of these treatments could reduce some negative effects of muscle soreness induced by eccentric exercise. The application of warm-up aimed to decrease viscosity of muscle tissues and stretching aimed to reduce passive tension. These two treatments were performed before eccentric exercise, and massage was performed after exercise with the aim of increasing blood flow and reducing waste products. The results, however, were not consistent as the maximal force, the flexion of elbow angle, and the creatine kinase level in blood were reduced, while other soreness parameters such as soreness sensation, extension elbow angle, and myoglobin in blood did not change. Therefore, the combination of these treatments did not reduce the severity of muscle soreness any more than any individual treatment. In other studies, warm-up was reported to be effective in reducing the severity of muscle soreness and functional loss (Nosaka & Clarkson, 1997), massage was effective in reducing soreness sensation (Bale & James, 1991; Smith & Jackson, 1990; Tiidus & Shoemaker, 1995), while stretching did not show any effect at all (High et al., 1989; Johansson et al., 1999; Lund et al., 1998a).

3.8 The effects of stretching on performance

Stretching is expected to increase flexibility, and, consequently, enhance sport performance (Gleim & McHugh, 1997). The effects of stretching on several performance parameters have been investigated including muscle strength, power, and endurance, as well as the efficiency of exercise such as running economy. However, recent research (Behm, Button, & Butt, 2001; Church et al., 2001; Fowles et al., 2000; Handel, Horstmann, Dickhuth, & Gulch, 1997; Young & Behm, 2003) still questions whether these interventions provide any benefit for performance (see Table 3.5).

Table 3.5 The effects of stretching on performance.

References	Trial design	Samples	Interventions	Outcome measures	Main results
Static stretching					
Kokkonen et al. (1998)	CBT	15 men & 15 women (hamstrings).	20 min stretching (5 stretches, 3 times assisted, 3 times unassisted, hold 15 s, rest 15 s).	1. Sit & reach score. 2. Maximum strength (1RM).	S: ↑ ROM (16%). S: ↓ strength (7.3%).
Fowles et al. (2000)	CBT	8 men, 4 women.	13 x 135 s static stretches, total 30 min.	1.MVC. 2.Twicth interpolation with EMG. 3. Twitch characteristics at pre, immediately post, 5, 15, 30, 45, 60 min post-stretching.	S: ↓ MVC (28, 21,13,12,10, and 9% (by the time to collect data)). S: ↓ Motor unit activation & EMG after treatment but recovered by 15 min.
Knudson et al.(2001)	CBT	10 men & 10 women (quadriceps, hamstrings, plantarflexors).	3 x 15 s static stretch.	1. Peak velocities. 2. Duration of concentric phase. 3. Duration of eccentric phase. 4. Smallest knee angle. 5.Jump height.	NS

Table 3.5 (continued) The effects of stretching on performance.

References	Trial design	Samples	Interventions	Outcome measures	Main results
Nelson et al. (2001)	Pre-Post	10 men and 5 women	One active and 3 passive stretching for 15 min.	Peak torque at 1.05, 1.57, 2.62, 3.67, and 4.71 rad.s^{-1}.	S: ↓ strength at 1.05 rad.s^{-1} (7.2%) and 1.57 rad.s^{-1} (4.5%).
Behm et al. (2001)	CBT	12 men	Quadriceps stretching (45s held, 15 s rest, for 5 sets).	1. MVC. 2. EMG. 3. Evoked torque. 4. Tetanic torque.	S: ↓ MVC (12%), muscle inactivation (2.8%), EMG (20%), evoked force (11.7%).
Cornwell et al. (2002)	CCT	10 men.	3 x 30 s static stretch.	1.Active muscle stiffness. 2.EMG. 3.Jump height.	S: ↓ jump height (7.4%). S: ↓ active stiffness (2.8%).
Young et al. (2003)	CBT	13 men, 4 women (quadriceps and plantarflexors).	2 x 30 s for each muscle.	1. Concentric force. 2. Concentric jump height. 3. Concentric rate of force developed. 4. Drop jump height.	S: ↓ concentric force (4%).
Laur et al. (2003)	CBT	16 men and 16 women (hamstrings).	3 x 20 s static stretching.	Perceived exertion.	S: ↑ perceived exertion.
Ballistic stretching					
Nelson et al. (2001)	CBT	11 male and 11 female college students.	Ballistic stretch: 15 bob up and down once per min.	1. Sit & reach score. 2. Maximum strength (1RM).	S: ↑ ROM (7.5%). S: ↓ strength (7.3%).

Table 3.5 (continued) The effects of stretching on performance.

References	Trial design	Samples	Interventions	Outcome measures	Main results
PNF stretching (short term)					
Wiktorsson-Moller et al. (1983)	CBT	8 healthy males.	PNF: isometric contraction 4-6 s, relax 2 s, passive stretching 8 s.	1. ROMs of lower extremities. 2.hamstrings and quadriceps strength.	S:↑ ROM of ankle dorsiflexion and plantarflexion, hip flexion, extension, abduction, knee flexion.
Church et al. (2001)	CBT	40 women.	1. Static stretching. 2. PNF.	1.Vertical jump. 2. ROM.	PNF S: ↓ jump height (3%).
PNF stretching (long term)					
Wilson et al. (1992)	CCT	16 male weightlifters (*n* = 9 in experiment group, *n* = 7 in control group).	Flexibility training (6-9 rep) of upper extremities, 10-15 min per session, twice a week for 8 weeks.	1.Rebound bench press (RBP). 2.Purely concentric bench press (PCBP).	S: ↑ ROM (3%). S: ↑ RBP (5.4%). S: ↓ SEC stiffness (7.2%).
Worrell et al. (1994)	CCT	19 participants with short hamstrings (one leg static – one leg PNF)	Static-15 s held, 15 s rest PNF-5 s isometric, 5 s rest 4 rep per day, 5 days/week, 3 weeks	1. ROM 2. Con/ecc strength	S: ↑ strength Ecc-60 & 120°.s^{-1}. Con- 120°.s^{-1}.

Table 3.5 (continued) The effects of stretching on performance.

References	Trial design	Samples	Interventions	Outcome measures	Main results
Hunter and Marshall (2002)		60 participants (15 per group).	Static tretching (3 x 20 s) and PNF (submaximal contraction 10 s).	1.Drop jump. 2.Countermovement jump.	NS

CCT = controlled clinical trial; RCT = randomised controlled trial; PPT = pre- & post-test trial; CBT, counterbalance trial; ROM = range of motion; EMG = electromyography; SEC = series elastic components; S = significant; NS = non-significant; MVC = maximum voluntary contraction; con = concentric contraction; ecc = eccentric contraction; RM = repetitive maximum.

3.8.1 Muscle strength, power, and endurance

The acute effects of static stretching, ballistic stretching, and PNF can reduce muscle strength as determined by maximum lifting capacity (Church et al., 2001; Kokkonen et al., 1998; Nelson & Kokkonen, 2001) and isometric contraction force (Fowles et al., 2000). Nelson et al. (Nelson, Guillory et al., 2001) reported that a decrease in muscle strength for slow velocities of movement after static stretching, and decreases in performance of functional high velocity movements, such as jumping, after static stretching (Cornwell et al., 2002; Knudson et al., 2001; Nelson, Guillory et al., 2001; Young & Behm, 2003). The negative acute effect of stretching on performance is probably explained by the change in neuromuscular transmission and/or biomechanical properties of muscle. Several studies of the effect of stretching on performance have demonstrated a reduction of performance associated with a decrease in neural activation (H-reflex amplitude) (Avela et al., 1999; Fowles et al., 2000; Vujnovich & Dawson, 1994). Some stretching research has reported an increase in muscle compliance as investigated by range of motion (Halbertsma et al., 1996; McNair & Stanley, 1996; Taylor et al., 1995; Warren et al., 1976; Wiktorsson-Moller et al., 1983), active muscle stiffness (Cornwell et al., 2002; Wilson et al., 1992), and passive stiffness (Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996).

A study by Fowles et al. (2000) assessed strength performance after prolonged stretch (13 maximal stretches, two minutes and 15 seconds hold, and five seconds rest) by measuring force, EMG activity, and passive stiffness. Strength was lost maximally immediately after stretch (28%) and lasted more than one hour after stretch (9%). Interestingly, muscle activation and EMG activity was significantly depressed after stretching but was recovered by 15 minutes. Passive stiffness recovered quickly after stretching at 15 minutes but did not fully recover within one hour. The results imply that the impaired muscle activation was responsible for strength loss after prolonged stretching in the early phase while impaired contractile force was responsible for strength loss throughout the entire period. This implication is consistent with the results of several studies where the mechanism responsible for a decrease in performance after acute stretching is likely to be caused by neuromuscular inhibition (Behm et al., 2001; Cornwell et al., 2002). Behm et al. (2001) investigated the effects of static stretching (held for 45 seconds, and rest for 15 seconds for five times) on voluntary and evoked force, and EMG activity of quadriceps. Maximal voluntary and evoked contraction decreased similarly by 12%, and muscle activation and EMG activity decreased 2.8% and 20%, respectively. Similarly, Cornwell et al. (2002) reported that static stretching of gastrosoleus (180 s) reduced jump height by 7.4% but active stiffness was reduced by only 2.8%. Other studies reported that static stretching reduced jumping performance (knee bend) by 3% (Knudson et al., 2001; Young & Behm, 2003). A reduction in jumping performance was consistent with a reduction of EMG activity (Young & Behm, 2003), but there were no changes in biomechanical variables (vertical velocity, knee angle, duration of concentric and eccentric phases) (Knudson et al., 2001). The prolonged stretch in Fowles et al.’s (2000) study (75 seconds for 13 times) might have increased muscle compliance more than in any other study as there was evidence that static stretching held for 90 seconds for five times could decrease muscle stiffness (Magnusson, Simonsen, Aagaard, Sorensen et al., 1996; Magnusson, Simonson et al., 1996). The shorter duration could not change passive muscle properties (Halbertsma et al., 1996; Magnusson et al., 2000) therefore, the change in muscle compliance and muscle inactivation in Fowles et al.’s (2000) study caused more strength loss (28%) than that reported by Behm et al. (2001) (12%).

The detrimental effects of acute stretching exercise on muscular endurance was shown by Laur et al. (2003) when the application of acute stretching reduced the maximal number of repetitions performed with a submaximal load, and also produced higher perceived exertion scores. Although the magnitude of reduction was small, it was statistically significant.

Interestingly, studies on the effects of long-term stretching reported a positive effect of stretching on performance (Handel et al., 1997; Wilson et al., 1992; Worrell et al., 1994). Three weeks of flexibility training in both PNF and static stretching increased peak torque of hamstrings eccentrically (at 60°/s and 120°/s) and concentrically (at 120°/s only) (Worrell et al., 1994). PNF training (contract-relax technique) for eight weeks increased maximum torque of knee flexors and extensors (1997). The increase in muscle strength of knee flexors was significant at all velocities and might be due to the contraction phase of the PNF stretching technique showing the same effect as isometric muscle training. Therefore, knee flexors, which are used in normal activity less than knee extensors, showed more increase in muscle strength. More functionally, Wilson et al. (1992) reported that eight weeks of static flexibility training increased rebound bench press performance by 5.4%, in accordance with a decrease in active muscle stiffness by 7.2%. The authors proposed that flexibility-induced performance enhancement might result from increased musculotendinous compliance facilitating the use of energy strain in stretch short-cycle activities. In contrast with the acute stretching, Hunter and Marshall (2002) reported that the combination of static and PNF training for ten weeks did not result in a detrimental effect on countermovement jump and drop jump, but helped to increase knee joint range of motion (Hunter & Marshall, 2002). Therefore, flexibility training of at least three weeks is beneficial to some performance factors as indicated by increased range of motion and muscle strength. Unfortunately, there is no research on the effects of flexibility training on neuromuscular activity.

There are no studies on the effects of acute stretching after prolonged flexibility training. It would be questionable whether the negative effects of acute stretching will attenuate the positive effects of flexibility training because the athletes, who commonly undertake flexibility training, also perform stretching before competition. There are also no studies on the effects of dynamic stretching on performance. As research (Behm et al., 2001; Church et al., 2001; Fowles et al., 2000; Handel et al., 1997; Young & Behm, 2003) reported the detrimental effects on athletic performance of all stretching techniques (static, PNF, and ballistic), dynamic stretching might be a useful protocol to increase flexibility without decreasing athletic performance.

3.8.2 The efficiency of exercise

Flexibility is considered to play an important role in the efficiency of movement (Gajdosik et al., 1990) by enabling the use of elastic potential energy in muscle (Gleim & McHugh, 1997). The more compliant muscle-tendon unit needs more contractile force to transmit to the joint and, consequently, causes a greater delay in external force generation (Gleim & McHugh, 1997). A stiffer muscle would provide a more efficient transmission of contractile force production (Gleim & McHugh, 1997), but this contradicts the aim of stretching, which intends to increase muscle-tendon unit compliance.

Craib et al. (1996) reported that less flexible runners have showed a reduced aerobic demand during running (better running economy). The positive and significant correlation between range of motion and the aerobic demand of running presented in only two movements (external hip rotation and dorsiflexion) accounted for 47% of the variance observed in running economy. Craib et al.'s (1996) study was cross-sectional, did not control the training programme of the runners, and did not consider other factors that might have influenced running economy such as kinematic, anthropometric, physiological, and cellular variables. In contrast, the flexibility training of hip flexors (three weeks) (Godges, MacRae, & Engelke, 1993) and lower leg muscles (quadriceps, hamstrings, and gastrosoleus) (ten weeks) (Nelson, Kokkonen, Eldredge, Cornwell, & Glickman-Weiss, 2001) resulted in increased range of motion but had no effect on running economy. None of the published papers in this area reported the effects of flexibility on running time, stride length, stride frequency, or the perception of fatigue. The optimal level of flexibility (static and dynamic) for running performance needs to be researched.

3.9 The effects of stretching on injury prevention

3.9.1 Rate of injury

Despite performance of stretching generally being recommended before exercise to reduce the risk of injury, a recent review of the effects of stretching on the incidence of injury indicated inconclusive results (Weldon & Hill, 2003). The inconclusive results might be due to exercise-related injury being a complex phenomenon with physiological, psychological, and environmental factors involved. The majority of research in this area has been retrospective and does not provide a clear relationship between flexibility and injury (Dubravcic-Simunjak, Pecina, Kuipers, Moran, & Haspl, 2003; Gleim & McHugh, 1997; Watson, 2001).

In one prospective study, Van Mechelen et al. (1993) provided a standardised program of stretching exercises to runners and assessed the number of injuries after 16 weeks. There was no reduction in injury incidence per 1000 hours of running between the experimental (standardised program of stretching exercise) and the control (no stretching information) groups. In a study of army recruits (Pope, Herbert, & Kirwan, 1998; Pope, Herbert, Kirwan, & Graham, 2000), a preexercise stretching programme of 11-12 weeks did not reduce the risk of exercise-related injury. Fitness and age (Pope et al., 2000), and the early detection of symptoms of overuse injuries (van Mechelen et al., 1993) were more important factors for injury rather than the stretching exercise. An increase in range of motion (or static flexibility) resulting from stretching may not be necessary for a majority of sports such as running and swimming which do not require extreme ranges of motion. Dynamic flexibility might be more important because it represents the resistance of the muscle-tendon unit during movement. However, there is no research on the relationship between dynamic flexibility and rate of injury.

3.9.2 Muscle damage

In contrast to the general belief that stretching helps reduce the risk of muscle damage from unaccustomed eccentric exercise, previous research reported that both prolonged static and ballistic stretching (60 s for two stretches for each muscle) induced muscle damage (Smith et al., 1993). Moreover, no research has reported any benefit from stretching on the severity of muscle damage or muscle soreness either before (High et al., 1989; Johansson et al., 1999; Lund et al., 1998a) or after (High et al., 1989) exercise. The effects of stretching before and after eccentric exercise have been reviewed recently (Herbert & Gabriel, 2002). The lack of evidence for the benefits of stretching may be due to previous research only investigating static stretching. If the decrease in passive muscle stiffness is the key to reducing the severity of muscle damage, static stretching in these studies (High et al., 1989; Johansson et al., 1999;

Lund et al., 1998a) was not held long enough to induce a decrease in passive stiffness (most studies used stretches held less than 90 s). Interestingly, a study by McNair et al. (2000) reported that passive dynamic stretching did reduce muscle stiffness, while a study by McHugh et al. (1999) reported that passive stiffness was related to the severity of muscle damage measured by strength loss, pain, muscle tenderness, and creatine kinase activity. Therefore, any stretching technique that can reduce passive stiffness might help to reduce the severity of muscle damage. To date, there are no published papers reporting the effects of dynamic stretching on the severity of muscle damage. The effects of other stretching techniques such as ballistic and PNF on the severity of muscle damage have also not been determined.

3.10 Summary and conclusions

Despite stretching commonly being performed before exercise to enhance performance and reduce the risk of injury, there is limited scientific data to support the suggested benefits of stretching. Static and ballistic stretching have been shown to have detrimental effects on muscle strength and functional performances such as jumping, and to have inconclusive effects on the incidence of injury, no effects on the severity of muscle damage. Even though research has indicated that stretching is an effective treatment to increase static flexibility (range of motion), the effects on dynamic flexibility (muscle stiffness) are inconclusive given the variation of the length of hold and the number of repetitions used in studies. The aim of stretching is to increase flexibility, but does flexibility help to enhance performance? The ideal flexibility for the performance of each sports activity is different. Compliant muscle might be beneficial to eccentric contraction while stiffer muscle might be more suitable for concentric and isometric contractions. Does flexibility help to reduce the rate of injury? The majority of research does not support this statement. In fact, the majority of movement in sports requires repetitive movements within the normal range of motion. An increase in range of motion, therefore, is not necessary. The aim to reduce resistance during repetitive movement might be more beneficial in terms of increasing quality of movement and reducing the risk of overuse injury. Practically, the optimal level of flexibility is required because the increase in flexibility (more compliant muscle) might not benefit performance but may help to reduce the risk of injury. The compliant muscle-tendon unit absorbs and requires more energy to shorten, and consequently delays and reduces external force production. Nevertheless, the increase in ability to absorb energy in the compliant muscle might help to reduce the mechanical overload on muscle fibres, and consequently reduce the risk of muscle injury and the severity of muscle damage. Further research is needed to investigate the appropriate stretching techniques and the optimal level of flexibility which can maintain or improve performance, or which can prevent injury.

3.11 Recommendations

In order to clarify the effects of stretching, further research is recommended to:

- Provide information on the relationship of dynamic flexibility, performance, and rate of injury.
- Examine the effects of several stretching techniques such as ballistic, PNF, and dynamic stretching on dynamic flexibility and neuromuscular sensitivity.
- Compare the effects of several stretching techniques such as static, ballistic, PNF, and dynamic stretching on different types of performance, the severity of muscle soreness, running economy, and rate of injury.
- Study the effects of acute stretching after long-term flexibility training.
- Provide more information on the appropriate flexibility level to enhance performance and reduce the risk of injury.

CHAPTER 4

PREVENTATIVE STRATEGIES FOR EXERCISE INDUCED MUSCLE DAMAGE

Weerapong, P., Hume, P. A., & Kolt, G. S. (2004). Preventative strategies for exercise-induced muscle damage. *Critical Reviews in Physical and Rehabilitation Medicine, 16,* 133-150.

4.1 Summary

Eccentric exercise is part of regular rehabilitation and sports training. Unaccustomed eccentric exercise causes muscle damage that presents as delayed soreness, strength and range of motion loss, swelling, and increased passive stiffness. These symptoms reduce the ability to exercise and might be harmful if further exercise is continued. Several interventions such as warm-up, stretching, massage, acupuncture, anti-inflammatory drugs, and oestrogen supplements have been researched in order to find interventions that successfully alleviate the severity of muscle damage. The results are inconclusive due mainly to the variety of exercise-induced muscle damage protocols, the types of intervention protocols, and the doses of application. From a practical point of view prevention strategies are preferred by practitioners because they reduce time lost from training, reduce the cost of treatment, and reduce the risk of further injury. For that reason, this article emphasises the mechanism of initial events and the factors involving the severity of muscle damage. Research on the prevention of eccentric exercise-induced muscle damage is reviewed and discussed. Appropriate preventative strategies on muscle damage from eccentric exercise are suggested.

4.2 Introduction

Muscle damage resulting from exercise commonly occurs when the exercise involves an unaccustomed “lengthening” eccentric contraction. This type of exercise is difficult to avoid for several reasons. First, rehabilitation practitioners often prescribe eccentric exercise in the initial stage of treatment in rehabilitation programmes because it elicits less fatigue and pain than concentric contraction at the same workload (Hollander et al., 2003; Horstmann et al., 2001). Second, coaches usually combine concentric and eccentric contraction in training programmes because this provides greater strength gains than concentric exercise alone, particularly when the eccentric exercise is emphasised (Hilliard-Robertson, Schneider, Bishop, & Guilliams, 2003). As well, eccentric contraction is also a part of regular exercise when smooth movement is required. Unaccustomed eccentric exercise appears to result in muscle damage and, consequently, causes soreness sensation and strength and functional movement loss. The symptoms of muscle damage usually subside within a week and might not affect daily living in healthy individuals. However, when the symptoms of muscle damage occur in patients undergoing rehabilitation, or in athletes, this might perturb the rehabilitation or training programme, as well as sport performance. Several researchers have investigated interventions thought to reduce and/or prevent the severity of muscle damage. From a clinical point of view, preventative intervention is preferred because it reduces the cost of treatment, time lost from training or rehabilitation, and the likelihood of sustaining further injury, and it also maintains the ability to exercise. Therefore, this chapter reviews the initial mechanisms of muscle damage, analyses related factors, and proposes possible strategies for prevention. Previous research related to preventative strategies is discussed.

In this article, the term “eccentric exercise” is used to indicate the type of exercise that induces muscle damage. The term “severity of muscle damage” indicates the magnitude of the symptoms after eccentric exercise, including soreness sensation, strength and range of motion loss, swelling, and tenderness. In terms of muscle damage from eccentric exercise, soreness sensation has been the major concern in earlier research (High et al., 1989; Hilbert et al., 2003; Johansson et al., 1999) because the symptoms after eccentric exercise are usually termed “delayed-onset muscle soreness”.

However, other symptoms, such as strength and range of motion loss, need to be considered as they might have more impact on athletes or patients than soreness sensation, and they may take longer to recover. This chapter considers the other symptoms of muscle damage such as strength and range of motion loss, swelling, tenderness, and stiffness, along with soreness sensation after exercise-induced muscle damage.

Several reviews of eccentric exercise-induced muscle damage have been published in the past 15 years, that contain information on the mechanisms and aetiology of exercise-induced muscle fibre injury, muscle function and adaptation after exercise-induced muscle damage, treatment strategies, and performance factors (Armstrong, 1990; Armstrong et al., 1991; Cheung et al., 2003; Clarkson & Hubal, 2002; Clarkson et al., 1992; Clarkson & Sayers, 1999; Friden & Lieber, 1992; Tiidus, 1999). No reviews, however, have dealt with preventative strategies for muscle damage from eccentric exercise.

Literature for this review was located using three electronic databases (PubMed, SPORT Discus, and ProQuest 5000 International) in addition to manual journal searches. The electronic databases provided access to biomedical and sports-related journals, serial publications, books, theses, conference papers, and related research published since 1985. The key phrases used to search the databases included: delayed onset muscle soreness (DOMS), mechanisms of DOMS, sports performance and DOMS, DOMS treatments, DOMS and prevention, DOMS and exercise, and repeated bout effect. Articles not published in English and/or in scientific journals, articles that focused on the psychological effects of DOMS, or articles on the effects of treatments after performing exercise-induced muscle damage were not included in this review. The criteria for inclusion were:

- Articles must have used normal, healthy participants. Age, gender, and fitness differences were not excluding factors
- Articles must have discussed the possible mechanisms of initial events of DOMS, factors inducing DOMS, and the impacts on exercise performance
- Articles must have investigated preventative interventions in reducing the severity of DOMS.

4.3 Signs and symptoms of muscle damage

Even though eccentric contractions result in less fatigue and pain than concentric contractions immediately after exercise (Hollander et al., 2003; Horstmann et al., 2001), eccentric contractions have been shown to produce specific and long-lasting effects on soreness sensation and muscle functions (Newham, Mills, Quigley, & Edwards, 1983; Sayers, Knight, & Clarkson, 2003; Whitehead, Allen, Morgan, & Proske, 1998). It is well known that eccentric exercise causes muscle damage (Armstrong et al., 1991; Clarkson & Hubal, 2002; Clarkson & Sayers, 1999; McHugh, Connolly, Eston, Kremenic et al., 1999). The specific and long-lasting effects resulting from lengthening contractions (a contraction that happens when an external torque is greater than an internal torque within muscle) include delayed onset soreness sensation, prolonged strength loss, reduced range of motion, and increased passive stiffness. Several reviews have described the nature and time course of these changes (Clarkson et al., 1992; Ebbeling & Clarkson, 1989). In brief, soreness sensation appears 24 hours after exercise, peaks at 2-3 days after exercise, and slowly recovers but does not fully subside until 8-10 days after exercise (Clarkson et al., 1992). Strength dramatically reduces immediately after exercise, and slowly recovers to the level of 80% of the preexercise strength ten days after exercise (Clarkson et al., 1992). Range of motion reduces as the damaged muscles shorten spontaneously (decreased relaxed muscle length) and are unable to fully contract voluntarily (decreased active range of motion). The spontaneous muscle shortening occurs immediately after exercise and peaks on day three after exercise. Muscle loses the ability to contract maximally immediately after exercise and gradually recovers. The loss of range of motion measurements appear to return to baseline within ten days after exercise (Clarkson et al., 1992). The increase in passive stiffness peaks on day two after exercise and returns to preexercise values on day ten (Howell et al., 1993). To illustrate the changes, the time course of soreness sensation, range of motion, muscle strength, and passive stiffness from earlier literature is presented in Figure 4.1.

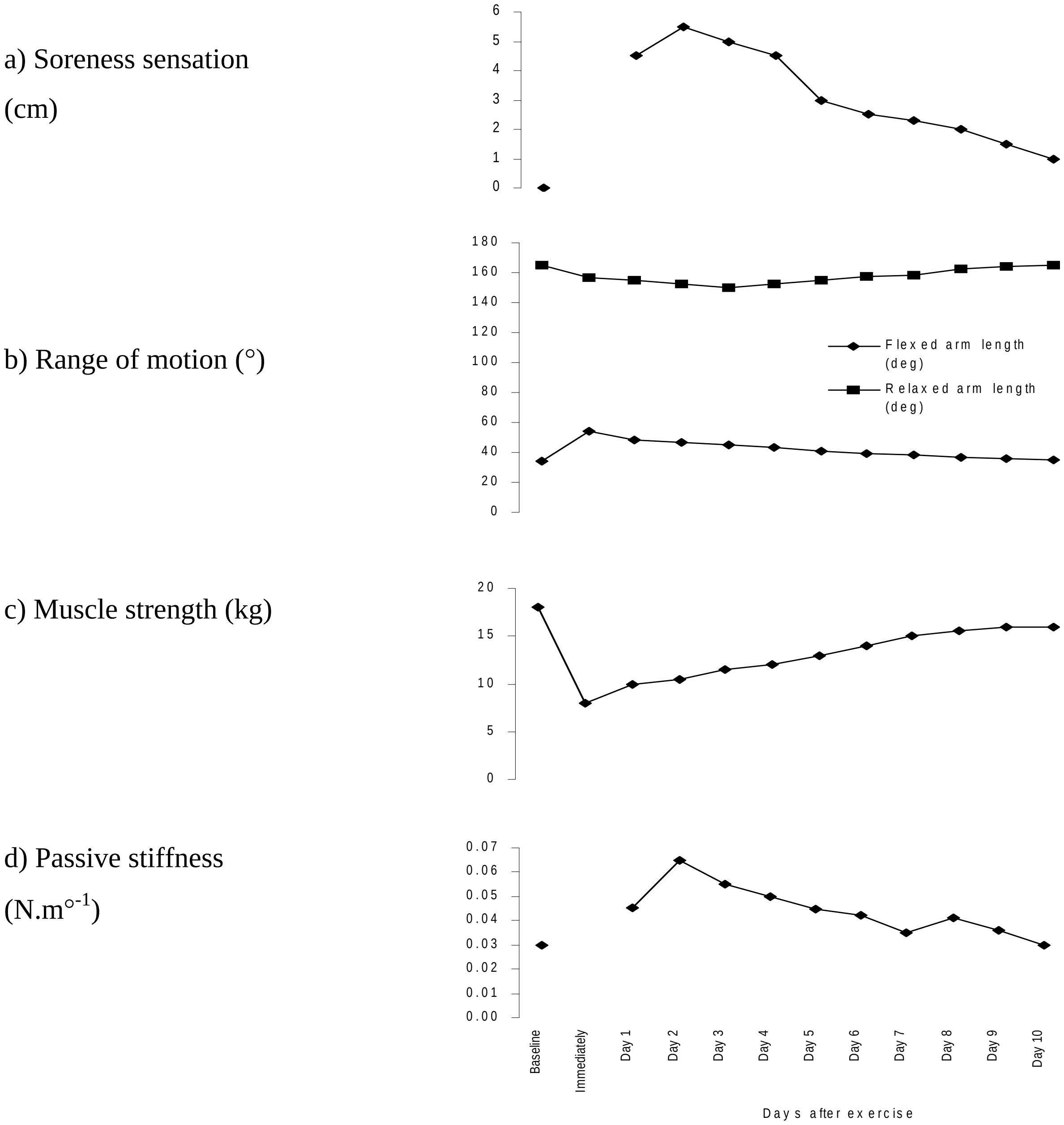


Figure 4.1 The parameters indicating the severity of muscle soreness (soreness sensation, range of motion, muscle strength, and passive stiffness) in elbow flexor muscles. Data was compared between preexercise, immediately post exercise, and follow-up for 10 days. Data were modified from Clarkson et al. (1992) and Howell et al. (1993).

Careful interpretation of the data in Figure 4.1 is needed as they are based on muscle damage to the elbow flexors (from two sets of 35 maximal eccentric contractions with five minutes rest between each set) (Clarkson et al., 1992; Howell et al., 1993). The time course and the severity of muscle damage in other muscles with different exercise-induced muscle damage protocols would be slightly different (Brown, Child, Donnelly, Saxton, & Day, 1996; McHugh, Connolly, Eston, Kremenic et al., 1999; Vickers, 2001). It is clear, however, that the adverse effects of eccentric exercise last longer than ten days and have an impact on functional movements.

4.4 Impact of muscle damage on sports performance

Strength, range of motion, and a feeling of competence are important factors in performing sports and exercise. Unaccustomed eccentric exercise causes adverse effects on these factors (Bourgeois, MacDougall, MacDonald, & Tarnopolsky, 1999; Brown et al., 1996; Chleboun et al., 1995; Cleak & Eston, 1992; Crenshaw, Karlsson, Styf, Backlund, & Friden, 1995; McHugh, Connolly, Eston, Kremenic et al., 1999). Moreover, soreness sensation is aggravated during movements especially when the eccentric contraction is involved. Eccentric exercise, therefore, may cause significant reduction in performance during training and competition and/or increase the risk of further injury. The impact of muscle damage from eccentric exercise on athletic performance in terms of biomechanics has been reviewed recently by Cheung et al. (2003). The perception of functional impairment, joint kinematics, strength and power, altered recruitment patterns, and injury risk factors in healthy adults were emphasised.

Despite obvious changes in soreness sensation and biomechanical properties of muscle, reviews on the impact of muscle damage resulting from eccentric exercise on physiological and metabolism parameters are controversial. Dolezal et al. (2000) reported an increase in resting metabolic rate at 24 hours (18%) and 48 hours (11%) after eccentric exercise. The muscle damage was induced by using a leg press exercise for eight sets at the subjects’ 6-repetitive maximum. The symptoms of muscle damage were similar in trained and untrained groups except that the untrained group showed a higher magnitude in creatine kinase and soreness sensation. Walsh et al. (2001), however, reported that after eccentric exercise (eccentric cycling for 30 minutes), muscle oxygen utilisation and local oxygen transport (measured by near infrared spectroscopy), and muscle fibre respiration at rest (measured from biopsy) of the vastus lateralis muscle were unchanged.

In addition, previous research has reported an increase in physiological and metabolic responses from healthy, untrained men indicating exercise stress parameters (i.e., minute volume, breathing frequency, respiratory exchange ratio, heart rate, rating of perceived exertion, venous blood lactate concentration, and plasma cortisol concentration) during cycling two days after eccentric exercise (Gleeson, Blannin, Walsh, Field, & Pritchard, 1998; Gleeson, Blannin, Zhu, Brooks, & Cave, 1995). Even though the changes in these physiological parameters indicated an earlier onset of fatigue and more stressful exercise than in normal circumstances, the cycling efficiency as measured by oxygen uptake (Gleeson et al., 1998; Gleeson et al., 1995) and time to exhaustion (Gleeson et al., 1998) were not different. The results of the Gleeson et al. (1998; 1995) study might be due to the muscle damage in these two studies being induced by bench stepping (involving mainly the quadriceps) (Hamill & Knutzen, 1995). The subsequent exercise test for the efficiency was cycling (involving all muscles of the legs, i.e., quadriceps, gluteus maximus, gastrocnemious, hamstrings, dorsiflexors) (Hamill & Knutzen, 1995). In fact, if athletes perform eccentric exercise with several muscles during training, it is possible that the exercise efficiency on the subsequent day might be impaired. This notion is supported by Calbet et al. (2001) who reported an increase in running cost (8%) 48 hours after a duathlon (5 kilometres running, 16 kilometres cycling, and 2 kilometres running). However, seven days after the duathlon, the running economy had nearly returned to the baseline.

4.5 The importance of preventative strategies to reduce muscle damage

Coaches, athletes, and medical practitioners are well aware of the symptoms of muscle damage after eccentric exercise because it clearly affects subsequent exercise or performance (i.e., strength, power, range of motion, and probably exercise economy). If muscle damage does occur, the following problems are of importance:

- How long is an appropriate recovery period?
- What is (are) the main factor(s) to determine when an athlete can start their training again?
- How much does the treatment cost?
- Is an athlete likely to get a related injury such as a muscle tear if the rest time is not enough?
- Are there any long-term effects from muscle damage if such damage happens repeatedly?
- How much do the symptoms of muscle damage affect performance?
- Is a reduction of soreness sensation a good indicator to recommence training? Muscle weakness requires longer recovery and might have more impact on performance than soreness sensation.

To avoid the above problems, preventative strategies are required. A comprehensive understanding of the initial mechanisms of exercise-induced muscle damage is necessary to investigate appropriate intervention. This chapter, therefore, reviews briefly the initial event of exercise-induced muscle damage and the factors that contribute to the magnitude of muscle damage.

4.6 The mechanisms and factors contributing to muscle damage

4.6.1 Initial events of muscle damage from lengthening exercise

Many theories have been proposed to explain muscle damage including inflammation (Smith, 1991), muscle spasm (deVries & Housh, 1996), metabolism (Evans & J. Cannon, 1991), and mechanical theories (Armstrong, 1984). Recently, several authors reviewed the initial events of exercise-induced muscle damage (Clarkson & Hubal, 2002; Lieber & Friden, 1999; Morgan & Allen, 1999; Proske & Morgan, 2001) and stated that the mechanical theory appears to be the most likely explanation.

Friden and Lieber (1992) hypothesised that high tension from eccentric contraction might stretch or break the intermediate filaments (desmin and dystropin). The intermediate filaments are responsible for maintaining the sarcomeres in register/alignment (Morgan & Allen, 1999) and may fail when the nonhomogeneous lengthening of sarcomeres occurs (Armstrong et al., 1991). The disruption of these intermediate filaments would expose degraded Z-proteins and globular proteins, and then activated lysosomal enzymes, causing further degradation and eventually Z-disk dissipation (Faulkner et al., 1993). Changes in ultrastructure such as focal damage have been observed using electron microscopy (Stupka et al., 2000). Clarkson and Hubal (2002) described the changes in ultrastructural integrity as “Z-line streaming, Z-lines out of register, loss in thick myofilaments, loss in mitochondria in areas that showed abnormalities, and disturbed arrangement of filaments at the A-band” (p. S53). In addition, Abraham (1977) reported an increase in the ratio of hydroxyproline/creatinine (OHP/Cr) in 24 hr urine collection which indicated the disruption of the connective tissue elements in the muscles and/or their attachments. In an animal study, however, Hasselman et al. (1995) showed the disruption of both muscle fibres and connective tissue only at a high intensity of exercise (actively stretched at 10 cm/s to 90% of the force required to fail). A lower intensity of exercise (70% and 80% of the same relative force) showed only evidence of muscle fibre disruption.

Proske and Morgan (2001) described the series of events leading to muscle damage from eccentric exercise suggesting that lengthening contraction with mechanical overload from eccentric exercise caused overstretched sarcomeres. The sarcomeres are disrupted and cause the optimum length to shift to a longer length. When the disrupted sarcomeres are spread widely membrane damage occurs. The damage of muscle fibres in series (sarcomeres) and membrane (connective tissues) causes local contracture and increases in stiffness. The damage of muscle fibres and connective tissues lead to several symptoms of muscle damage such as strength and range of motion loss, tenderness, oedema, and soreness (Proske & Morgan, 2001).

4.6.2 Factors related to the magnitude of muscle damage

Several researchers have investigated the factors involved in the magnitude of muscle damage (Abraham, 1977; MacIntyre et al., 2000; Prior et al., 2001; Stupka et al., 2000). Information that has been reported is useful for coaches and health care practitioners in designing eccentric exercise programs at early stages of training or rehabilitation sessions in order to minimise muscle damage.

A number of factors have been linked to the severity of muscle damage. An individuals' characteristics such as muscle strength or physical fitness level are not related to the response to eccentric exercise-induced muscle damage (Clarkson & Hubal, 2002). Genetics, as investigated in research on identical twins, is also not a predisposing factor in the severity of muscle damage (Giulbin & Gaffney, 2002). Muscle properties, such as passive stiffness, have been reported as having a positive relationship with the magnitude of muscle damage (McHugh, Connolly, Eston, Kremenic et al., 1999). In a study by Leivseth et al. (1993), the stiffer muscles demonstrated less sarcomere numbers and muscle length and increased mechanical energy loss during passive movement than did normal muscles. Therefore, stiff muscles might reduce the ability to withstand elongation and may be more likely to develop injuries (which generally occur during muscle elongation) (Leivseth et al., 1993). Nevertheless, there are no published studies on the relationship between other properties of muscle on the severity of muscle damage such as active stiffness, tendon stiffness, creep, and stress relaxation.

Several mechanical factors involving exercise prescription have been investigated such as velocity (Kulig, Powers, Shellock, & Terk, 2001), intensity (Nosaka & Newton, 2002b ; Nosaka, Newton, & Sacco, 2002a; Tiidus & Ianuzzo, 1983), duration of exercise (Tiidus & Ianuzzo, 1983), resting period (Teague & Schwane, 1995), starting position (Newham, Jones, Ghosh, & Aurora, 1988; Nosaka & Sakamoto, 2001), and muscle attachment (Prior et al., 2001). Kulig et al. (2001) compared fast and slow eccentric movement of elbow flexors at the same work load (60% 1RM) and duration (144 seconds). Fifty-eight percent of participants reported soreness sensation in the arm that performed fast movement while none of the participants reported soreness sensation in the arm that performed a slow protocol. The results are questionable due to the exercise muscles (elbow flexors) and the exercise protocol (Fast: 2 seconds eccentric movement plus 2 seconds concentric movement per one repetition, 36 repetitions; Slow: 10 seconds eccentric movement plus 2 seconds concentric movement per one repetition, 12 repetitions). Elbow flexors consist of both one-joint (brachialis) and two-joint (biceps brachii) muscles that are recruited differently at different velocities. The brachialis was more active during the slow movement protocol while the biceps brachii were more active during the fast protocol (Kulig et al., 2001). Kulig et al. (2001) did not compare the same muscle when comparing the two velocities. The number of repetitions might be a more important factor in the severity of muscle damage than the velocity of exercise. As a result, the recommendation to decrease the velocity of eccentric exercise to reduce the severity of muscle soreness is questionable.

At the same exercise velocity, biarticular and monoarticular muscles respond to the same eccentric exercise protocol differently. Prior et al. (2001) investigated muscle injuries in four muscles (vastus lateralis, vastus medialis, vastus intermediate, and rectus femoris) by using magnetic resonance. The four muscles were activated equally during eccentric exercise (similar muscle transverse relaxation of water protons [T_2]) but the rectus femoris showed a greater level of muscle injury on the following days (greater delayed T_2) than the other muscles. The results showed that the biarticular muscle was likely to experience more damage than the monoarticular muscle when exercised at the same velocity. Prior et al. (2001) proposed that the greater injury to rectus femoris might have been due to the position of exercise. In this study, participants sat on the seat and exercised their quadriceps eccentrically. The seated position might restrict the ability of rectus femoris to transfer force between the hip and knee joints. Interestingly, the results also indicated that the level of muscle activation during exercise was not a unique determinant of muscle injury.

The starting position of exercise and/or the initial muscle length is another factor thought to be involved in the magnitude of muscle damage. Newham et al. (1988) and Nosaka and Sakamoto (2001) compared two different starting positions with the same range of motion (short and long muscle range, 50-130° and 100-180°, respectively). The participants reported more soreness and demonstrated more strength and range of motion loss, and more swelling in the long muscle length condition than in the short muscle length condition. The images from the magnetic resonance and ultrasound also showed greater damage in the long muscle length condition (Nosaka & Sakamoto, 2001). At a longer muscle length, both bicep brachii and brachialis contributed to torque while at the shorter length, brachialis had the greater contribution (Kawakami et al., 1994). As a result, the more muscle involved in the exercise protocol, the more muscle damage that occurred.

Other factors that may affect the magnitude of muscle damage include intensity of exercise, number of repetitions, and the length of rest period between each set. Previous research on the severity of muscle damage has agreed that intensity of exercise is the most important factor (Nosaka & Newton, 2002b; Nosaka et al., 2002a; Tiidus & Ianuzzo, 1983). In a study by Nosaka and Newton (2002b), the high intensity of exercise (100% of maximum isometric contraction) produced more severity of muscle damage than the low intensity of exercise (50% of maximum isometric contraction) as measured by isometric muscle strength, range of motion, upper arm circumference, and plasma creatine kinase activity. High intensity of exercise also required longer periods of time to recover. In a study by Nosaka et al. (2002a), at the high intensity of exercise (maximal eccentric contraction), an increase in the number of repetitions (12, 24, and 60 repetitions) did not affect soreness sensation but did produce more severity of muscle damage as indicated by an increase in strength and range of motion loss as well as the level of plasma creatine kinase (Nosaka et al., 2002a). In a study by Tiidus and Ianuzzo (1983), high intensity and short duration exercise (80% 10RM, 170 repetitions) resulted in greater serum enzyme activities (LDH, CK, and GOT) and soreness sensation than for low intensity and long duration exercise (30% 10RM, 545 repetitions). Nevertheless, the rest period between each eccentric exercise (0 second, 15 seconds, 5 minutes, and 10 minutes) did not affect the severity of muscle damage (Teague & Schwane, 1995). Therefore, increases in intensity and duration of exercise seem to produce more severity of muscle damage, with intensity having a more pronounced effect.

4.7 Preventative strategies to reduce muscle damage

Several interventions have been proposed as preventative or as treatment strategies for eccentric exercise-induced muscle damage including acupuncture (Barles et al., 2000), ultrasound (Ciccone et al., 1991; Craig et al., 1999), cryotherapy (Eston & Peters, 1999), compression (Chleboun et al., 1995; Kraemer et al., 2001), anti-inflammatory drugs (Hasson et al., 1993), hyperbaric oxygen therapy (Mekjavic et al., 2000), warm-up (High et al., 1989; Nosaka & Clarkson, 1997), stretching (High et al., 1989; Johansson et al., 1999; Lund et al., 1998a), and massage (Lightfoot et al., 1997; Rodenburg et al., 1994; Smith, Keating et al., 1994; Tiidus & Shoemaker, 1995). These strategies as treatments have been reviewed extensively (Cheung et al., 2003; Connolly, Sayers, & McHugh, 2003; Herbert & Gabriel, 2002; Kendall & Eston, 2002; McHugh, 2003; Tiidus, 1997) but none have been evaluated in terms of effectiveness as preventative interventions.

4.7.1 Repeated bout of eccentric exercise

The "repeated bout effect" is believed to be the most effective strategy in preventing muscle damage from eccentric exercise. The repeated bout effect refers to "the protective adaptation to a single bout of eccentric exercise" (McHugh, 2003). The protective adaptation is known to markedly reduce the severity of muscle damage as indicated by soreness sensation, and strength and range of motion loss, when these symptoms of the second bout of eccentric exercise are compared with the initial bout (Chen, 2003; Chen & Hsieh, 2000; Cleary, Kimura, Sitler, & Kendrick, 2002; Nosaka & Newton, 2002c).

The repeated bout effect has been shown to produce a rapid adaptation of muscle properties which happen while the damage process is still occurring (within one day after the initial bout of eccentric exercise) (Chen & Hsieh, 2001; Nosaka & Newton, 2002c; Paddon-Jones, Muthalib, & Jenkins, 2000; Smith, Fulmer et al., 1994). However, how long the protective effect lasts is uncertain. Byrnes et al. (1985) and Nosaka et al. (1991) reported that the repeated bout effect did not last longer than six weeks. Lund et al. (1998b) stated from their pilot work that the repeated bout effect did not appear after 8-10 weeks (data was not shown in the article). Nosaka et al. (2001a) reported that the protective effect of elbow flexors (24 maximal eccentric actions) lasted for at least six months (see Table 4.1). Interestingly, the effectiveness of the repeated bout seems to be greater the sooner it was repeated after the initial bout of eccentric exercise. Repeated exercise when muscle has not fully recovered (Day 1, 2, 3, 4, and 6 after the initial bout) has been shown to neither exaggerate the damage nor delay the recovery rate (Chen & Hsieh, 2000, 2001; Mair et al., 1995; Nosaka & Newton, 2002c;

Paddon-Jones et al., 2000; Smith, Fulmer et al., 1994). These results can be interpreted as indicating that there are some rapid adaptations which fully protect muscle damage from the second bout of eccentric exercise. The results from previous research have shown that the sooner the repeated bout is performed the less severe the symptoms of muscle damage will be (see Table 4.1).

Several studies have shown that the initial bout of eccentric exercise produced a protective adaptation on muscle regardless of the severity of muscle damage of the initial bout (Brown, Child, Day, & Donnelly, 1997; Chen & Hsieh, 2000; Nosaka, Sakamoto, Newton, & Sacco, 2001b; Paddon-Jones et al., 2000). A small number of contractions (as little as two maximal eccentric contractions) showed a slightly protective adaptation on the following 24 maximal eccentric contractions (performed two weeks later) (Nosaka et al., 2001b). It should be noted, that the two eccentric contractions protocol of the initial bout did not produce muscle damage. However, the effectiveness of the repeated bout effect of the two contractions was less than the six and 24 maximal eccentric contractions (Nosaka et al., 2001b). Interestingly, there were no significant differences of the repeated bout effects between the six and the 24 maximal eccentric contractions. Therefore, the low intensity of the initial bout as indicated by low repetitions such as 16% (Paddon-Jones et al., 2000), 20% (Brown et al., 1997), 25% (Nosaka et al., 2001b), 42% (Chen & Hsieh, 2000), and 60% (Brown et al., 1997) of the subsequent bout is effective in producing the repeated bout effect as indicated by less swelling, and range of motion and strength loss. Nevertheless, in the studies of Nosaka et al. (2001b) and Paddon-Jones and Quigley (1997), the low volume of eccentric exercise did not reduce the soreness sensation when compared with the first bout (soreness level of the first bout was 1.1 cm.) (Nosaka et al., 2001b; Paddon-Jones & Quigley, 1997). This might be due to the low volume of the first bout of exercise being too small to cause soreness sensation.

There is evidence that high intensity exercise (maximal contraction) is a more pronounced factor on the repeated bout effect than the number of repetitions. For example, Nosaka et al. (2002b) reported that low intensity eccentric training (50% of 1-RM) did not produce the repeated bout effect on the subsequent maximal bout.

Therefore, high intensity of the initial bout eccentric exercise is necessary for providing repeated bout effects.

The repeated bout effect appears to be specific to the exercised muscle (Eston, Finney, Baker, & Baltzopoulos, 1996) but not specific to the pattern of exercise (Eston, Lemmey, McHugh, Byrne, & Walsh, 2000; Rowlands, Eston, & Tilzey, 2001). In a study by Eston et al. (1996), a knee extensors exercise (100 maximal eccentric contractions) showed protective effects on downhill running two weeks later. In two studies, downhill running showed a repeated bout effect on subsequent downhill running regardless of the stride length (Eston et al., 2000; Rowlands et al., 2001). However, in the Connolly et al. (2002) study, there was no evidence of crossover effects to the contralateral limb. Therefore, to maximise the effectiveness of the protective adaptation on the repeated bout without severe damage of muscle from the initial bout, the initial eccentric exercise programme should be high intensity (maximal eccentric contraction), have low repetitions (at least 15% of the repetitions of the subsequent exercise), and be specific to the muscle group. The subsequent bout should be repeated within two to six weeks (after muscle weakness recovers).

Rcently, several authors have reviewed the possible adaptative mechanisms of the repeated bout effect (Clarkson & Hubal, 2002; Connolly et al., 2002; McHugh, 2003). The proposed mechanisms have included neural, mechanical, and cellular adaptations (McHugh, 2003). Neural adaptation could occur by an increase in motor unit activation, a change in motor unit recruitment, and an increased synchrony of motor firing (McHugh et al., 2001). Mechanical adaptation could occur by increasing muscle stiffness from the adaptation in the cytoskeletal proteins responsible for maintaining the alignment and structure of sarcomeres (McHugh, 2003). Cellular adaptation could occur by a longitudinal addition of sarcomeres, an adaptation in inflammatory response, and an adaptation to maintain excitation-contraction coupling (McHugh, 2003).

Table 4.1.. Time course of muscle adaptation of the repeated bout effect (the exercise bouts occurred at least two weeks apart).

Timing	Exercise intensity		Repeated bout effect (% reduction from the initial bout)				
	Muscles	Intensity of eccentric exercise	Soreness	Maximum isometric contraction	Range of motion	Circumference	Image
2 weeks (McHugh et al., 2001)	Hamstrings	6 sets of 10 repetitions at 60% MVC	83% s	0% s	-	-	-
3 weeks (Byrnes et al., 1985)	Quadriceps	30 min downhill running (slope -10°)	58% s	-	-	-	-
6 weeks (Byrnes et al., 1985)	Quadriceps	30 min downhill running (slope -10°)	64% s	-	-	-	-
8 weeks (Foley, Jayaraman, Prior, Pivarnik, & Meyer, 1999)	Elbow flexors	5 sets of 10 repetitions at 110% of 10RM	30% s	-	-	-	20% s
9 weeks (Byrnes et al., 1985)	Quadriceps	30 min downhill running (slope -10°)	20%	-	-	-	-
6 months (Nosaka et al., 2001a)	Elbow flexors	24 maximal eccentric contractions	30% s	24% s	s	43%s	35% s
9 months (Nosaka et al., 2001a)	Elbow flexors	24 maximal eccentric contractions	13%	20% s	s	20%	ns
12 months (Nosaka et al., 2001a)	Elbow flexors	24 maximal eccentric contractions	20%	11%	ns	0%	ns

Note. MVC = Maximum voluntary contraction, RM = Repetitive maximum, ns = nonsignificant,
s = significant

The neural adaptation theory is the most widely investigated of the three theories. Some research has supported this theory by reporting a reduction of fast-twitch motor unit activation during the subsequent exercise bout (three days later) (Chen, 2003).

Much research, however, has not supported this theory. For example, McHugh et al. (2001) reported that EMG per unit torque and median frequency showed no difference between the initial and the repeated bout of eccentric exercises (two weeks apart). Therefore, there was no evidence of an increase in motor unit recruitment or activation. Nosaka et al. (2002b) reported the repeated bout effect in electrically stimulated eccentric contraction. The changes in symptoms of muscle damage (maximal voluntary isometric force, range of motion, plasma creatine kinase and aspirate aminotransferase activities, upper arm circumference, muscle thickness from ultrasonography, and muscle soreness) from the subsequent electrically stimulated eccentric contractions were smaller than for the symptoms from the initial electrically stimulated eccentric contraction (two weeks later). The electrical stimulation was applied at the same muscle sites by the same conditions for the initial and the subsequent bouts. The peak force of the stimulation was the same between the two bouts. As a result, the pattern of motor unit activation was exactly the same between bouts but the symptoms of muscle damage were different. The lack of crossover effect did not support the central neural adaptation theory (Connolly et al., 2002).

There is little published on the mechanical theory of the repeated bout effects but it has been suggested that mechanical adaptation of muscle might present as a change in muscle stiffness (McHugh, 2003). The change in active stiffness contributes to changes of the stretch reflex, level of muscle activation, and passive joint properties (McNair & Stanley, 1996), while the change in passive stiffness contributes to changes of cross-links between the actin and myosin filaments, and series and elastic components of muscle (Gajdosik, 2001). There have been no reports on the effects of eccentric exercise on active stiffness, either through a single bout or as a training effect. Howell et al. (1993) reported a change in passive stiffness after eccentric exercise. Passive stiffness increased nearly two-fold immediately after elbow eccentric exercise and remained elevated for three days (see Figure 4.1). A rapid decrease in passive stiffness then occurred on days 4-6 but the degree of stiffness did not return to the baseline completely until ten days after eccentric exercise. At this period of time, while passive stiffness was still higher than the normal level, the repeated bout effect had already occurred. Therefore, one might conclude that a high level of passive stiffness produces a protective effect on the subsequent bout of eccentric exercise. This notion is contrary to that of McHugh et al. (1999) who reported that the stiff participants experienced more severity of muscle damage than the compliant participants. McHugh (2003) also stated in his recent review that “less stiffness was thought to enable greater sarcomere shortening thereby avoiding sarcomere strain” (p. 92). In fact, the increase of passive stiffness after eccentric exercise was thought to be one of the protective effects preventing muscle from further damage (Clarkson & Sayers, 1999). Therefore, more evidence on the mechanical theory is needed to investigate the effects of eccentric training on muscle stiffness, the time course of elevated passive stiffness after eccentric exercise, and the repeated bout effect on passive stiffness.

The cellular theory is another theory used to explain the repeated bout effect (McHugh, 2003). Cellular adaptation after initial eccentric exercise is thought to be due to the longitudinal addition of sarcomeres as shown by the shift of optimal length to the right (longer muscle length) (Brockett, Morgan, & Proske, 2001). The change in sarcomere numbers connected in the series of muscle fibres was considered the most plastic property of muscle and these could be changed within days after changes in activity patterns (Morgan, Brockett, Gregory, & Proske, 2002). However, there are a number of questions that need to be addressed:

- What is the least amount of time needed to produce an increase in the number of sarcomeres?
- Can the longitudinal addition of sarcomeres happen within a day after eccentric exercise (because the repeated bout effect occurred 24 hours after the initial bout) (Chen & Hsieh, 2001)?
- Can the longitudinal addition of sarcomeres occur due to low intensity stimulation (because the repeated bout effect occurred when the initial bout was low intensity (8% of the subsequent bout) (Nosaka et al., 2001b)?
- How long does the shift to the longer length of muscle last (because the repeated bout effect lasts for six months) (Nosaka et al., 2001a)?

Armstrong et al. (1983) proposed another theory that is used to explain the repeated bout effect. This theory is based on the idea that the high mechanical lengthening overload on muscle fibres from the initial bout results in damage only to the weak muscle fibres. Therefore, the strong fibres remain. The existence of strong fibres might be responsible for reducing severity of muscle damage for the subsequent bout of eccentric exercise. As a result, repeated bout effects (the less severity of muscle damage) can be seen as early as the first day after the initial bout (Chen & Hsieh, 2001). In the study by Foley et al. (1999), the prolonged loss in muscle volume (7-10%, 2-8 weeks after the initial bout) was thought to be a result of extinction of weak fibres within the muscle compartment. The shift to the longer muscle length as reported by Brockett et al. (2001) might be the length of the remaining strong fibres, not from the longitudinal addition of sarcomere. The protective mechanism which lasted for six months, as reported by Nosaka et al. (2001a), might be a consequence of the time that muscle has built up weak fibres and returned to a normal combination between the weak and strong fibres. Therefore, the protective effect from the strong fibres alone disappeared. The decrease in motor unit activation (30%) and median frequency (20%) reported by Chen (2003) might also be the result of the vanishing of the weak fibres. The pronounced effect of the high intensity (maximal contractions), not the number of repetitions, might be another reason to support this theory because the high intensity of exercise is more likely to damage the weak fibres than the number of repetitions.

4.7.2 Training

In an early report, Hill (1951) stated that training was the only way to reduce the severity of muscle damage. Contrary to Hill's (1951) suggestion, a single bout of eccentric exercise provides a protective effect on a subsequent bout (known as the repeated bout effect), regular eccentric exercise training, however, seems not to provide a protective benefit on the severity of muscle damage on subsequent eccentric exercise.

The published literature on the effect of training on severity of muscle damage does not support the common belief that training can prevent muscle damage from eccentric exercise. Concentric training was reported to increase the susceptibility of muscle damage (Gleeson, Eston, Marginson, & McHugh, 2003; Ploutz-Snyder, Tesch, & Dudley, 1998; Whitehead et al., 1998). The protective effect of eccentric training, however, is still uncertain. Balnave et al. (1993) reported that downhill running once a week for eight weeks reduced muscle damage. When eccentric training was compared with concentric training, Nosaka et al. (2002a) reported no difference in the symptoms of muscle damage between groups. If concentric training did increase the severity of muscle damage as reported by other studies (Gleeson et al., 2003; Ploutz-Snyder et al., 1998; Whitehead et al., 1998), eccentric training in the Nosaka et al. (2002a) study might have provided the same results because the data were not compared with a nontraining group.

An increase in muscle stiffness may be the mechanism responsible for increasing the severity of muscle damage after training. A positive relationship has been reported between active stiffness and isometric and concentric (r = 0.6-0.8) but not eccentric contraction (Wilson et al., 1994). Similarly, a positive relationship has been reported between passive stiffness and isometric (r = 0.6) (Kubo et al., 2001) and concentric contraction (r = 0.5) (Gajdosik, 2002). There have been no reports on the relationship between passive stiffness and eccentric contraction. Even though a stiff musculotendinous unit is more efficient in force transmission generated by the muscle to the bone, stiff muscles might be more susceptible to the strain imposed by the lengthening contraction than complaint muscle. Stiff muscles have been reported to reduce the ability of muscle fibres to absorb energy (Leivseth et al., 1993). The reduced ability of muscle fibres to absorb energy during lengthening contractions might cause more strain on the myofibril than on the compliant muscles. Moreover, a positive relationship has been reported between passive stiffness and the severity of muscle damage. McHugh et al. (1999) attributed passive stiffness as tendon-aponeurosis extensibility and stated that the aponeurosis rupture was responsible for greater damage in stiff muscles than in compliant muscles. Therefore, any intervention that increases muscle stiffness might also increase the susceptibility of the muscle to damage. Klinge et al. (1997) reported that isometric training of the hamstring muscles increased both strength (43%) and passive stiffness (data were not shown). In the same way, eccentric training was reported to increase active stiffness (Pousson, van Hoecke, & Boubel, 1990). Although these studies did not directly investigate the effect of training on the severity of muscle damage, their results could indicate that stiffness might be a mechanism responsible for the more vulnerable musculotendinous unit after training.

4.7.3 Preexercise activities (warm-up, stretching, and massage)

Warm-up, stretching, and massage are commonly recommended before exercise or competition to improve performance and reduce the risk of injury. Unfortunately, the scientific data to support this belief are not widely evident. Warm-up has been shown to elevate muscle temperature (Magnusson et al., 2000), increase muscle blood flow (Tiidus & Shoemaker, 1995), and to increase neurological excitability (Shellock & Prentice, 1985). Stretching has been shown to increase flexibility (Smith, 1994). Research has demonstrated that massage can increase muscle blood flow (Hansen & Kristensen, 1973), muscle temperature (Drust et al., 2003), and muscle flexibility (Nordschow & Bierman, 1962), and reduce tissue adhesion (Braverman & Schulman, 1999; Nordschow & Bierman, 1962). The benefits of such preexercise activities might help to reduce injury risk factors. Unfortunately, there are limited published studies in this area.

A search of the literature identified only one published research paper on the effects of warm-up on the severity of muscle damage (Nosaka & Clarkson, 1997). Nosaka and Clarkson (1997) found that both high (100 repetitions of maximal concentric contraction) and low (100 repetitions of minimal concentric contraction) intensities of warm-up could reduce the magnitude of muscle damage as indicated by reduced soreness sensation, strength and range of motion loss, swelling, and creatine kinase activity. The authors proposed that warm-up might help to increase muscle temperature and circulation, and consequently, increase muscle and connective tissue elasticity. The proposed mechanism that warm-up could increase musculotendinous unit elasticity (reduce muscle stiffness) was not investigated in this study. If passive stiffness is the mechanism responsible for the severity of muscle damage, the results from this study indicate that the warm-up programme might need to address specific muscles because a general warm-up (running) was not shown to affect passive stiffness (Magnusson et al., 2000). If active stiffness is the mechanism responsible for the severity of muscle damage, a general warm-up might help to reduce the severity of muscle damage. As McNair and Stanley (1996) reported, a reduction of active stiffness was achieved after jogging. To date, there are no published papers on the effects of general warm-up on the magnitude of muscle damage and the effects of a specific warm-up on passive stiffness.

Several authors have investigated the effects of preexercise stretching on the magnitude of muscle damage (High et al., 1989; Johansson et al., 1999; Lund et al., 1998a; Wessel & Wan, 1994), and a recent review has summarised the findings (Herbert & Gabriel, 2002). Unfortunately, most researchers found that static stretching did not help to reduce the magnitude of muscle damage. The ineffectiveness of stretching on the severity of muscle damage might be that stretching has no effects on passive (Halbertsma et al., 1996; Klinge et al., 1997; Magnusson, 1998) and active (Cornwell & Nelson, 1997) stiffness. There was no research on the effects of other types of stretching (e.g., ballistic, proprioceptive neuromuscular facilitation, dynamic stretching) on muscle stiffness and the severity of muscle damage.

No published studies have reported the effects of preexercise massage on the severity of muscle damage. Massage can increase muscle temperature (Drust et al., 2003) and blood flow (Hansen & Kristensen, 1973), which might help to increase muscle compliance and reduce muscle stiffness. Nevertheless, the only research on the effects of massage on passive stiffness did not support this claim (Stanley et al., 2001). Massage involves several techniques such as effleurage, petrissage, and friction. Each technique is used for different purposes and provides different effects. Stanley et al. (2001) used an effleurage technique for ten minutes on the hamstring muscles and did not find any change in passive stiffness. Generally, massage therapists use effleurage techniques to stimulate the parasympathetic nervous system and evoke the relaxation response. Therefore, the massage technique used in Stanley et al.’s study might not have been appropriate to produce changes in passive stiffness. Petrissage and friction are techniques aim to mobilise deep muscle tissue or the skin and subcutaneous tissue and increase local circulation. From a clinical point of view, these techniques might be more appropriate in reducing muscle stiffness, and consequently, reducing the severity of muscle damage.

There was only one published research on the combined effects of warm-up, stretching, and massage on the severity of muscle damage. Rodenburg et al. (1994) reported that the combination of warm-up and stretching before eccentric exercise, and massage after exercise, was effective in reducing the severity of muscle damage. Unfortunately, the researchers did not compare the effect of each treatment alone. The results of Rodenburg et al. (1994) suggest the benefit of warm-up only being given because stretching showed no effect on the severity of muscle damage at all. Massage was also unlikely to provide any benefit because it was administered immediately after eccentric exercise. Immediately after eccentric exercise, muscle does not need more blood flow to eliminate waste products such as lactic acid because eccentric exercise does not produce a lot of waste products (Armstrong et al., 1991; Clarkson & Hubal, 2002; Clarkson & Sayers, 1999; McHugh, Connolly, Eston, Kremenic et al., 1999). Therefore, it was not clear which intervention provided the benefit for preventing muscle damage in Rodenburg et al. (1994) study.

4.7.4 Pharmaceutical substances

The role of several chemical substances (e.g., anti-inflammatory drugs, antioxidants, herbs, nutritional supplements) has been researched in relation to preventing or reducing the severity of muscle damage. The use of anti-inflammatory drugs such as aspirin, diclofenac, ibuprofen, naproxen, and ketoprofen was emphasised in the literature recently reviewed by Connolly et al. (2003). Comparison on the effectiveness of different anti-inflammatory drugs on the severity of muscle damage was difficult due to the variety of anti-inflammatory drugs used, doses, time to provide drugs, intensity and protocol of eccentric exercise-induced muscle damage, and the initial characteristic of participants. Research on the prophylactic effects of the anti-inflammatory drugs [ibuprofen (Hasson et al., 1993), naproxen (Bourgeois et al., 1999), and flurbiprofen (Semark, Noakes, St Clair Gibson, & Lambert, 1999)], however, indicated that only ibuprofen (400 mg three doses in 24 hours for a total of 1200 mg) showed preventative effects on eccentric exercise-induced muscle damage by reducing soreness sensation and strength and range of motion loss (Hasson et al., 1993). Interestingly, the prophylactic application of ibuprofen in this study showed a faster effect in relieving the symptoms of muscle damage than did the after-exercise therapeutic treatment.

There have been attempts to investigate the effects of several substances used to prevent the symptoms of muscle damage such as fish oil (Lenn et al., 2002), ethanol (Clarkson & Reichsman, 1990), herbs (Vickers, Fisher, Smith, Wyllie, & Lewith, 1997), and pollen extract (Krotkiewski, Brzezinska, Liu, Grimby, & Palm, 1994). Fish oil was thought to decrease the inflammatory process (Lenn et al., 2002). Ethanol was thought to reduce a leakage of muscle proteins after eccentric exercise (Clarkson & Reichsman, 1990). A herb (Arnica) is commonly used for many types of soft tissue trauma (Vickers et al., 1997). Unfortunately, none of these substances was shown to be effective in reducing the severity of muscle damage (Clarkson & Reichsman, 1990; Lenn et al., 2002; Vickers et al., 1997). Pollen extract is a free radical-scavenging preparation which was thought to attenuate or eliminate tissue destruction (Krotkiewski et al., 1994). Pollen extract was reported to reduce soreness sensation and lower the concentration of lipid peroxides (reduce the level of free radicals). However, Krotkiewski et al. (Krotkiewski et al., 1994) did not investigate the other symptoms of muscle damage such as strength and range of motion loss.

4.7.5 Oestrogen

Several researchers have reported less severity of muscle soreness in females than in males (Dannecker, Koltyn, Riley, & Robinson, 2003; MacIntyre et al., 2000; Stupka et al., 2000). Reduced severity of muscle soreness in women was also found in research which compared women who were contraceptive users with eumenorrheic nonoral contraceptive users (Carter, Dobridge, & Hackney, 2001; Thompson, Hyatt, De Souza, & Clarkson, 1997). This observation has been explained as being related to the protective effects of oestrogen on cardiac and smooth muscles in premenopausal females when they are compared with age-matched males (Kendall & Eston, 2002). The protective effects of oestrogen on muscle damage might be that oestrogen defends against oxygen free radicals and reduces the susceptibility of muscle membrane to being damaged (Tiidus, 2003). Oestrogen also acts as a membrane stabiliser and gene regulator (Kendall & Eston, 2002). The potential protective role of oestrogen on muscle damage from eccentric exercise has been reviewed recently (Kendall & Eston, 2002; Tiidus, 2003).

It is important to note that the majority of previous research on the effects of oestrogen on muscle damage reported only a reduction in soreness perception. When other indirect parameters such as strength, range of motion, circumference, and serum creatine kinase activity in the oral contraceptive and nonoral contraceptive user groups were compared, no differences of these parameters were found (Thompson et al., 1997). Therefore, the effectiveness of estrogen on preventing muscle damage is questionable. Moreover, research in this area compared males and females who might actually have more differences in muscle properties than the effects of estrogen alone. The comparison between the oral contraceptive and nonoral contraceptive users might show the effects of oestrogen at different levels. If estrogen provides a long-term protective effect, women who are nonoral contraceptive users still have high estrogen levels during the regular menstrual cycle that might provide the chronic protective effects on muscle damage. Tiidus (2003) recommended that research should compare the menopausal women who received oestrogen supplement with those who received nonoestrogen supplement. However, menopausal women are considered a special population who might have a change in muscle properties due to aging. Further research that compares male participants who are provided with oestrogen with male participants who do not receive oestrogen is needed. Such research might lead to more understanding on the effects of oestrogen on muscle damage and lead to more practical use of the hormone.

4.8 Practical recommendations for the health care practitioner

To prevent and/or reduce the severity of muscle damage from unaccustomed eccentric exercise, based on the previous research, medical practitioners and coaches should recommend to their patients in rehabilitation programmes or to athletes that they perform a specific warm-up regularly on muscles that are predominantly used in the rehabilitation programme or sport. A “light” specific eccentric exercise following a specific warm-up (at least 15% of the number of repetitions of the real eccentric exercise at maximum contraction, low velocity, and short muscle length position) that produces a “low” severity of muscle damage is recommended. This light eccentric exercise will produce a repeated bout effect that also helps to reduce the severity of muscle damage. The next eccentric exercise session should be two weeks later in order to recover from strength loss. If eccentric training needs to be performed before two weeks, the intensity of eccentric exercise should be low (50-60% of maximal isometric voluntary contraction).

It is important to note, that when chemical substances are administered to prevent or relieve the symptoms of muscle damage, one should be aware of the side effects that might be harmful. Long-term use of anti-inflammatory drugs has resulted in an increased risk of stomach ulcers, kidney failure, and liver damage (Hasson et al., 1993). Oestrogen might be beneficial for female patients or athletes, but the effects on male patients or athletes are uncertain. For these reasons, the use of chemical substances might not be an appropriate strategy in reducing the severity of muscle damage.

CHAPTER 5

THE EFFECTS OF ACTIVE DYNAMIC WARM-UP, PASSIVE DYNAMIC STRETCHING, AND MASSAGE ON STIFFNESS, RANGE OF MOTION, MAXIMUM VOLUNTARY ISOMETRIC STRENGTH, AND SORENESS IN HAMSTRING MUSCLES BEFORE AND OVER FOUR DAYS AFTER ECCENTRIC EXERCISE.

Weerapong, P., Hume, P. A., & Kolt, G. S. (2004). The effects of active dynamic warm-up, passive dynamic stretching, and massage on stiffness, range of motion, maximum voluntary isometric strength, and soreness in hamstring muscles before and over four days, after eccentric exercise. Manuscript submitted for publication.

5.1 Summary

Objectives: To examine the effects of active dynamic warm-up, passive dynamic stretching, and massage on stiffness, range of motion, maximum voluntary isometric strength, and soreness in hamstring muscles before and over four days, after eccentric exercise.

Methods: Thirty healthy adult males were randomised to warm-up (100 active dynamic knee flexion/extensions at 120°/s), passive dynamic stretching (at 5°/s), or effleurage and petrissage massage (20 minutes) intervention groups. On Day 1 stiffness, range of motion, maximum voluntary isometric strength, and soreness measures were recorded for the knee flexor muscles before and after each intervention. On Day 2 the procedures were repeated with a bout of eccentric exercise immediately after the intervention. All measures were repeated on Days 3, 4 and 5. Mean changes in measures from Day 1 baseline were expressed as effect sizes (ES). Data were presented as change scores between stretching or massage interventions and warm-up.

Results: Warm-up showed a moderate effect between pre and post intervention in reducing stiffness (0.5 ES) and muscle strength (0.3 ES) compared to the baseline. Warm-up also showed less strength and flexion range of motion loss after eccentric exercise and faster recovery over the data collection period compared with stretching and massage groups.

Conclusions: Warm-up was more effective than stretching or massage in strength and range of motion loss of knee flexor muscles after eccentric exercise. Athletes are encouraged to perform active dynamic warm-up before eccentric exercises in sport performance.

5.2 Introduction

Muscle damage induced from unaccustomed eccentric exercise is a significant problem for coaches and athletes because it causes pain and diminishes muscle function and ability to participate in sport especially at the beginning of sports season (Cheung et al., 2003). Various researchers have agreed that the sequence of muscle damage events consists of the mechanical rupture of intramuscular structures, leading to calcium homeostasis disturbances, followed by inflammation before recovery occurs (Armstrong et al., 1991; Clarkson & Hubal, 2002; Evans & Cannon, 1991; Friden & Lieber, 1992). Symptoms of muscle damage considered in this paper are loss of muscle strength, increased soreness sensation, decreased range of motion, increased muscle stiffness, and perturbed athletic performance (Weerapong, Hume, & Kolt, 2004a). These adverse effects of eccentric exercise last longer than 10 days (Weerapong et al., 2004a) and are likely to increase the risk of further sports injury.

A number of factors have been linked to the severity of muscle damage including passive stiffness (Weerapong et al., 2004). Participants who had stiffer muscle (determined by passive stiffness) experienced more severe muscle damage (McHugh, Connolly, Eston, Kremenic et al., 1999). The stiffer muscles demonstrated less sarcomere numbers and muscle length and increased mechanical energy loss during passive movement than did complaint muscles (Leivseth et al., 1993). Therefore, stiff muscles might reduce the ability to withstand elongation and may be more likely to develop injuries which generally occur during muscle elongation. As exercise-induced muscle damage is initiated by high tensile force from eccentric exercise on the elongated muscle fibres, interventions that help to reduce muscle stiffness may help to reduce mechanical overload on muscle fibres, reduce the rupture of intramuscular structures, and consequently, reduce the severity of muscle damage. Therefore, There is limited research on the effectiveness of preexercise activities on passive stiffness or severity of muscle damage (Weerapong et al., 2004).

Sport practitioners typically use a combination of warm-up and stretching which are based on experience or anecdotal notes from coaches and atheletes. Reasoning behind these preexercise strategies is often based on possibly invalid generalizations of scientific findings. The systemic investigation of isolated preexercise activity is required to examine the effectiveness of preexercise activities.

Therefore, this study aimed to examine the effects of active dynamic warm-up, passive dynamic stretching, and massage on stiffness, range of motion, maximum voluntary isometric strength, and soreness in hamstring muscles before and over four days, after eccentric exercise.

Warm-up has been defined as active continuous movements of a muscle (Nosaka & Clarkson, 1997). Active dynamic warm-up was used as a control group in this study because of practical issue. Passive dynamic stretching has been defined as passive continuous movements (McNair et al., 2000). Passive dynamic movement can reduce muscle passive stiffness (McNair et al., 2000), which is related to the severity of muscle damage (McHugh, Connolly, Eston, Kremenic et al., 1999). There are no data on the effects of passive dynamic stretching on the severity of eccentric exercise-induced muscle damage. Massage has been defined as “a mechanical manipulation of body tissues with rhythmical pressure and stroking for the purpose of promoting health and well-being”(Cafarelli & Flint, 1992) (pp. 1). Effleurage and petrissage techniques were used in this study as they are commonly used to increase muscle compliance. There are no data on the effects of massage on compliance of the effects of preexercise massage on eccentric exercise-induced muscle damage prevention.

5.3 Methods

5.3.1 Participants

Thirty healthy males who had no musculoskeletal injury in the last two years were randomised into one of three groups: massage; stretching; warm-up. On Day 1 the participants read an information package and signed a consent form approved by the Auckland University of Technology Ethics Committee. The participants’ height and weight were collected using scales and a stadiometer (SECA 220, Birmingham, England).

5.3.2 Procedure

The experimental protocol is presented in Figure 5.1. Throughout five consecutive days, the participants were asked to attend the laboratory at the same time each day. To follow the influence of massage, stretching, and warm-up, muscle soreness, range of motion, muscle strength, and passive muscle stiffness were measured in the same sequence as Day 1. Participants were asked to minimise the use of their legs to perform heavy activity, and to record their physical activity using the International Physical Activity Questionnaire (IPAQ) (2001), over the five days of the study. Day 1 represented the effects of intervention on muscle properties, Day 2 represented the acute effects of intervention on muscle damage, and Days 3, 4 and 5 represented the long term effects of intervention on muscle damage.

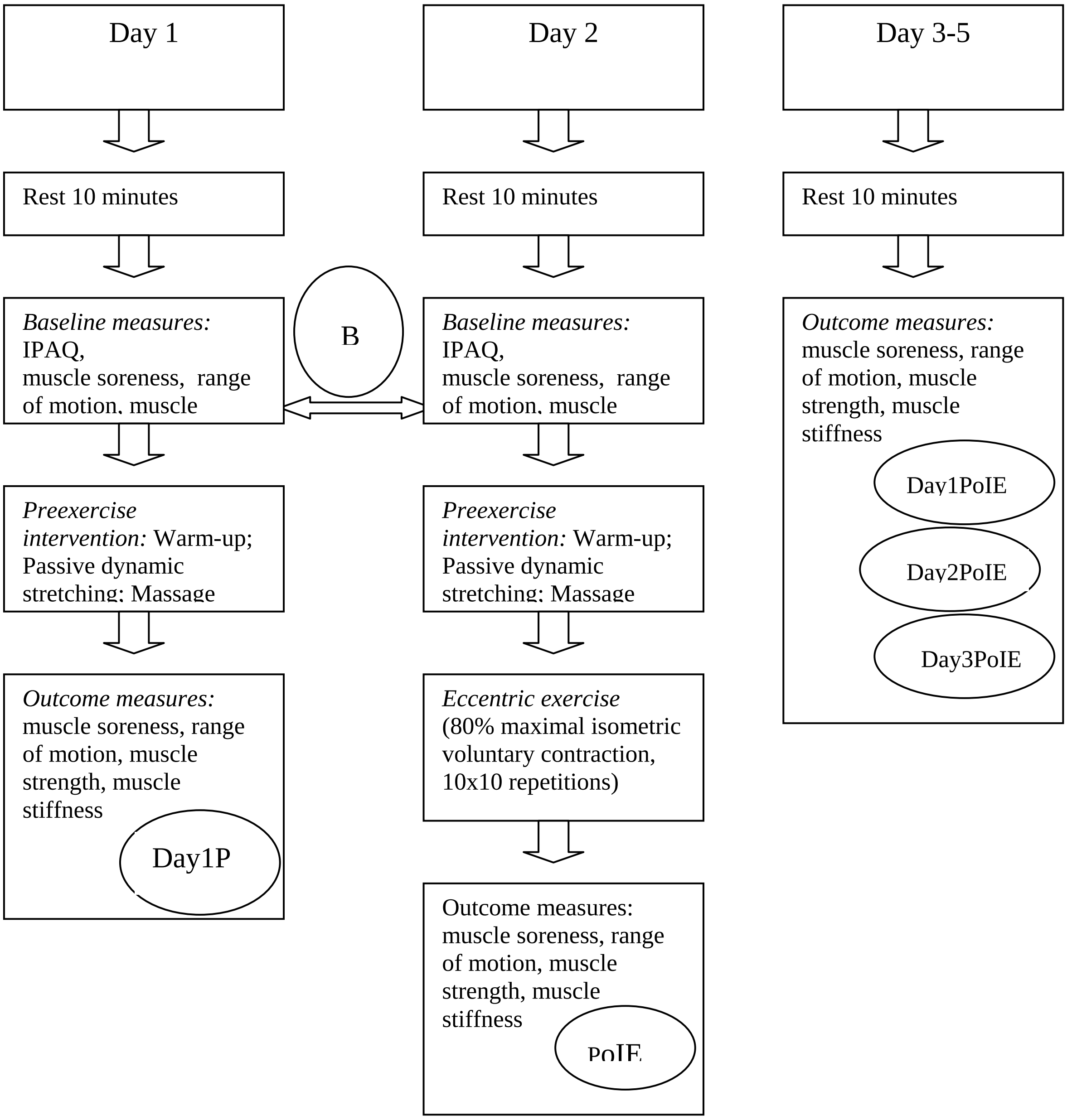
5.3.3 Interventions

5.3.3.1 Warm-up control

In the active dynamic warm-up (control) condition, the participants sat on a KinCom dynamometer seat (Chattecx Corporation, Chattanooga, Tennessee) with their trunk, pelvis, and distal thigh of the non-dominant leg strapped to the chair. The non-dominant foot was placed on the stool. The dominant thigh was strapped with an adjustable support to the chair and the lower leg was strapped to the dynamometer arm, allowing free knee flexion and extension. Participants were then asked to move their dominant knee joint 100 times with minimal force (less than 15%MVC) from full extension to a 90° flexed position by using the dynamometer at 120°/s (Nosaka & Clarkson, 1997).

5.3.3.2 Passive dynamic stretching intervention

In the passive dynamic stretching condition, the participants’ knee joint was moved ten times passively from 90° flexion to full extension using the KinCom dynamometer at 5°/s (McNair et al., 2000). The participants were asked to completely relax throughout the experimental period of three minutes.



IPAQ = International Physical Activity Questionnaire, B = Baseline (the average of Day 1 and Day 2 pre-intervention measures), PoI = Postintervention, PoIE = Postintervention and eccentric exercise

Figure 5.1 Experimental protocol.

5.3.3.3 Massage intervention

Participants were administered effleurage and petrissage for 20 minutes on their hamstring muscles before performing eccentric exercise. Participants lay prone on the bed with pillow support under their non-dominant ankle. The massage techniques used in this study were based on the basic Swedish remedial system (De Domenico & Wood, 1997). The same qualified physiotherapist with ten years experience carried out all massage interventions on the hamstring muscles with the massage sequence steps (two minutes each) as follows:

1. Superficial stroking
2. Palmar kneading
3. Squeeze kneading
4. Alternating palmar kneading
5. Reinforced kneading
6. Repeated sequence steps 2-5
7. Deep stroking

5.3.3.4 Eccentric exercise intervention

The eccentric exercise intervention required the participants to flex their knee against the KinCom dynamometer which extended in the isokinetic eccentric mode at 120°/s. The participants' lower leg moved to the flexed position concentrically. Each participant performed ten repetitions per set for ten sets with two minutes between each set (Howell et al., 1993). Visual feedback of the force during eccentric exercise was given to the participants to ensure consistency in the requested 80% maximum isometric contraction exercise intensity.

5.3.4 Measures

5.3.4.1 Muscle soreness

Participants rated muscle soreness (0 - 10 cm) during isometric contraction by drawing a mark across a printed visual analogue scale line 10 cm in length, anchored at the beginning by the descriptor "No soreness at all" and at the end by "Worst soreness imaginable" (Ebbeling & Clarkson, 1989).

5.3.4.2 Range of motion

The non-dominant leg knee angle in standing, during active maximal knee flexion and during relaxed hanging knee extension were measured using a universal goniometer (Clarkson, 2000). The head of the greater trochanter, the joint space between the lateral condyle of the femur and tibial tuberosity, and the lateral malleolus were marked to help goniometer placement for angle measurement.

5.3.4.3 Isometric torque

Maximum isometric torque (N.m) was assessed using the KinCom dynamometer. The hamstring muscles were tested with the participant in a sitting position with their arms crossed over their chest. The non-dominant lower leg was strapped to the dynamometer two centimetres proximal to the malleoli (Klinge et al., 1997). Participants were asked to bend their knee maximally at their own pace. Torque was measured at a knee angle of 45° with the hip flexed at 90° (McHugh et al., 2001). Participants performed three maximum voluntary contractions, separated by rest intervals of 30 seconds (Byrne & Eston, 2002). The average maximal isometric torque of the three contractions was calculated (Nosaka & Clarkson, 1997).

5.3.4.4 Passive muscle stiffness

The measurement of passive stiffness of the hamstrings has been described in detail elsewhere (Magnusson, Simonsen, Aagaard, Sorensen et al., 1996). Briefly, the participants sat on the KinCom dynamometer as described in the warm-up intervention but the participants' hip joint of the non-dominant leg was positioned at 30° above the horizontal line with a support under the distal thigh. In this position, the participants' hip joint was flexed at 120°. The KinCom dynamometer was set at zero degrees parallel to the horizontal plane. The gravity and limb weight were calibrated according to the guidelines in the dynamometer's instruction manual (Magnusson et al., 2000). The dynamometer was programmed to extend the knee joint passively in the sagittal plane at 5°/s (McNair et al., 2000) from 80° below the horizontal plane to 80% of individual full extension (Klinge et al., 1997). The full extension angle was determined by the investigator passively manually extending the participants' leg to an angle producing a firm feeling of resistance to further movement (Clarkson, 2000). The 80% of maximal knee extension was chosen so that the obtained data was primarily from the passive properties of the muscle unit without involvement of the tendon, the posterior capsular constraints, or force from muscle contraction (Klinge et al., 1997).

Passive force, joint angle, and velocity were recorded at 100 Hz using a LabView data acquisition programme. Participants were allowed to rest for 30 seconds between trials in order to minimize the effect of previous passive tests (Klinge et al., 1997).

Passive stiffness was defined as resistance to stretch of the hamstrings muscles during passive knee extension (i.e., passive resistance or passive force to the change in the length displacement) (Magnusson et al., 2000). Passive stiffness was expressed as the average slope of the torque-angle curve at the final one-third of knee extension range of motion (Magnusson, 1998).

5.3.5 Statistical analyses

Data are reported as mean and standard deviation. Change scores between interventions are reported as 90% confidence limit intervals, effect sizes, and *P*-values. Baseline values were calculated by averaging the Day 1 preintervention and the Day 2 preintervention values of each group. Mean maximum isometric torque and passive stiffness were calculated as a percentage of the baseline value.

Change scores were calculated as differences from baseline for group mean post-intervention values and group mean post-eccentric exercise values. Effect sizes (ES) were calculated by dividing the change scores by the standard deviation of Day 1 pre-intervention and the Day 2 pre intervention values for all the groups. The thresholds of trivial, small, moderate, and large effects were 0.2, 0.6, 1.2, and 2.0, respectively (Hopkins, 2002b). The magnitude of the effect-size statistic for soreness sensation, range of flexion and extension motion, maximum isometric torque, and passive stiffness for each intervention was assessed comparing baseline (B) and post-intervention (PoI), immediately post-intervention and eccentric exercise (ImPoIE), and for the three consecutive days after the eccentric exercise (D1PoIE, D2PoIE, D3PoIE). A positive value means the change score after each intervention is more than the baseline. A negative value means the change score after the intervention is less than baseline.

Confidence intervals (CI) were calculated using the "Calculating likely (confidence) limits and likelihood for true values" spreadsheet (Hopkins, 2002a). A 90% CI was used because it represents adequate precision for making probabilistic inferences. The probability that the true value was less than the lower confidence limit and more than the upper confidence limit were both only 0.05, which can be interpreted as very unlikely (Peterson et al., 2004).

Paired t-tests were used to test for differences in B and PoI, ImPoIE, D1PoIE, D2PoIE, and D3PoIE within each treatment group, and unpaired t-tests were used to test for group effects for each of the measures of stiffness, range of motion, maximum voluntary isometric strength, and soreness.

5.4 Results

Participant characteristics of age, weight, height, and the level of physical activity are presented in Table 5.1. The level of physical activity recorded each day during the data collection period with the IPAQ was not different between groups.

Table 5.1.. Mean (± *SD*) participant characteristics and IPAQ scores for the five testing days.

Group	Age (years)	Mass (kg)	Height (cm)	IPAQ (min)				
				Day 1*	Day 2**	Day 3**	Day 4**	Day 5**
Warm-up (*n* = 10)	26.9 (4.9)	75.0 (8.4)	178.0 (6.2)	163.5 (138.6)	29.5 (33.0)	27.0 (28.7)	29.5 (40.9)	31.0 (46.9)
Stretching (*n* = 10)	25.4 (5.3)	74.7 (8.7)	175.2 (6.7)	214.0 (188.8)	40.0 (49.2)	25.0 (43.8)	33.0 (40.6)	12.0 (25.3)
Massage (*n* = 10)	26.0 (4.4)	78.2 (12.3)	177.9 (9.5)	223.5 (154.3)	49.0 (66.1)	44.0 (53.2)	26.0 (36.0)	38.0 (46.9)

* was one week before testing, ** was one day before testing.

5.4.1 The effectiveness of interventions on muscle properties

Summaries of variables that identify muscle properties and symptoms of muscle damage are presented in Table 5.2-5.3. Immediately after the intervention, the warm-up group resulted in a small effect in reducing isometric muscle strength (ES = -0.28, 0.32, 0.43) and passive stiffness (ES = 0.49, 0.57, 0.70) immediately after intervention compared with baseline, stretching and massage groups, respectively. The knee flexion range of motion underwent a small increase in the stretching group when compared with the warm-up group (ES = 0.47). There was no change in other variables in massage and stretching groups.

Table 5.2 Mean (± *SD*) of soreness sensation, range of motion (active knee flexion and relaxed knee extension), maximal isometric torque, passive stiffness, maximum tension, and stress relaxation for warm-up (*n*=10), stretching (*n*=10), and massage (*n*=10) participants.

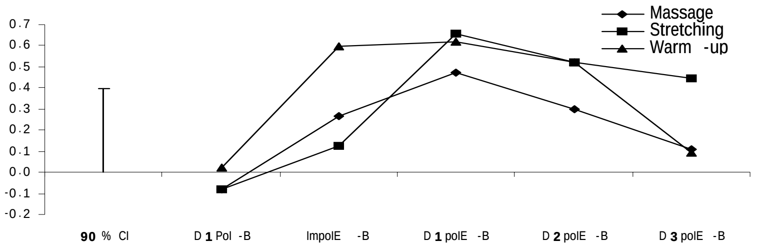
Variables	Interventions	B	Day1PoI	PoIE	Day 1PoIE	Day 2PoIE	Day 3PoIE
Soreness sensation (cm)	Massage	4.4 (3.0)	4.2 (3.4)	5.1 (3.0)	5.6 (2.5)	5.2 (2.6)	4.7 (2.7)
	Stretching	3.5 (2.4)	3.3 (2.5)	3.8 (2.4)	5.2 (2.5)	4.8 (2.7)	4.6 (2.7)
	Warm-up	3.1 (2.4)	3.1 (2.5)	4.6 (2.2)	4.7 (2.7)	4.4 (2.3)	3.3 (2.2)
Range of motion – Active knee flexion (°)	Massage	62 (6)	62 (5)	72 (7)	68 (7)	66 (5)	67 (5)
	Stretching	63 (6)	61 (6)	72 (8)	71 (5)	68 (7)	68 (4)
	Warm-up	62 (4)	63 (6)	67 (9)	64 (9)	62 (7)	63 (5)
Range of motion – Relaxed knee extension (°)	Massage	168 (3)	169 (2)	167 (3)	169 (3)	168 (3)	169 (2)
	Stretching	169 (3)	168 (3)	167 (4)	168 (3)	167 (2)	168 (4)
	Warm-up	170 (3)	170 (4)	169 (3)	170 (3)	169 (3)	170 (3)
Isometric torque (N.m)	Massage	383 (66)	385 (58)	317 (67)	319 (75)	348 (91)	344 (81)
	Stretching	387 (61)	396 (50)	342 (57)	330 (43)	340 (66)	336 (64)
	Warm-up	384 (100)	365 (101)	351 (82)	359 (31)	367 (110)	389 (106)
Passive stiffness (N.m.$^{\circ 1}$)	Massage	1.21 (0.40)	1.15 (0.44)	1.27 (0.45)	1.32 (0.54)	1.36 (0.61)	1.23 (0.54)
	Stretching	1.04 (0.19)	1.02 (0.33)	1.13 (0.26)	1.16 (0.26)	1.17 (0.23)	1.19 (0.31)
	Warm-up	1.19 (0.29)	1.04 (0.28)	1.14 (0.28)	1.14 (0.35)	1.18 (0.42)	1.13 (0.44)

Note. PoI = Postintervention, B = Baseline (the average of Day 1 and Day 2 preintervention measures), PoIE = Postintervention and eccentric exercise, CI = confidence interval limits, ES = Effect size statistic

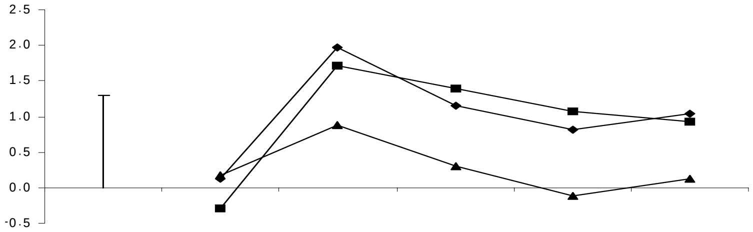
Table 5.3 Change scores between-interventions, 90%CI, and *p*-value of Day 1, Day 2 and Day 3 compared with warm-up group in passive stiffness, range of motion, maximum voluntary isometric strength, and soreness for stretching (*n*=10) and massage (*n*=10) groups compared with warm-up (*n*=10) group.

Variable	Intervention	Statistics	PoI-B	Immediately PoIE-B	Day 1 PoIE-B	Day 2 PoIE-B	Day 3 PoIE-B
Soreness sensation	Massage-Warm-up	ES	-0.10	-0.33	-0.15	-0.22	-0.02
		90%CI	-0.3, 0.1	-0.6, 0.0	-0.6, 0.3	-0.7, 0.3	-0.5, 0.5
		P-value	0.45	0.06	0.55	0.43	0.96
	Stretching-Warm-up	ES	-0.10	-0.47	-0.03	0.00	0.35
		90%CI	-0.3, 0.1	-0.9, -0.1	-0.5, 0.6	-0.5, 0.5	-0.2, 0.9
		P-value	0.49	0.06	0.91	0.99	0.27
Range of motion – Flexion	Massage-Warm-up	ES	-0.05	1.09	0.85	0.93	0.92
		90%CI	-0.5, 0.5	-3.8, 6.0	0.0, 1.7	-0.4, 2.2	0.3, 1.5
		P-value	0.87	0.70	0.11	0.23	0.02
	Stretching-Warm-up	ES	-0.47	0.83	1.09	1.19	0.80
		90%CI	-1.2, 0.3	-0.7, 2.4	0.0, 0.2	0.0, 2.3	0.3, 1.3
		P-value	0.31	0.36	0.11	0.09	0.01
Range of motion – Extension	Massage-Warm-up	ES	0.07	0.03	0.14	0.24	0.10
		90%CI	-0.3, 0.4	0.0, 0.1	-1.5, 1.8	-0.2, 0.7	-0.1, 0.3
		P-value	0.73	0.39	0.88	0.40	0.32
	Stretching-Warm-up	ES	-0.15	-0.11	-0.17	0.04	-0.14
		90%CI	-0.5, 0.2	-1.1, 0.9	-0.8, 0.4	0.0, 0.1	-1.2, 0.9
		P-value	0.47	0.86	0.62	0.43	0.81
Isometric torque	Massage-Warm-up	ES	0.32	-0.47	-0.53	-0.24	-0.62
		90%CI	0.0, 0.6	-1.0, 0.1	-1.1, 0.0	-1.0, 0.5	-1.1, -0.1
		P-value	0.06	0.15	0.12	0.58	0.03
	Stretching-Warm-up	ES	0.43	0.15	-0.37	-0.34	-0.69
		90%CI	0.1, 0.7	-0.7, 0.4	-0.8, 0.1	-1.0, 0.3	-1.3, -0.1
		P-value	0.02	0.63	0.19	0.38	0.05
Passive stiffness (linear phase)	Massage-Warm-up	ES	0.57	0.66	1.04	1.00	0.47
		90%CI	-0.2, 1.3	-0.5, 1.8	-0.4, 2.5	-0.4, 2.4	-0.7, 1.7
		P-value	0.20	0.35	0.23	0.22	0.50
	Stretching-Warm-up	ES	0.70	0.80	1.15	1.11	1.46
		90%CI	-0.2, 1.7	0.0, 1.6	0.1, 2.2	0.1, 2.2	0.4, 2.5
		P-value	0.21	0.10	0.06	0.08	0.03

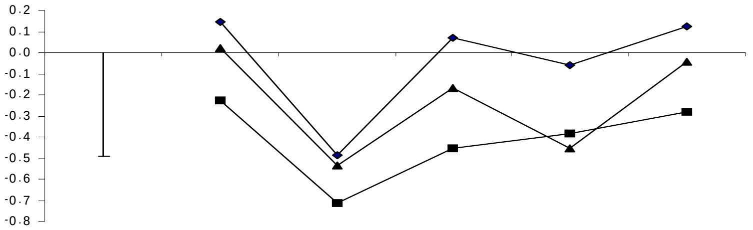
Note. PoI = Postintervention, B = Baseline (the average of Day 1 and Day 2 preintervention measures), PoIE = Postintervention and eccentric exercise, CI = confidence interval limits, ES = Effect size statistic



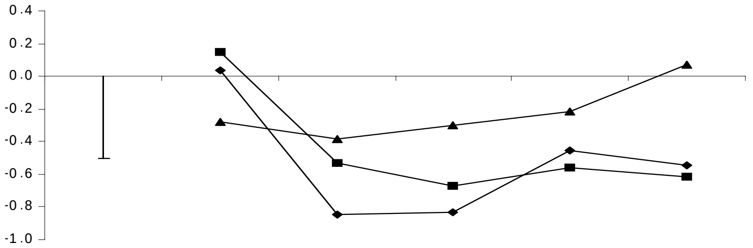
a) soreness sensation



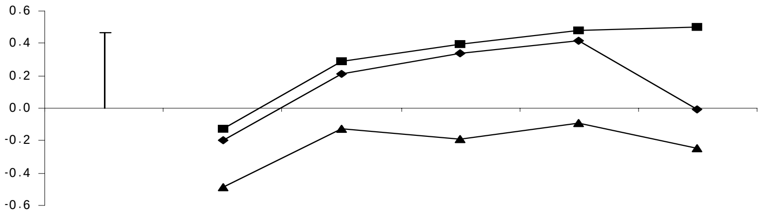
b) range of motion-active knee flexion



c) range of motion-active knee extension



d) maximum isometric torque



e) passive stiffness

Figure 5.2 Comparison of the magnitude of the effect-size statistic for soreness sensation, range of flexion and extension motion, maximum isometric torque, and passive stiffness of each intervention when comparing baseline (B) and postintervention (PoI), immediately postintervention and eccentric exercise (ImPoIE), and three consecutive days after the eccentric exercise (D1PoIE, D2PoIE, D3PoIE).

Notes: Day 1 represented the effects of intervention on muscle properties, Day 2 represented the acute effects of intervention on muscle damage, and Days 3, 4 and 5 represented the long term effects of intervention on muscle damage.

5.4.2 The effectiveness of interventions on the severity of muscle damage

The peak of muscle soreness and strength loss occurred one day after eccentric exercise in all intervention groups. Immediately after exercise, participants in the massage and stretching groups experienced slightly less soreness sensation than the warm-up group (ES = 0.33 to 0.47). The soreness sensation of the warm-up and massage groups returned to the baseline values on day three after eccentric exercise, while the soreness sensation of the stretching group was still above the warm-up group at the end of the five-day data collection period (ES = 0.35) (see Figure 5.2a). Throughout the follow-up period, strength loss in the warm-up group showed a better recovery than the massage and stretching groups (ES = 0.15 to 0.69) (see Figure 5.2d).

Knee flexion range of motion in the massage and stretching groups increased more than in the warm-up group (ES = 0.80 to 1.09) over the period of the study. There were trivial effects the massage group for knee extension range of motion when compared with the warm-up group on day two after eccentric exercise (see Figures 5.2b and 5.2c).

Passive stiffness in the massage and stretching groups was higher than in the warm-up group (ES = 0.47 to 1.46) after eccentric exercise throughout the data collection period (see Figures 5.2e).

5.5 Discussion

Preexercise activities aim to prevent injury by increasing joint range of motion (Gleim & McHugh, 1997) and muscle compliance.

The outcome after each intervention was compared with the baseline (pre and postcomparison) and the control group (warm-up), the results were comparatively similar. Therefore, only the results from the comparison between passive dynamic stretching and massage and warm-up groups were discussed.

Only the dynamic stretching group showed small effects of increasing knee flexion joint range of motion compared with the warm-up group (ES = 0.47). Active knee flexion range of motion is related to the ability of hamstring muscles to contract and is limited by the compliance of the quadriceps muscles. A repetitive passive movement may help to enhance viscoelastic responses (McNair et al., 2000) of both flexor and extensor muscles. This acute adaptation to passive continuous motion is also used in rehabilitation to maintain or increase range of motion.

In this study, the maximum isometric torque and passive stiffness in the warm-up group was reduced more than the massage and stretching groups (moderate effect). This might be due to warm-up causing the muscle-tendon units of the hamstring muscles to become more compliant, resulting in a reduction of passive stiffness. The more compliant muscle might, theoretically, delay the force transmitted to the joint (Gleim & McHugh, 1997), and require more energy to contract.

The mechanisms responsible for the decrease in passive stiffness could be the combination of increased muscle temperature (Magnusson et al., 2000) and muscle blood flow (Shoemaker et al., 1997), as well as the continuous movement of connective tissues (McNair et al., 2000). Previous research in-vitro showed that warm muscle (40°C) appeared to be more compliant and less susceptible to failure when compared with cooler muscle (35°C) (Noonan & Best, 1993). Interestingly, the general warm-up (30 minutes running at 75% of maximum O_2 uptake), which increased hamstring muscle temperature by 4°C in the Magnusson et al. (2000) study did not affect the viscoelastic properties of muscles. Similarly, passive continuous movement alone as presented by the stretching group in Magnusson et al. (1998), and in the present study, did not affect hamstring muscle properties. This research suggests that the combination of the increase in muscle temperature and continuous movement is required to reduce passive stiffness. For example, an increase in muscle temperature as well as the increase in muscle blood flow might help to reduce the resistance from muscle structures (i.e., cross-links between actin and myosin filaments, connections between endosarcomeric and exosarcomeric cytoskeletals, and perimysium) (Gajdosik, 2001). The continuous movement might help to redistribute the mobile components within the collagen framework such as polysaccharides and water (McNair et al., 2000). As a result, a specific warm-up is an effective protocol in reducing passive stiffness where passive stiffness is defined as the passive resistance or passive force to the change in the length displacement (Magnusson et al., 2000).

The results in this study showed that the participants in the warm-up group demonstrated less functional loss and faster recovery, as determined by knee flexion range of motion, maximal isometric torque, and passive stiffness, than the stretching and massage groups. This result is consistent with the report of Nosaka et al. (1997) for elbow flexor muscles. The mechanisms for reducing the severity of muscle damage might be muscle fibres would be more flexible and more tolerant to mechanical overload and, as a result, would show minimal symptoms of muscle damage. Unfortunately, there are no published studies to support this suggestion.

5.5.1 Limitations of the study and suggested further research

The results of this study must be considered in light of four potential limitations. First, data should ideally be collected using a cross-over design to reduce individual response effects, however, this was not possible due to the effect of repeated bouts of eccentric exercise inducing muscle damage. The random assignment of participants to warm-up, stretching, or massage groups attempted to minimize the effects of individual responses on the effect of the intervention. Second, there was no non-treatment control group. Active dynamic warm-up was chosen as a comparison treatment control group because it is common for athletes to jog as their warm-up. The KinCom dynamometer movement was used instead of free jogging in an attempt to keep the amount of warm-up and the intensity consistent between participants. Thirdly, there was a small sample size. Pilot work indicated a high reliability (intraclass correlation coefficient > 0.90) for all measures. Participants were asked to come to laboratory at the same time each day for five consecutive days. The measurements took two hours for the first day and one hour for the following days. The exercise protocol aimed to damage muscle and caused soreness, strength and range of motion loss on the following days. Because of these practical limitations, a number of thirty participants (10 participants per group) was recruited. Finally, the participants were not blind to the intervention, which may have led to variation in any psychological effects (which were not measured).

Further study on the effects of other types of preexercise activities such as ballistic and PNF stretching, or friction technique massage on passive muscle stiffness and the severity of muscle damage is required. Examination of the effect of preexercise activities on active stiffness, and the relationship between active stiffness and the severity of muscle damage, is recommended.

5.6 Conclusions

Active dynamic warm-up was more effective than dynamic passive stretching or effleurage and petrissage massage in reducing stiffness of knee flexor muscles after eccentric exercise. Athletes should be encouraged to perform active dynamic warm-up before eccentric exercises in sport performance.

CHAPTER 6

A COMPARISON OF PREEXERCISE INTERVENTIONS (DYNAMIC STRETCHING, AND MASSAGE) ON LEG STIFFNESS AND JUMPING AND SPRINTING PERFORMANCE

Weerapong, P., Hume, P. A., & Kolt, G. S. (2004). A comparison of preexercise interventions (dynamic stretching and massage) on leg stiffness and jumping and sprinting performance. Manuscript submitted for publication.

6.1 Summary

This study examined the effects of two preexercise interventions (dynamic stretching, and massage) on leg stiffness and performance (jumping and sprinting). Twelve adult males with no musculoskeletal problems were randomised into six groups that were combinations of stretching, massage, or warm-up intervention presentation. Each group performed one of the three interventions each day over three weeks. The warm-up group, which was used as a control group, jogged at a self-selected velocity for five minutes. The stretching group performed dynamic stretching exercises for the lower musculature as demonstrated to them by a qualified sport scientist. The massage group received a 10-minute effleurage, petrissage, and tapotement intervention on their legs by a qualified physiotherapist. Jumping height and sprinting time were measured three times: immediately after intervention, at 10 minutes after intervention, and 20 minutes after intervention. The vertical leg stiffness was calculated during the jumping take-off using the force over the displacement during contact with the ground. Data were analysed by using effect size statistics (ES) and confidence limit interval (CI). No clinically significant differences in variables (jumping height, leg stiffness, and sprinting time) were found at any data collection time except for the participants in the dynamic stretching group who showed small positive effects (ES = 0.28, CI = -.12 to .67) on jumping height. For this reason, dynamic stretching is a preferable preexercise activity when compared with massage or warm-up (jogging). Further research is needed to elucidate whether dynamic stretching can substitute static stretching to enhance sports performance.

6.2 Introduction

Sport practitioners typically use a combination of warm-up and stretching which are based on experience or anecdotal notes from coaches and atheletes. Reasoning behind these preexercise strategies is often based on possibly invalid generalizations of scientific findings. The systemic investigation of isolated preexercise activity is required to examine the effectiveness of preexercise activities.

Despite the expected benefits of warm-up, stretching, and massage to enhance performance, recent research does not support the notion that these preparatory exercises benefit performance. Gray and Nimmo (2001) found that low intensity warm-up (40% $\dot{V}O_{2max}$) had no effects on cycling time to the point of exhaustion (high intensity). Warm-up is a common practice before exercise and the literature did not show any detrimental effects of warm-up, warm-up. Therefore, low intensity jogging as a warm-up for five minutes (Gray & Nimmo, 2001) was selected for the control group in this study.

Static, ballistic, and proprioceptive neuromuscular facilitation (PNF) stretching result in decreased force production (Shrier, 2004) after stretching. Dynamic stretching, which uses functional movements, has also been used prior to exercise and training (Fletcher & Jones, 2004). A search of the literature found only one published paper showed that dynamic stretching decreased sprinting time immediately after intervention (Fletcher & Jones, 2004). There was no research on dynamic stretching on other types of performance such as jumping and no research on time effects.

In the study by Wiktorsson-Moller et al. (1983), 6 to 15 minutes of petrissage (a massage technique that is applied with firm pressure on the tissues) reduced muscle strength of hamstring concentric force and quadriceps isometric force. However, the tests of muscle strength in this study may not be suitable for monitoring performance. Performance measures such as jumping or sprinting are recommended to monitor the effects of intervention (Murphy & Wilson, 1997). Another study on the effects of preexercise massage focused on sprinters (Harmer, 1991) and reported that massage had no effects on mean stride frequencies. In the Harmer (1991) study, however, the outcome measure might be considered inappropriate, as stride frequency needs to be combined with stride length to determine performance. Therefore, the studies on the effects of preexercise massage on performance are limited.

Sports and clinical biomechanists are interested in the role of stiffness on performance and injury. A high level of leg vertical stiffness was found to benefit some performance such as jumping and sprinting (Harrison, Keane, & Coglan, 2004). It should be noted that in Harrison et al.'s study, sprinters and endurance runners had the same level of neuromechanical potentiation of muscle contraction as showed by the same level of stretch-shortening performance indices (countermovement jump/squat jump and drop-rebound jump/squat jump). Sprinters, however, performed higher jump height and lower sprinting time than the endurance runners, using a stiffer leg spring. According to the authors, muscle stiffness control was a key factor for the better performance (Harrison et al., 2004). The ability to control vertical leg stiffness resulted from the training programme. It is interesting to investigate whether preexercise activities contribute to the ability to control leg vertical stiffness and help to enhance performance.

This study compared the effectiveness of two preexercise interventions (dynamic stretching and massage) with warm-up on leg stiffness and jump and sprint performance in twelve healthy male participants.

6.3 Methods

6.3.1 Participants

Twelve healthy males aged 28.0 (5.6) years, mass 73.7 (8.0) kg, and height 175.3 (7.6) cm (mean (*SD*)) participated in the study. The study received approval from the Auckland University of Technology Ethics Committee. Participants read the participant information package that outlined the experimental procedure, and signed the consent form. All participants were asked to refrain from other exercise for one week before the test day and to come to the laboratory at the same period of time for four days (one day per week) over a period of four weeks. The participants were also asked to wear the same shoes for each testing day.

6.3.2 Test protocol

All participants completed a familiarization session where they were informed of the experimental procedure, had the correct techniques of jumping and sprinting demonstrated to them, and became familiar with the equipment and testing protocol. Participants practiced jumping and sprinting until they felt comfortable with the outcome measure protocols. Body weight and height were collected using scales and a stadiometer (SECA 220, Birmingham, England).

Participants were randomly assigned into one of six groups in order to minimise learning effects. The interventions were assigned in order in each group (warm-up-stretching-massage, warm-up-massage-stretching, stretching-warm-up-massage, stretching-massage-warm-up, massage-warm-up-stretching, massage-stretching-warm-up). Each group performed one of the three interventions (warm-up, dynamic stretching, or massage) each day. Twelve participants completed all three interventions at the end of data collection period.

The participants rested for ten minutes before receiving an intervention (warm-up, dynamic stretching, or massage). The outcome measures (jumping height and sprinting time from three countermovement jumps and, then, three 30 m sprints) were collected three times: immediately after the intervention, and 10 minutes, and 20 minutes post-intervention. The experimental setting is presented in Figure 6.1.

6.3.3 Interventions

6.3.3.1 Warm-up intervention

Participants performed warm-up by using a standard beep test (Australian Sports Commission, 1998) with a beep sounding at nine-second intervals (level one, repeated for five times) for a period of five minutes. Participants ran in an indoor stadium at their pace but were asked to run the same distance during each beep. The distance each participant ran on Day 1 was marked on the floor, and they ran the same distance on the following two days.

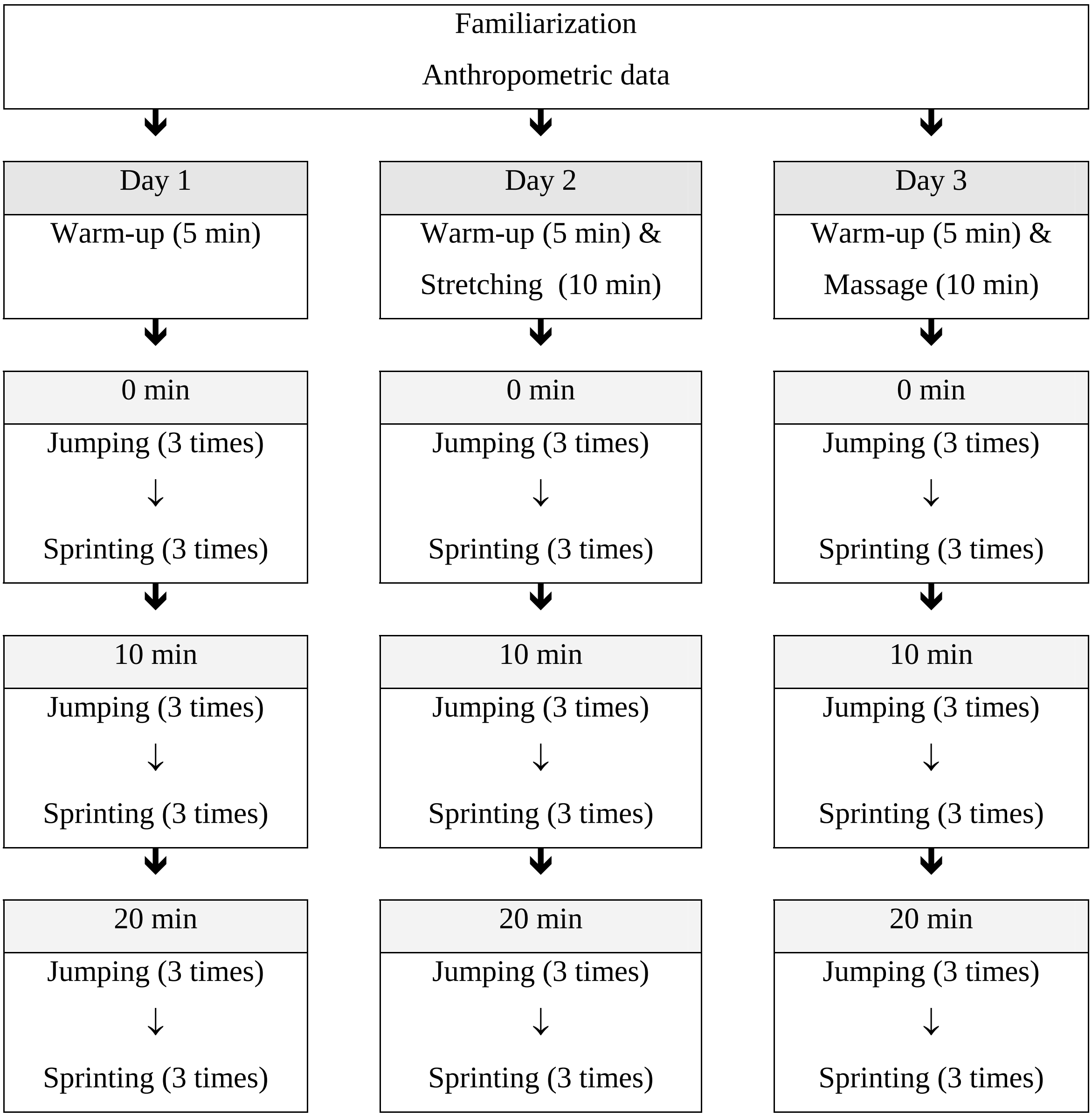


Figure 6.1 The experimental setting for one group

6.3.3.2 Warm-up and dynamic stretching interventions

Participants performed warm-up for five minutes as previously described before performing dynamic stretching, defined as movements of a series of joints as a result of muscle contractions throughout the range of motion (Fletcher & Jones, 2004). The dynamic stretching was designed to stretch all leg musculature, and participants were instructed by a qualified sport scientist to complete the stretching sequences as follows:

45 s x cycling calf stretch

From the starting position of trunk bent with both hands and feet on the floor, the participant bent the right hip and knee until the right heel was moved above the floor (right toes were still on the floor). The right leg was then straightened and the same procedure was repeated for the left leg (see Figure 6.2a).

45 s x lateral movement hamstrings stretch

From standing with feet shoulder width apart the participant bent the right hip and knee to 90°, the left hip was abducted with the knee straight, and the foot was pointed laterally. Body mass was then transferred to the right leg and the left leg was stretched, and then body mass transferred back again to the left leg and the right leg stretched. The procedure of stretching each leg was alternated slowly for 45 s (see Figure 6.2b).

45 s x swinging hamstrings stretch

The participant stood with one side of the body facing the wall, one hand holding a wall frame, and both feet shoulder width apart. Participant then moved the right hip into flexion and extension. Hip flexion and extension movements were alternated slowly for 45 s. Then, the participant stood with the left side to the wall and the procedure was repeated for the left leg for 45 s (see Figure 6.2c).

45 s x lateral movement adductors stretch

The participant stood facing the wall with both hands holding the wall frame. Both feet were shoulder width apart. The participant moved the right hip into abduction then adduction. Hip abduction and adduction movements were alternated slowly for 45 s. The procedure was repeated for the left leg for 45 s (see Figure 6.2d).

45 s x horizontal adductors and abductor stretch

From standing with feet slightly more than shoulder width apart the participant bent both hips and knees to 90° then transferred body mass to the right leg to stretch the left leg, and transferred body mass to the left leg to stretch the right leg. The procedure for each leg was alternated slowly for 45 s (see Figure 6.2e).

Forward walking lunge

From standing with feet shoulder width apart the participant stepped the right leg forward then bent the right hip and knee to 90° with the left hip slightly extended and the knee flexed to 90°. Participants then took a step forward and repeated the same procedure for the left leg (see Figure 6.2f). The total distance covered for this movement was for 35 m.

Backward walking lunge

From standing with feet shoulder width apart the participant extended the right hip and stepped backward with the right leg. Participants then bent the left knee until the left hip and knee were flexed to 90°, the right hip was in neutral position and the right knee was flexed to 90°. Next, participants took a step backward and moved the left leg toward the right leg before starting to stretch the left leg in the same procedure (see Figure 6.2g). The total distance covered for this movement was for 35 m.

Heels up (slow)

From standing with feet shoulder width apart the participant stepped the right leg forward. Participants then bent the left knee up to the buttocks with the left hip straight while the right leg on the floor. Next, participants extended the left knee and stepped the left leg forward. The procedure was repeated for the right leg (see Figure 6.2h). The total distance covered for this movement was for 35 m.

Knees up (slow)

From standing with feet shoulder width apart, the participant stepped the right leg forward. Participants then bent the left hip and knee up to the chest. Next, participants extended the left knee and stepped the left leg forward. The procedure was repeated for right leg (see Figure 6.2i). The total distance covered for this movement was for 35 m.

Crossover stepping

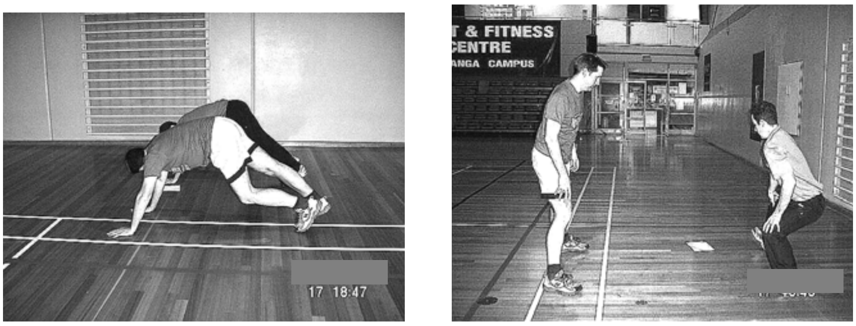
From standing with feet shoulder width apart the participant stepped the right leg sideward. Participants then flexed the left hip and stepped across the right leg sideward from the front. Next, participants extended the left hip and stepped the left leg sideward from the back. The procedure was repeated for the right leg (see Figure 6.2j). The total distance covered for this movement was for 35 m.

Two widths: heels up (fast)

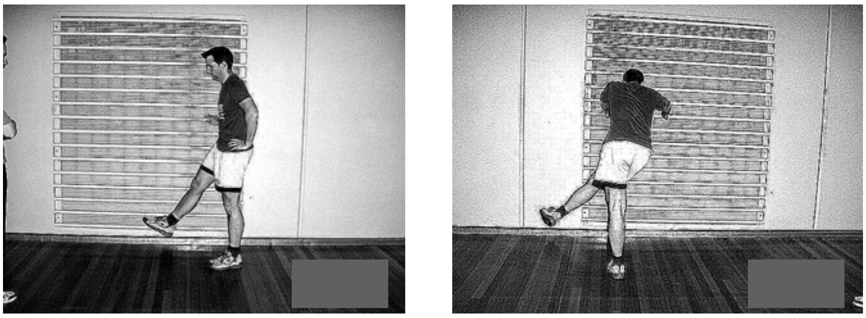
The same movement as heels up (slow) was completed but at a faster velocity and for 35 m (see Figure 6.2h).

Two widths: knees up (fast)

The same movement as knees up (slow) was completed but at a faster velocity and for 35 m (see Figure 6.2i).

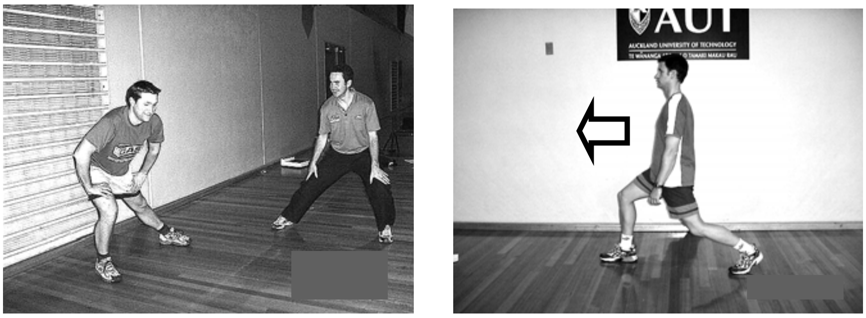

[redacted] [redacted]

a) Cycling calf stretch b) Lateral movement hamstrings stretch


[redacted] [redacted]

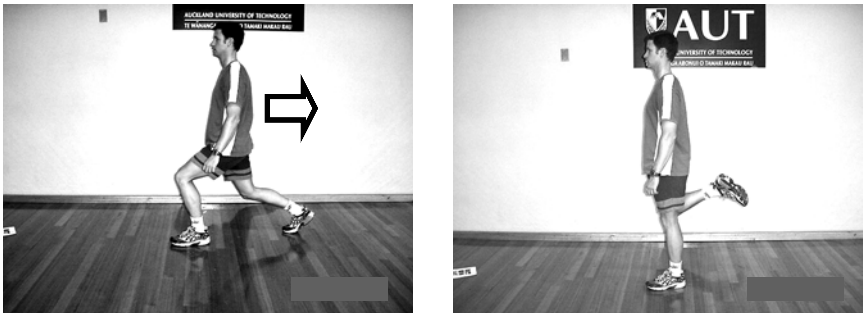
c) Swinging hamstrings stretch d) Lateral movement adductors stretch

Figure 6.2 Dynamic stretching protocol.



e) Horizontal adductors and abductors stretch

f) Forward walking lunge



g) Backward walking lunge

h) Heels up (slow)

i) Knees up (slow)

j) Crossover stepping

Figure 6.2 (continued) Dynamic stretching protocol.

6.3.3.3 Warm-up and massage intervention

Participants performed warm-up for five minutes as previously described. They then lay on their back on the massage plinth with pillow support under their knees while receiving ten minutes of effleurage, petrissage, and tapotement on their leg muscles. The basic Swedish remedial system (De Domenico & Wood, 1997, p. 85-88) was used in this study because it is a common practice. The same qualified physiotherapist (with ten years experience) administered all massage interventions on both legs with the massage sequences as follows (one time in each sequence meant the whole leg or the whole muscle, each sequence was performed for 30s):

1. Superficial stroking on the leg muscles four times
2. Palmar kneading on the quadriceps muscles three times
3. Palmar kneading on the hamstrings muscles three times
4. Superficial stroking on the lower leg muscles three times
5. Thumb kneading on the tibialis anterior three times
6. Palmar kneading on the calf muscles three times
7. Tapotement on the quadriceps four times
8. Tapotement on the hamstrings four times
9. Tapotement on the calf muscles four times
10. Superficial stroking on the leg muscles three times

6.3.4 Outcome measures

6.3.4.1 Measurement of vertical jump

Participants jumped vertically maximally from a force platform (Kistler 5233A, Switzerland) for three trials. Participants were allowed to rest one minute between jumps. Participants performed a counter movement jump by placing hands on the waist while bending the knees, and then jumping as high as possible. The degree of knee-bend was self-determined by each participant in order to jump as high as possible (Harman, Rosenstein, Frykman, & Rosenstein, 1990). The results of three attempts were averaged to calculate jump height to minimize possible crossover effects or progressive error (Church et al., 2001). The output from the force platform was collected at 500 Hz for further analysis using the LabView programme (National Instruments). Jumping impulse was calculated from the area under the force-time curve (Hamill & Knutzen, 1995) when the force was more than the participant's body mass. The jumping velocity at take-off ($V_{take\text{-}off}$) was calculated by dividing jumping impulse by body mass (Hamill & Knutzen, 1995). Jumping height (J_{ht}) was calculated as: $J_{ht} = (V_{take\text{-}off})^2/2g$ where g was the gravitational constant ($9.81\ m.s^{-2}$) (Hamill & Knutzen, 1995).

6.3.4.2 Measurement of leg stiffness

A model of a spring-mass was used to analyze the control of vertical leg spring stiffness (Harrison & Gaffney, 2004). The vertical leg stiffness was defined as the ratio of the force in the spring (ΔF) to the displacement of the spring (ΔL) at the instant that the leg spring was maximally compressed (Harrison & Gaffney, 2004). The proportion of the peak vertical reaction force and the maximal vertical displacement of the centre of mass show the simplest calculation of leg stiffness during contact with the ground (Butler et al., 2003; McMahon & Cheng, 1990).

To calculate leg stiffness from a vertical jump, the equation was manipulated by using Newton's second law of motion which states: $F = m*a$, where F = vertical force from the force platform; m = body mass; a = acceleration. Since $a = dv/dt$ this equation can be written as: $F = m* (dv/dt)$. If each side is multiplied by dt, the result is: $F*dt = d (m*v)$ or $F*dt = mv_{final} - mv_{initial}$.

The velocity of the person at the initiation of the jump is zero (Hamill & Knutzen, 1995), therefore the equation is: $F*dt = mv_{final}$, where F*dt. This is known as impulse (N.s) which is the area under the force-time curve and was obtained from the force platform output. Therefore, the velocity of the centre of mass at take-off (v) can be calculated as: $v = impulse/m$.

The displacement of the centre of mass (s) was calculated as: $s = v*t$, where t = time. The displacement was calculated from the area under the velocity-time curve.

The mechanical work (W) results from the force applied to the object, and the displacement of the object during the force application was calculated from: $W = F*s$.

The power of jumping at take-off (P) was calculated as: $P = W/t$ or the derivative of force-displacement curve.

The power per body mass (kg) was used to calculate the eccentric-concentric phase of the counterbalance jump (the point where the power per body mass changes from negative to positive is the eccentric to concentric transition point). This occurs when the knees joint bends maximally (eccentric) before take-off by pushing the knees to extend (concentric).

Leg stiffness (k) was calculated from: $k = \Delta F/\Delta L$, where ΔF is the force at the eccentric-concentric transition point; ΔL is the distance of the change of centre of mass from the eccentric-concentric transition point to the take-off point (when F is zero).

6.3.4.3 Measurement of sprinting test

Participants ran 30 m through photoelectric cells which detected time at the beginning and at the end of the 30 m (Krustrup, Mohr, & Bangsbo, 2002). Participants were allowed to rest one minute between sprintings. The results of three attempts were averaged to calculate sprinting time (Church et al., 2001).

6.4 Statistical analysis

To compare the effects of warm-up, dynamic stretching, and massage on jump height and sprinting time, paired-t tests and effect sizes (ES, fractions of baseline between-subject standard deviations) with the precision of estimation at 90% confidence limits were used. The 90%CI was used because it represented adequate precision for making probabilistic inferences. The probability that the true value was less than the lower confidence limit and more than the upper confidence limit were both only 0.05, which can be interpreted as very unlikely. The thresholds of the effect for trivial, small, moderate, and large magnitude were 0.2, 0.6, 1.2, and 2.0, respectively (Hopkins, 2002b). Data were analysed in log transformation because it provided the most accurate estimate of the percent change or difference. The relationships between leg stiffness and jump height and sprinting time were analysed with Pearson correlations.

6.5 Results

The mean and standard deviations of jump height, leg stiffness, and sprinting time at 0, 10, and 20 minutes after intervention are presented in Table 6.1.

Table 6.1 Mean and standard deviations of jumping height (m), leg stiffness (N.m^{-1}), and sprinting time (s).

Intervention		Jumping height (m)			Leg stiffness (N.m^{-1})			Sprinting time (s)		
		0 min	10 min	20 min	0 min	10 min	20 min	0 min	10 min	20 min
Stretching	Mean	0.22	0.21	0.21	47.82	48.80	48.25	4.54	4.59	4.61
(*n*=12)	*SD*	0.06	0.06	0.06	14.87	17.00	18.52	0.35	0.37	0.35
Massage	Mean	0.20	0.20	0.20	48.62	50.52	50.93	4.63	4.58	4.63
(*n*=12)	*SD*	0.05	0.04	0.04	18.82	18.96	20.12	0.37	0.38	0.39
Warm-up	Mean	0.20	0.20	0.20	50.41	49.54	52.16	4.57	4.58	4.59
(*n*=12)	*SD*	0.04	0.04	0.05	17.83	16.35	21.25	0.35	0.37	0.37

No clinically significant differences in variables (jumping height, leg stiffness, and sprinting time) were found in the massage and dynamic stretching groups at any data collection time, except for participants in the dynamic stretching group showing small positive effects on jumping height (see Table 6.2). The magnitude of the ES were progressively declined throughout the data collecting period (ES = 0.28 to 0.17). The lower confidence limits did not reach -0.2 which meant that dynamic stretching had low probability to be harmful. The upper confidence limits were 0.4 to 0.7 which meant that dynamic stretching was likely to be beneficial. No clinical significance in other variables (leg stiffness and sprinting time) was found in the dynamic stretching group.

There was a moderate but significant negative correlation (-0.43 to -0.49) between leg stiffness and performance (jumping height and sprinting time) (see Table 6.3).

Table 6.2 Effects sizes, 90%CI, % change, and *p*-value when compared between stretching (*n* = 12) or massage (*n* = 12) interventions with warm-up (*n* = 12).

Measures	Statistics (log transformed)	Stretching - warm-up			Massage - warm-up		
		0 min	10 min	20 min	0 min	10 min	20 min
Jumping height	ES	0.28	0.23	0.17	-0.10	0.12	0.13
	90%CI	-0.12, 0.67	0.01, 0.46	-0.09, 0.43	-0.38, 0.17	-0.14, 0.38	-0.13, 0.39
	% change	5.69	5.33	4.70	-2.06	2.67	3.65
	P-value	0.24	0.09	0.26	0.51	0.44	0.38
Leg stiffness	ES	-0.10	-0.05	-0.17	-0.11	0.01	-0.06
	90%CI	-0.45, 0.25	-0.36, 0.26	-0.42, 0.09	-0.30, 0.08	-0.30, 0.33	-0.41, 0.28
	% change	-3.78	-1.82	-6.81	-4.29	0.45	-2.56
	P-value	0.51	0.82	0.35	0.41	0.80	0.81
Sprinting time	ES	-0.10	0.03	0.08	0.17	0.00	0.11
	90%CI	-0.28, 0.08	0.15, 0.21	-0.06, 0.21	-0.07, 0.40	-0.16, 0.16	-0.10, 0.33
	% change	-0.73	0.23	0.60	1.21	-0.01	0.86
	P-value	0.33	0.76	0.31	0.22	1.0	0.36

Table 6.3 Pearson correlations between leg stiffness, and jumping height and sprinting time.

Leg stiffness		Jump height	Sprinting time
0 min	Correlation	-0.430	-0.455
	P-value	0.009	0.015
10 min	Correlation	-0.467	-0.459
	P-value	0.004	0.014
20 min	Correlation	-0.493	-0.464
	P-value	0.002	0.013

6.6 Discussion

This study is the first published investigation to present the effects of dynamic stretching on jumping height, and the effects of massage on jumping height. In this study results at 0, 10, and 20 minutes after intervention were also reported. The main finding in this study was that dynamic stretching increased jumping height immediately after interventions and slightly declined throughout 20 minutes data collection period. Among the 12 participants three participants (25%) showed a reduction of 14%, two participants (16%) showed no change (0%), and seven participants (59%) showed an increase in jumping performance of 17%. A mixed response from participants was also reported in Knudson et al. (2001) who suggested that it was an expected effect of self-administered stretching in the real world. Our results showed that dynamic stretching enhanced jumping height in majority of participants (59%). There were no clinical significant changes in leg stiffness following any interventions.

The present study was limited by a small sample size. The study, therefore, was designed as a counterbalance design, which eliminated of the individual response effects because the data from each participant was compared with their own performance after dynamic stretching and massage with their data from the warm-up. The study was also designed to minimise learning effects by allowing familiarization session and arranged the order of the interventions into six groups, and ensured that an equal number of participants received the same order of intervention. The time gap between each intervention was one week, which allowed the participants to achieve a full recovery from the previous test. The participants were asked not to perform any rigorous exercise during data collection period. The tests were conducted at the same time each day and at the same day of the week. As well, pilot work showed a high reliability between days (CV = 3-7% for jumping height and 1-3% for sprinting time). Therefore, our results should represent the true effects of dynamic stretching and massage on jumping performance in healthy male participants.

Recent research has reported detrimental effects up to 30% on performance for static, ballistic, and PNF stretching (Shrier, 2004). In our study, jumping height increased 6%, 5%, and 5% at 0, 10, and 20 minutes after intervention, respectively, when comparing dynamic stretching with the warm-up intervention. Data were presented as a percentage of log transformation that can be applied to individual who has different jumping ability.

There was only one published paper on the effects of dynamic stretching on performance. Fletcher and Jones (2004) found that dynamic stretching enhanced performance by decreasing sprinting time. The mechanism responsible for the decrease in sprinting time in Fletcher and Jones’ study maybe due to an increase in core temperature after muscle contractions or an increase in coordination after repetitive movements. In our study, however, dynamic stretching increased only jumping height but showed no effects on sprinting time. The reason may be due to sprinting time in Fletcher and Jones’ study was measured immediately after dynamic stretching while sprinting time in our study was measured five minutes after intervention. Jumping height in this study was measured immediately after intervention and the increase in jumping height lasted long for 20 minutes. Therefore, the mechanism of dynamic stretching responsible for increasing jumping height may be different from the decrease in sprinting time in Fletcher and Jones’ study.

The reason why dynamic stretching enhanced jumping height but had no effects on sprinting time in this study is still unclear. The ability to control muscle stiffness has been shown to enhance jumping and sprinting performance (Harrison et al., 2004). In our study, however, we did not found effects dynamic stretching and massage on leg vertical stiffness. Average leg vertical stiffness in this study was 48-52 $N.m^{-1}$ for the twelve participants, which was consistent with a previous report (Arampatzis, Schade, Walsh, & Bruggemann, 2001). Our study, however, found a significant negative correlation between leg stiffness and jumping height and sprinting time i.e., a stiffer limb resulted in lower jump heights and shorter sprinting times. This result was consistent with a study by Harrison et al.(2004) that athletes who performed better jumping and sprinting used a significant stiffer leg spring than the other group.

Dynamic stretching may enhance performance through the biomechanical, physiological, and neurological mechanisms because the nature of this type of stretching is repetitive active movement throughout range of motion (Fletcher & Jones, 2004). Participants may achieve the ability to control muscle stiffness after a repetitive active movement of leg musculature. Repetitive active dynamic contraction of muscle throughout the range of motion is expected to increase muscle temperature and muscle blood flow. Movement, without holding at end range of motion, may not reduce neuromuscular sensitivity but help to enhance coordination and proprioceptive sensation (Weerapong, Hume, & Kolt, 2004).

Results in our study showed no clinical significance in sprinting time which was consistent with that of Harmer (1991) who found that massage had no effects on stride length in sprinters. The non-clinical significant results might be due to the fact that neither dynamic stretching nor massage had any effects on sprinting. Alternately, sprinting might not have been sensitive enough to detect the effects of the intervention. Another explanation might relate to the fact that sprinting was performed after jumping. The time gap between the intervention and sprinting was four minutes. If the effects of dynamic stretching and massage lasted less than five minutes then they would not have had any impact on sprinting during sport performance.

6.7 Conclusions

The results in the present study showed that only the dynamic stretching group showed a clinical significantly higher jumping height throughout the data collection period. The reason may be due to the nature of dynamic stretching is a repetitive active movement throughout range of motion which may help to enhance performance by biomechanical (ability to control muscle stiffness), physiological (increase muscle temperature and blood flow), and neurological (increase coordination and proprioception) mechanisms. The reason why these benefits affected only on jumping but not sprinting is still unclear. There is very limited published paper on dynamic stretching, therefore, further study of dynamic stretching on biomechanical, physiological, and neurological mechanisms and the effects of dynamic stretching in more variety of sport performance in professional athletes is recommended.

CHAPTER 7

GENERAL DISCUSSION

7.1 Discussion

In this PhD thesis, chapters 2-6 are presented as manuscripts submitted to international peer-review journals. Each journal prescribes length, therefore, this chapter is presented to clarify and elaborate the ideas on the thesis work that can not be included in a journal manuscript.

The definition of each intervention is different in Study A and Study B, depending on the objective of the study. The warm-up intervention in Study A was operationally defined as the active dynamic movement of hamstrings which has been called a specific warm-up. The warm-up intervention in Study B was operationally defined as running which has been called a general warm-up. The warm-up intervention was used as a control group because it is commonly performed before exercise and sport competition. The stretching intervention in Study A was operationally defined as passive dynamic movement, a movement which was reported to reduce passive stiffness (McNair et al., 2000). The stretching intervention in Study B was operationally defined as the active dynamic movements of leg muscles which has been called dynamic stretching. This type of stretching has been used before exercise (Shellock & Prentice, 1985) but there are no published studies on its effects on performance. The massage intervention applied in Study A consisted of effleurage and petrissage. The aim of these two techniques was to increase muscle compliance. The massage intervention used in Study B consisted of effleurage, petrissage, and tapotement, and these techniques were applied with the aim of increasing muscle compliance and stimulating neural activity. The summary of the interventions and definitions are presented in Table 7.1.

The first study of this PhD thesis compared three preexercise interventions, warm-up (active dynamic movement), stretching (passive dynamic movement), and massage (effleurage and petrissage), on passive stiffness and the severity of muscle damage. Active dynamic movement was applied with the aim of increasing muscle blood flow and increasing muscle temperature. Passive dynamic movement was applied with the aim to increase muscle compliance. Massage was applied with the aim of increasing muscle blood flow and increasing muscle temperature, and increasing muscle compliance. Passive stiffness was investigated as a responsible mechanism of preexercise interventions because previous research reported a positive relationship between passive stiffness and the severity of muscle damage (McHugh, Connolly, Eston, Kremenic et al., 1999). A decrease in passive stiffness was expected to reduce mechanical overload on muscle fibres and reduce muscle damage.

Table 7.1 Interventions and definitions in Study A and B.

Intervention	Definitions	
	Study A	Study B
Warm-up	Active dynamic movement (specific warm-up)	Running (general warm-up)
Stretching	Passive dynamic movement (passive dynamic stretching)	Active dynamic movement (active dynamic stretching)
Massage	Effleurage and petrissage techniques.	Effleurage, petrissage, and tapotement techniques.

Only active dynamic movement appeared to reduce passive stiffness (ES -0.49, 90% confidence limits ±0.26) and functional loss (-0.38 ±0.32 and -0.88 ±0.67 for strength and range of motion, respectively) from muscle damage from eccentric exercise. Active dynamic movement might reduce passive stiffness by increasing muscle blood flow and temperature. A repetitive movement of active dynamic movement might help to reorganize the mobile components within the collagen framework such as polysaccharides and water (McNair et al., 2000).

A reduction in passive stiffness before eccentric exercise might help to reduce the severity of muscle damage. The possible mechanisms, however, have not been investigated in this thesis. These mechanisms were based on information from previous research which reported that greater strain on muscle produces greater muscle damage (Morgan & Allen, 1999), and that stiff muscle showed greater muscle damage than compliant muscle (McHugh, Connolly, Eston, Kremenic et al., 1999). A compliant muscle-tendon unit might help to reduce the severity of muscle damage by: 1) increasing the optimal length of muscle fibres from the length-tension curve; 2) increasing the ability to distribute the force across the muscle-tendon unit from eccentric exercise; 3) rearranging connective tissue to provide more protection; 4) increasing tolerance to lengthening; and 5) increasing the ability to absorb energy. It should be noted, that no published studies exist that provide direct evidence to support these mechanisms. These mechanisms have been suggested based on indirect literature and need to be investigated further.

Five particular theories are suggested to explain why the complaint muscle showed less severity of muscle damage. Firstly, when muscle is lengthened beyond the optimal length, the sarcomeres become more unstable (Morgan & Allen, 1999). Each sarcomere is stretched until it is unable to support the tension. Within a muscle at a given length, shorter sarcomeres are stretched more and become disrupted after repeated lengthening (Brockett, Morgan, Gregory, & Proske, 2002). The area of disrupted sarcomeres spreads throughout muscle fibres and leads to muscle damage (Brockett et al., 2002). This hypothesis is supported by the finding that eccentric exercise at a longer muscle length (stretch position) caused more muscle damage than the shorter length (Nosaka & Sakamoto, 2001). Newham et al. (1988) and Nosaka and Sakamoto (2001) compared two different starting positions with the same range of motion (short and long muscle range, 50-130° and 100-180°, respectively). The participants reported more soreness and demonstrated more strength and range of motion loss, and more swelling in the long muscle length condition than in the short muscle length condition. The images from magnetic resonance and ultrasound also showed greater damage in the long muscle length condition than the short muscle length condition (Nosaka & Sakamoto, 2001). Therefore, if an intervention helps to increase optimal length of muscle, it may help to reduce muscle damage from eccentric exercise.

A second theory is that more compliant muscle might distribute the force acting on muscle fibres and the tendon-aponeurosis complex better than in a stiffer muscle. Even though the literature contains no direct evidence to support this hypothesis, previous research has reported that a compliant muscle (measured using the same protocol as our study) showed less severity of muscle damage after eccentric exercise (McHugh, Connolly, Eston, Kremenic et al., 1999).

A third theory is that perimysium plays an important role in preventing over-stretching of muscle fibre bundles. Purslow (1989) suggested that perimysium is arranged in different directions at different sarcomere lengths, and that perimysium collagen networks can be rearranged in some circumstances such as when muscle is immobilised (Williams & Goldspink, 1984). When muscle was immobilised in a lengthened position, the angle of the collagen fibres was arranged at a less acute angle than when muscle was in shortened position (Williams & Goldspink, 1984). The rearrangement of perimysium might affect the compliance of muscle. Therefore, any intervention that causes an acute adaptation of the parallel elastic components by rearranging connective tissue of muscle might help to reduce muscle damage.

The fourth theory suggests that compliant muscle fibres might be more tolerant to lengthening load. The increase in stretch tolerance resulted in increasing muscle compliance (range of motion) after static stretching (Halbertsma et al., 1996; Klinge et al., 1997). Stretch tolerance is defined as the individual's limitation of movement (Halbertsma et al., 1996). Although there is no clear evidence to support this hypothesis, the increase in stretch tolerance may help to increase the ability of muscle fibres to sustain mechanical overload and, therefore, reduce muscle damage.

The final theory to explain why compliant muscle shows less severity of muscle damage is that compliant muscle-tendon units may increase elastic energy storage and utilisation more than stiff connective tissues (McNair & Stanley, 1996; Weldon & Hill, 2003). Therefore, compliant connective tissues may absorb force from eccentric exercise more than stiff connective tissues and transfer this force from connective tissues to muscle fibres less than stiff connective tissue (Anderson & Pandy, 1993). Unfortunately, there were no published studies to support this mechanism in humans. A study by Ettema (1996) reported that complaint muscle showed greater total energy storage in series elastic components and better mechanical efficiency than stiff muscle in rat gastrocnemius muscle. Ettema's (1996) study, however, investigated mechanical efficiency and efficiency of storage and release of series elastic energy in skeletal muscle during the stretch-shortening cycle. Mechanical efficiency and efficiency of storage and release of parallel elastic components during eccentric contraction have not been investigated yet.

The ideal preexercise activity should be both prevent injury and enhance performance. From the result of study A, a specific warm-up or dynamic active movement showed the best preventative result among the three interventions. So in study B, the effects of active dynamic movement and massage on leg stiffness and jump and sprint performance were investigated. The results showed that neither active dynamic movement nor massage affected leg stiffness and jump height and sprint time when compared with a control warm-up only group. Active dynamic movement showed a small positive effect while massage showed a trivial negative effect when compared with warm-up. For this reason, active dynamic movement is a preferable preexercise activity when compared with massage and warm-up only.

The small positive effects of active dynamic movement on jump and sprint performance might be the result of the nature of movement similar to a combination between warm-up and stretching. The active contraction of muscles helps to increase muscle temperature and blood circulation (warm-up effects) and the repetitive movement throughout ROM helps to increase muscle compliance (stretching effect). Movement, without holding at end range of motion, may not reduce neuromuscular sensitivity. As there are no published studies on the effects of dynamic movement on biomechanics (e.g., active and passive stiffness and ROM) and neurological mechanisms (e.g., H-reflex), further research is needed to elucidate the benefits of dynamic stretching on flexibility, muscle properties, and neuromuscular sensitivity. The information from such research will be beneficial to sport and health practitioners to help make decisions on whether active dynamic movement should be performed instead of static stretching (recent research reported that static stretching showed the detrimental effects on performance and had no effects on eccentric exercise-induced muscle damage prevention [see Chapter 3]).

From the results of the two studies, active dynamic movement was shown to be the best intervention when compared with passive dynamic movement or massage interventions because it showed preventative effects on eccentric exercise-induced muscle damage (Study A) and showed a small positive effects on performance (Study B).

7.2 Practical applications

The results of this thesis showed positive effects of active dynamic movement in minimizing muscle damage and enhancing performance. Active dynamic movement helped to minimize function loss as indicated by muscle strength and range of motion but did not help to minimize soreness sensation as massage did. Previous research showed that posteccentric exercise massage also helped to reduce soreness sensation. To minimize the obvious symptoms of muscle damage after eccentric exercise athletes should perform active dynamic movement before eccentric exercise and receive massage 1-2 hours after eccentric exercise (Farr et al., 2002).

As there is very limited research on preexercise dynamic stretching, more research is needed to confirm these benefits and investigate the mechanisms. It should be noted that the individual responses is an important issue. A majority of participants (59%) in this study showed an increase in jumping height while some participants (25%) showed a detrimental effect after dynamic stretching. These results are consistent with studies by Fletcher and Jones (2004) and Knudson et al. (2001). Therefore, coaches and sports practitioners should watch for individual athlete responses and provide appropriate stretching protocols for each of them.

Dynamic stretching was found to benefit the stretch-shortening cycle (SSC) as indicated by jumping in this PhD thesis and sprinting in Fletcher and Jones' study (Fletcher & Jones, 2004). It was interesting that, in the present study, the increase in jumping height lasted for over 20 minutes, but sprinting time was not affected at all. Sprinting time in this study was measured five minutes after the interventions while sprinting time in Fletcher and Jones' study was measured immediately after the intervention. Given limited evidence, dynamic stretching may provide more benefit in performances of countermovement jumps that require more eccentric action and muscle compliance, rather than sprinting which has a rapid switch from eccentric to concentric action. Some researchers have claimed that complaint muscle performs SSC movement (rebound bench press) better than stiff muscle (Wilson et al., 1992). Alternatively a reduction of muscle stiffness after static stretching partly accounted for the decrease in countermovement jump performance (Cornwell et al., 2002). Active stiffness was measured in both studies. Dynamic stretching may benefit other activities that do not require SSC movement such as cycling, swimming through physiological, neurological, and psychological mechanisms such as an increase in muscle temperature and blood flow, increase in muscle coordination and proprioception, as well as the awareness of exercise in the central nervous system.

From the results of this PhD thesis, it is recommended that athletes start the preexercise programme with a general warm-up which should be similar to sports or exercise activities such as slow swimming if the athletes are swimmers. General warm-up will help to prepare the muscles, body, and mind for exercise. Then, active dynamic movement or dynamic stretching should be performed to help prevent muscle damage and probably help enhance performance. The repetitive movement should be performed by the muscle used in upcoming sports or exercise. Static stretching should not be performed immediately before exercise as it shows detrimental effects on performance and no effects on eccentric exercise induced muscle damage prevention (Shrier, 2004). Cool-down and static stretching should be performed after training or exercise. Static stretching will help to increase range of motion and reorganize muscle fiber after exercise (McNair et al., 2000). Long term static stretching training showed no detrimental effects on performance (Wilson et al., 1992). Massage may be provided 1- 2 hours after exercise to minimize soreness sensation and for relaxation (Farr et al., 2002).

7.3 Limitations

There were some potential limitations on the two studies reported in this thesis. Study A only focused on passive stiffness because of technical limitations. The pilot study indicated that it was impossible to perform massage and eccentric exercise, and measure passive stiffness and active stiffness in one muscle without moving the participants several times. The time taken to perform and measure all of these parameters was also considered to take too long especially when the participants were asked to come to the laboratory for five consecutive days and required to come at the same time each day (two hours for the first two days and one hour for three subsequent days). There was no study on the correlation between active muscle stiffness and severity of muscle damage. Therefore, passive stiffness of hamstring muscle was chosen for study because it has been found that the participants with stiffer muscle, as determined by passive stiffness of hamstring muscles, showed more severity of muscle damage than participants with more compliant muscle (McHugh, Connolly, Eston, Kremenic et al., 1999).

The design of Study A could not be a cross-over with the participants in all interventions due to the repeated bout effects of eccentric exercise. A cross-over design should be used to eliminate the individual responses and provide less variable results. Nosaka et al. (2001a), however, reported on the occurrence of the repeated bout effects of eccentric exercise up to six months after completing the initial exercise. Therefore, a randomised controlled trial design was used in Study A due to a time constraint.

In Study B, the participants were healthy adult males who were not familiar with the technical aspects of optimising jumping and sprinting activities. Initially, soccer players from local clubs and Auckland University of Technology (AUT) were contacted. However, those players did not respond to the recruitment initiation due to a requirement of study design that the players needed to come to the AUT stadium once a week for four consecutive weeks. The time to collect data needed to be early morning and during the day as a consequence of the stadium's schedule. Therefore, AUT students were recruited to be participants. Even though the AUT participants were allowed to familiarize themselves in a session prior to the beginning of the research study, the variation of outcome measures (jumping height and sprinting time) is likely to be higher than for those of athletes.

Jumping height was calculated indirectly from flight time using force platform data. Data were also collected using a video recorder to compare jumping height with the data from the force platform. A high correlation of jump height (R = 0.81) between data calculated from flight time (force platform) and the change of centre of mass (video recorder) was found. The changes to ankle, knee, and hip ROM and centre of mass (COM) using the video tape would be analysed as evidence if there was any significant change in the outcome measures after intervention. As a consequence of the finding of a nonsignificant increase in jump height (6%) after dynamic stretching, data from one subject who showed the highest increase in jump height was selected to analyse the change in ROM and COM by using the SiliconCoach-Pro programme1. However, there

---

1 SiliconCoach-Pro programme is a computer programme which allows kinematic data analysis from video images.

were no differences in both ROM and COM. This finding was consistent with a study by Knudson et al. (2001) who reported a nonsignificant reduction in jump height (3%) after static stretching and nonsignificant changes in kinematic variables (duration of eccentric and concentric phase and knee angle). It also took 15 hours to analyze data from the video tape for just one participant. Data from the force platform, therefore, were selected to be analyzed.

7.4 Recommendations

This thesis reported the effects of warm-up, stretching, and massage on performance and the severity of muscle damage. Active dynamic movement was the best activity for athletes to maintain performance and reduce the severity of muscle damage from eccentric exercise. Further studies are recommended as followed:

- The relationship between active stiffness and the severity of muscle damage. This study will provide information on the relationship between contractile components, series elastic components, and reflex of muscle properties and severity of muscle damage.

- The effects of other types of stretching (e.g., ballistic and PNF) and general warm-up (e.g., jogging) on active and passive stiffness. This study will provide information on the best intervention for reducing muscle stiffness. A cross-over design is preferable to minimise individual response effects.
- The effects of active dynamic movement on active stiffness. This study will provide information on the effects of active dynamic movement on contractile components, series elastic components, and reflex of muscle properties.

- The effects of active dynamic movement on specific performance in athletes. This study will provide practical information for coaches and athletes on whether active dynamic movement is beneficial to sport performance.
- The relationship of active and passive stiffness to leg stiffness. This study will provide information on the relationship between stiffness of muscle properties and functional stiffness measurement.

- The relationship between active or passive stiffness and rate of injury. This study will provide practical information on the relationship between dynamic muscle flexibility and injury prevention.
- Neurological and psychological effects of active dynamic movement on performance. This study will provide information on how active dynamic movement affects performance and prevents muscle damage from eccentric exercise. Such information will help researchers and coaches in modeling a proper preexercise activity.

7.4 Conclusions

Active dynamic stretching (warm-up in Study A) was the most effective intervention among the three interventions studied (active dynamic movement, passive dynamic movement, and massage) in reducing the functional loss (range of motion and strength loss) from exercise-induced muscle damage by reducing passive stiffness. The benefit of active dynamic movement on performance (jump height) is still unclear as the results of this thesis showed only small positive effects on jump but not on sprint performance. Among the three preexercise interventions investigated in this thesis, the active dynamic movement was the best preexercise strategy for reducing the severity of muscle damage and maintaining/enhancing sports performance.

REFERENCES

Abraham, W. (1977). Factors in delayed muscle soreness. *Medicine & Science in Sports & Exercise, 9*, 11-20.

Anderson, F., & Pandy, M. (1993). Storage and utilization of elastic strain energy during jumping. *Journal of Biomechanics, 26*, 1413-1427.

Appell, H., Soares, J., & Durate, J. (1992). Exercise, muscle damage and fatigue. *Sports Medicine, 13*, 108-115.

Arampatzis, A., Schade, F., Walsh, M., & Bruggemann, G. (2001). Influence of leg stiffness and its effect on myodynamic jumping performance. *Journal of Electromyography and Kinesiology, 11*, 355-364.

Armstrong, R. (1984). Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. *Medicine & Science in Sports & Exercise, 16*, 529-538.

Armstrong, R. (1990). Initial events in exercise-induced muscular injury. *Medicine & Science in Sports & Exercise*, *22*, 429-435.

Armstrong, R., Ogilvie, R., & Schwane, J. (1983). Eccentric exercise-induced injury to rat skeletal muscle. *Journal of Applied Physiology, 54*, 80-93.

Armstrong, R., Warren, G., & Warren, J. (1991). Mechanisms of exercise-induced muscle fibre injury. *Sports Medicine, 12*, 184-207.

Arnett, M. (2002). Effects of prolonged and reduced warm-up on diurnal variation in body temperature and swim performance. *Journal of Strength and Conditioning Research*, *16*, 256-261.

Avela, J., Kyrolainen, H., & Komi, P. (1999). Altered reflex sensitivity after repeated and prolonged passive muscle stretching. *Journal of Applied Physiology, 86*, 1283-1291.

Bale, P., & James, H. (1991). Massage, warmdown and rest as recuperative measures after short term intense exercise. *Physiotherapy in Sport, 13,* 4-7.

Balke, B., Anthony, J., & Wyatt, F. (1989). The effects of massage treatment on exercise fatigue. *Clinical Biomechanics, 1,* 189-196.

Balnave, C., & Thompson, M. (1993). Effect of training on eccentric exercise-induced muscle damage. *Journal of Applied Physiology, 75*, 1545-1551.

Barles, P., Robinson, J., Allen, J., & Baxter, G. (2000). Lack of effect of acupuncture upon signs and symptoms of delayed onset muscle soreness. *Clinical Physiology, 20*, 449-456.

Behm, D., Button, D., & Butt, J. (2001). Factors affecting force loss with prolonged stretching. *Canadian Journal of Applied Physiology, 26*, 261-272.

Bell, A. (1964). Massage and physiotherapist. *Physiotherapy, 50*, 406-408.

Benjamin, P., & Lamp, S. (1996). *Understanding sports massage*. Champaign, IL: Human Kinetics.

Benz, R., Friden, J., & Leiber, R. (1998). Simultaneous stiffness and force measurements reveal subtle injury to rabbit soleus muscles. *Molelular and Cellular Biochemistry, 179*, 147-158.

Bishop, D., Bonetti, D., & Dawson, B. (2001). The effect of three different warm-up intensities on kayak ergometer performance. *Medicine & Science in Sports & Exercise, 33*, 1026-1032.

Black, C., Vickerson, B., & McCully, K. (2003). Noninvasive assessment of vascular function in the posterior tibial artery of healthy humans. *Dynamic Medicine, 2*, 1.

Blood, R. (2000). *Massage therapy at Ironman Canadian Triathalon*. Retrieved 23, 07, 2001, from http//www.amtamassage.org/journal/su_00_journal/su_00_sports_med.html

Boone, T., & Cooper, R. (1995). The effect of massage on oxygen consumption at rest. *American Journal of Chinese Medicine, 13*, 37-41.

Bourgeois, J., MacDougall, D., MacDonald, J., & Tarnopolsky, M. (1999). Naproxen does not alter indices of muscle damage in resistance-trained men. *Medicine & Science in Sports & Exercise, 31*, 4-9.

Braverman, D., & Schulman, R. (1999). Massage techniques in rehabilitation medicine. *Physical Medicine and Rehabilitation Clinics of North America, 10*, 631-649.

Brockett, C., Morgan, D., Gregory, J., & Proske, U. (2002). Damage to different motor units from active lengthening of the medial gastrocnemious muscle of the cat. *Journal of Applied Physiology, 92*, 1104-1110.

Brockett, C., Morgan, D., & Proske, U. (2001). Human hamstring muscles adapt to eccentric exercise by changing optimum length. *Medicine & Science in Sports & Exercise, 33*, 783-790.

Brown, S., Child, R., Day, S., & Donnelly, A. (1997). Exercise-induced skeletal muscle damage and adaptation following repeated bouts of eccentric muscle contractions. *Journal of Sports Sciences, 15*, 215-222.

Brown, S., Child, R., Donnelly, A., Saxton, J., & Day, S. (1996). Changes in human skeletal muscle contractile function following stimulated eccentric exercise. *European Journal of Applied Physiology, 72*, 515-521.

Burke, D., Culligan, C., & Holt, L. (2000). The theoretical basis of proprioceptive neuromuscular facilitation. *Journal of Strength and Conditioning Research, 14,* 496-500.

Burke, D., Holt, L., Rasmussen, R., MacKinnon, N., Vossen, J., & Pelham, T. (2001). Effects of hot or cold water immersion and modified proprioceptive neuromuscular facilitation flexibility exercise on hamstring length. *Journal of Athletic Training, 36,* 16-19.

Butler, R., Crowell, H., & Davis, I. (2003). Lower extremity stiffness: implications for performance and injury. *Clinical Biomechanics, 18,* 511-517.

Byrne, C., & Eston, R. (2002). The effect of exercise-induced muscle damage on isometric and dynamic knee extensor strength and vertical jump performance. *Journal of Sports Sciences, 20,* 417-425.

Byrnes, W., Clarkson, P., White, J., Hsieh, S., Frykman, P., & Maughan, R. (1985). Delayed onset muscle soreness following repeated bouts of downhill running. *Journal of Applied Physiology, 59,* 710-715.

Cafarelli, E., & Flint, F. (1992). The role of massage in preparation for and recovery from exercise. *Sports Medicine, 14,* 1-9.

Calbet, J., Chavarren, J., & Dorado, C. (2001). Running economy and delayed onset muscle soreness. *Journal of Sports Medicine and Physical Fitness, 41,* 18-26.

Callagan, M. (1993). The role of massage in the management of the athlete: a review. *British Journal of Sports Medicine, 27,* 28-33.

Carter, A., Dobridge, J., & Hackney, A. (2001). Influence of estrogen on markers of muscle tissue damage following eccentric exercise. *Fiziologiia Cheloveka, 27,* 133-137.

Cavagna, G., & Citterio, G. (1974). Effects of stretching on the elastic characteristics and the contractile component of frog striated muscle. *Journal of Physiology, 239,* 1-14.

Chen, T. (2003). Effects of a second bout of maximal eccentric exercise on muscle damage and electromyographic activity. *European Journal of Applied Physiology, 89,* 115-121.

Chen, T., & Hsieh, S. (2000). The effects of repeated maximal voluntary isokinetic eccentric exercise on recovery from muscle damage. *Research Quarterly for Exercise and Sport, 71,* 260-266.

Chen, T., & Hsieh, S. (2001). Effects of a 7-day eccentric training period on muscle damage and inflammation. *Medicine & Science in Sports & Exercise, 33,* 1732-1738.

Cheung, K., Hume, P., & Maxwell, L. (2003). Delayed onset muscle soreness: treatment strategies and performance factors. *Sports Medicine, 33,* 145-164.

Chleboun, G., Howell, J., Baker, H., Ballard, T., Graham, J., Hallman, H., et al. (1995). Intermittent pneumatic compression effect on eccentric exercise-induced swelling, stiffness, and strength loss. *Archives of Physical Medicine and Rehabilitation, 76*, 744-749.

Church, J., Wiggins, M., Moode, E., & Crist, R. (2001). Effects of warm-up and flexibility treatments on vertical jump performance. *Journal of Strength and Conditioning Research, 15*, 332-336.

Ciccone, C., Leggin, B., & Callamaro, J. (1991). Effects of ultrasound and trolamine salicylate phonophoresis on delayed-onset muscle soreness. *Physical Therapy, 71*, 675-678.

Cinque, C. (1989). Massage for cyclists: the winning touch. *Physician and Sportsmedicine, 17*, 167-170.

Clarkson, H. (2000). *Musculoskeletal assessment: Joint range of motion and manual muscle strength* (2nd ed.). Philadelphia: Lippincott Williams & Wilkins.

Clarkson, P., & Hubal, M. (2002). Exercise-induced muscle damage in humans. *American Journal of Physical Medicine and Rehabilitation, 81(Suppl.),* 52-69.

Clarkson, P., Nosaka, K., & Braun, B. (1992). Muscle function after exercise-induced muscle damage and rapid adaptation. *Medicine & Science in Sports & Exercise, 24*, 512-520.

Clarkson, P., & Reichsman, F. (1990). The effect of ethanol on exercise-induced muscle damage. *Journal of Studies on Alcohol, 51*, 19-23.

Clarkson, P., & Sayers, S. (1999). Etiology of exercise-induced muscle damage. *Canadian Journal of Applied Physiology, 24*, 234-248.

Cleak, M., & Eston, R. (1992). Muscle soreness, swelling, stiffness and strength loss after intense eccentric exercise. *British Journal of Sports Medicine, 26*, 267-272.

Cleary, M., Kimura, I., Sitler, M., & Kendrick, Z. (2002). Temporal pattern of the repeated bout effect of eccentric exercise on delayed-onset muscle soreness. *Journal of Athletic Training, 37*, 32-36.

Connolly, D., Reed, B., & McHugh, M. (2002). The repeated bout effect: does evidence for a crossover effect exist? *Journal of Sports Sciences and Medicine, 1,* 80-86.

Connolly, D., Sayers, S., & McHugh, M. (2003). Treatment and prevention of delayed onset muscle soreness. *Journal of Strength and Conditioning Research, 17,* 197-208.

Corley, M., Ferriter, J., Zeh, J., & Gifford, C. (1995). Physiological and psychological effects of back rubs. *Applied Nursing Research, 8*, 39-43.

Cornelius, W., Jensen, R., & Odell, M. (1995). Effects of PNF stretching phases on acute arterial blood pressure. *Canadian Journal of Applied Physiology, 20*, 222-229.

Cornwell, A., & Nelson, A. (1997). The acute effects of passive stretching on active musculotendinous stiffness. *Medicine & Science in Sports & Exercise, 29(Suppl.),* S281.

Cornwell, A., Nelson, A., & Sidaway, B. (2002). Acute effects of stretching on the neuromechanical properties of the triceps surae muscle complex. *European Journal of Applied Physiology, 86*, 428-434.

Craib, M., Mitchell, V., Fields, K., Cooper, T., Hopewell, R., & Morgan, D. (1996). The association between flexibility and running economy in sub-elite male distance runners. *Medicine & Science in Sports & Exercise, 28,* 737-743.

Craig, J., Bradley, J., Walsh, D., Baxter, G., & Allen, J. (1999). Delayed onset muscle soreness: Lack of effect of therapeutic ultrasound in humans. *Archives of Physical Medicine and Rehabilitation, 80*, 318-323.

Crenshaw, A., Karlsson, S., Styf, J., Backlund, T., & Friden, J. (1995). Knee extension torque and intramuscular pressure of the vastus lateralis muscle during eccentric and concentric activities. *European Journal of Applied Physiology and Occupational Physiology, 70*, 13-19.

Dannecker, E., Koltyn, K., Riley, J., & Robinson, M. (2003). Sex differences in delayed onset muscle soreness. *Journal of Sports Medicine and Physical Fitness, 43*, 78-84.

Danneskiold-Samsoe, B., Christiansen, E., Lund, B., & Anderson, R. (1982). Regional muscle tension and pain (fibrositis): effects of massage and myoglobin in plasma. *Scandinavian Journal of Medicine & Science in Sports, 15*, 17-20.

De Domenico, G., & Wood, E. (1997). *Beard's massage* (4th ed.). Philadelphia: W.B. Saunders.

Delaney, J., Leong, K., Watkins, A., & Brodie, D. (2002). The short-term effects of myofascial trigger point massage therapy on cardiac autonomic tone in healthy subjects. *Journal of Advanced Nursing, 37*, 364-371.

deVries, H., & Housh, T. (1996). *Physiology of exercise: for physical education, athletics and exercise science*. Madison, WI: Brown & Benchmark.

Dolezal, B., Potteiger, J., Jacobsen, D., & Benedict, S. (2000). Muscle damage and resting metabolic rate after acute resistance exercise with an eccentric overload. *Medicine & Science in Sports & Exercise, 32,* 1202-1207.

Dolgener, F., & Morien, A. (1993). The effect of massage on lactate disappearance. *Journal of Strength and Conditioning Research, 7*, 159-162.

Draper, D., & Prentice, W. (2002). Therapeutic Ultrasound. In W. Prentice (Ed.), *Therapeutic modalities for physical therapists* (2nd ed., pp. 270-304). New York: McGraw-Hill.

Drust, B., Atkinson, G., Gregson, W., French, D., & Binningsley, D. (2003). The effects of massage on intra muscular temperature in the vastus lateralis in humans. *International Journal of Sports Medicine, 24,* 395-399.

Dubravcic-Simunjak, S., Pecina, M., Kuipers, H., Moran, J., & Haspl, M. (2003). The incidence of injuries in elite junior figure skaters. *American Journal of Sports Medicine, 31,* 511-517.

Dubrosky, V. (1982). Changes in muscle and venous blood flow after massage. *Soviet Sports Reviews, 4,* 56-57.

Dubrosky, V. (1983). The effect of massage on athletes' cardiorespiratory systems. *Soviet Sports Reviews, 5,* 48-49.

Ebbeling, C., & Clarkson, P. (1989). Exercise-induced muscle damage and adaptation. *Sports Medicine, 7,* 207-234.

Enoka, R., Hutton, R., & Eldred, E. (1980). Changes in excitability of tendon tap and Hoffmann reflexes following voluntary contractions. *Electroencephalogram and Clinical Neurophysiology, 48,* 664-672.

Ernst, E. (1998). Does post-exercise massage treatment reduce delayed onset muscle soreness? A systematic review. *British Journal of Sports Medicine, 32,* 212-214.

Eston, R., Finney, S., Baker, S., & Baltzopoulos, V. (1996). Muscle tenderness and peak torque changes after downhill running following a prior bout of isokinetic eccentric exercise. *Journal of Sports Sciences, 14,* 291-299.

Eston, R., Lemmey, A., McHugh, P., Byrne, C., & Walsh, S. (2000). Effect of stride length on symptoms of exercise-induced muscle damage during a repeated bout of downhill running. *Scandinavian Journal of Medicine & Science in Sports, 10,* 199-204.

Eston, R. G., & Peters, D. (1999). Effects of cold water immersion on the symptoms of exercise induced muscle damage. *Journal of Sports Sciences, 17,* 231-238.

Ettema, G. (1996). Mechanical efficiency and efficiency of storage and release of series elastic energy in skeletal muscle during stretch-shorten cycles. *Journal of Experimental Biology, 199,* 1983-1997.

Evans, W., & Cannon, J. (1991). The metabolic effects of exercise-induced muscle damage. *Exercise and Sport Sciences Reviews, 19,* 99-125.

Evans, W. J., & Cannon, J. G. (1991). The metabolic effects of exercise-induced muscle damage. *Exercise and Sport Sciences Reviews, 18,* 99-125.

Farr, T., Nottle, C., Nosaka, K., & Sacco, P. (2002). The effects of therapeutic massage on delayed onset muscle soreness and muscle function following downhill walking. *Journal of Science and Medicine in Sport, 5*, 297-306.

Faulkner, J., Brooks, S., & Opiteck, J. (1993). Injury to skeletal muscle fiber during contractions: conditions of occurrence and prevention. *Physical Therapy, 73,* 911-921.

Field, T., Grizzle, N., Scafidi, F., & Schanberg, S. (1997). Massage and relaxation therapies' effects on depressed adolescent mothers. *Adolescence, 31*, 903-1003.

Fletcher, I., & Jones, B. (2004). The effects of different warm-up stretch protocols on 20 meter sprint performance in trained rugby union players. *Journal of Strength and Conditioning Research, 2004,* 885-888.

Foley, J., Jayaraman, R., Prior, B., Pivarnik, J., & Meyer, R. (1999). MR measurements of muscle damage and adaptation after eccentric exercise. *Journal of Applied Physiology, 87*, 2311-2318.

Fowles, J., Sale, D., & MacDougall, J. (2000). Reduced strength after passive stretch of the human plantarflexors. *Journal of Applied Physiology, 89*, 1179-1188.

Fraser, J., & Kerr, J. (1993). Psychological effects of back massage on elderly institutionalized patients. *Journal of Advanced Nursing, 18*, 238-245.

Friden, J., & Lieber, R. (1992). Structural and mechanical basis of exercise-induced muscle injury. *Medicine & Science in Sports & Exercise, 24*, 521-530.

Gajdosik, R. (2001). Passive extensibility of skeletal muscle: review of the literature with clinical implications. *Clinical Biomechanics, 16*, 87-101.

Gajdosik, R. (2002). Relationship between passive properties of the calf muscles and plantarflexion concentric isokinetic torque characteristics. *European Journal of Applied Physiology, 87*, 220-227.

Gajdosik, R., Giuliani, C., & Bohannon, R. (1990). Passive compliance and length of the hamstring muscles of the healthy men and women. *Clinical Biomechanics, 5,* 23-29.

Galloway, S., Watt, J., & Sharp, C. (2004). Massage provision by physiotherapists at major athletics events between 1987 and 1998. *British Journal of Sport Medicine, 38*, 235 - 237.

Gam, A., Warming, S., Larsen, L., Jensen, B., Hoydalsmo, O., Allon, I., et al. (1998). Treatment of myofascial trigger-points with ultrasound combined with massage and exercise-a randomized controlled trial. *Pain, 77,* 73-79.

Giulbin, J., & Gaffney, P. (2002). Identical twins are discordant for markers of eccentric exercise-induced muscle damage. *International Journal of Sports Medicine, 23,* 471-476.

Gleeson, M., Blannin, A., Walsh, N., Field, C., & Pritchard, J. (1998). Effect of exercise-induced muscle damage on the blood lactate response to incremental exercise in humans. *European Journal of Applied Physiology and Occupational Physiology, 77,* 292-295.

Gleeson, M., Blannin, A., Zhu, B., Brooks, S., & Cave, R. (1995). Cardiorespiratory, hormonal and haematological responses to submaximal cycling performed 2 days after eccentric or concentric exercise bouts. *Journal of Sports Sciences, 13,* 471-479.

Gleeson, N., Eston, R., Marginson, V., & McHugh, M. (2003). Effects of prior concentric training on eccentric exercise induced muscle damage. *British Journal of Sports Medicine, 37,* 119-125.

Gleim, G., W, & McHugh, M. P. (1997). Flexibility and its effects on sports injury and performance. *Sports Medicine, 24,* 289-299.

Goats, G. C. (1994). Massage-the scientific basis of an ancient art: Part 1. The techniques. *British Journal of Sports Medicine, 28,* 149-152.

Godges, J., MacRae, P., & Engelke, K. (1993). Effects of exercise on hip range of motion, trunk muscle performance, and gait economy. *Physical Therapy, 73,* 468-477.

Gollhofer, A., Schopp, A., Rapp, W., & Stroinik, V. (1998). Changes in reflex excitability following isometric contraction in humans. *European Journal of Applied Physiology, 77,* 89-97.

Gray, S., & Nimmo, M. (2001). Effects of active, passive or no warm-up on metabolism and performance during high-intensity exercise. *Journal of Sports Sciences, 19,* 693-700.

Groer, M., Mozingo, J., Droppleman, P., Davis, M., Jolly, M., Boynton, M., et al. (1994). Measures of salivary secretary immunoglobulin A and state anxiety after a nursing back rub. *Applied Nursing Research, 7,* 2-6.

Guissard, N., Duchateau, J., & Hainaut, K. (2001). Mechanisms of decreased motoneuron excitation during passive muscle stretching. *Experimental Brain Research, 137,* 163-169.

Gupta, S., Goswami, A., Sadhukhan, A., & Mathur, D. (1996). Comparative study of lactate removal in short term massage of extremities, active recovery and a passive recovery period after supramaximal exercise sessions. *International Journal of Sports Medicine, 217,* 106-110.

Guyton, A., & Hall, J. (2000). *Textbook of medical physiology* (10th ed.). Philadelphia, PA: W.B. Saunders.

Halbertsma, J., van Bolhuis, A., & Goeken, L. (1996). Sport stretching: effects on passive muscle stiffness of short hamstrings. *Archives of Physical Medicine and Rehabilitation, 77,* 688-692.

Hamill, J., & Knutzen, K. (1995). *Biomechanical basis of human movement.* Baltimore, MD: Williams & Wilkins.

Handel, M., Horstmann, T., Dickhuth, H., & Gulch, R. (1997). Effects of contract-relax stretching training on muscle performance in athletes. *European Journal of Applied Physiology, 76*, 400-408.

Hansen, T., & Kristensen, J. (1973). Effect of massage, shortwave diathermy and ultrasound upon 133Xe disappearance rate from muscle and subcutaneous tissue in the human calf. *Scandinavian Journal of Medicine & Science in Sports, 5,* 179-182.

Harman, E., Rosenstein, M., Frykman, P., & Rosenstein, R. (1990). The effects of arms and countermovement on vertical jumping. *Medicine & Science in Sports & Exercise, 22,* 825-833.

Harmer, P. (1991). The effect of pre-performance massage on stride frequency in sprinters. *Journal of Athletic Training, 26*, 55-58.

Harris, C., Wilcox, A., Smith, G., Quinn, C., & Lawson, L. (1990). The effect of delayed onset muscular soreness (DOMS) on running kinematics. *Medicine & Science in Sports & Exercise, 22(Suppl.),* 34.

Harrison, A., & Gaffney, S. (2004). Effects of muscle damage on stretch-shortening cycle function and muscle stiffness control. *Journal of Strength and Conditioning Research, 18,* 771-776.

Harrison, A., Keane, S., & Coglan, J. (2004). Force-velocity relationship and stretch-shortening cycle function in sprint and endurance athletes. *Journal of Strength and Conditioning Research, 18*, 473-479.

Hasselman, C., Best , T., Seaber, A., & Garrett, W. J. (1995). A threshold and continuum of injury during active stretch of rabbit skeletal muscle. *American Journal of Sports Medicine, 23*, 65-73.

Hasson, S., Cone, M., Ellison, C., Goehrs, L., Hall, L., Van Vyven, E., et al. (1992). Effect of retrograde massage on muscle soreness and performance. *Physical Therapy, 72(Suppl.),* 100.

Hasson, S., Daniels, J., Divine, J., Niebuhr, B., Richmond, S., Stein, P., et al. (1993). Effects of ibuprofen use on muscle soreness, damage, and performance: a preliminary investigation. *Medicine & Science in Sports & Exercise, 25,* 9-17.

Hawley, J., Williams, M., Hamling, G., & Walsh, R. (1989). Effects of a task-specific warm-up on anaerobic power. *British Journal of Sports Medicine, 23,* 233-236.

Hemmings, B. (2000a). Psychological and immunological effects of massage after sport. *British Journal of Therapy and Rehabilitation, 7,* 516-519.

Hemmings, B. (2000b). Sports massage and psychological regeneration. *British Journal of Therapy and Rehabilitation, 7*, 184-188.

Hemmings, B., Smith, M., Gradon, J., & Dyson, R. (2000). Effects of massage on physiological restoration, perceived recovery, and repeated sports performance. *British Journal of Sports Medicine, 34*, 109-115.

Henricson, A., Fredriksson, K., Persson, I., Pereira, R., & Westlin, N. (1984). The effects of heat and stretching on the range of hip motion. *Journal of Orthopaedic & Sports Physical Therapy, 6*, 110-115.

Herbert, R., & Gabriel, M. (2002). Effects of stretching before and after exercising on muscle soreness and risk of injury: systemic review. *British Journal of Sports Medicine, 325*, 1-5.

Hernandez-Reif, M., Field, T., Krasnegor, J., & Theakston, H. (2001). Lower back pain is reduced and range of motion increased after massage therapy. *International Journal of Neuroscience, 106*, 131-145.

High, D., Howley, E., & Franks, B. (1989). The effects of static stretching and warm-up on prevention of delayed-onset muscle soreness. *Research Quarterly for Exercise and Sport, 60*, 357-361.

Hilbert, J., Sforzo, G., & Swensen, T. (2003). The effects of massage on delayed onset muscle soreness. *British Journal of Sports Medicine, 37*, 72-75.

Hill, A. (1951). The mechanics of voluntary muscle. *Lancet, 261*, 947-954.

Hilliard-Robertson, P., Schneider, S., Bishop, S., & Guilliams, M. (2003). Strength gains following different combined concentric and eccentric exercise regimens. *Aviation, Space, and Environmental Medicine, 74*, 342-347.

Hollander, D., Durand, R., Trynicki, J., Larock, D., Castracane, V., Hebert, E., et al. (2003). RPE, pain, and physiological adjustment to concentric and eccentric contractions. *Medicine & Science in Sports & Exercise, 35*, 1017-1025.

Hone, L., Siler, W., & Schwane, J. (1990). Does delayed muscle soreness affect oxygen consumption and selected gait parameters during running? *Medicine & Science in Sports & Exercise, 22(Suppl.),* 34.

Hopkins, W. (2002a). *Calculating likely (confidence) limits and likelihoods for true values (Excel spreadsheet)*. Retrieved 5 June 2003 from sportsci.org/resource/stats/xcl.xls

Hopkins, W. (2002b, 6/06/02). *A scale of magnitudes for effects statistics*. Retrieved 5 June 2003, from sportsci.org/resource/stats/effectmag.html

Horstmann, T., Mayer, F., Maschmann, J., Niess, A., Roecker, K., & Dickhuth, H. (2001). Metabolic reaction after concentric and eccentric endurance-exercise of the knee and ankle. *Medicine & Science in Sports & Exercise, 33*, 791-795.

Hovind, H., & Nielsen, S. (1974). Effect of massage on blood flow in skeletal muscle. *Scandinavian Journal of Medicine & Science in Sports, 6*, 74-77.

Howell, J., Chleboun, G., & Conaaster, R. (1993). Muscle stiffness, strength loss, swelling and soreness following exercise-induced injury in humans. *Journal of Physiology, 464*, 183-196.

Hunter, D., Conveney, V., & Spriggs, J. (2001). Investigation into the effect of static stretching on the active stiffness and damping characteristics of the ankle joint plantar flexors. *Physical Therapy in Sport, 2*, 15-22.

Hunter, J., & Marshall, R. (2002). Effects of power and flexibility training on vertical jump technique. *Medicine & Science in Sports & Exercise, 34*, 478-486.

International Physical Activity Questionnaire. (2001, May, 2002). Retrieved 11 July 2002, from http://www.ipaq.ki.se/dloads/IPAQ_S7S_FINAL_MAY_01.PDF

Ironson, G., Field, T., Scafidi, F., Hashimoto, M., Kumar, M., Kumar, A., et al. (1996). Massage therapy is associated with enhancement of the immune system's cytotoxic capacity. *International Journal of Neuroscience, 84*, 205-217.

Johansson, P., Lindstrom, L., Sundelin, G., & Lindstrom, B. (1999). The effects of preexercise stretching on muscular soreness, tenderness and force loss following heavy eccentric exercise. *Scandinavian Journal of Medicine & Science in Sports, 9*, 219-225.

Kaada, B., & Torsteinbo, O. (1989). Increase of plasma b-endorphin in connective tissue massage. *General Pharmacy, 20*, 487-489.

Kawakami, Y., Nakazawa, K., Fujimoto, T., Nozaki, D., Miyashita, M., & Fukunaga, T. (1994). Specific tension of elbow flexor and extensor muscles based on magnetic resonance imaging. *European Journal of Applied Physiology and Occupational Physiology, 68*, 139-147.

Kendall, B., & Eston, R. (2002). Exercise-induced muscle damage and the potential protective role of estrogen. *Sports Medicine, 32*, 103-123.

Kjaer, M. (2004). Role of extracellular matrix in adaptation of tendon and skeletal muscle to mechanical loading. *Physiological Reviews, 84*, 649-698.

Klinge, K., Magnusson, S., Simonsen, E., Aagaard, P., Klausen, K., & Kjaer, M. (1997). The effects of strength and flexibility training on skeletal muscle electromyographic activity, stiffness, and viscoelastic stress relaxation response. *American Journal of Sports Medicine, 25*, 710-716.

Knight, C., Rutledge, C., Cox, M., Acosta, M., & Hall, S. (2001). Effect of superficial heat, deep heat, and active exercise warm-up on the extensibility of the plantar flexors. *Physical Therapy, 81*, 1206-1214.

Knudson, D., Bennett, K., Corn, R., Leick, D., & Smith, C. (2001). Acute effects of stretching are not evident in the kinematics of the vertical jump. *Journal of Strength and Conditioning Research, 15*, 98-101.

Kokkonen, J., Nelson, A., & Cornwell, A. (1998). Acute muscle stretching inhibits maximal strength performance. *Research Quarterly for Exercise and Sport, 69,* 411-415.

Kraemer, W., Bush, J., Wickham, R., Denegar, C., Gomez, A., Gotshalk, L., et al. (2001). Influence of compression therapy on symptoms following soft tissue injury from maximal eccentric exercise. *Journal of Orthopaedic & Sports Physical Therapy, 31*, 282-290.

Krotkiewski, M., Brzezinska, Z., Liu, B., Grimby, G., & Palm, S. (1994). Prevention of muscle soreness by pretreatment with antioxidants. *Scandinavian Journal of Medicine & Science in Sports, 4*, 191-199.

Krustrup, P., Mohr, M., & Bangsbo, J. (2002). Activity profile and physiological demands of top-class soccer assistant refereeing in relation to training status. *Journal of Sports Sciences, 20,* 861-867.

Kubo, K., Kanehisa, H., & Fukunaga, T. (2001). Is passive stiffness in human muscles related to the elasticity of tendon structures? *European Journal of Applied Physiology, 85*, 226-232.

Kubo, K., Kanehisa, H., & Fukunaga, T. (2002). Effects of resistance and stretching training programmes on the viscoelastic properties of human tendon structures in vivo. *Journal of Physiology, 538*, 219-226.

Kulig, K., Powers, C., Shellock, F., & Terk, M. (2001). The effects of eccentric velocity on activation of elbow flexors: evaluation by magnetic resonance imaging. *Medicine & Science in Sports & Exercise, 33*, 196-200.

Labyak, S., & Metzger, B. (1997). The effects of effleurage backrub on the physiological components of relaxation: a meta-analysis. *Nursing Research, 46*, 59-62.

Laur, D., Anderson, T., Geddes, G., Crandall, A., & Pincivero, D. (2003). The effects of acute stretching on hamstring muscle fatigue and perceived exertion. *Journal of Sports Sciences, 21*, 163-170.

Leivadi, S., Hernandez-Reif, M., Field, T., O'Rourke, M., D'Arienzo, S., Lewis, D., et al. (1999). Massage therapy and relaxation effects on university dance students. *Journal of Dance Medicine and Science, 3,* 108-112.

Leivseth, G., Tindall, A., Lovaas, E., & Reikeraas, O. (1993). Change in muscle stiffness caused by reduced joint mobility. *Scandinavian Journal of Medicine & Science in Sports, 3*, 28-36.

Lenn, J., Uhl, T., Mattacola, C., Boissonneault, G., Yates, J., Ibrahim, W., et al. (2002). The effects of fish oil and isoflavones on delayed onset muscle soreness. *Medicine & Science in Sports & Exercise, 34,* 1605-1613.

Lieber, R., & Friden, J. (1999). Mechanisms of muscle injury after eccentric contraction. *Journal of Science and Medicine in Sport, 2*, 253-265.

Lightfoot, J., Char, D., McDermont, J., & Goya, C. (1997). Immediate post-exercise massage does not attenuate delayed onset of muscle soreness. *Journal of Strength and Conditioning Research, 11*, 119-124.

Longworth, J. (1982). Psychophysiological effects of slow stroke back massage in normotensive females. *Advances in Nursing Science, 4*, 44-61.

Lund, H., Vestergaard-Poulsen, P., Kanstrup, I., & Sejrsen, P. (1998a). The effects of passive stretching on delayed onset muscle soreness, and other detrimental effects following eccentric exercise. *Scandinavian Journal of Medicine & Science in Sports, 8*, 216-221.

Lund, H., Vestergaard-Poulsen, P., Kanstrup, I., & Sejrsen, P. (1998b). Isokinetic eccentric exercise as a model to induce and reproduce pathophysiological alterations related to delayed onset muscle soreness. *Scandinavian Journal of Medicine & Science in Sports, 8*, 208-215.

Lund, I., Lundberg, T., Kurosawa, M., & Uvnas-Moberg, K. (1999). Sensory stimulation (massage) reduces blood pressure in unanaesthesized rats. *Journal of Autonomic Nervous System, 78*, 30-37.

MacIntyre, D., Reid, W., Lyster, D., & McKenzie, D. (2000). Different effects of strenuous eccentric exercise on the accumulation of neutrophils in muscle in women and men. *European Journal of Applied Physiology, 81 (1-2)*, 47-53.

Magnusson, S. (1998). Passive properties of human skeletal muscle during stretch manoeuvres. *Scandinavian Journal of Medicine & Science in Sports, 8*, 65-77.

Magnusson, S., Aagaard, P., Larsson, B., & Kjaer, M. (2000). Passive energy absorption by human muscle-tendon unit is unaffected by increase in intramuscular temperature. *Journal of Applied Physiology, 88*, 1215-1220.

Magnusson, S., Simonsen, E., Aagaard, P., Dyhre-Poulsen, P., McHugh, M., & Kjaer, M. (1996). Mechanical and physical responses to stretching with and without preisometric contraction in human skeletal muscle. *Archives of Physical Medicine and Rehabilitation, 77*, 373-378.

Magnusson, S., Simonsen, E., Aagaard, P., Gleim, G., & McHugh, M. (1995). Viscoelastic response to repeated static stretching in the human hamstring muscle. *Medicine & Science in Sports & Exercise, 5*, 342-347.

Magnusson, S., Simonsen, E., Aagaard, P., & Kjaer, M. (1996). Biomechanical responses to repeated stretches in human hamstring muscle in vivo. *American Journal of Sports Medicine, 24*, 622-628.

Magnusson, S., Simonsen, E., Aagaard, P., Sorensen, H., & Kjaer, M. (1996). A mechanism for altered flexibility in human skeletal muscle. *Journal of Physiology, 497*, 291-298.

Magnusson, S., Simonson, E., Dyhre-Poulsen, P., Aagaard, P., Mohr, T., & Kjaer, M. (1996). Viscoelastic stress relaxation during static stretch in human skeletal muscle in the absence of EMG activity. *Scandinavian Journal of Medicine & Science in Sports, 6,* 323-328.

Mair, J., Mayr, M., Muller, E., Koller, A., Haid, C., Artner-Dworzak, E., et al. (1995). Rapid adaptation to eccentric exercise-induced muscle damage. *International Journal of Sports Medicine, 16,* 352-356.

McHugh, M. (2003). Recent advances in the understanding of the repeated bout effect: the protective effect against muscle damage from a single bout of eccentric exercise. *Scandinavian Journal of Medicine & Science in Sports, 13*, 88-97.

McHugh, M., Connolly, D., Eston, R., & Gleim, G. (2000). Electromyographic analysis of exercise resulting in symptoms of muscle damage. *Journal of Sports Sciences, 18,* 163-172.

McHugh, M., Kremenic, I., Fox, M., & Gleim, G. (1998). The role of mechanical and neural restraints to joint range of motion during passive stretch. *Medicine & Science in Sports & Exercise, 30*, 928-932.

McHugh, M. P., Connolly, D., Eston, R., Gartman, E., & Gleim, G. (2001). Electromyographic analysis of repeated bouts of eccentric exercise. *Journal of Sports Sciences, 19,* 163-170.

McHugh, M. P., Connolly, D. A., Eston, R. G., Kremenic, I., Nicholas, S. J., & Gleim, G. W. (1999). The role of passive muscle stiffness in symptoms of exercise-induced muscle damage. *American Journal of Sports Medicine, 27,* 594-599.

McHugh, M. P., Connolly, D. A. J., Eston, R. G., & Gleim, G. W. (1999). Exercise-induced muscle damage and potentials mechanisms for the repeated bout effect. *Sports Medicine, 27,* 157-170.

McHugh, M. P., Magnusson, S., Gliem, G., & Nicholas, J. (1992). Viscoelastic stress relaxation in human skeletal muscle. *Medicine & Science in Sports & Exercise, 24,* 1375-1382.

McMahon, T., & Cheng, G. (1990). The mechanics of running: how does stiffness couple with speed? *Journal of Biomechanics, 23(Suppl 1.),* 65-78.

McNair, D. M., Lorr, M., & Droppleman, L. F. (1971). *Profile of Mood State manual*. San Diego: Educational and Industrial Testing Service.

McNair, P., Dombroski, E., Hewson, D., & Stanley, S. (2000). Stretching at the ankle joint: Viscoelastic responses to holds and continuous passive motion. *Medicine & Science in Sports & Exercise, 33,* 354-358.

McNair, P., & Stanley, S. (1996). Effect of passive stretching and jogging on the series muscle stiffness and range of motion of the ankle joint. *British Journal of Sports Medicine, 30,* 313-318.

Mekjavic, I., Exner, J., Tesch, P., & Eiken, O. (2000). Hyperbaric oxygen therapy does not affect recovery from delayed onset muscle soreness. *Medicine & Science in Sports & Exercise, 32*, 558-563.

Mohr, K., Pink, M., Elsner, C., & Kvitne, R. (1998). Electromyographic investigation of stretching: the effect of warm-up. *Clinical Journal of Sport Medicine, 8,* 215-220.

Monedero, J., & Donne, B. (2000). Effect of recovery interventions on lactate removal and subsequent performance. *International Journal of Sports Medicine, 21*, 593-597.

Moore, M., & Kukulka, C. (1991). Depression of Hoffmann reflexes following voluntary contraction and implications for proprioceptive neuromuscular facilitation therapy. *Physical Therapy, 71*, 321-333.

Morelli, M., Chapman, C., & Sullivan, S. (1999). Do cutaneous receptors contribute to the changes in the amplitude of the H-reflex during massage? *Electromyography and Clinical Neurophysiology, 39,* 441-447.

Morelli, M., Seaborne, D., & Sullivan, S. (1990). Changes in H-reflex amplitude during massage of triceps surae in healthy subjects. *Journal of Orthopaedic & Sports Physical Therapy, 12*, 55-59.

Morelli, M., Seaborne, D., & Sullivan, S. (1991). H-reflex modulation during manual muscle massage of human triceps surae. *Archives of Physical Medicine and Rehabilitation, 72*, 915-919.

Morgan, D., & Allen, D. (1999). Early events in stretch-induced muscle damage. *Journal of Applied Physiology, 87*, 2007-2015.

Morgan, D., Brockett, C., Gregory, J., & Proske, U. (2002). The role of the length-tension curve in the control of movement. *Advances in Experimental Medicine and Biology, 508*, 489-494.

Muir, I., Chesworth, B., & Vandervoort, A. (1999). Effect of a static calf-stretching exercise on the resistive torque during passive ankle dorsiflexion in healthy subjects. *Journal of Orthopaedic & Sports Physical Therapy, 29,* 421-424.

Murphy, A., & Wilson, G. (1997). The ability of tests of muscular function to reflect training-induced changes in performance. *Journal of Sports Sciences, 15,* 191-200.

Nelson, A., Guillory, I., Cornwell, A., & Kokkonen, J. (2001). Inhibition of maximal voluntary isokinetic torque production following stretching is velocity-specific. *Journal of Strength and Conditioning Research, 15,* 241-246.

Nelson, A., & Kokkonen, J. (2001). Acute ballistic muscle stretching inhibits maximal strength performance. *Research Quarterly for Exercise and Sport, 72*, 415-419.

Nelson, A., Kokkonen, J., Eldredge, C., Cornwell, A., & Glickman-Weiss, E. (2001). Chronic stretching and running economy. *Scandinavian Journal of Medicine & Science in Sports, 11,* 260-265.

Newham, D., Jones, D., Ghosh, G., & Aurora, P. (1988). Muscle fatigue and pain after eccentric contractions at long and short length. *Clinical Science, 74,* 553-557.

Newham, D., Mills, K., Quigley, B., & Edwards, R. (1983). Pain and fatigue after concentric and eccentric exercise contractions. *Clinical Science, 64,* 55-62.

Noonan, T., & Best, T. (1993). Thermal effects on skeletal muscle tensile behaviour. *American Journal of Sports Medicine, 21*, 517-525.

Nordschow, M., & Bierman, W. (1962). The influence of manual massage on muscle relaxation: effect on trunk flexion. *Journal of American Physical Therapy, 42,* 653-657.

Nosaka, K., & Clarkson, P. (1997). Influence of previous concentric exercise on eccentric exercise-induced muscle damage. *Journal of Sports Sciences, 15,* 477-483.

Nosaka, K., Clarkson, P., McGuiggin, M., & Byrne, J. (1991). Time course of muscle adaptation after high force eccentric exercise. *European Journal of Applied Physiology and Occupational Physiology, 63*, 70-76.

Nosaka, K., & Newton, M. (2002a). Concentric or eccentric training effect on eccentric exercise-induced muscle damage. *Medicine & Science in Sports & Exercise, 34,* 63-69.

Nosaka, K., & Newton, M. (2002b). Difference in the magnitude of muscle damage between maximal and submaximal eccentric loading. *Journal of Strength and Conditioning Research, 16*, 202-208.

Nosaka, K., & Newton, M. (2002c). Repeated eccentric exercise bouts do not exacerbate muscle damage and repair. *Journal of Strength and Conditioning Research, 16,* 117-122.

Nosaka, K., Newton, M., & Sacco, P. (2002a). Delayed-onset muscle soreness does not reflect the magnitude of eccentric exercise-induced muscle damage. *Scandinavian Journal of Medicine & Science in Sports, 12*, 337-346.

Nosaka, K., Newton, M., & Sacco, P. (2002b). Responses of human elbow flexor muscles to electrically stimulated forced lengthening exercise. *Acta Physiologica Scandinavica, 174,* 137-145.

Nosaka, K., & Sakamoto, K. (2001). Effect of elbow joint angle on the magnitude of muscle damage to the elbow flexors. *Medicine & Science in Sports & Exercise, 33*, 22-29.

Nosaka, K., Sakamoto, K., Newton, M., & Sacco, P. (2001a). How long does the protective effect on eccentric exercise-induced muscle damage last? *Medicine & Science in Sports & Exercise, 33*, 1490-1495.

Nosaka, K., Sakamoto, K., Newton, M., & Sacco, P. (2001b). The repeated bout effect of reduced-load eccentric exercise on elbow flexor muscle damage. *European Journal of Applied Physiology, 85,* 34-40.

Osternig, L., Robertson, R., Troxel, R., & Hansen, P. (1987). Muscle activation during proprioceptive neuromuscular facilitation (PNF) stretching techniques. *American Journal of Physical Medicine and Rehabilitation, 66*, 298-307.

Paddon-Jones, D., Muthalib, M., & Jenkins, D. (2000). The effects of a repeated bout of eccentric exercise on indices of muscle damage and delayed onset muscle soreness. *Journal of Science and Medicine in Sport, 3,* 35-43.

Paddon-Jones, D., & Quigley, B. (1997). Effect of cryotherapy on muscle soreness and strength following eccentric exercise. *International Journal of Sports Medicine, 18*, 588-593.

Peterson, C., Wilson, B., & Hopkins, W. (2004). Effects of modified-implement training on fast bowling in cricket. *Journal of Sports Sciences, 22,* 1035-1039.

Ploutz-Snyder, L., Tesch, P., & Dudley, G. (1998). Increased vulnerability to eccentric exercise-induced dysfunction and muscle injury after concentric training. *Archives of Physical Medicine and Rehabilitation, 79,* 58-61.

Pope, M., Phillips, R., Haugh, L., Hsieh, C., MacDonald, L., & Haldeman, S. (1994). A prospective randomized three-week trial of spinal manipulation, transcutaneous muscle stimulation, massage and corset in the treatment of subacute low back pain. *Spine, 19*, 2571-2577.

Pope, R., Herbert, R., & Kirwan, J. (1998). Effects of ankle dorsiflexion range and pre-exercise calf muscle stretching on injury risk in army recruits. *Australian Journal of Physiotherapy, 44*, 165-177.

Pope, R., Herbert, R., Kirwan, J., & Graham, B. (2000). A randomised trial of pre-exercise stretching for prevention of lower-limb injury. *Medicine & Science in Sports & Exercise, 32*, 271-277.

Pousson, M., van Hoecke, J., & Boubel, F. (1990). Changes in elastic characteristics of human muscle induced by eccentric exercise. *Journal of Biomechanics, 23*, 343-348.

Prior, B., Jayaraman, R., Reid, R., Cooper, T., Foley, J., Dudley, G., et al. (2001). Biarticular and monoarticular activation and injury in human quadriceps muscle. *European Journal of Applied Physiology, 85*, 185-190.

Proske, U., & Morgan, D. (2001). Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. *Journal of Physiology, 537,* 333-345.

Purslow, P. (1989). Strain-induced reorientation of an intramuscular connective tissue network: implications for passive muscle elasticity. *Journal of Biomechanics, 21,* 21-31.

Puustjarvi, K., Airaksinen, O., & Pontinen, P. (1990). The effects of massage in the patients with chronic tension headache. *Acupuncture & Electro-Therapeutics Research, 15*, 159-162.

Ribot-Ciscar, E., Tardy-Gervet, M., Vedel, J., & Roll, J. (1991). Post-contraction changes in human muscle spindle resting discharge and stretch sensitivity. *Experimental Brain Research, 86,* 673-678.

Rinder, A., & Sutherland, C. (1995). An investigation of the effects of massage on quadriceps performance after exercise fatigue. *Complementary Therapies in Nursing and Midwifery, 1,* 99-102.

Rodenburg, J., Steenbeek, D., Schiereck, P., & Bar, P. (1994). Warm-up, stretching and massage diminish harmful effects of eccentric exercise. *International Journal of Sports Medicine, 15*, 414-419.

Rosenbaum, D., & Hennig, E. (1995). The influence of stretching and warm-up exercises on Achilles tendon reflex activity. *Journal of Sports Sciences, 13,* 481-490.

Rowlands, A., Eston, R., & Tilzey, C. (2001). Effect of stride length manipulation on symptoms of exercise-induced muscle damage and the repeated bout effect. *Journal of Sports Sciences, 19,* 333-340.

Sady, S., Wortman, M., & Blanke, D. (1982). Flexibility training: ballistic, static or proprioceptive neuromuscular facilitation? *Archives of Physical Medicine and Rehabilitation, 63*, 261-263.

Samples, P. (1987). Does "sport massage" have a role in sport medicine? *Physician and Sportsmedicine, 15,* 177-183.

Sayers, S., Knight, C., & Clarkson, P. (2003). Neuromuscular variables affecting the magnitude of force loss after eccentric exercise. *Journal of Sports Sciences, 21,* 403-410.

Semark, A., Noakes, T., St Clair Gibson, A., & Lambert, M. (1999). The effect of a prophylactic dose of flurbiprofen on muscle soreness and sprinting performance in trained subjects. *Journal of Sports Sciences, 17,* 197-203.

Shellock, F., & Prentice, W. (1985). Warming-up and stretching for improved physical performance and prevention of sports-related injuries. *Sports Medicine, 2,* 267-278.

Shoemaker, J., Tiidus, P., & Mader, R. (1997). Failure of manual massage to alter limb blood flow: measures by Doppler Ultrasound. *Medicine & Science in Sports & Exercise, 29,* 610-614.

Shrier, I. (2004). Does stretching improve performance?: a systematic and critical review of the literature. *Clinical Journal of Sport Medicine, 14*, 267-273.

Silver, F., Freeman, J., & Seehra, G. (2003). Collagen self-assembly and the development of tendon mechanical properties. *Journal of Biomechanics, 36*, 1529-1553.

Smith, C. (1994). The warm-up procedure: to stretch or not to stretch. A brief review. *Journal of Orthopaedic & Sports Physical Therapy, 19*, 12-17.

Smith, L. (1991). Acute inflammation: the underlying mechanism in delayed onset muscle soreness? *Medicine & Science in Sports & Exercise, 23*, 542-551.

Smith, L. (1992). Causes of delayed onset muscle soreness and the impact on athletic performance: a review. *Journal of Applied Sport Science Research, 6*, 135-141.

Smith, L., Brunetz, M., Chenier, T., McCammon, M., Houmard, J., Franklin, M., et al. (1993). The effects of static and ballistic stretching on delayed onset muscle soreness. *Research Quarterly for Exercise and Sport, 64*, 103-107.

Smith, L., Fulmer, M., Holbert, D., McCammon, M., Houmard, J., Frazer, D., et al. (1994). The impact of a repeated bout of eccentric exercise on muscular strength, muscle soreness and creatine kinase. *British Journal of Sports Medicine, 28*, 267-271.

Smith, L., Keating, M., Holbert, D., Sprattt, D., McCammon, M., Smith, S., et al. (1994). The effects of athlete massage on delayed onset muscle soreness, creatine kinase and neutrophil count: a preliminary report. *Journal of Orthopaedic and Sports Physical Therapy, 19*, 93-99.

Smith, M., & Jackson, C. (1990). Delayed onset muscle soreness (DOMS), serum creatine kinase (SCK), and creatine kinase-MB (%CK-MB) related to performance measurements in football. *Medicine & Science in Sports & Exercise, 22(Suppl.),* 34.

Spernoga, S., Uhl, T., Arnold, B., & Gansneder, B. (2001). Duration of maintained hamstring flexibility after one-time, modified hold-relax stretching protocol. *Journal of Athletic Training, 36*, 44-48.

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *The State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.

Stanley, S., Purdam, C., Bond, T., & McNair, P. (2001, July). *Passive tension and stiffness properties of the ankle plantar flexors: the effects of massage.* Paper presented at the XVIIIth Congress of the International Society of Biomechanics, Zurich.

Starkey, J. (1976). Treatment of ankle sprains by simultaneous use of intermittent compression and ice packs. *American Journal of Sports Medicine, 4*, 141-144.

Sterne, J., & Smith, G. (2001). Sifting the evidence--what's wrong with significance tests? *BMJ, 322*, 226-231.

Stewart, I., & Sleivert, G. (1998). The effects of warm-up intensity on range of motion and anaerobic performance. *Journal of Orthopaedic & Sports Physical Therapy, 27*, 154-161.

Stupka, N., Lowther, S., Chorneyko, K., Bourgeois, J., Hogben, C., & Tarnopolsky, M. (2000). Gender differences in muscle inflammation after eccentric exercise. *Journal of Applied Physiology, 89*, 2325-2332.

Sullivan, S., Williams, L., Seaborne, D., & Morelli, M. (1991). Effects of massage on alpha motoneuron excitability. *Physical Therapy, 71*, 555-560.

Tappan, F., & Benjamin, P. (1998). *Tappan's handbook of healing massage techniques: classic, holistic, and emerging methods*. Stamford, TP: Appleton & Lange.

Taylor, B., Waring, C., & Brashear, T. (1995). The effects of therapeutic application of heat or cold followed by static stretch on hamstring muscle length. *Journal of Orthopaedic & Sports Physical Therapy, 21*, 1995.

Taylor, D., Brooks, D., & Ryan, J. (1997). Viscoelastic characteristics of muscle: passive stretching versus muscular contractions. *Medicine & Science in Sports & Exercise, 29*, 1619-1624.

Taylor, D., Dalton, J., Seaber, A., & Garrett, W. (1990). Viscoelastic properties of muscle-tendon units: the biomechanical effects of stretching. *American Journal of Sports Medicine, 18*, 300-309.

Teague, B., & Schwane, J. (1995). Effect of intermittent eccentric contractions on symptoms of muscle microinjury. *Medicine & Science in Sports & Exercise, 27*, 1378-1384.

Terry, P., Lane, A., Lane, H., & Keohane, L. (1999). Development and validation of a mood measure for adolescents. *Journal of Sports Sciences, 17*, 861-872.

Thacker, S., Gilchrist, J., Stroup, D., & Kimsey, C. J. (2004). The impact of stretching on sports injury risk: a systematic review of the literature. *Medicine & Science in Sports & Exercise, 36*, 371-378.

Thayer, R. E. (1967). Measurement through self-report. *Psychological Reports, 20*, 663-678.

Thigpen, L., Moritani, T., Ythiebaud, R., & Hargis, J. (1985). The acute effects of static stretching on alpha motoneuron excitability. In W. Winter, R. Norman, R. Wells, K. Hayes & A. Patla (Eds.), *Biomechanics IX-A* (pp. 353-357). Champaign, IL: Human Kinetics.

Thompson, H., Hyatt, J., De Souza, M., & Clarkson, P. (1997). The effects of oral contraceptives on delayed onset muscle soreness following exercise. *Contraception, 56*, 59-65.

Tiidus, P. (1997). Manual massage and recovery of muscle function following exercise: a literature review. *Journal of Orthopaedic & Sports Physical Therapy, 25,* 107-112.

Tiidus, P. (1999). Massage and ultrasound as therapeutic modalities in exercise-induced muscle damage. *Canadian Journal of Applied Physiology, 24,* 267-278.

Tiidus, P. (2003). Influence of estrogen on skeletal muscle damage, inflammation, and repair. *Exercise and Sport Sciences Reviews, 31,* 40-44.

Tiidus, P., & Ianuzzo, C. (1983). Effects of intensity and duration of muscular exercise on delayed soreness and serum enzyme activities. *Medicine & Science in Sports & Exercise, 15,* 461-465.

Tiidus, P., & Shoemaker, J. (1995). Effleurage massage, muscle blood flow and long term post-exercise recovery. *International Journal of Sports Medicine, 16,* 478-483.

Toft, E., Sinkjaler, T., Kalund, S., & Espersen, G. (1989). Biomechanical properties of the human ankle relation to passive stretch. *Journal of Biomechanics, 22,* 1129-1132.

van Mechelen, W., Hlobil, H., Kemper, H., Voorn, W., & de Jongh, H. (1993). Prevention of running injuries by warm-up, cool-down, and stretching exercise. *American Journal of Sports Medicine, 21,* 711-719.

Vanderby, R., & Provenzano, P. (2003). Collagen in connective tissue: from tendon to bone. *Journal of Biomechanics, 36,* 1523-1527.

Vickers, A. (2001). Time course of muscle soreness following different types of exercise. *BMC Musculoskeletal Disorder, 2,* 5.

Vickers, A., Fisher, P., Smith, C., Wyllie, S., & Lewith, G. (1997). Homoeopathy for delayed onset muscle soreness: a randomised double blind placebo controlled trial. *British Journal of Sports Medicine, 31,* 304-307.

Viitasalo, J., Niemela, K., Kaapola, R., Korjus, T., Levola, M., Mononen, H., et al. (1995). Warm under water water-jet massage improves recovery from intense physical exercise. *European Journal of Applied Physiology, 71,* 431-438.

Vujnovich, A., & Dawson, N. (1994). The effects of therapeutic muscle stretch on neural processing. *Journal of Orthopaedic & Sports Physical Therapy, 20,* 145-153.

Walsh, B., Tonkonogi, M., Malm, C., Ekblom, B., & Sahlin, K. (2001). Effect of eccentric exercise on muscle oxidative metabolism in humans. *Medicine & Science in Sports & Exercise, 33,* 436-441.

Warren, C., Lehmann, J., & Koblanski, J. (1976). Heat and stretch procedures: an evaluation using rat tail tendon. *Archives of Physical Medicine and Rehabilitation, 57,* 122-126.

Watson, A. (2001). Sports injuries related to flexibility, posture, acceleration, clinical defects, and previous injury, in high-level players of body contact sports. *International Journal of Sports Medicine, 22*, 222-225.

Weber, M., Servedio, F., & Woodall, W. (1994). The effects of three modalities on delayed onset muscle soreness. *Journal of Orthopaedic & Sports Physical Therapy, 20*, 236-242.

Weerapong, P., Hume, P., & Kolt, G. (2004a). Preventative strategies for exercise-induced muscle damage. *Critical Reviews in Physical & Rehabilitation Medicine, 16*, 133-149.

Weerapong, P., Hume, P., & Kolt, G. (2004b). Stretching: mechanisms and benefits on sport performance and injury prevention. *Physical Therapy Reviews, 9*, 189-206.

Weinberg, R., Jackson, A., & Kolodny, K. (1988). The relationship of massage and exercise to mood enhancement. *The Sport Psychologist, 2*, 202-211.

Weldon, S., & Hill, R. (2003). The efficacy of stretching for prevention of exercise-related injury: a systematic review of the literature. *Manual Therapy, 8*, 141-150.

Wenos, J., Brilla, L., & Morrison, M. (1990). Effect of massage on delayed onset muscle soreness. *Medicine & Science in Sports & Exercise, 22(Suppl.)*, 4.

Wessel, J., & Wan, A. (1994). Effect of stretching on the intensity of delayed-onset muscle soreness. *Clinical Journal of Sport Medicine, 4*, 83-87.

Whitehead, N., Allen, T., Morgan, D., & Proske, U. (1998). Damage to human muscle from eccentric exercise after training with concentric exercise. *Journal of Physiology, 512*, 615-620.

Wiemann, K., & Hahn, K. (1997). Influences of strength, stretching and circulatory exercises on flexibility parameters of the hamstrings. *International Journal of Sports Medicine, 18*, 340-346.

Wiktorsson-Moller, M., Oberg, B., Ekstrand, J., & Gillquist, J. (1983). Effects of warming up, massage, and stretching on range of motion and muscle strength in the lower extremity. *American Journal of Sports Medicine, 11*, 249-252.

Williams, P., & Goldspink, G. (1984). Connective tissue changes in immobilised muscle. *Journal of Anatomy, 138*, 343-350.

Wilson, G., Elliott, B., & Wood, G. (1992). Stretch shorten cycle performance enhancement through flexibility training. *Medicine & Science in Sports & Exercise, 21*, 116-123.

Wilson, G., Murphy, A., & Pryor, J. (1994). Musculotendinous stiffness: Its relationship to eccentric, isometric and concentric performance. *Journal of Applied Physiology, 76*, 2714-2719.

Witvrouw, E., Mahieu, N., Danneels, L., & McNair, P. (2004). Stretching and injury prevention: an obscure relationship. *Sports Medicine, 34*, 443-449.

Wolfe, R., & Cumming, G. (2004). Communicating the uncertainty in research findings: confidence intervals. *Journal of Science and Medicine in Sport*, *7*, 138-143.

Worrell, T., Smith, T., & Winegardner, J. (1994). Effects of hamstring stretching on hamstring muscle performance. *Journal of Orthopaedic & Sports Physical Therapy, 20*, 154-159.

Yackzan, L., Adams, C., & Francis, K. (1984). The effects of ice massage on delayed muscle soreness. *American Journal of Sports Medicine, 12*, 159-165.

Young, W., & Behm, D. (2003). Effects of running, static stretching and practice jumps on explosive force production and jumping performance. *Journal of Sports Medicine and Physical Fitness, 43*, 21-27.

Zehr, E. (2002). Considerations for the use of the Hoffmann reflex in exercise studies. *European Journal of Applied Physiology, 86*, 455-468.

Zeitlin, D., Keller, S., Shiflett, S., Schleifer, S., & Bartlett, J. (2000). Immunological effects of massage therapy during academic stress. *Psychosomatic Medicine, 62*, 83-87.

Zelikovski, A., Kaye, C., Fink, G., Spitzer, S., & Shapiro, Y. (1993). The effects of the modified intermittent sequential pneumatic device (MISPD) on exercise performance following an exhaustive exercise bout. *British Journal of Sports Medicine, 27*, 255-259.

Zito, M., Driver, D., Parker, C., & Bohannon, R. (1997). Lasting effects of one bout of two 15-second passive stretches on ankle dorsiflexion range of motion. *Journal of Orthopaedic & Sports Physical Therapy, 26*, 214-221.

APPENDICES

Appendix A: Participant Information Package (Study A)

AUCKLAND UNIVERSITY OF TECHNOLOGY
TE WĀNANGA ARONUI O TAMAKI MAKAU RAU

PARTICIPANT INFORMATION PACKAGE

Contact person:

Pornratshanee Weerapong, PhD Candidate, Auckland University of Technology, Private Bag 92006, Tel: (09) 9179999 x 7848 Email: pornratshanee.weerapong@aut.ac.nz

Title: Preparation strategies for exercise: the effects of warm-up, dynamic stretching, and massage on muscle properties and the severity of delayed onset muscle soreness.

Introduction:

You are invited to take part in the above mentioned research project. Your participation in this testing is voluntary. You are free to withdraw consent and discontinue participation at anytime without influencing any present and/or future involvement with the Auckland University of Technology.

Your consent to participate in this research will be indicated by your signing and dating the consent form. Signing the consent form indicates that you have freely given your consent to participate, and that there has been no coercion or inducement to participate.

Aim of Study

The project aims to examine possible mechanisms for warm-up, dynamic stretching, and manual massage in terms of biomechanical (evaluated by muscle stiffness and range of motion), and the severity of delayed onset muscle soreness (DOMS) in healthy participants.

Participants

Healthy males, age 20-40 years, who have not taken part in a weight-training programme or regularly perform strenuous leg exercise for occupational or recreational purposes within the past year.

Criteria for exclusion

Participants who have previous injuries on leg muscles and/or have musculoskeletal problems. Participants who take part in a weight-training programme or regularly perform strenuous leg exercise for occupational or recreational purposes within the past year will be excluded.

Location

Room AA126, Akoranga Campus, Auckland University of Technology

Time

By participating in Study A you will be required to give up one hour of your time on the first two days and 30 minutes per day on day three to five (five consecutive days).

Methods

Data will be collected during both the experimental and control conditions using Visual Analogue Scale (VAS) and punctate methods for muscle soreness, KinCom dynamometer for muscle strength and muscle stiffness, and goniometer for range of motion.

You will be randomly assigned to receive one of these treatments (warm-up, dynamic stretching, and massage).

You will be required to exercise knee flexor muscles eccentrically by using KinCom Dynamometer. The eccentric exercise bout will consists of knee flexion exercises at 80% maximum isometric contraction, 10 repetitions per set for ten sets. You will be allowed to rest for one minute between each set.

You will have the following variables measured: soreness of knee flexors, passive muscle stiffness of knee flexors, isometric muscle strength of knee flexors, electromyography of knee flexors, and range of motion of knee flexion and extension before intervention as baseline data, immediately after intervention, and immediately after eccentric exercise.

Passive muscle stiffness of knee flexors, isometric muscle strength of knee flexors electromyography of knee flexors, range of motion of knee flexion and extension, soreness sensation will be measured everyday for four consecutive days, at the same time each day.

The information collected will allow us to determine if preexercise activities (warm-up, dynamic stretching, and massage) can reduce the risk of knee flexors injury.

Benefits of the study

Possible long term effects of injury prevention, and thereby a potential reduction in chronic pain, cost of injury treatment, and time lost from training activities.

Possible risks of the study

There is a possible injury, however this risk is an equivalent to that for normal participation in physical training. Taking part in this research will not cost you.

Results

You will receive an individualised report and a copy of the final report (with no participants identified).

Any concerns regarding the nature of this project should be notified in the first instance to Patria Hume, the Project Supervisor. If you have any other questions please feel free to contact Patria Hume or Pornratshanee Weerapong at any time.

Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEC, Madeline Banda, madeline.banda@aut.ac.nz, 917 9999 ext 8044.

Approved by the Auckland University of Technology Ethics Committee on

12 December 2002 AUTEC Reference number 02/173

Appendix B: Consent to Participation in Research (Study A)

AUCKLAND UNIVERSITY OF TECHNOLOGY
TE WĀNANGA ARONUI O TAMAKI MAKAU RAU

Consent to Participation in Research

Title of Project: Preparation strategies for exercise: the effects of warm-up, dynamic stretching, and massage on muscle soreness.

Project Supervisor: Associate Professor Patria Hume (PhD)

Researcher: Pornratshanee Weerapong (MSc), Auckland University of Technology Private Bag 92006, Auckland. Phone: 9179999 ext 7848

- I have read and understood the information provided about this research project.
- I have had an opportunity to ask questions and to have them answered.
- I understand that I may withdraw myself or any information that I have provided for this project at any time prior to completion of data collection, without being disadvantaged in any way.
- I agree to take part in this research.

Participant signature:..

Participant name:…………………………………………….

Date: …………………………………….

Project Supervisor Contact Details: Associate Professor Patria Hume (PhD)

Auckland University of Technology

Private Bag 92006, Auckland.

Phone: 9179999 ext 7306

Approved by the Auckland University of Technology Ethics Committee on 12 December 2002 AUTEC Reference number 02/173

Appendix C: Approval Letter for the Pilot Study (Study A) from the Auckland University of Technology Ethics Committee

To: Patria Hume

From: Madeline Banda

Date: 28 November 2005

Subject: 01/72 The effects of massage on relaxation, flexibility, and the severity of delayed onset muscle soreness - pilot

Dear Patria

- Your application for ethics approval was considered by AUTEC at their meeting on 27 August.
- Your application was approved for a period of two years until August 2003.
- You are required to submit the following to AUTEC:
- A brief annual progress report indicating compliance with the ethical approval given.
- A brief statement on the status of the project at the end of the period of approval or on completion of the project, whichever comes sooner.
- A request for renewal of approval if the project has not been completed by the end of the period of approval.
- The Committee wishes you well with your research.

Please include the application number and study title in all correspondence and telephone queries.

Yours sincerely

Madeline Banda (Executive Secretary AUTEC)

Appendix D: Approval Letter for the Main Study (Study A) from the Auckland University of Technology Ethics Committee

To: Patria Hume

From: Madeline Banda

Date: 12 December 2002

Subject: 02/173 Preparation strategies for exercise: The effects of warm up, stretching, and massage on muscle soreness

Dear Patria

- Your application for ethics approval was considered by AUTEC at their meeting on 09/12/02.
- Your application was approved for a period of two years until December 2004.
- You are required to submit the following to AUTEC:
- A brief annual progress report indicating compliance with the ethical approval given.
- A brief statement on the status of the project at the end of the period of approval or on completion of the project, whichever comes sooner.
- A request for renewal of approval if the project has not been completed by the end of the period of approval.

Please note that the Committee grants ethical approval only. If management approval from an institution/organisation is required, it is your responsibility to obtain this.

The Committee wishes you well with your research.

Please include the application number and study title in <u>all</u> correspondence and telephone queries.

Yours sincerely

Madeline Banda (Executive Secretary AUTEC)

Appendix E: International Physical Activity Questionnaire: Short Last 7 Days Self-Administered Format

In answering the following questions,

- Vigorous physical activities refer to activities that take hard physical effort and make you breathe much harder than normal.
- Moderate activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal.

1a. During the last 7 days, on how many days you do <u>vigorous</u> physical activities like heavy lifting, digging, aerobics, or fast bicycling?

Think about *only* those physical activities that you did for at least 10 minutes at a time.

……….. days per week ➔ 1b. How much time in total did you usually spend on one of those days doing vigorous physical activities?

☐ none

……………. hours……………minutes

2a. Again, think *only* about those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do <u>moderate</u> physical activities like carrying light loads, bicycling at a regular pace, or double tennis? Do not include walking.

……….. days per week ➔ 2b. How much time in total did you usually spend on one of those days doing moderate physical activities?

☐ none

……………. hours……………minutes

3a. During the last 7 days, on how many days did you <u>walk</u> for at least 10 minutes at a time? This includes walking at work and at home, walking to travel from place to place, and any other walking that you did solely for recreation, sport, exercise or leisure.

……….. days per week ➔ 3b. How much time in total did you usually spend walking on one of those days?

☐ none

……………. hours……………minutes

The last question is about the time you spent sitting on weekdays while at work, at home, while doing course work and during leisure time. This includes time spent sitting at a desk, visiting friends, reading travelling on bus or sitting or lying down to watch television.

---

4. During last 7 days, how much time in total did you usually spend sitting on a week day?

……………. hours……………minutes

This is the end of the questionnaire, thank you for participating.

Appendix F: International Physical Activity Questionnaire: Short Last 7 Days Self-Administered Format (Modification)

In answering the following questions,

- Vigorous physical activities refer to activities that take hard physical effort and make you breathe much harder than normal.
- Moderate activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal.

---

1. Yesterday, on how much time in total did you spend on <u>vigorous</u> physical activities like heavy lifting, digging, aerobics, or fast bicycling?

Think about *only* those physical activities that you did for at least 10 minutes at a time.

☐ none ……………. hours……………minutes

Please indicate …………………………………………

---

2. Again, think *only* about those physical activities that you did for at least 10 minutes at a time. Yesterday, on how much time in total did you spend on <u>moderate</u> physical activities like carrying light loads, bicycling at a regular pace, or double tennis? Do not include walking.

☐ none ……………. hours……………minutes

Please indicate …………………………………………

---

3. Yesterday, on how much time in total did you <u>walk</u> for at least 10 minutes at a time? This includes walking at work and at home, walking to travel from place to place, and any other walking that you did solely for recreation, sport, exercise or leisure.

☐ none ……………. hours……………minutes

Please indicate …………………………………………

The last question is about the time you spent sitting on weekdays while at work, at home, while doing course work and during leisure time. This includes time spent sitting at a desk, visiting friends, reading travelling on bus or sitting or lying down to watch television.

---

4. Yesterday, on how much time in total did you <u>sitting</u>?

……………. hours……………minutes

This is the end of the questionnaire, thank you for participating.

Appendix G: Data Collection Sheet (Study A)

Participant characteristics
Name……………………….……..… Date………………….. Time ……….…… Dominant leg L / R
Date of birth…………..… Occupation……………….. Weight………kg Height………..

Physical activity (please indicate) 1)…………………………… 2)………………………………
Frequency. ………hr/week for………… month (s)/year(s)
Medication……………………………………

Investigator only (Code…………)
Lever arm………….cm
MVC………….…Nm 80% MVC……. Nm

Knee support height………………cm
Muscle length……………... deg ROM for passive stiffness…………deg

Tasks	D1			D2			D3			D4			D5		
1. ☐ Participant information ☐ Consent form ☐ BW ☐ Height ☐ %BF (Triceps………./…………/………., Subscapularis……./…………/………., Supraspinal………./………/………., Abdominal……..…./………/……….)															
2. IPAQ															
3. ROM - Flexion (A/P) - Extension (A/P)															
4 Passive stiffness															
5 MVC & VAS															
6 ☐ Warm-up ☐ Stretching ☐ Massage															
7 ROM - Flexion (A/P) - Extension (A/P)															
8 Passive stiffness															
9 MVC &VAS															
10 Eccentric exercise (80%MVC, 10rep/set, 10sets)															

Appendix G (continued). Data Collection Sheet (Study A)

11 ROM - Flexion (A/P) - Extension (A/P)					
12 Passive stiffness					
13 MVC &VAS					

Please draw a line at the point that you feel

(D1-pre)

No Soreness at all Worst Soreness Imaginable

Please draw a line at the point that you feel
(D1-post)

No Soreness at all Worst Soreness Imaginable

Please draw a line at the point that you feel
(D2-pre)

No Soreness at all Worst Soreness Imaginable

Please draw a line at the point that you feel
(D2-post)

No Soreness at all Worst Soreness Imaginable

Please draw a line at the point that you feel
(D3)

No Soreness at all Worst Soreness Imaginable

Please draw a line at the point that you feel
(D4)

No Soreness at all Worst Soreness Imaginable

Please draw a line at the point that you feel
(D5)

No Soreness at all Worst Soreness Imaginable

Appendix H: Pilot Results from Study A

Pilot testing was conducted during June 2002. Two female and one male participants completed the experimental condition for reliability but only one female and one male completed the experimental protocol for four consecutive days. The other male completed the eccentric exercise protocol. None of the participants were regular exercisers.

1. Reliability of the experimental protocol

The test-retest for reliability was conducted in two ways: within one day and for four consecutive days. For the within-day test, the participants were moved out from the KinCom dynamometer after finishing the first test and lain down for 20 minutes before starting the retest in the same sequence. For the between days tests (four consecutive days), participants came to the laboratory at the same time each day and were tested in the same procedure.

1.2 Test-retest within 20 minutes

The outcome parameters of muscle properties and the severity of muscle damge were measured in three participants. After finishing the first test of all outcome measures, the participants rested for 20 minutes before starting the second test in the same sequence. All parameters (range of motion, maximal isometric contraction (MVC), and passive stiffness) showed a high correlation (ICC > 0.90) between test and retest. However, tenderness, which was evaluated by the punctate measurer, showed a very low correlation (ICC=-0.33). The means and standard deviations of each parameter are presented in Table H1.

Table H1 Means and standard deviations for DOMS parameters for test-retest reliability within one day for three participants

Participants	A		B		C		ICC
Trials	Test	Retest	Test	Retest	Test	Retest	
Parameters							
1. Tenderness (kg)	3.18(0.25)	3.32(0.50)	3.26(0.03)	3.16(0.18)	3.16(0.51)	3.17(0.41)	0.33
2. ROM (°)							
Active							
- Flexion	42(1)	43(1)	47(1)	45(2)	39(1)	41(1)	0.91
- Extension	178(1)	176(1.53)	184(1)	183(0)	181(0)	181(1)	0.99
Passive							
- Extension	172(1)	175(2)	183(1)	183(1)	179(2)	18(1)	0.97
3. MVC (N.m)	167.28	173.24	310.64	303.58	115.80	124.80	1.00
4. Passive stiffness							
- Maximum tension (N.m)	24.1(1.2)	24.3(0.9)	33.4(1.6)	32.9(1.2)	13.3(0.1)	12.7(0.2)	1.00
- Slope 1 (N.m$^{\circ-1}$) (initial portion)	0.24(0.03)	0.23(0.03)	0.21(0.00)	0.22(0.01)	0.14(0.01)	0.12(0.00)	0.98
- Slope 2 (N.m$^{\circ-1}$) (linear portion)	0.33(0.03)	0.34(0.04)	0.55(0.02)	0.49(0.01)	0.20(0.02)	0.25(0.00)	0.96
- Slope all (N.m$^{\circ-1}$)	0.28(0.02)	0.20(0.02)	0.29(0.01)	0.30(0.02)	0.15(0.00)	0.15(0.00)	1.00
- Stress relaxation (N.m)	-	-	6.23(0.59)	6.80(0.26)	2.09(0.03)	1.20(0.12)	0.99

1.2 Test-retest for four consecutive days

Two participants completed the test-retest protocols for four consecutive days. The intra-class correlation coefficients (ICC) showed a high value in all parameters except tenderness (ICC = 0.45). The means and standard deviations of each parameter are presented in Table H2.

1.3 The severity of DOMS

One male completed the eccentric exercise-induced DOMS protocol at 60%MVC, 10 repetitions per set for six sets, and was followed up for four consecutive days. The exercise intensity was sufficient to induce symptoms of muscle damage as indicated by increasing soreness sensation, increasing active flexed knee range of motion, reducing passive knee extension, decreasing muscle strength, and increasing passive muscle stiffness (see Table H3 and Figure H1). The soreness sensation was highest on day two after exercise. The active knee flexion increased (26%) and passive knee extension decreased (15%) maximally on day two. Muscle strength decreased immediately (22%) after exercise and reached the highest loss on day two (27%). Interestingly, passive stiffness decreased (24%) immediately after eccentric exercise. The decrease of passive stiffness might be due to the increase of tissue temperature as a result from eccentric exercise (Nadel, Bergh, & Saltin, 1972). However, the passive stiffness increased up to 33% on Day 1 after exercise and returned to baseline value on day three.

1.4 Conclusions

The test-retest reliability of all outcome measures showed a high correlation within one day (ICC>0.90) and between the four days (four consecutive days) (ICC>0.98). However, the tenderness measurement showed a low correlation both within one day and between four days. This might be the result of the definition of “the first feeling of discomfort”, which was used to indicate tenderness (McHugh, Connolly, Eston, Gartman, & Gleim, 2001; McHugh et al., 1999; Teague & Schwane, 1995), not being clear enough. Thus, in the main study the participants will be asked to report soreness sensation by using the Visual Analogue Scale (VAS) only.

The severity of muscle damage in this study was less than in the study by McHugh et al. (2001, 1999) as indicated by less severity of pain and tenderness. This might be due to using a lower speed of exercise (90°/s) than McHugh et al.’s protocol (150°/s). The speed of exercise was reduced to 90°/s because the participant could not finish the first set of the exercise protocol (a total of six sets) and caused a velocity error in the KinCom. Moreover, the participant could not control the intensity of exercise consistently (as seen by the set tracer at 60%MVC on the KinCom’s screen). In this pilot study, the peak force (60%MVC), the lengthening distance (knee extension 80°-

0°), and exercise velocity (90°/s) were appropriate because the participant was able to exercise for ten repetitions per set for six sets. Thus, the main study will increase the number of contractions from six to seven sets in order to ensure that the symptoms of muscle damage occur.

Table H2 Means and standard deviations for DOMS parameters for test-retest reliability between days from two participants

Days Parameters	1	2	3	4	ICC
1. Tenderness (N)					
Participant B	3.26(0.30)	3.13(0.35)	3.08(0.20)	3.37(0.45)	0.45
Participant C	3.38(0.36)	3.16(0.51)	3.03(0.27	3.49(0.39)	
2. ROM (°)					
- Flexion	47(1)	45(1)	47(1)	45(1)	0.99
Participant B	39(0)	39(1)	40(0)	40(0)	
Participant C					
- Extension	184(1)	183(1)	183(0)	183(0)	0.98
Participant B	182(1)	181(0)	182(1)	181(1)	
Participant C					
3. Muscle length (°)					
Participant B	183(1)	182(2)	183(1)	182(1)	0.98
Participant C	180(2)	180(1)	181(1)	180(0)	
4. MVC (N.m)					
Participant B	311	309	300	301	1.00
Participant C	116	129	128	132	
5. Passive stiffness					
- Maximum tension (N.m)					
Participant B	33.4(1.7)	32.6(1.9)	31.3(2.5)	33.3(2.8)	1.00
Participant C	13.3(0.7)	12.7(0.3)	13.5(0.3)	12.3(0.1)	
- Slope 1 (N.m$^{\circ-1}$) (initial portion)					
Participant B	0.21(0.00)	0.21(0.02)	0.22(0.02)	0.22(0.01)	0.99
Participant C	0.14(0.01)	0.14(0.01)	0.12(0.00)	0.11(0.00)	
- Slope 2 (N.m$^{\circ-1}$) (linear portion)					
Participant B	0.55(0.02)	0.49(0.04)	0.51(0.07)	0.56(0.08)	0.98
Participant C	0.20(0.02)	0.24(0.02)	0.29(0.01)	0.20(0.01)	
- Slope all (N.m$^{\circ-1}$)					
Participant B	0.29(0.01)	0.29(0.01)	0.30(0.03)	0.31(0.02)	1.00
Participant C	0.15(0.00)	0.15(0.00)	0.16(0.00)	0.14(0.00)	
- Stress relaxation (N.m)					
Participant B	6.23(0.59)	6.16(0.25)	6.08(1.37)	5.16(0.52)	1.00
Participant C	2.09(0.25)	1.58(0.20)	2.04(0.23)	1.76(0.46)	

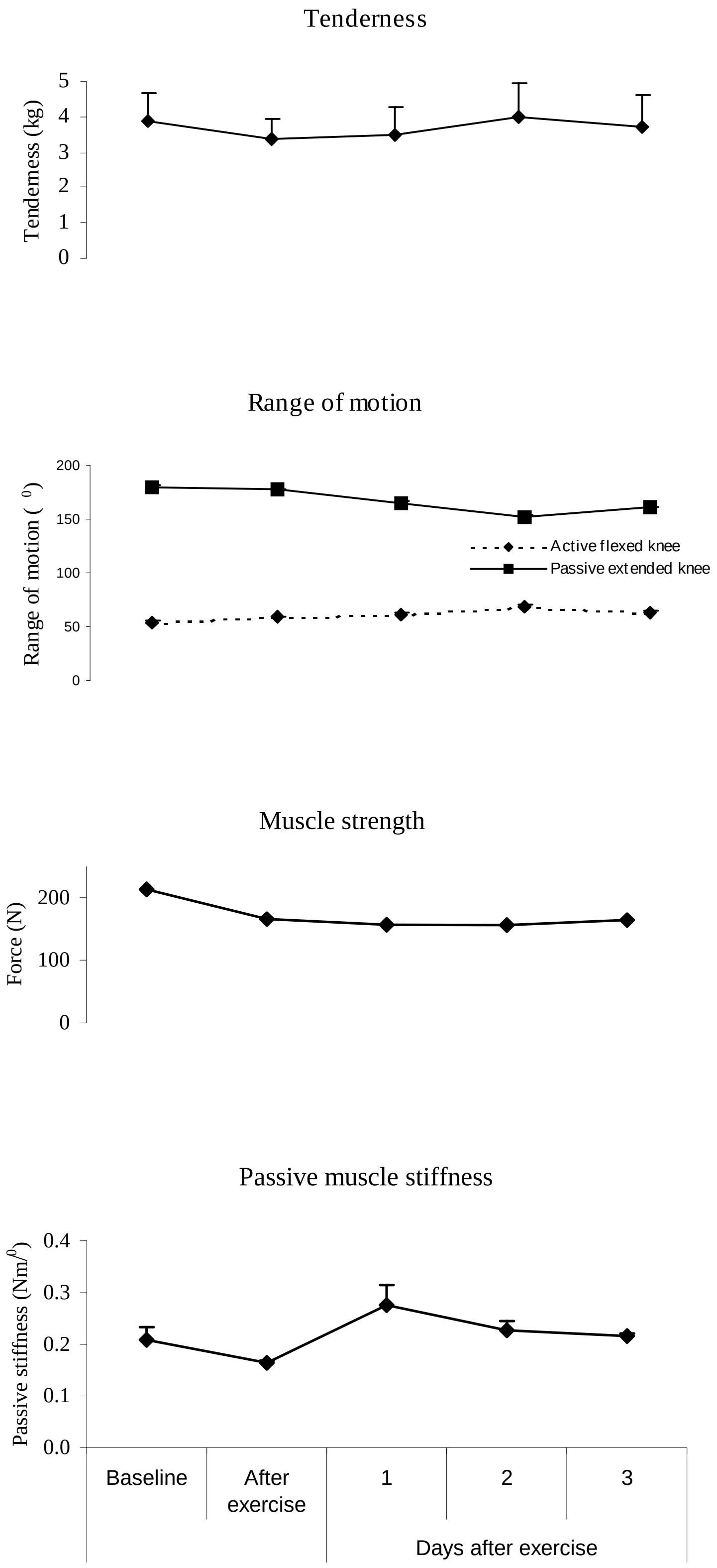
Table H3 Means and standard deviations for the severity for DOMS parameters for four consecutive days from one participant

Days			Days after exercise		
DOMS parameters	Baseline	After exercise	1	2	3
1. Muscle soreness (cm)	0.00	0.00	1.30	3.40	0.50
2. Tenderness (kg)	3.9(0.8)	3.4(0.6)	3.5(0.8)	4.0(0.9)	3.7(0.9)
3. Range of motion					
- Active flexed knee (°)	54.5(0.6)	58.5(0.6)	61.0(1.2)	69.0(1.2)	62.5(1.5)
- Passive extend knee (°)	180(0)	178(2)	165(0)	152(2)	160(1)
4. MVC (N.m)*	213	166	157	156	164
5. Passive muscle stiffness (N.m$^{\circ-1}$)	0.21(0.02)	0.16(0.00)	0.28(0.04)	0.23(0.02)	0.22(0.00)

* The maximum contraction from three trials is presented as the maximum voluntary contraction.

The participant in the pilot study did not complete the International Physical Activity Questionnaire (IPAQ) for the baseline physical activity and the modified IPAQ during the three follow-up days. In the main study, all participants will be asked to complete the IPAQ on Day 1 and the modified IPAQ on day two to four in order to monitor their physical activities.

In this pilot study, EMG data were not analyzed. The EMG collected during passive stiffness measurement was determined visually at the same time during data collection to ensure that the participants were completely relaxed. If there was a spike of EMG over the baseline, the measurement was then repeated.


Tenderness

Range of motion

Muscle strength

Passive muscle stiffness

Figure H1. The change in severity of DOMS parameters for four consecutive days for one participant

References

McHugh, M. P., Connolly, D., Eston, R., Gartman, E., & Gleim, G. (2001). Electromyographic analysis of repeated bouts of eccentric exercise. *Journal of Sports Sciences*, *19*, 163-170.

McHugh, M. P., Connolly, D. A., Eston, R. G., Kremenic, I., Nicholas, S. J., & Gleim, G. W. (1999). The role of passive muscle stiffness in symptoms of exercise-induced muscle damage. *American Journal of Sports Medicine*, *27*, 594-599.

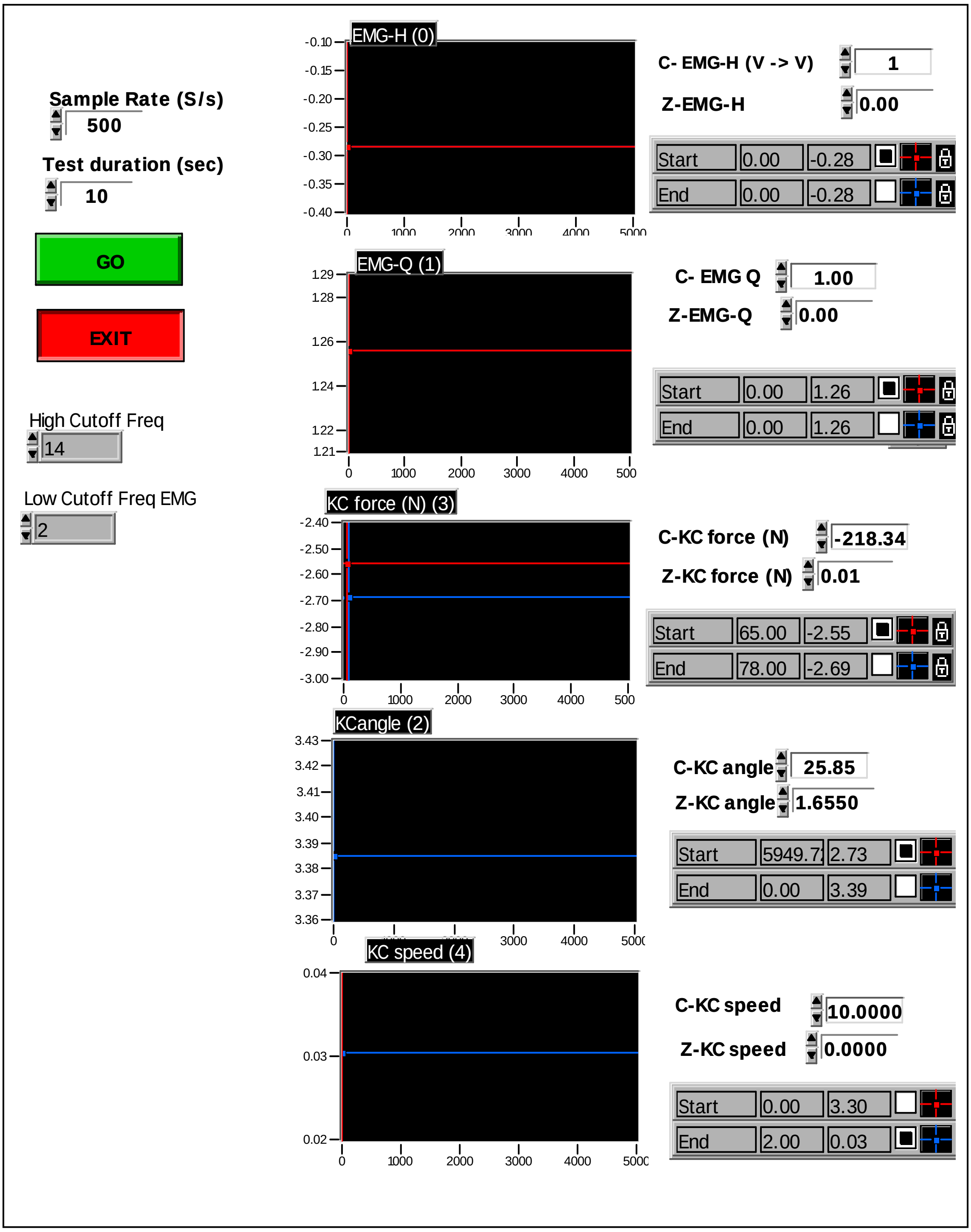
Nadel, E., Bergh, U., & Saltin, B. (1972). Body temperatures during negative work exercise. *Journal of Applied Physiology, 33*, 553-558.

Teague, B., & Schwane, J. (1995). Effect of intermittent eccentric contractions on symptoms of muscle microinjury. *Medicine & Science in Sports & Exercise, 27*, 1378-1384.

Appendix I: LabView for Data Collection and Analysis (Study A)

There are four Labview programmes used to collect and analyse data from the KinCom dynamometer in Study A. In each of these LabView programme, 40% of work is my own work, 40% is that of Associate Professor Patria Hume, and 20% is that of Karoline Cheung.

Figure I1 and I2 showed front panel and diagram, respectively, of the Labview programmes for data collection (passive stiffness). Figure I3 and I4 showed the front panel and diagram, respectively, of the Labview programmes for data analysis (passive stiffness and EMG of hamstrings and quadriceps during passive movement). Figure I5 and I6 showed the front panel and diagrams, respectively, of the Labview programmes for data analysis (maximum isometric contraction and EMG at maximum isometric contraction). Figure I7 and I8 showed the front panel and diagram, respectively, of the Labview programmes for data analysis (energy absorption during eccentric exercise).



Sample Rate (S/s) 500

Test duration (sec) 10

High Cutoff Freq 14

Low Cutoff Freq EMG 2

C- EMG-H (V -> V) 1

Z-EMG-H 0.00

Start 0.00 -0.28
End 0.00 -0.28

C- EMG Q 1.00

Z-EMG-Q 0.00

Start 0.00 1.26
End 0.00 1.26

C-KC force (N) -218.34

Z-KC force (N) 0.01

Start 65.00 -2.55
End 78.00 -2.69

C-KC angle 25.85

Z-KC angle 1.6550

Start 5949.7 2.73
End 0.00 3.39

C-KC speed 10.0000

Z-KC speed 0.0000

Start 0.00 3.30
End 2.00 0.03

Figure I1. Front panel of the Labview programme for data collection (passive stiffness and maximum isometric contraction) from Kincom dynamometer.

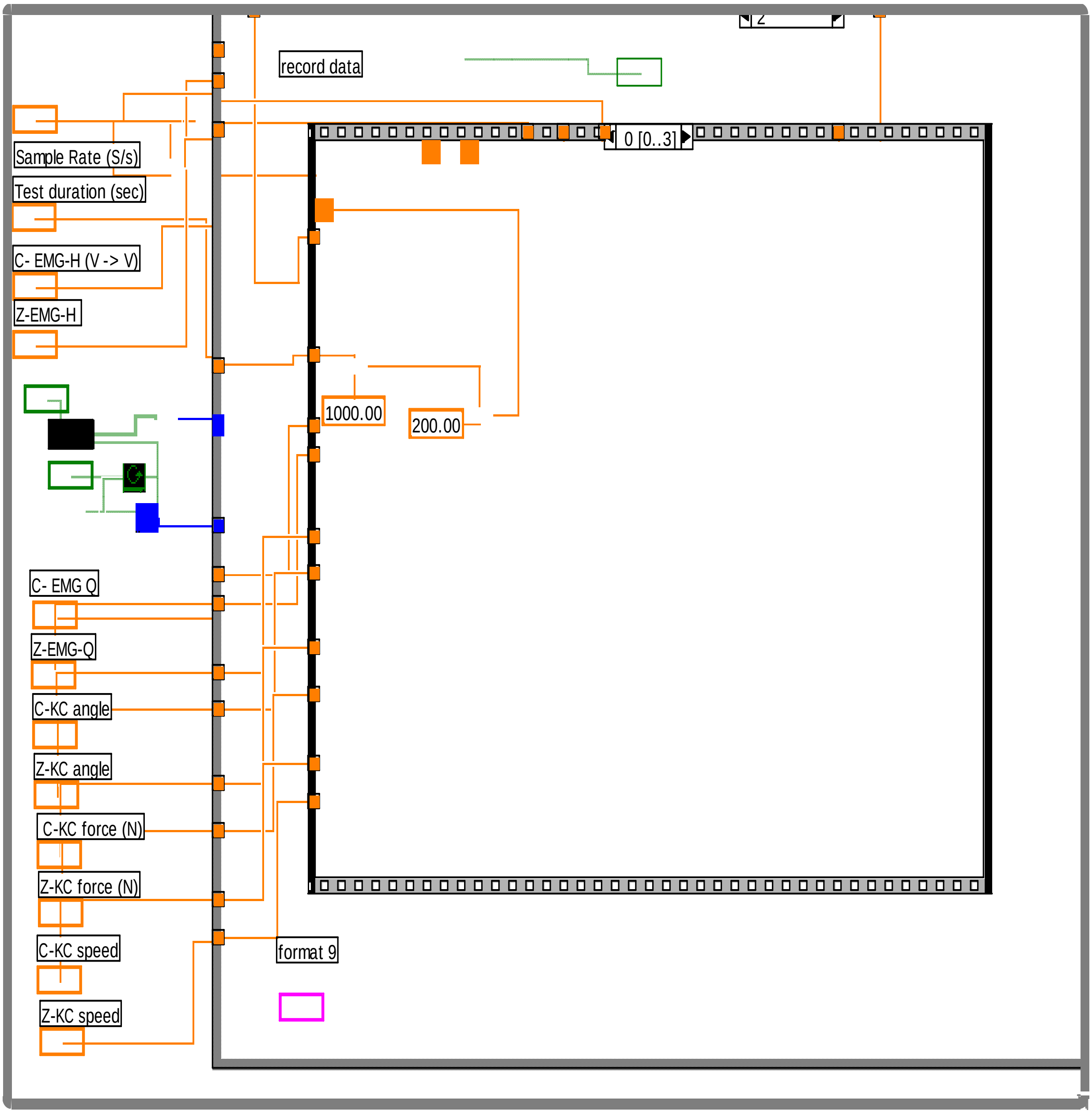


Figure I2. Diagram of the Labview programme for data collection (passive stiffness and maximum isometric contraction) from Kincom dynamometer (page 0).

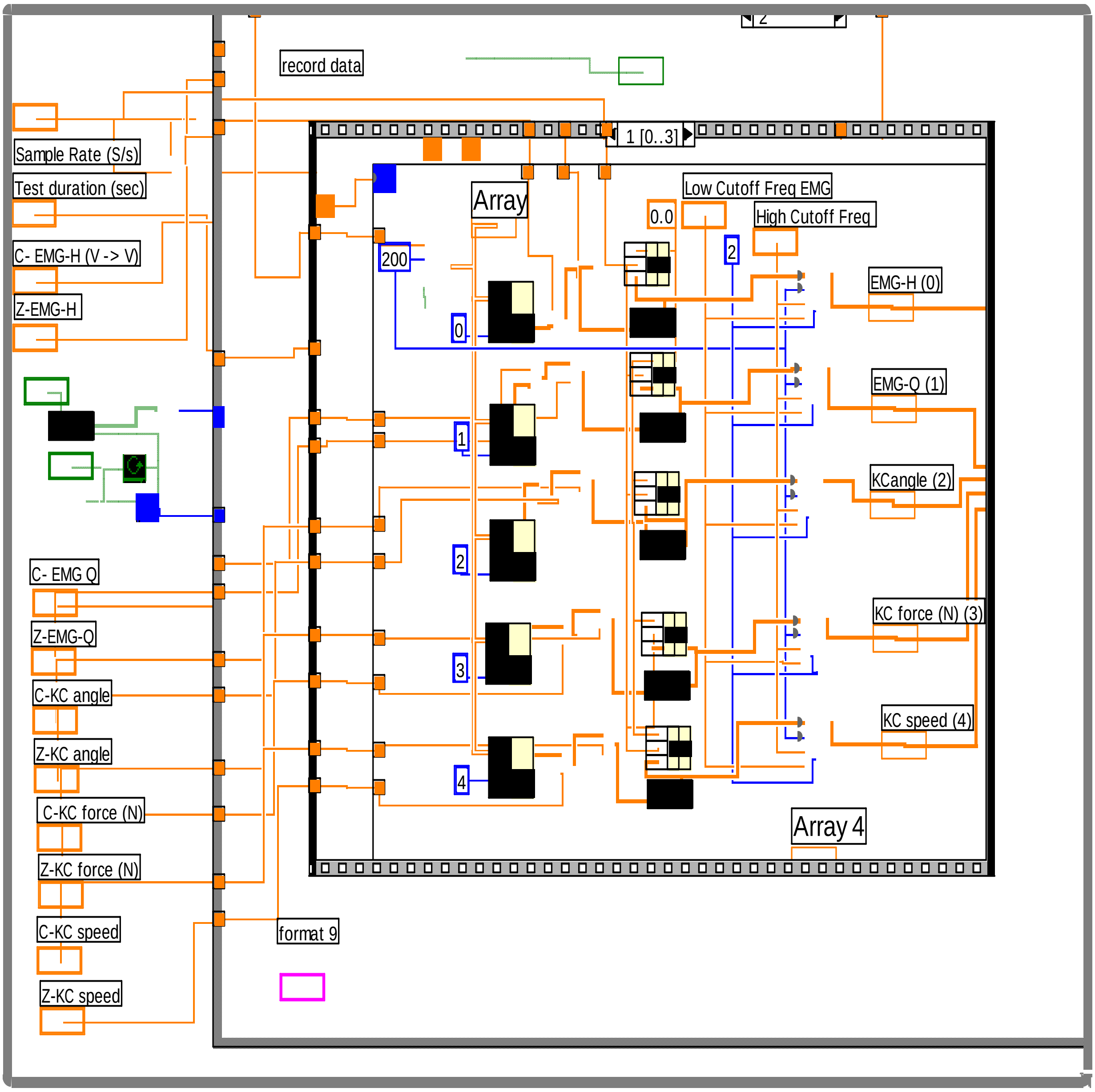


Figure I3. Diagram of the Labview programme for data collection (passive stiffness and maximum isometric contraction) from Kincom dynamometer (page 1).

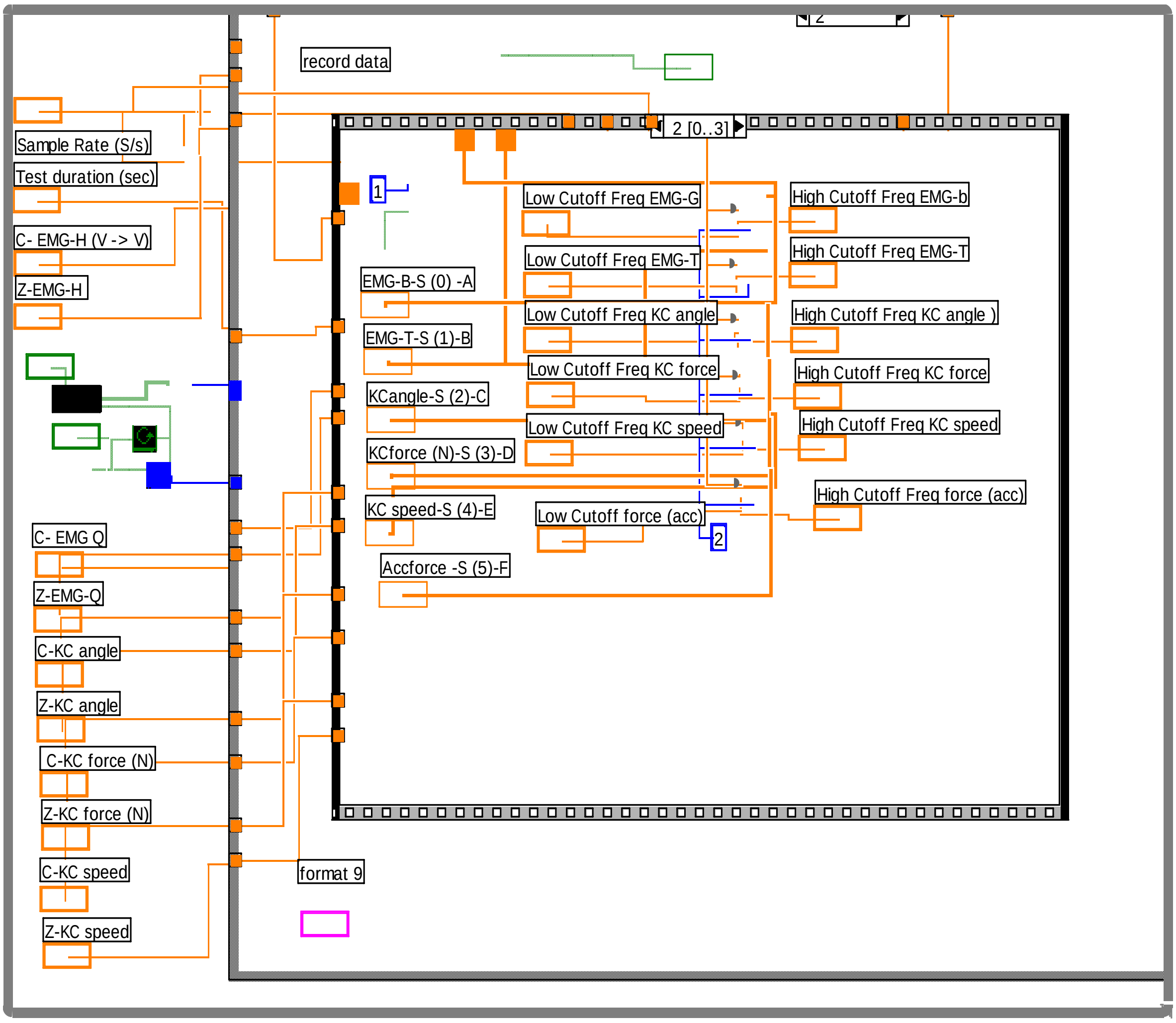


Figure I4. Diagram of the Labview programme for data collection (passive stiffness and maximum isometric contraction) from Kincom dynamometer (page 2).

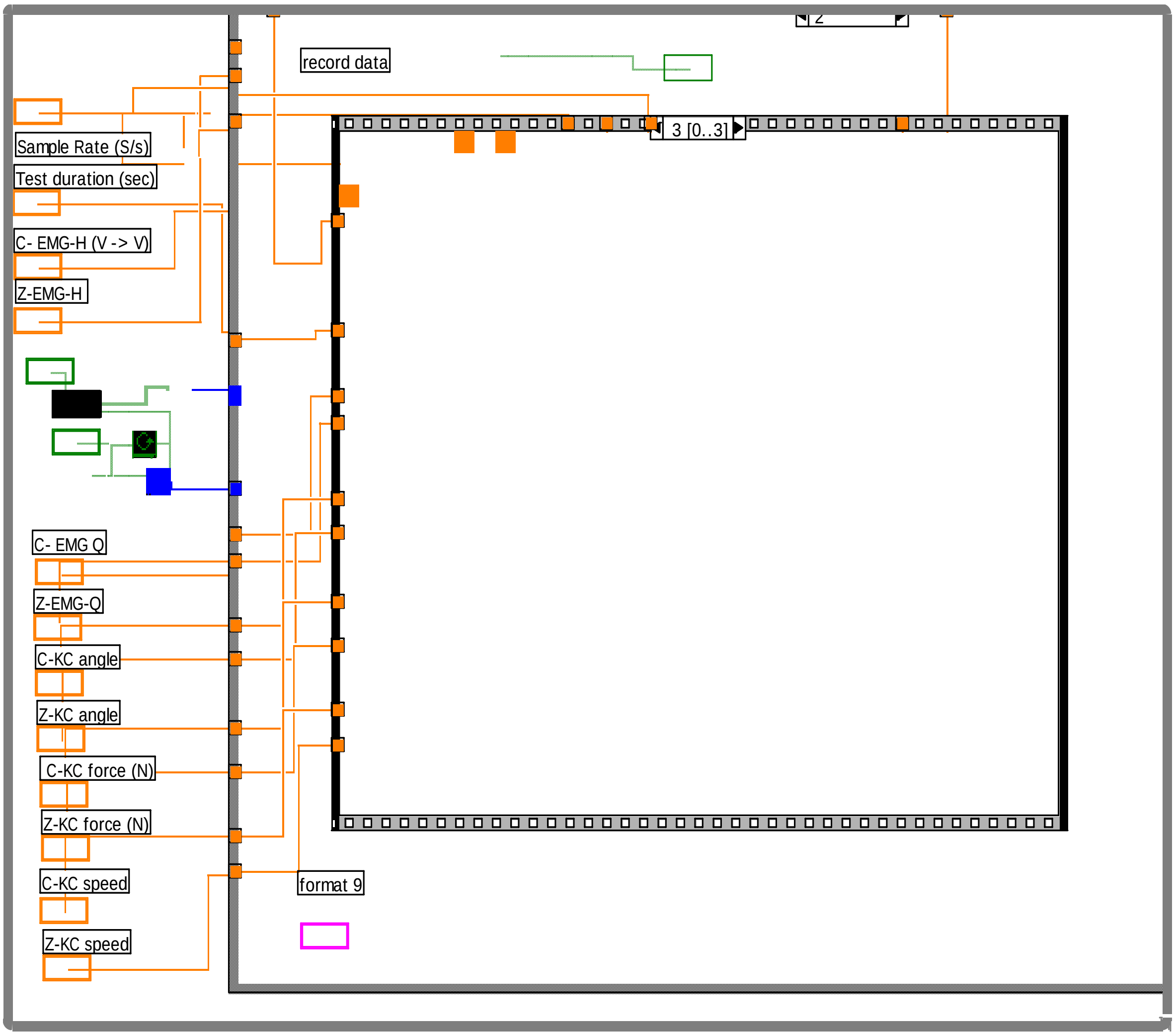
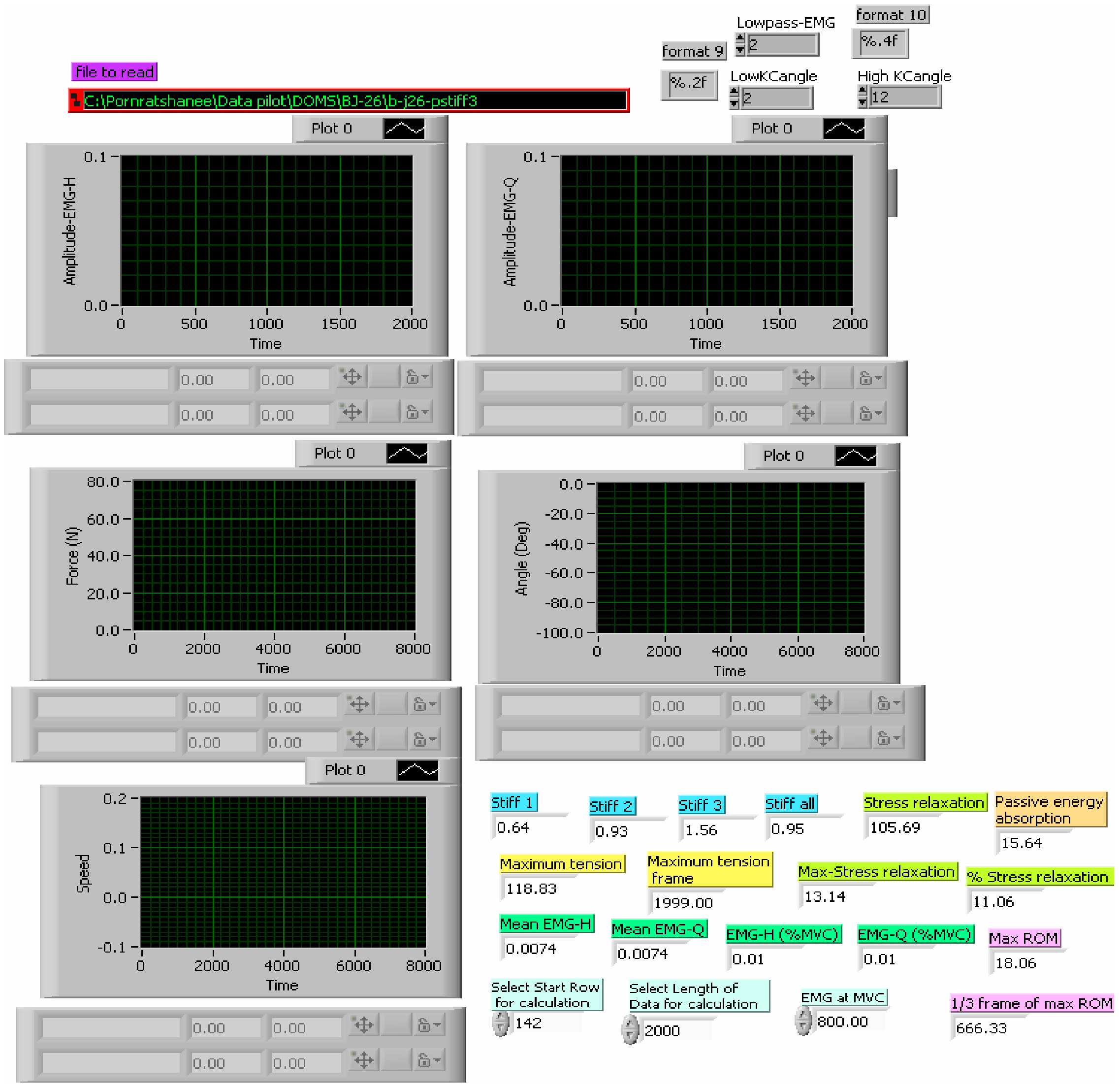


Figure I5. Diagram of the Labview programme for data collection (passive stiffness and maximum isometric contraction) from Kincom dynamometer (page 3).



File to read: C:\Pornratshanee\Data pilot\DOMS\BJ-26\b-j26-pstiff3

format 9: %.2f

Lowpass-EMG: 2

format 10: %.4f

LowKCangle: 2

High KCangle: 12

Stiff 1: 0.64

Stiff 2: 0.93

Stiff 3: 1.56

Stiff all: 0.95

Stress relaxation: 105.69

Passive energy absorption: 15.64

Maximum tension: 118.83

Maximum tension frame: 1999.00

Max-Stress relaxation: 13.14

% Stress relaxation: 11.06

Mean EMG-H: 0.0074

Mean EMG-Q: 0.0074

EMG-H (%MVC): 0.01

EMG-Q (%MVC): 0.01

Max ROM: 18.06

Select Start Row for calculation: 142

Select Length of Data for calculation: 2000

EMG at MVC: 800.00

1/3 frame of max ROM: 666.33

Figure I6. Front panel of the Labview programme for data analysis (passive stiffness and EMG of hamstrings and quadriceps during passive movement) from Kincom dynamometer.

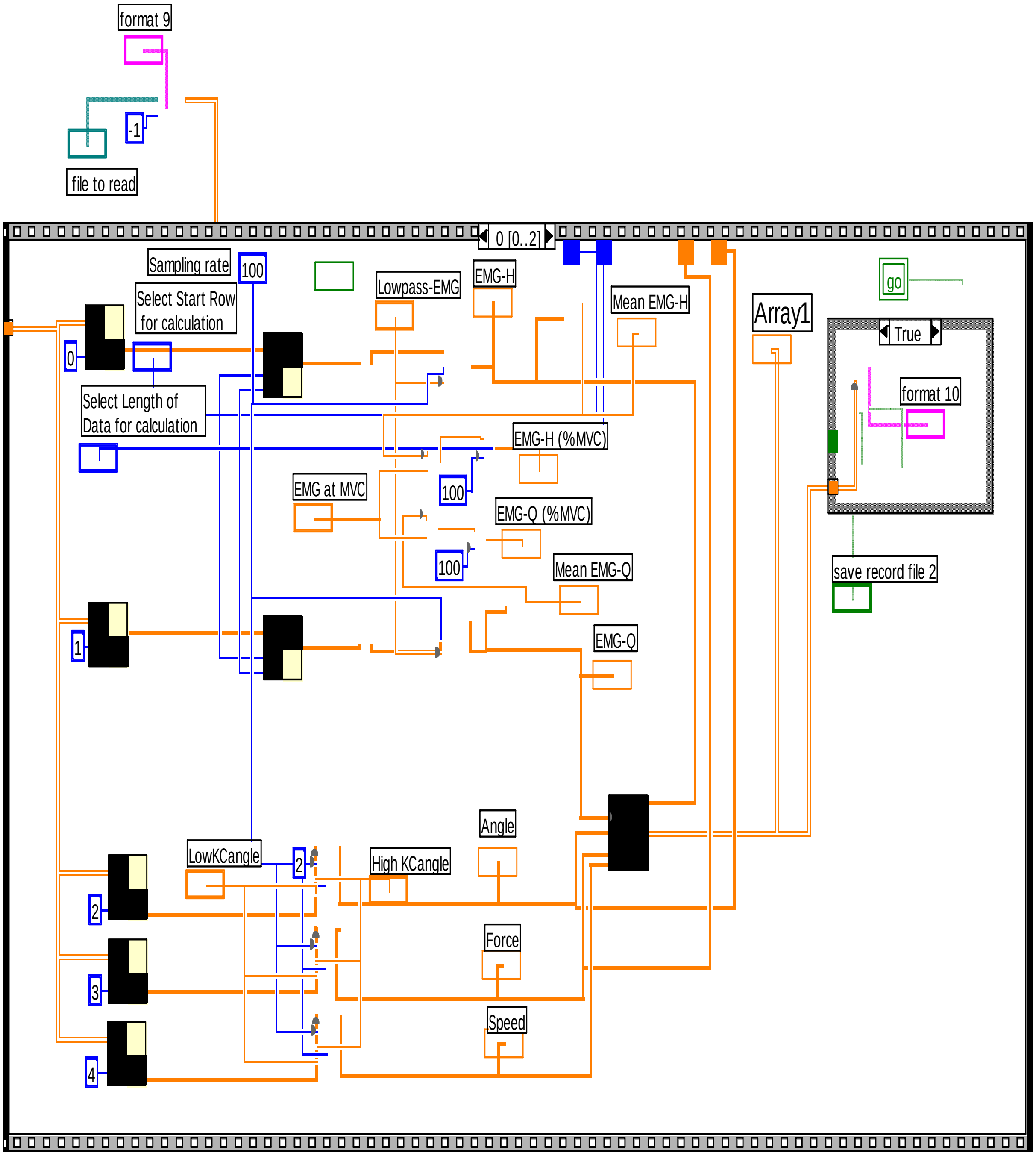


Figure I7. Diagram of the Labview programme for data analysis (passive stiffness and EMG of hamstrings and quadriceps during passive movement) from Kincom dynamometer (page 0).

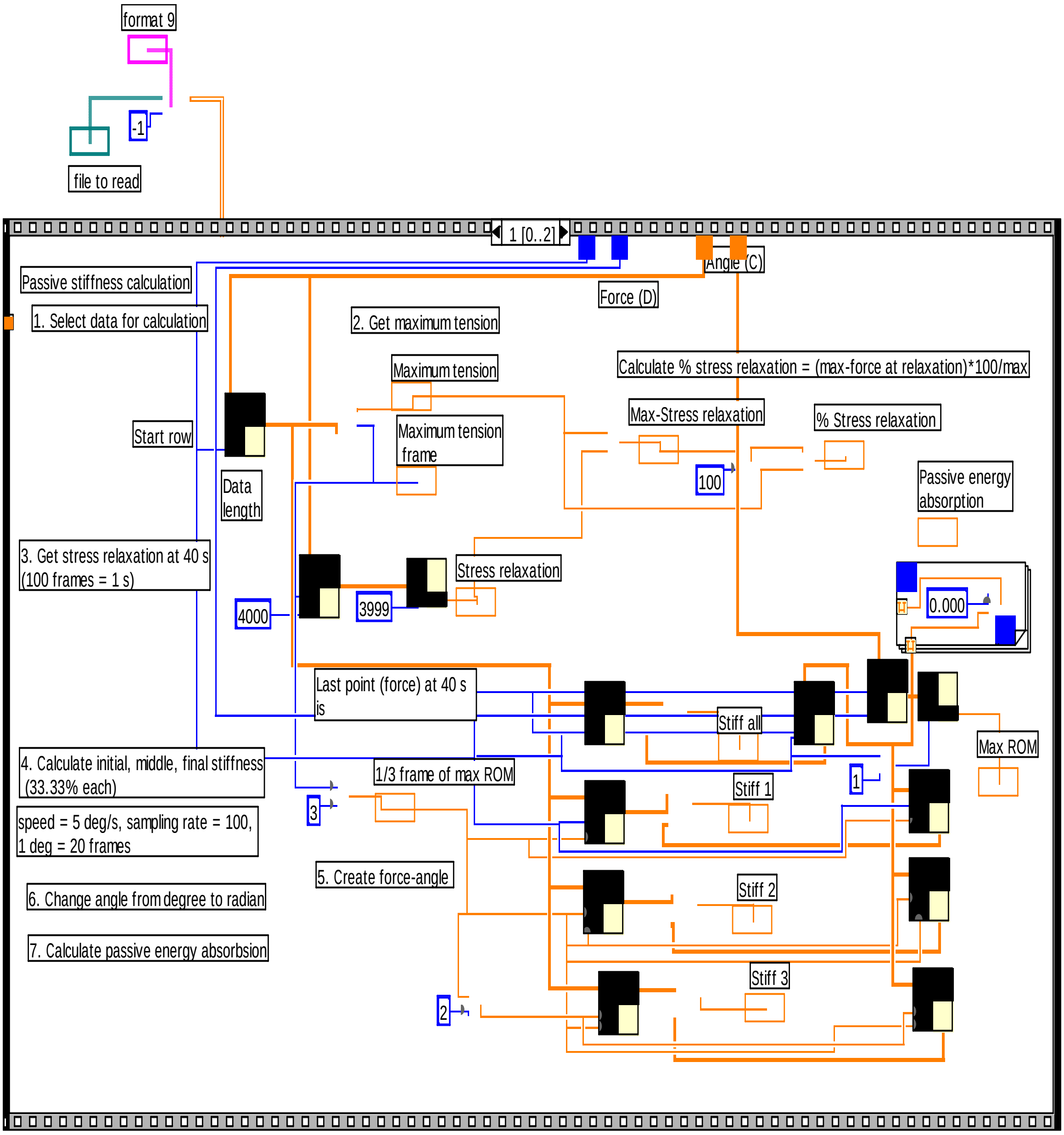
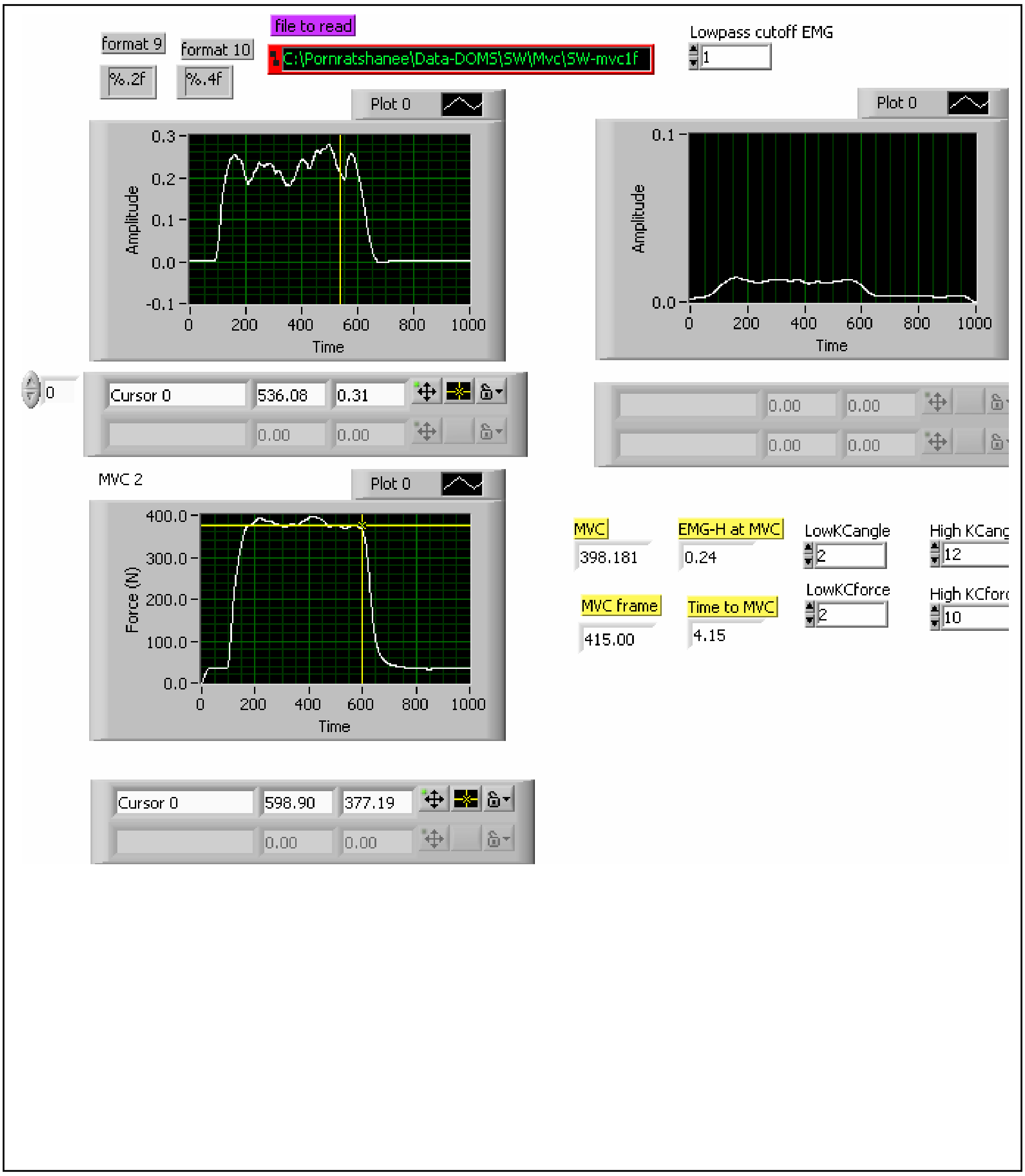


Figure I8. Diagram of the Labview programme for data analysis (passive stiffness and EMG of hamstrings and quadriceps during passive movement) from Kincom dynamometer (page 1).



format 9: %.2f

format 10: %.4f

file to read: C:\Pornratshanee\Data-DOMS\SW\Mvc\SW-mvc1f

Lowpass cutoff EMG: 1

Cursor 0 536.08 0.31

MVC 2

Cursor 0 598.90 377.19

MVC: 398.181

EMG-H at MVC: 0.24

LowKCangle: 2

High KCang: 12

MVC frame: 415.00

Time to MVC: 4.15

LowKCforce: 2

High KCforc: 10

Figure I9. Front panel of the Labview programme for data analysis (maximum isometric contraction and EMG of hamstrings at maximum isometric contraction) from Kincom dynamometer.

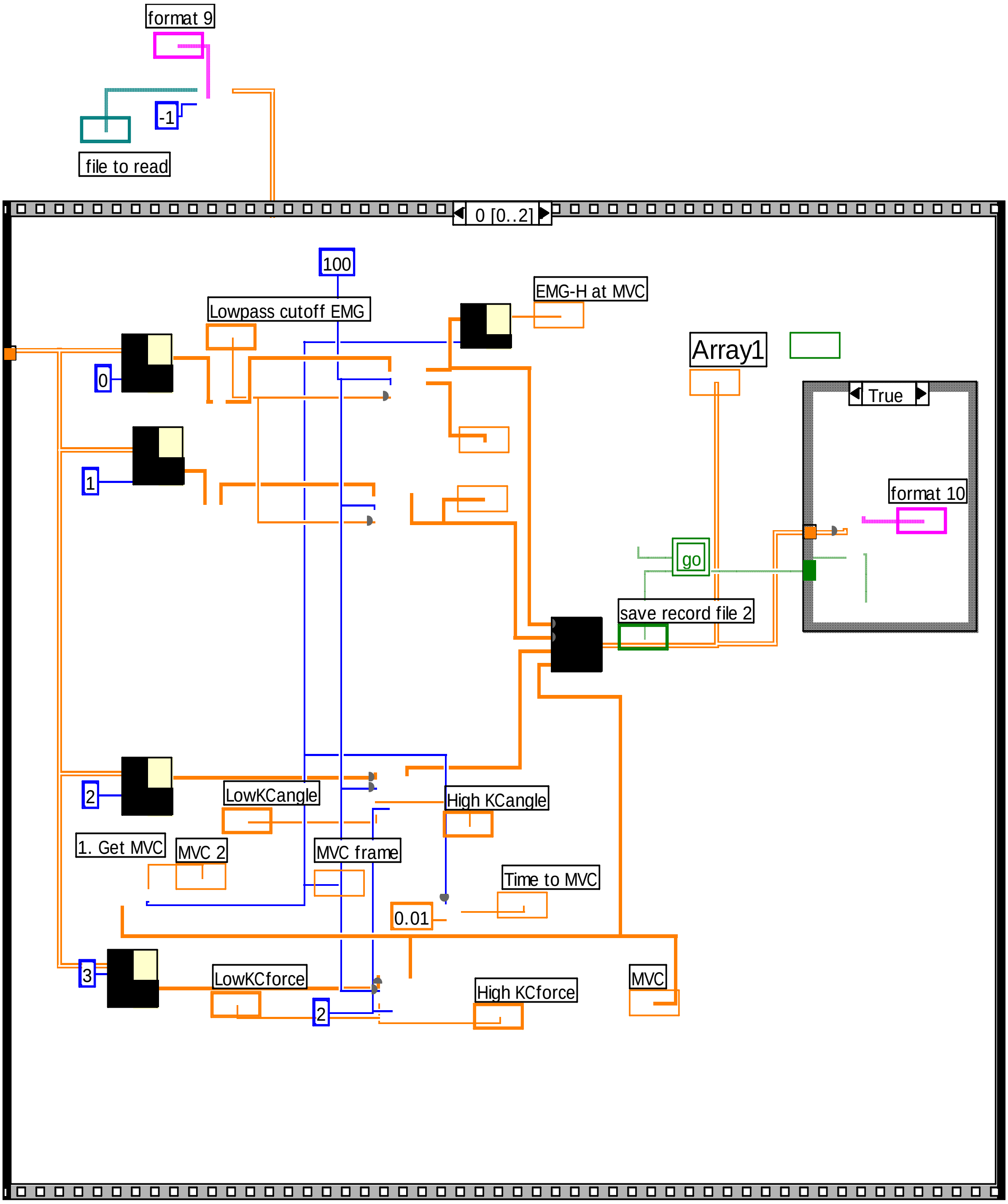
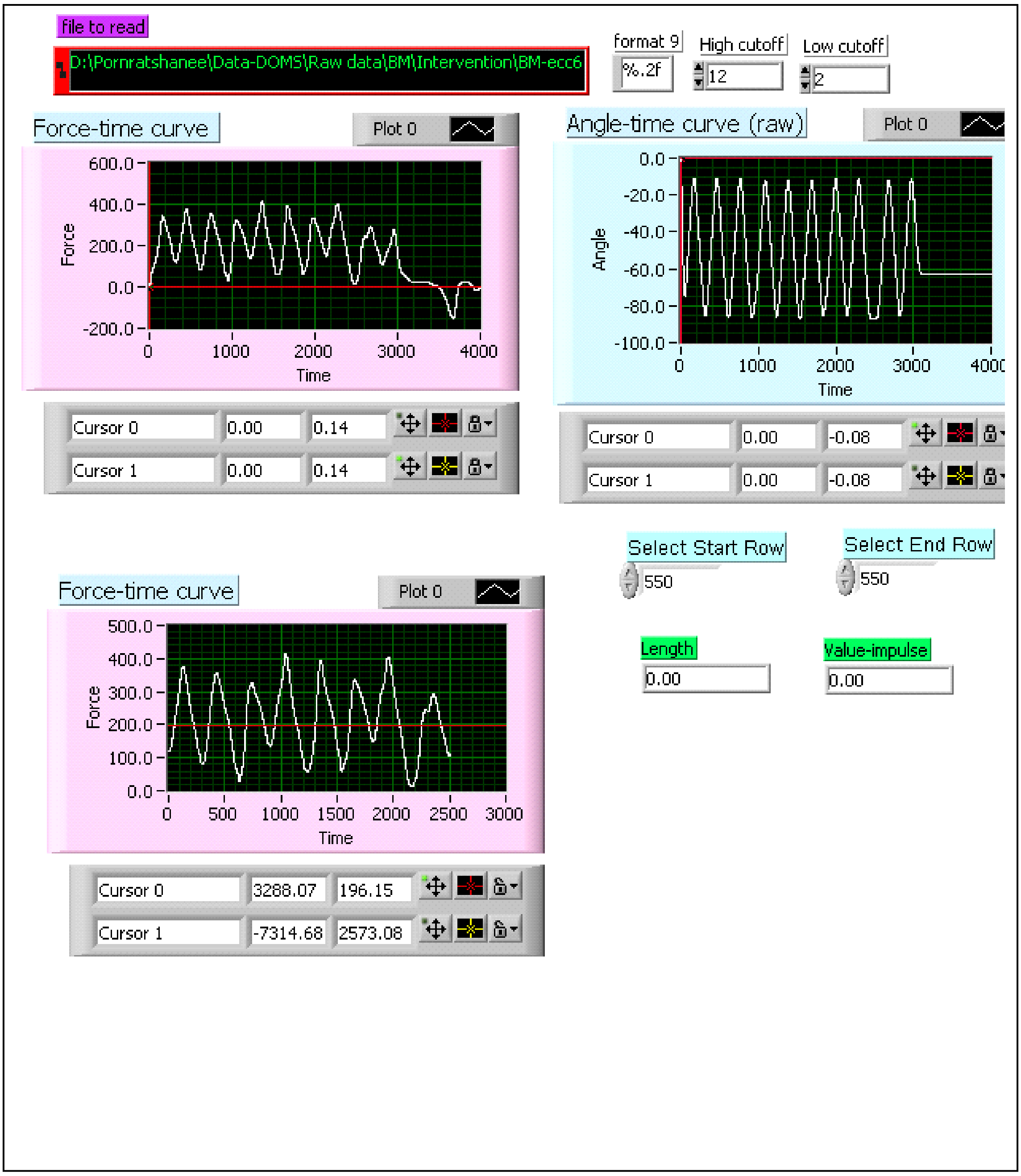


Figure I10. Diagram of the Labview programme for data analysis (maximum isometric contraction and EMG of hamstrings at maximum isometric contraction) from Kincom dynamometer.



File to read: D:\Pornratshanee\Data-DOMS\Raw data\BM\Intervention\BM-ecc6

format 9: %.2f

High cutoff: 12

Low cutoff: 2

Cursor 0 | 0.00 | 0.14

Cursor 1 | 0.00 | 0.14

Cursor 0 | 0.00 | -0.08

Cursor 1 | 0.00 | -0.08

Select Start Row: 550

Select End Row: 550

Length: 0.00

Value-impulse: 0.00

Cursor 0 | 3288.07 | 196.15

Cursor 1 | -7314.68 | 2573.08

Figure I11. Front panel of the Labview programme for data analysis (energy absorption during eccentric exercise) from Kincom dynamometer.

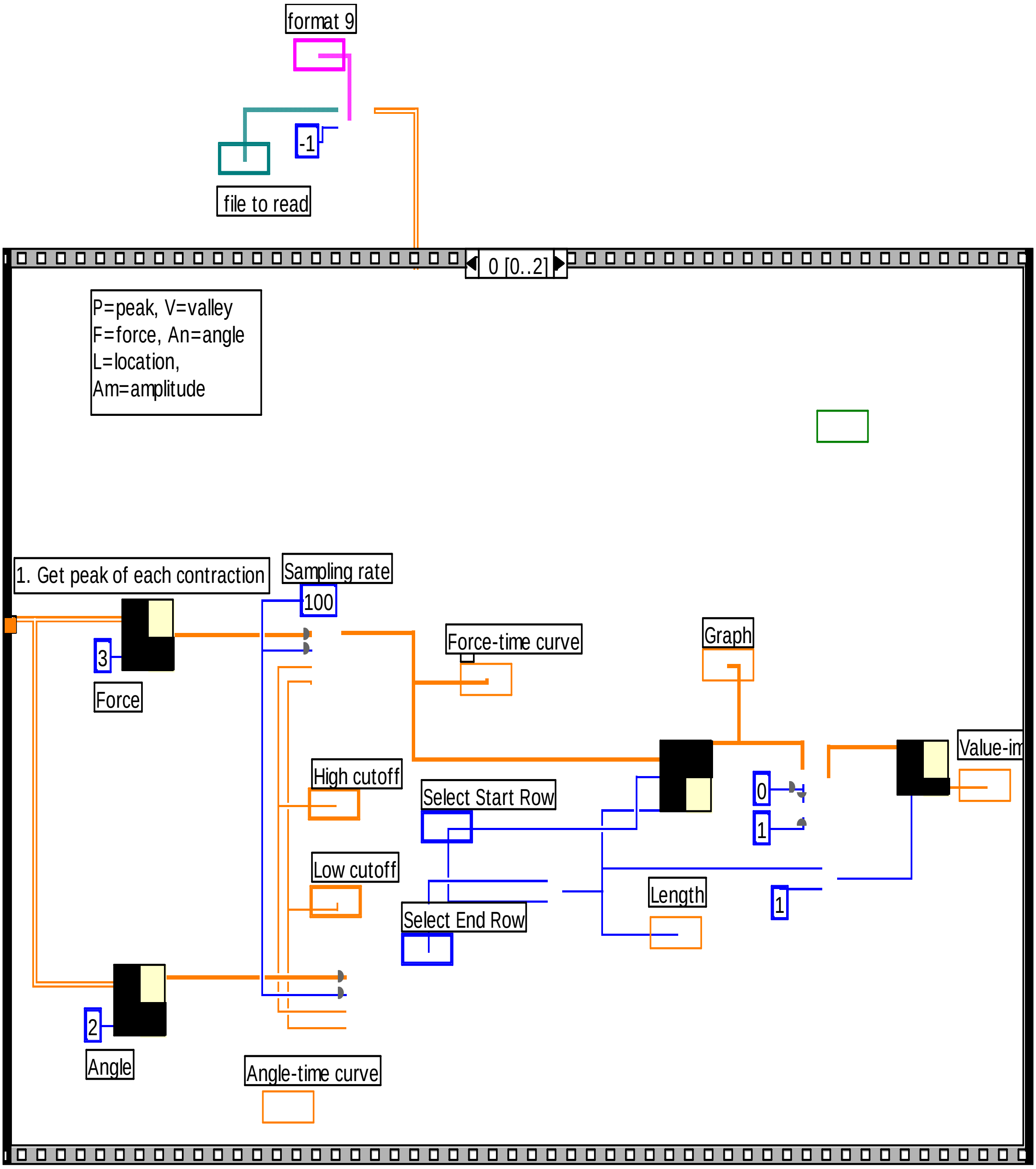


Figure I12. Diagram of the Labview programme for data analysis (energy absorption during eccentric exercise) from Kincom dynamometer.

Appendix J: Physical Activities Recording from the International Physical Activity Questionnaire

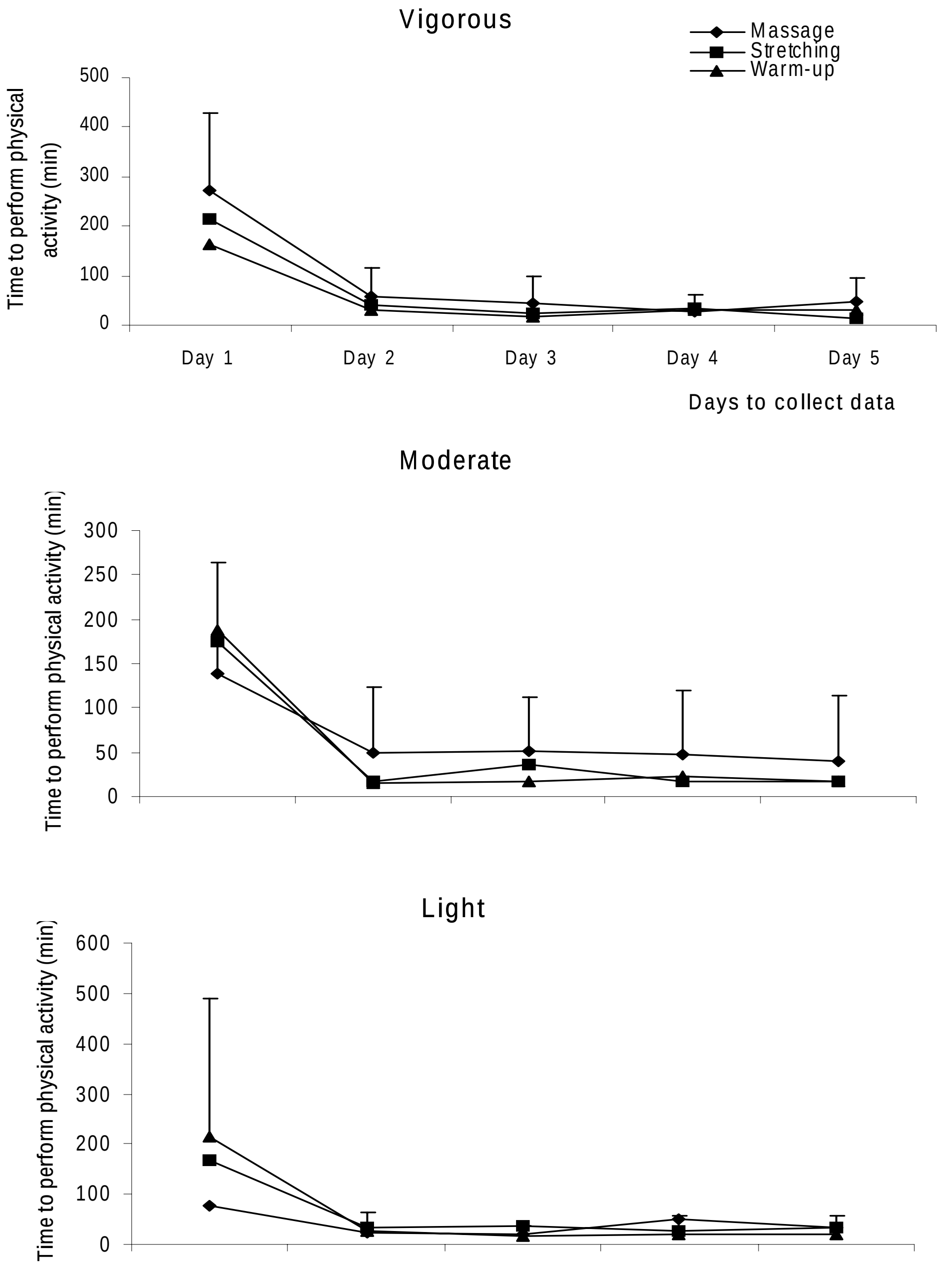


Figure J1. Physical activities recording from the International Physical Activity Questionnaire for five consecutive days during the data collection period. Day 1 presented physical activities (minutes) one week before testing. Day 2 to 5 presented physical activities (minutes) one day before testing.

Appendix K: Area Under Force-Time Curve During Eccentric Exercise

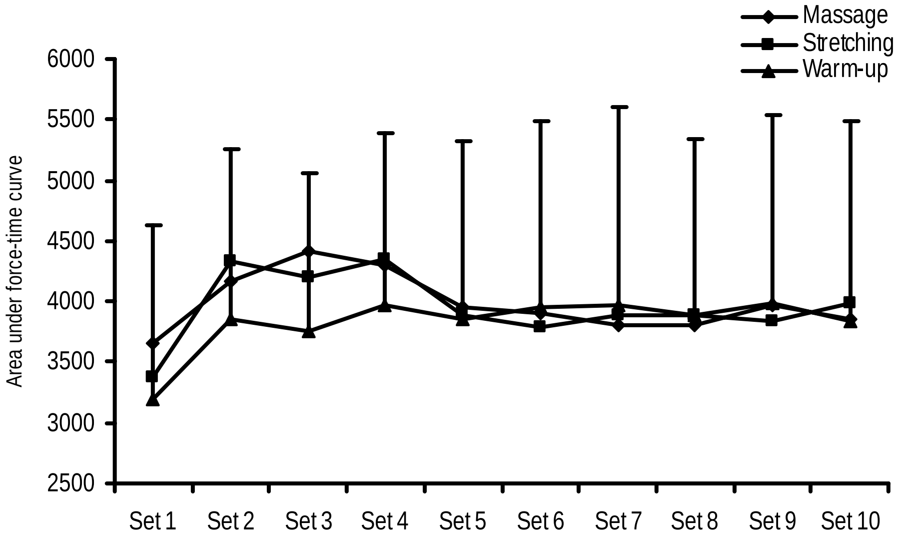


Figure K1. Area under force-time curve during eccentric exercise (80% of maximum voluntary contraction, 10 repetitions per set for 10 sets).

Appendix L: Participant Information Package (Study B)

AUCKLAND UNIVERSITY OF TECHNOLOGY
TE WĀNANGA ARONUI O TAMAKI MAKAU RAU

PARTICIPANT INFORMATION PACKAGE

Contact person:

Pornratshanee Weerapong, PhD Candidate, Auckland University of Technology, Private Bag 92006, Tel: (09) 9179999 x 7848 Email: pornratshanee.weerapong@aut.ac.nz

Title: Preparation strategies for exercise: the effects of warm-up, dynamic stretching, and massage on sport performance.

Introduction:

You are invited to take part in the above mentioned research project. Your participation in this testing is voluntary. You are free to withdraw consent and discontinue participation at anytime without influencing any present and/or future involvement with the Auckland University of Technology.

Your consent to participate in this research will be indicated by your signing and dating the consent form. Signing the consent form indicates that you have freely given your consent to participate, and that there has been no coercion or inducement to participate.

Aim of Study

The project aims to examine the effects of warm-up, dynamic stretching, and manual massage on sport performance (vertical jump and sprint test) in healthy male participants.

Participants

Healthy male, age 20-40years.

Criteria for exclusion

Participants who have previous injuries on leg muscles and/or have musculoskeletal problems.

.Location

Sport Performance Research Centre, Millennium Institute of Sports & Health, Albany

Time

By participating in this project you will be required to give up one hour per day for four days for four consecutive weeks.

Methods

Data will be collected during both the experimental and control conditions using force plate form for jumping height and ground reaction force and photoelectric cell for sprint time.

You will receive one of these treatments (warm-up, dynamic stretching, and massage) each day.

You will have measured jump height, sprint time, and kicking speed for three times; immediately after intervention, ten and 20 minutes post intervention.

The information collected will allow us to determine if determine if preexercise activities (warm-up, dynamic stretching, and massage) can increase sport performance (vertical jump, sprint test, and kicking speed).

All information that is collected will be kept confidential at all time. At no time will your data be made available to people outside of the research team. No material that could personally identify you will be used in any reports on this study. The data will be stored in a locked cabinet for a period of ten years following data collection.

Benefits of the study

This project will provide potential benefits for the selection of proper preexercise activities that will help to enhance performance.

Possible risks of the study

There is a possible injury, however this risk is an equivalent to that for normal participation in physical training.

Taking part in this research will not cost you.

Results

You will receive an individualised report and a copy of the final report (with no participants identified).

Any concerns regarding the nature of this project should be notified in the first instance to Patria Hume, the Project Supervisor. If you have any other questions please feel free to contact Patria Hume or Pornratshanee Weerapong at any time.

Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEC, Madeline Banda, madeline.banda@aut.ac.nz, 917 9999 ext 8044.

Approved by the Auckland University of Technology Ethics Committee on

12 December 2002 AUTEC Reference number 02/172

Appendix M: Consent to Participation in Research (Study B)

Consent to Participation in Research

Title of Project: Preparation strategies for exercise: the effects of warm-up, dynamic stretching, and massage on performance.

Project Supervisor: Associate Professor Patria Hume (PhD)

Researcher: Pornratshanee Weerapong (MSc), Auckland University of Technology Private Bag 92006, Auckland. Phone: 9179999 ext 7848

- I have read and understood the information provided about this research project.
- I have had an opportunity to ask questions and to have them answered.
- I understand that I may withdraw myself or any information that I have provided for this project at any time prior to completion of data collection, without being disadvantaged in any way.
- I agree to take part in this research.

Participant signature:..

Participant name:…………………………………………….

Date: …………………………………….

Project Supervisor Contact Details: Associate Professor Patria Hume (PhD)

Auckland University of Technology

Private Bag 92006, Auckland.

Phone: 9179999 ext 7306

Approved by the Auckland University of Technology Ethics Committee on 12 December 2002
AUTEC Reference number 02/172

Appendix N: Approval Letter for the Main Study (Study B) from the Auckland University of Technology Ethics Committee

MEMORANDUM

To: Patria Hume

From: Madeline Banda

Date: 12 December 2002

Subject: 02/172 Preparation strategies for exercise: The effects of warm up, stretching, and massage on performance

Dear Patria

- Your application for ethics approval was considered by AUTEC at their meeting on 09/12/12.
- Your application was approved for a period of two years until December 2004.
- You are required to submit the following to AUTEC:
- A brief annual progress report indicating compliance with the ethical approval given.
- A brief statement on the status of the project at the end of the period of approval or on completion of the project, whichever comes sooner.
- A request for renewal of approval if the project has not been completed by the end of the period of approval.

Please note that the Committee grants ethical approval only. If management approval from an institution/organisation is required, it is your responsibility to obtain this.

The Committee wishes you well with your research.

Please include the application number and study title in <u>all</u> correspondence and telephone queries.

Yours sincerely

Madeline Banda (Executive Secretary)

Appendix O: Data Collection Sheet (Study B)

Participant characteristics

Name…………………….……..… Date………………….. Time ……….……
Date of birth……………..… Occupation……………………..Weight………kg
Height………..cm

%BF (Triceps………./…………/………., Subscapularis……/…………/……….,
Supraspinal………./………/………., Abdominal……..…./………/……….)

Physical activity (please indicate) 1)………………… 2)……………………………
Frequency. ………hr/week for…………. month (s)/year(s)
medication…………………………

Parameters	Trials	Day 1 Weight………. Familiarisation	Day 2 Weight………..	Day 3 Weight……….	Day 4 Weight……
Immediately after intervention					
Jumping	A				
	B				
	C				
Sprinting	A				
	B				
	C				
10 min after intervention					
Jumping	D				
	E				
	F				
Sprinting	D				
	E				
	F				
20 min after intervention					
Jumping	G				
	H				
	I				
Sprinting	G				
	H				
	I				
Thank you.					

Appendix P: Pilot Results from Study B

Pilot testing was conducted during June 2002. One male participant completed the reliability test for three days. Within each day, the participant performed 3-jumping, 3-kicking, and 3-sprinting tests in order. Then, the participant repeated these performances again 10 minutes and 20 minutes after the first trial. The experimental protocol was repeated for three days. The means and standard deviations of each parameter are presented in Table P1. This pilot study aimed to test the possibility of the experimental protocol.

Table P1

Means and standard deviations (*SD*) for jumping, kicking and sprinting outcome parameters for one participant

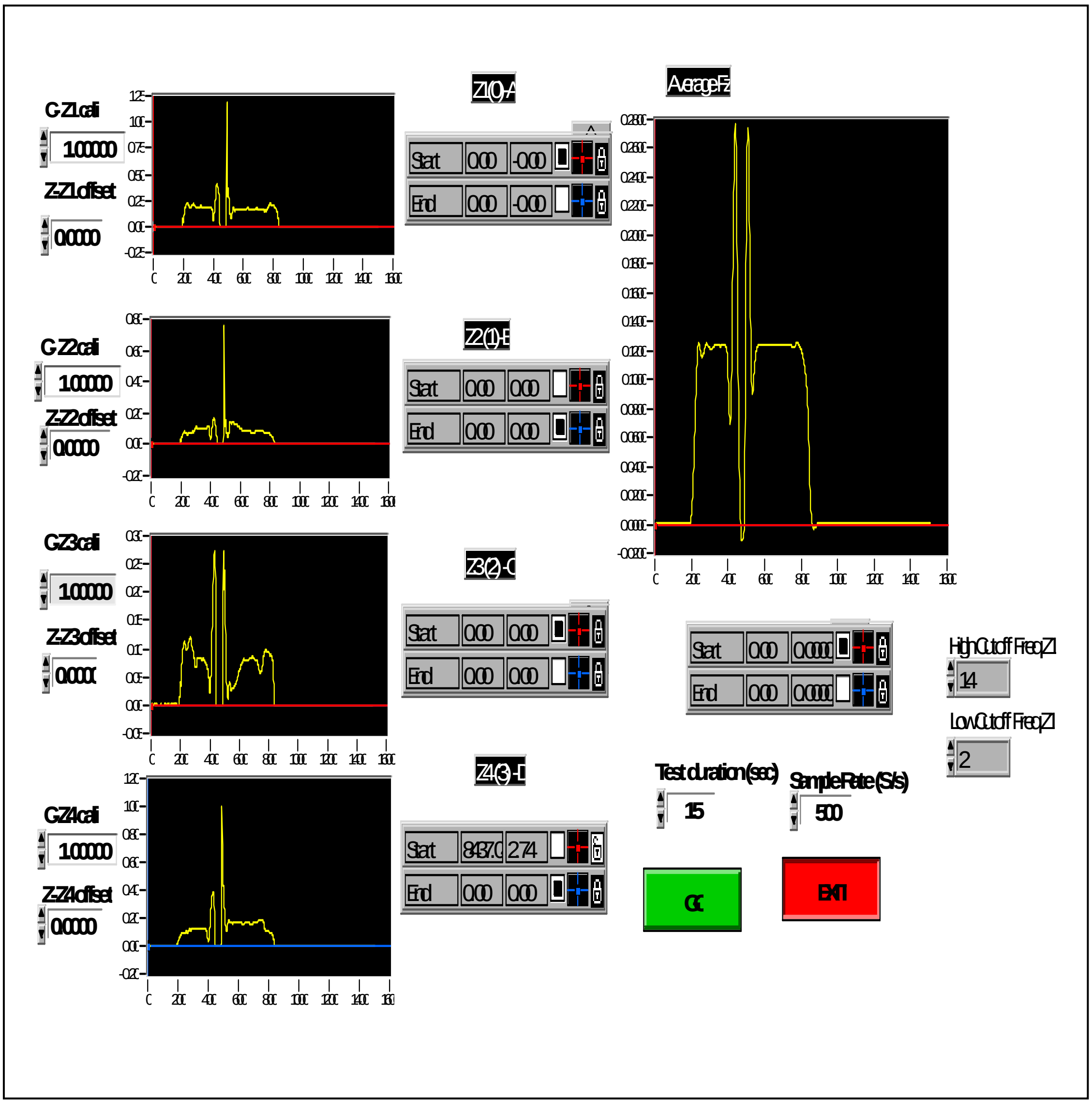
Parameters	Jumping												Kicking (m/s)			Sprinting (s)		
Day	Impulse (N.s)			Velocity (m/s)			Height (m)			Work done (J)								
Trials	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
1	158.8	160.8	167.7	2.2	2.3	2.3	0.25	0.26	0.28	18.0	18.5	20.1	29.8	30.0	31.8	1.91	1.97	1.95
2	163.4	167.0	166.7	2.3	2.3	2.3	0.27	0.28	0.28	19.1	20.0	19.9	29.0	30.7	30.6	1.92	1.98	1.94
3	156.9	168.4	175.1	2.2	2.4	2.5	0.25	0.28	0.31	17.6	20.3	22.0	29.3	31.5	31.0	1.91	1.91	1.94
4	170.2	164.0	169.2	2.4	2.3	2.4	0.29	0.27	0.29	20.8	19.3	20.6	27.3	29.6	31.3	1.9	1.92	1.94
5	170.8	174.6	171.0	2.4	2.5	2.4	0.29	0.31	0.29	20.9	21.8	20.9	30.5	33.4	31.1	1.88	1.97	1.87
6	162.0	168.0	169.4	2.3	2.4	2.4	0.26	0.28	0.29	18.8	20.2	20.5	30.8	33.2	31.5	1.87	1.91	1.87
7	165.9	169.5	174.7	2.3	2.4	2.5	0.28	0.29	0.31	19.7	20.6	21.8	27.9	33.3	30.3	-	1.94	1.86
8	170..4	172.0	179.5	2.4	2.4	2.5	0.29	0.30	0.30	20.8	21.2	23.1	28.0	33.1	30.4	-	1.98	1.88
9	164.7	169.9	170.1	2.3	2.4	2.4	0.27	0.29	0.29	19.42	20.7	20.8		33.4	29.8	-	1.98	1.88
Mean	164.8	168.3	171.5	2.3	2.4	2.4	0.27	0.28	0.29	19.5	20.3	21.1	29.1	32.0	30.9	1.90	1.95	1.90
SD	5.1	4.1	4.1	0.1	0.1	0.1	0.02	0.02	0.01	1.2	1.0	1.0	1.2	1.6	0.4	0.02	0.03	0.04

Appendix Q: LabView for Data Collection and Data Analysis (Study B)

There were five Labview programme used to collect and analyse data from the force platform in Study B. In each of these LabView programme, 60% of work is my own work, 20% is that of Associate Professor Patria Hume, and 20% is that of Karoline Cheung.

Figures Q1 and Q2 showed the front panel and diagram, respectively, of the Labview programme for data collection (jump height). Figures Q3 and Q4 showed the front panel and diagram, respectively, of the Labview programme for data analysis (jump height). Figures Q5 and Q6 showed the front panel and diagram, respectively, of the Labview programme for data analysis (leg stiffness during countermovement jump).

There were also two Labview programmes used to analyse data digitised from SiliconCoach programme in Study B. Figures Q7 and Q8 showed the front panel and diagram, respectively, of the Labview programme for data analysis (changes of center of mass). Figures Q9 and Q10 showed the front panel and diagram, respectively, of the Labview programme for data analysis (changes of range of motion).



G-Z1cali 100000

Z-Z1offset 00000

G-Z2cali 100000

Z-Z2offset 00000

G-Z3cali 100000

Z-Z3offset 0000(

G-Z4cali 100000

Z-Z4offset 00000

Test duration (sec) 15

Sample Rate (S/s) 500

High Cutoff Freq Z1 14

Low Cutoff Freq Z1 2

Figure Q1. Front panel of the Labview programme for data collection from force platform.

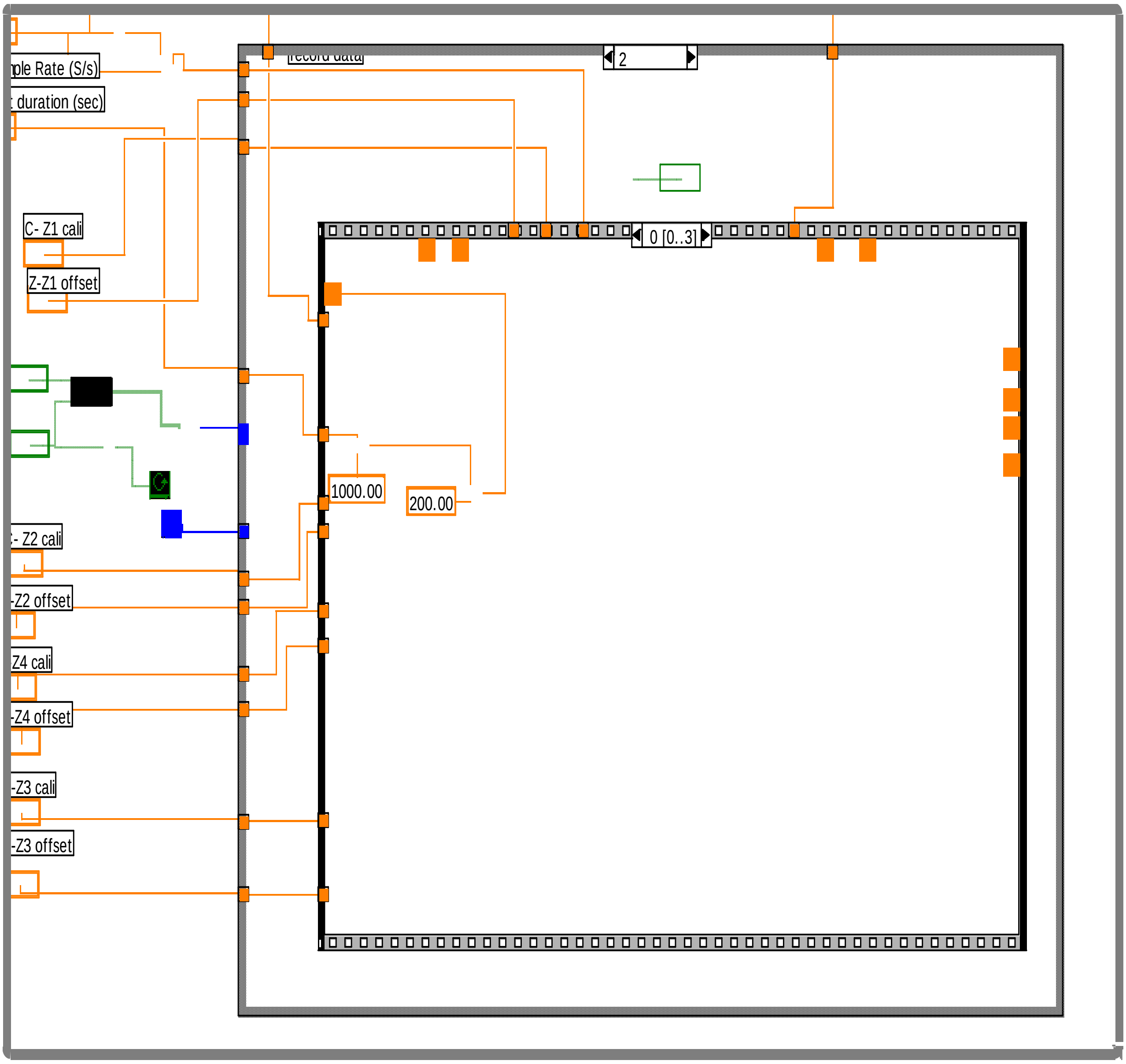


Figure Q2. Diagram of the Labview programme for data collection from force platform.

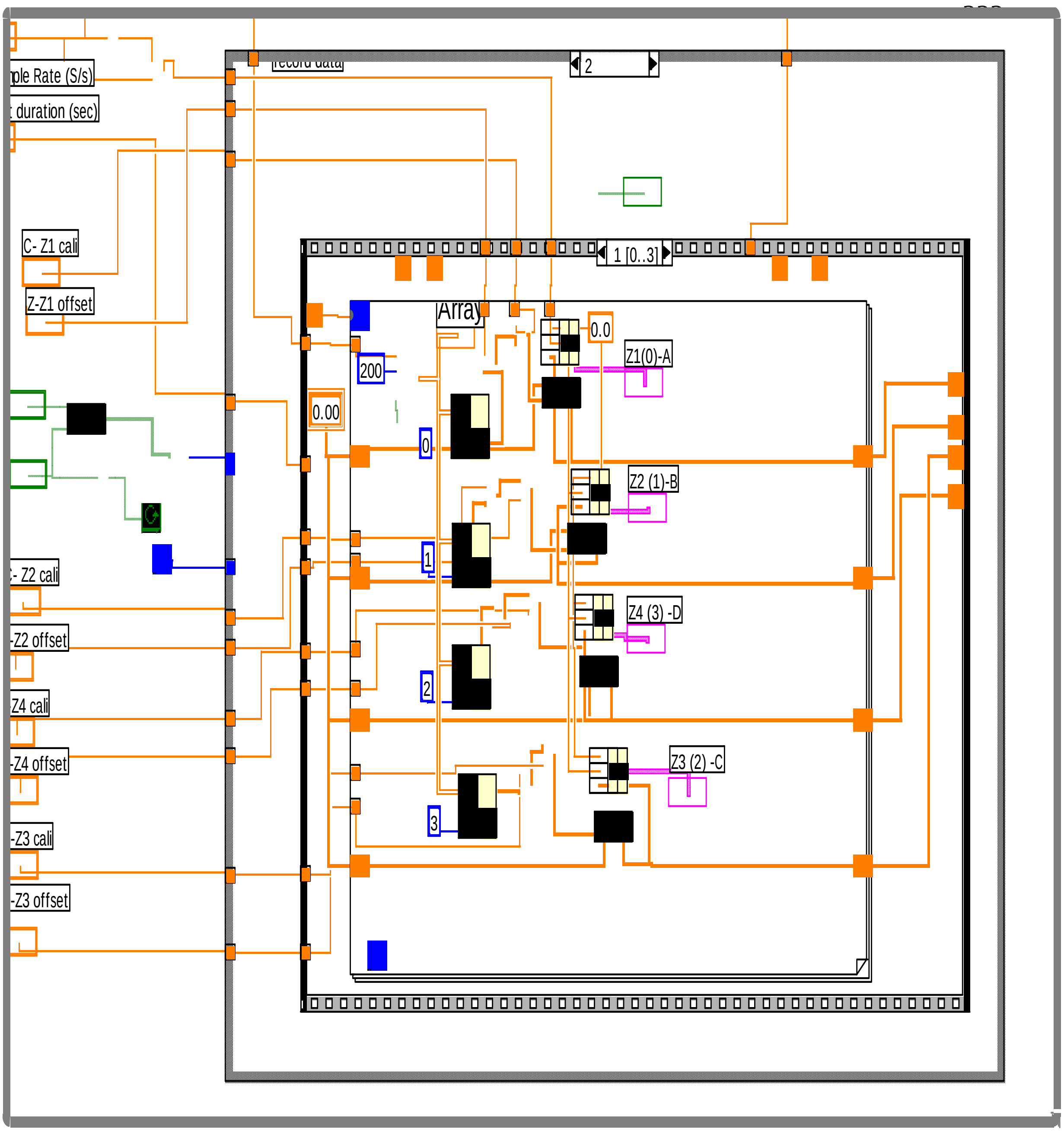


Figure Q3. Diagram of the Labview programme for data collection from force platform (page 1).

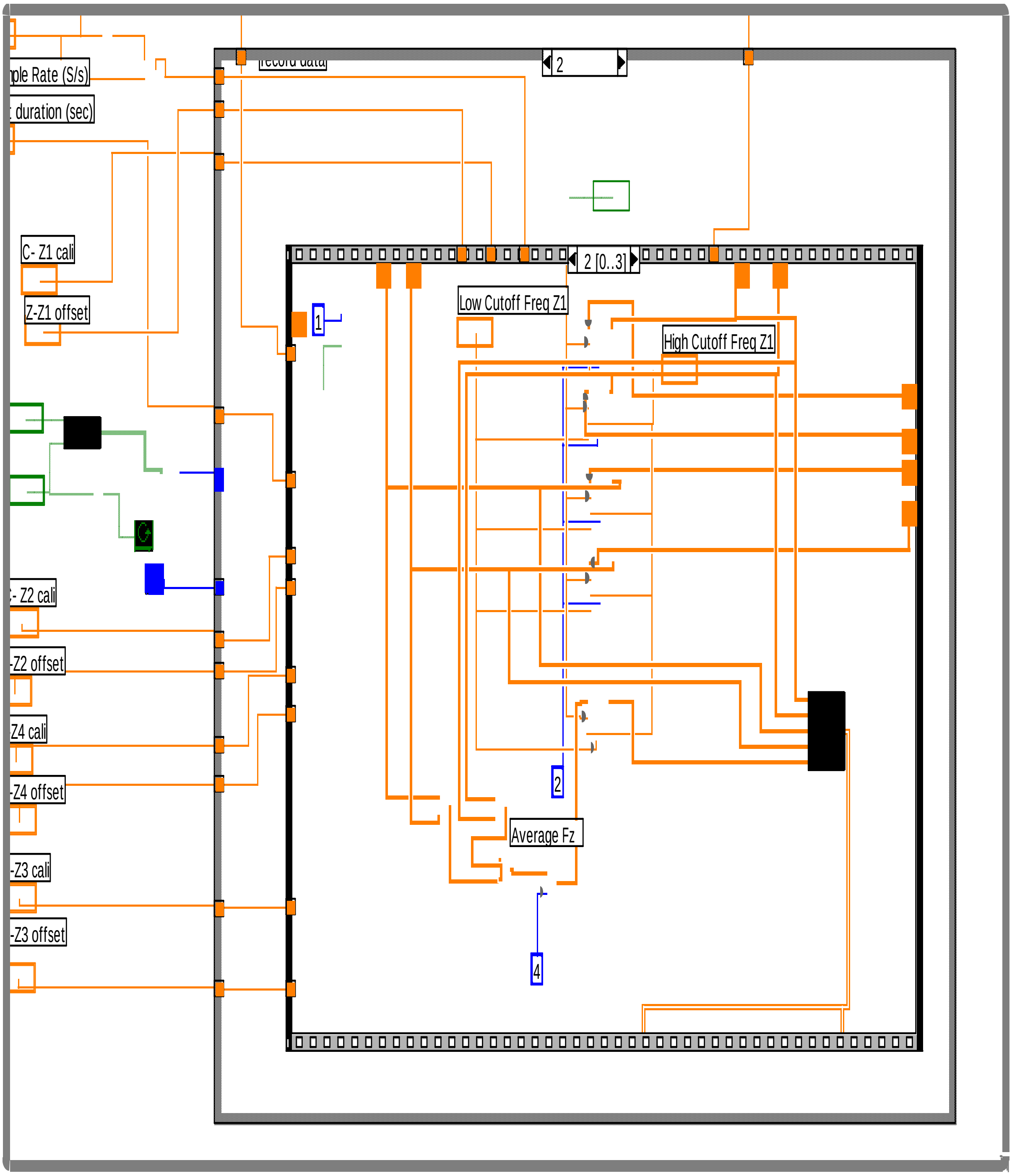


Figure Q4. Diagram of the Labview programme for data collection from force platform (page 2).

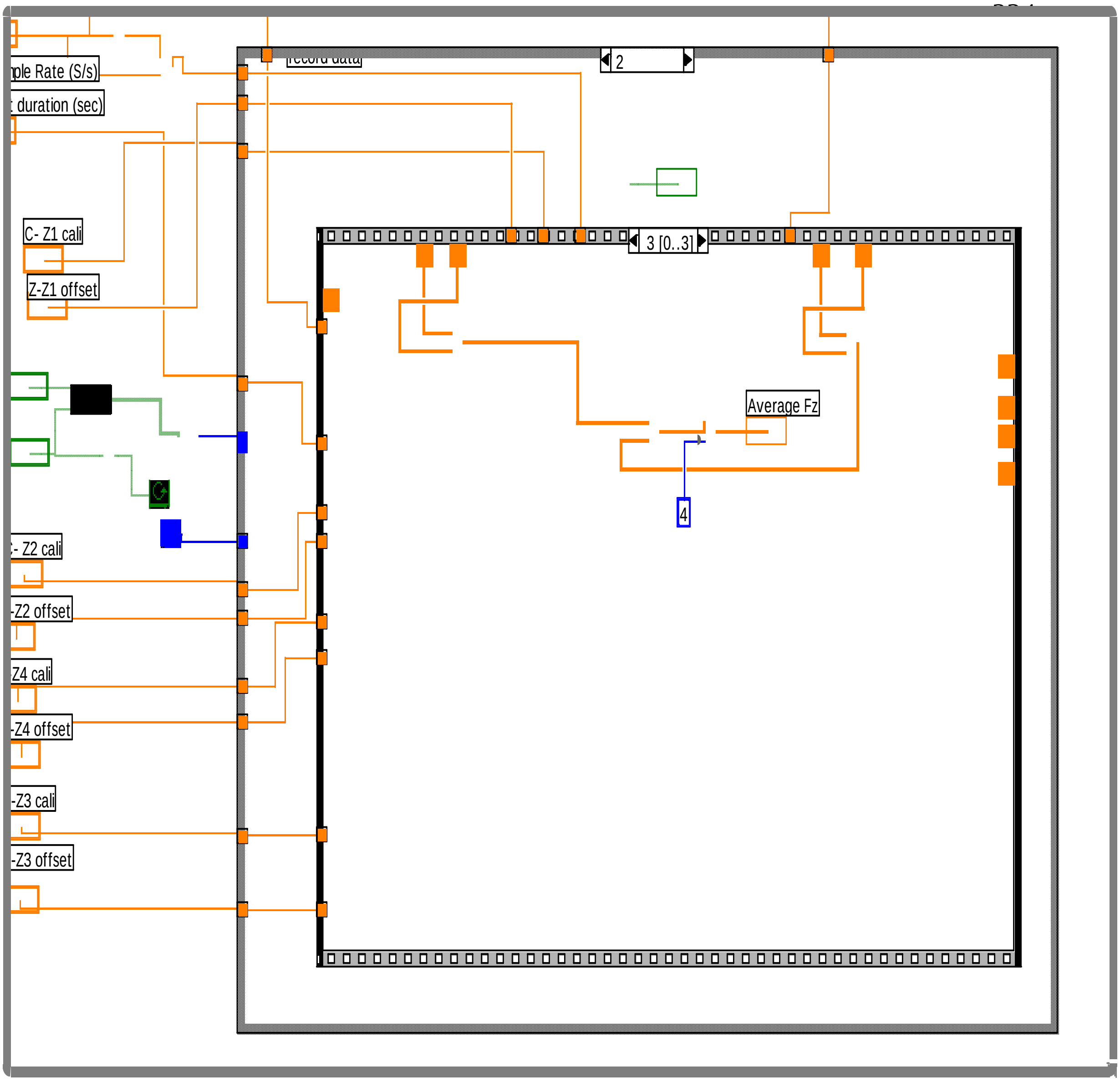
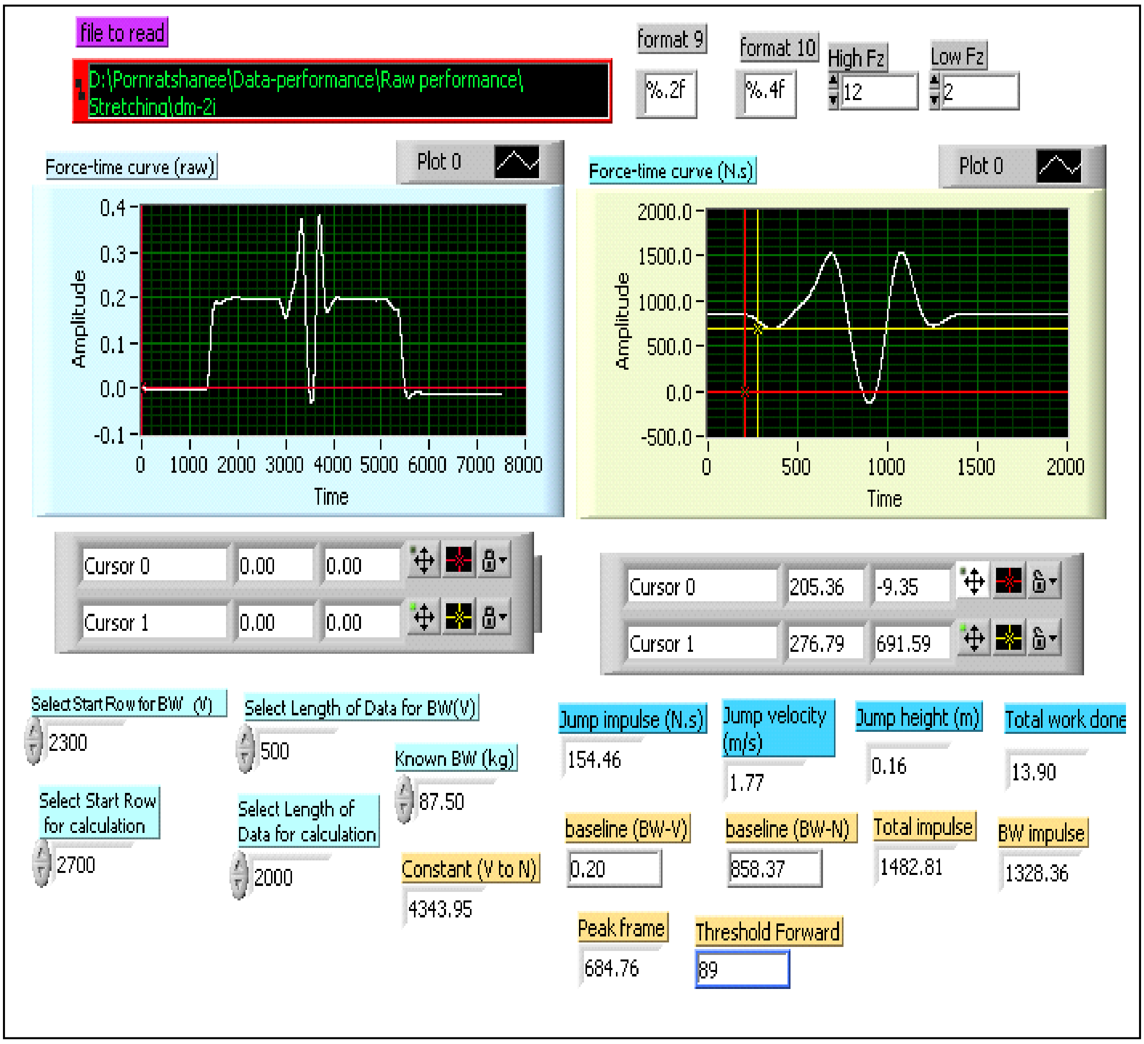


Figure Q5. Diagram of the Labview programme for data collection from force platform (page 3).



file to read: D:\Pornratshanee\Data-performance\Raw performance\Stretching\dm-2i

format 9: %.2f

format 10: %.4f

High Fz: 12

Low Fz: 2

Cursor 0	0.00	0.00
Cursor 1	0.00	0.00

Cursor 0	205.36	-9.35
Cursor 1	276.79	691.59

Select Start Row for BW (V): 2300

Select Length of Data for BW(V): 500

Select Start Row for calculation: 2700

Select Length of Data for calculation: 2000

Known BW (kg): 87.50

Constant (V to N): 4343.95

Jump impulse (N.s): 154.46

Jump velocity (m/s): 1.77

Jump height (m): 0.16

Total work done: 13.90

baseline (BW-V): 0.20

baseline (BW-N): 858.37

Total impulse: 1482.81

BW impulse: 1328.36

Peak frame: 684.76

Threshold Forward: 89

Figure Q6. Front panel of the Labview programme for data analysis (jump height) from force platform.

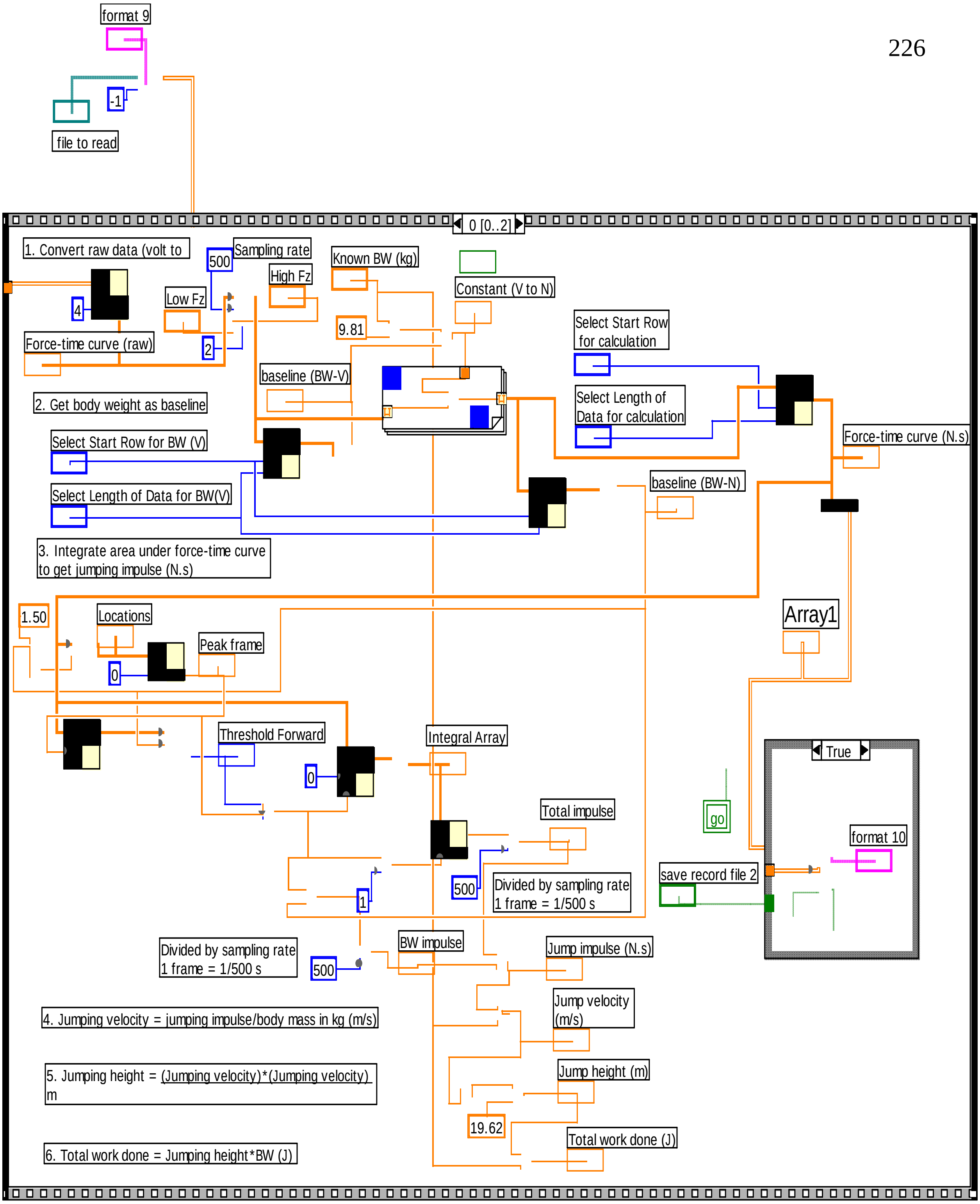
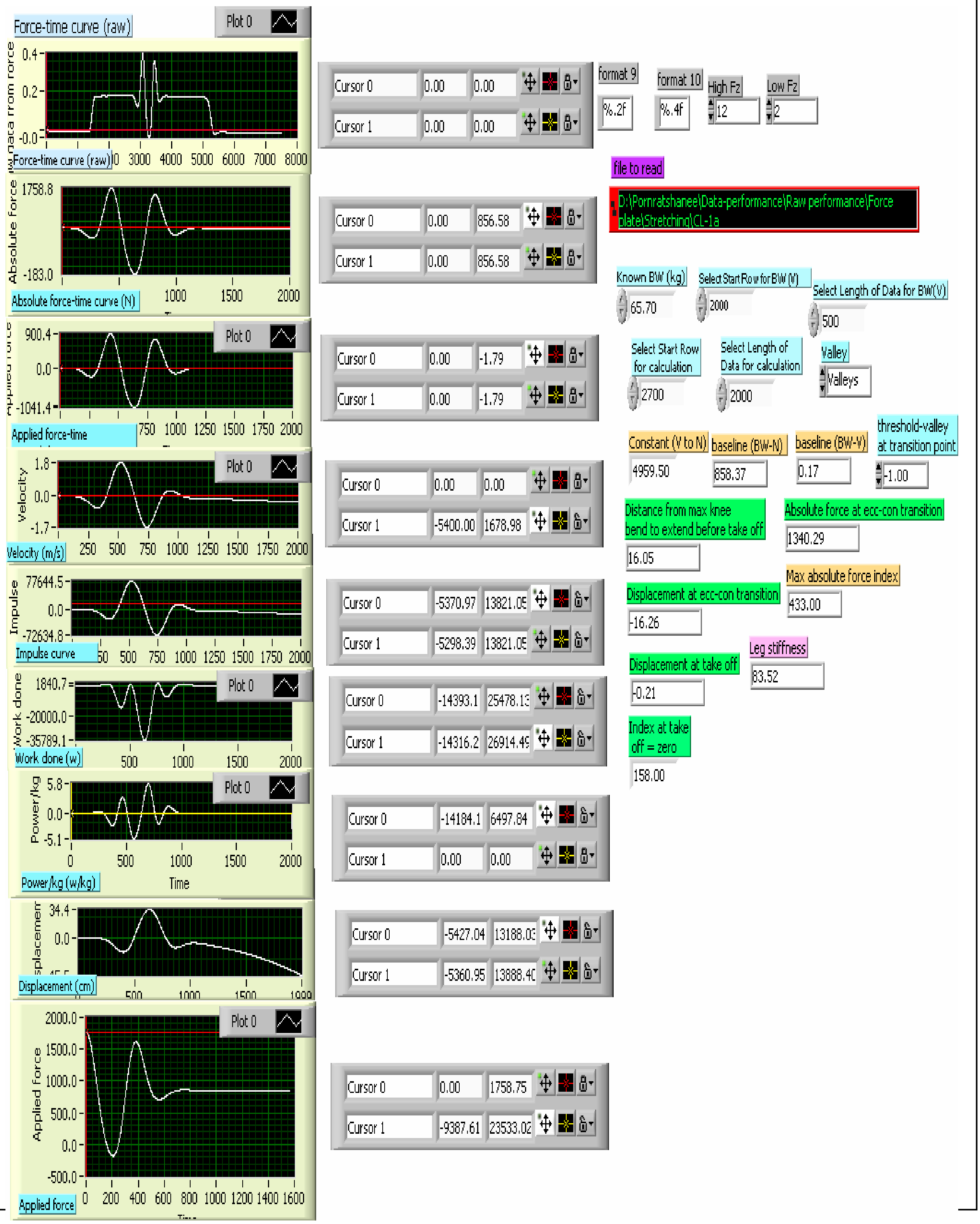


Figure Q7. Diagram of the Labview programme for data analysis (jump height) from force platform.



Cursor 0	0.00	0.00
Cursor 1	0.00	0.00

Cursor 0	0.00	856.58
Cursor 1	0.00	856.58

Cursor 0	0.00	-1.79
Cursor 1	0.00	-1.79

Cursor 0	0.00	0.00
Cursor 1	-5400.00	1678.98

Cursor 0	-5370.97	13821.05
Cursor 1	-5298.39	13821.05

Cursor 0	-14393.1	25478.1
Cursor 1	-14316.2	26914.4

Cursor 0	-14184.1	6497.84
Cursor 1	0.00	0.00

Cursor 0	-5427.04	13188.0
Cursor 1	-5360.95	13888.4

Cursor 0	0.00	1758.75
Cursor 1	-9387.61	23533.02

format 9: %.2f

format 10: %.4f

High Fz: 12

Low Fz: 2

file to read: D:\Pornratshanee\Data-performance\Raw performance\Force plate\Stretching\CL-1a

Known BW (kg): 65.70

Select Start Row for BW (V): 2000

Select Length of Data for BW(V): 500

Select Start Row for calculation: 2700

Select Length of Data for calculation: 2000

Valley: Valleys

Constant (V to N): 4959.50

baseline (BW-N): 858.37

baseline (BW-V): 0.17

threshold-valley at transition point: -1.00

Distance from max knee bend to extend before take off: 16.05

Absolute force at ecc-con transition: 1340.29

Max absolute force index: 433.00

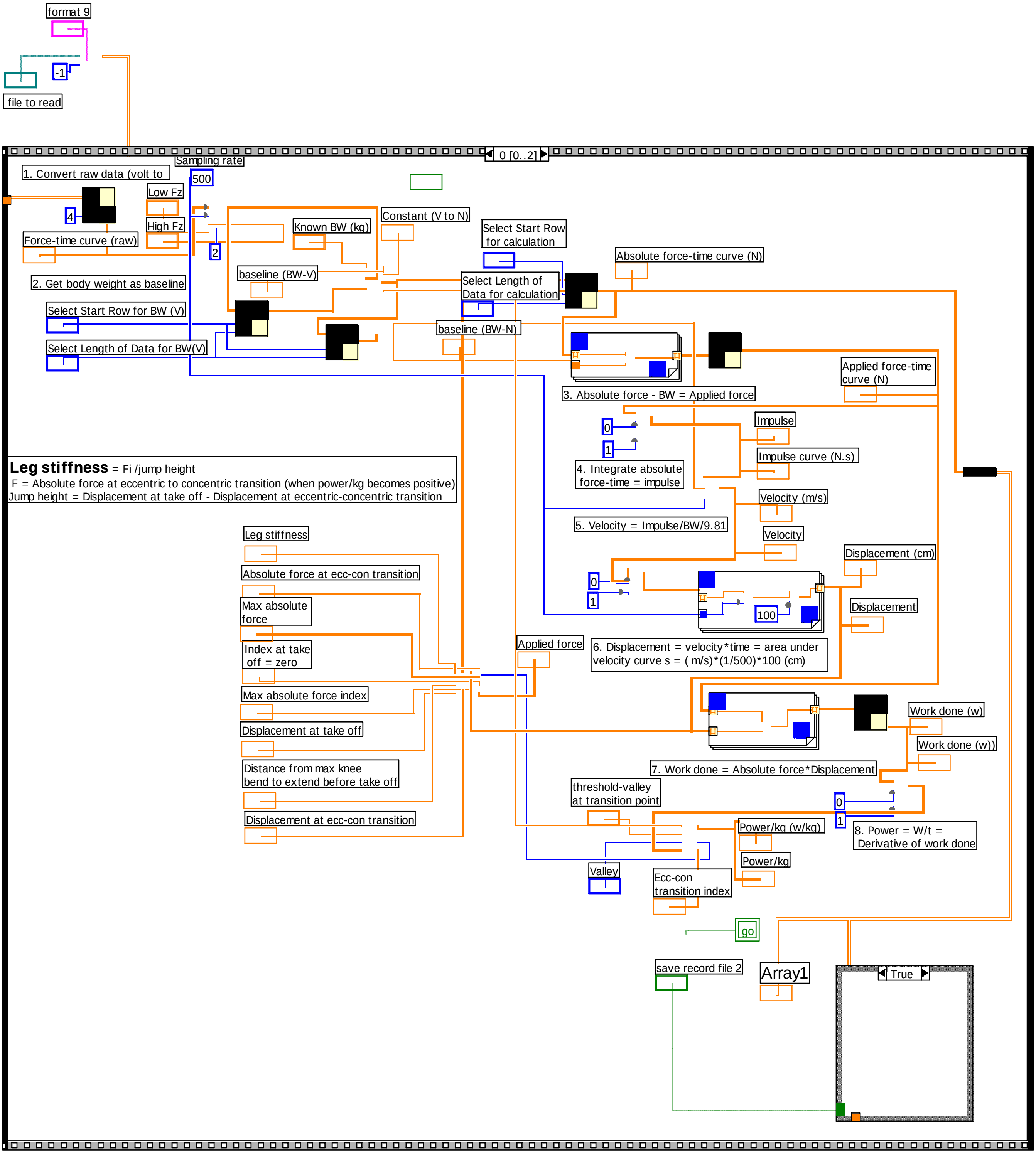
Displacement at ecc-con transition: -16.26

Leg stiffness: 83.52

Displacement at take off: -0.21

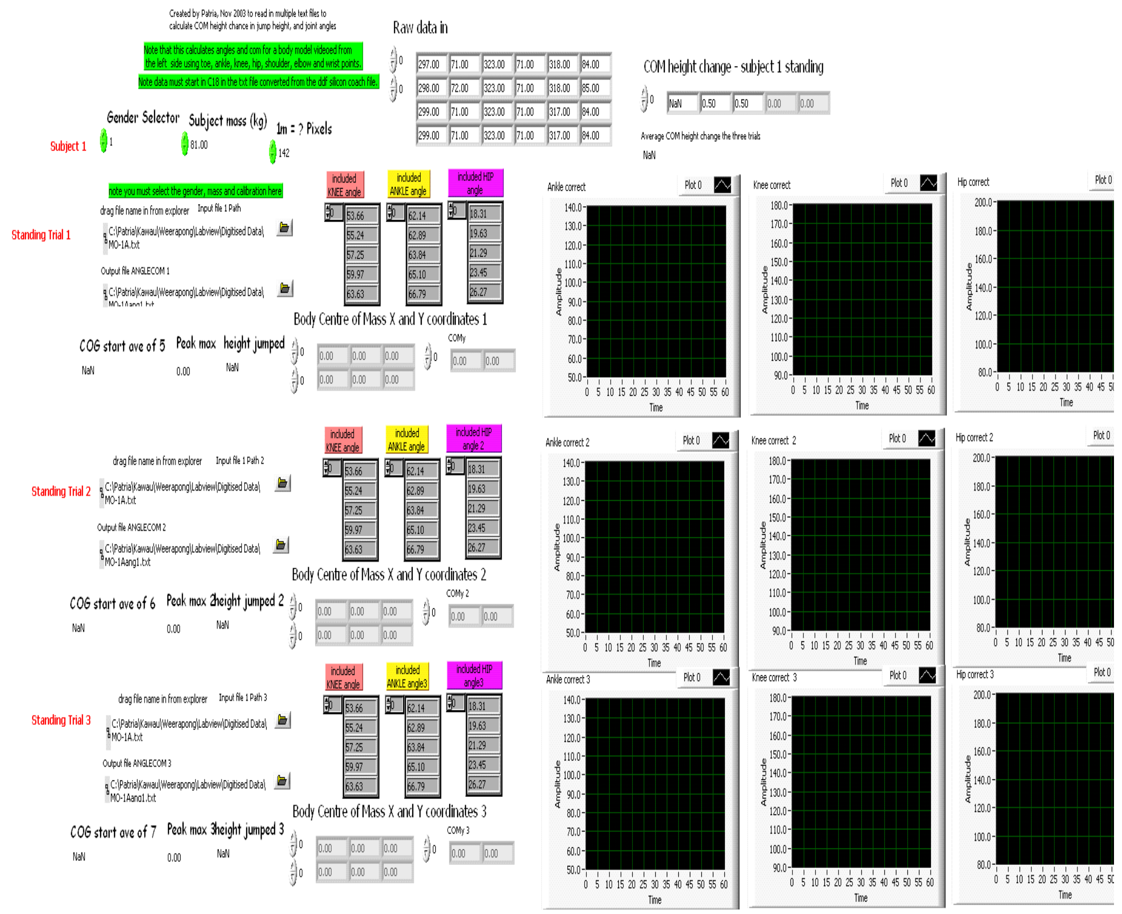
Index at take off = zero: 158.00

Figure Q8. Front panel of the Labview programme for data analysis (leg stiffness during countermovement jump) from force platform.



Leg stiffness = Fi /jump height
F = Absolute force at eccentric to concentric transition (when power/kg becomes positive)
Jump height = Displacement at take off - Displacement at eccentric-concentric transition

Figure Q9. Diagram of the Labview programme for data analysis (leg stiffness during countermovement jump) from force platform.



Created by Patria, Nov 2003 to read in multiple text files to calculate COM height chance in jump height, and joint angles

Note that this calculates angles and com for a body model videoed from the left side using toe, ankle, knee, hip, shoulder, elbow and wrist points.

Note data must start in C18 in the txt file converted from the ddf silicon coach file.

Raw data in

297.00	71.00	323.00	71.00	318.00	84.00
298.00	72.00	323.00	71.00	318.00	85.00
299.00	71.00	323.00	71.00	317.00	84.00
299.00	71.00	323.00	71.00	317.00	84.00

COM height change - subject 1 standing

NaN	0.50	0.50	0.00	0.00

Average COM height change the three trials: NaN

Subject 1

Gender Selector: 1

Subject mass (kg): 81.00

1m = ? Pixels: 142

note you must select the gender, mass and calibration here

Standing Trial 1

drag file name in from explorer — Input file 1 Path: C:\Patria\Kawau\Weerapong\Labview\Digitised Data\MO-1A.txt

Output file ANGLECOM 1: C:\Patria\Kawau\Weerapong\Labview\Digitised Data\MO-1Aang1.txt

included KNEE angle	included ANKLE angle	included HIP angle
53.66	62.14	18.31
55.24	62.89	19.63
57.25	63.84	21.29
59.97	65.10	23.45
63.63	66.79	26.27

Body Centre of Mass X and Y coordinates 1

0.00	0.00	0.00
0.00	0.00	0.00

COMy: 0.00, 0.00

COG start ave of 5: NaN

Peak max: 0.00

height jumped: NaN

Standing Trial 2

drag file name in from explorer — Input file 1 Path 2: C:\Patria\Kawau\Weerapong\Labview\Digitised Data\MO-1A.txt

Output file ANGLECOM 2: C:\Patria\Kawau\Weerapong\Labview\Digitised Data\MO-1Aang1.txt

included KNEE angle	included ANKLE angle	included HIP angle 2
53.66	62.14	18.31
55.24	62.89	19.63
57.25	63.84	21.29
59.97	65.10	23.45
63.63	66.79	26.27

Body Centre of Mass X and Y coordinates 2

0.00	0.00	0.00
0.00	0.00	0.00

COMy 2: 0.00, 0.00

COG start ave of 6: NaN

Peak max 2: 0.00

height jumped 2: NaN

Standing Trial 3

drag file name in from explorer — Input file 1 Path 3: C:\Patria\Kawau\Weerapong\Labview\Digitised Data\MO-1A.txt

Output file ANGLECOM 3: C:\Patria\Kawau\Weerapong\Labview\Digitised Data\MO-1Aang1.txt

included KNEE angle	included ANKLE angle3	included HIP angle3
53.66	62.14	18.31
55.24	62.89	19.63
57.25	63.84	21.29
59.97	65.10	23.45
63.63	66.79	26.27

Body Centre of Mass X and Y coordinates 3

0.00	0.00	0.00
0.00	0.00	0.00

COMy 3: 0.00, 0.00

COG start ave of 7: NaN

Peak max 3: 0.00

height jumped 3: NaN

Figure Q10. Front panel of the Labview programme for data analysis (changes of centre of mass) from video analysis using SiliconCoach programme.

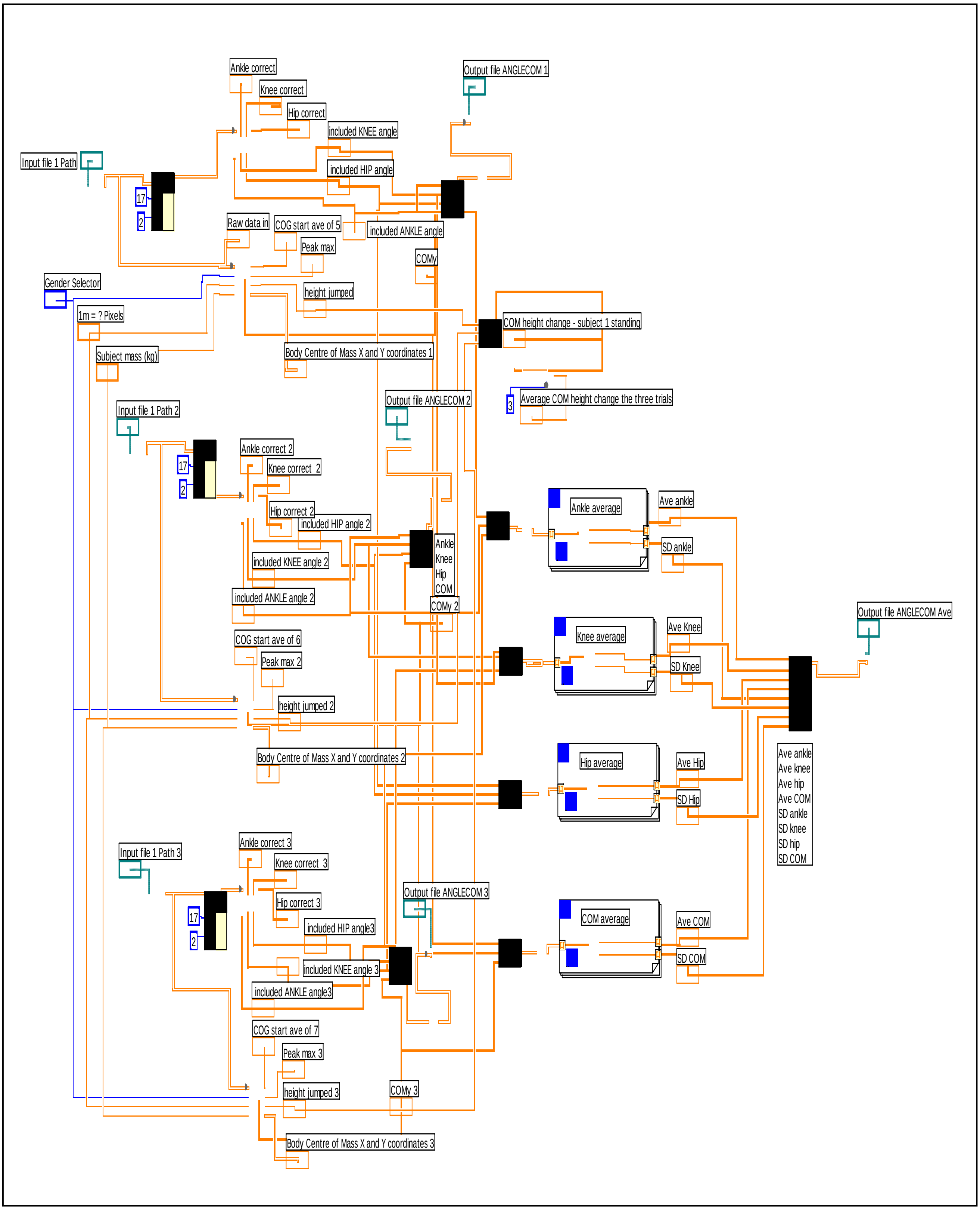
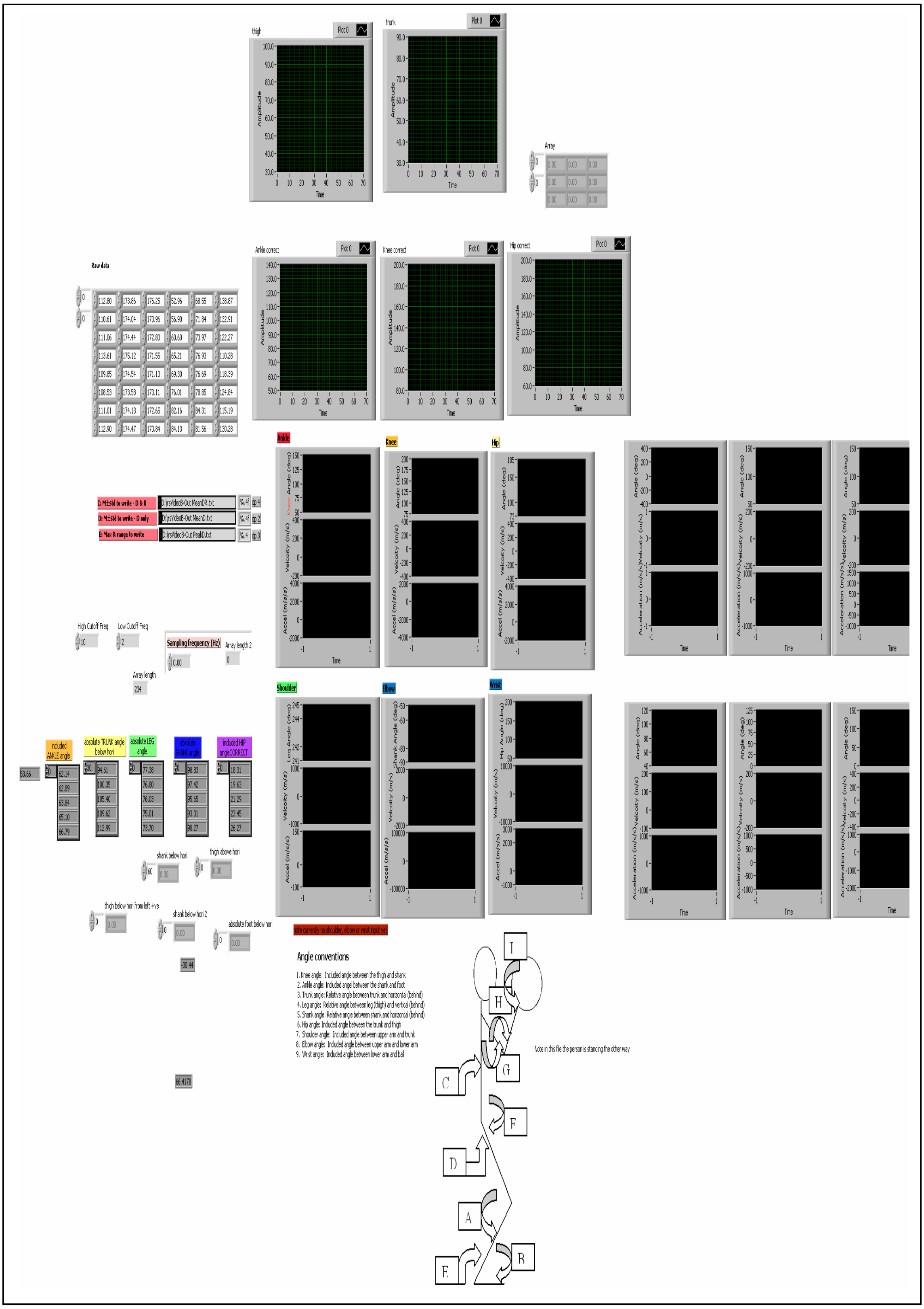


Figure Q11. Diagram of the Labview programme for data analysis (changes of centre of mass) from video analysis using SiliconCoach programme.



Raw data

112.80	173.86	176.25	52.96	68.55	138.87
110.61	174.04	173.96	56.90	71.84	132.91
111.06	174.44	172.80	60.60	73.97	122.27
113.61	175.12	171.55	65.21	76.93	110.28
109.85	174.54	171.10	69.30	76.69	118.39
108.53	173.58	173.11	76.01	78.85	124.84
111.01	174.13	172.65	82.16	84.31	115.19
112.90	174.47	170.84	84.13	81.56	130.28

C: M±std to write - D & R: D:\rsVideoB-Out MeanDR.txt %.4f dp 4

D: M±std to write - D only: D:\rsVideoB-Out MeanD.txt %.4f dp 2

E: Max & range to write: D:\rsVideoB-Out PeakD.txt %.4 dp 3

High Cutoff Freq: 10

Low Cutoff Freq: 2

Sampling frequency (Hz): 0.00

Array length 2: 0

Array length: 234

included ANKLE angle	absolute TRUNK angle below hori	absolute LEG angle	absolute SHANK angle	included HIP angleCORRECT
62.14	94.61	77.38	98.83	18.31
62.89	100.35	76.80	97.42	19.63
63.84	105.40	76.03	95.65	21.29
65.10	109.62	75.01	93.31	23.45
66.79	112.99	73.70	90.27	26.27

53.66

shank below hori: 60, 0.00

thigh above hori: 0, 0.00

thigh below hori from left +ve: 0, 0.00

shank below hori 2: 0, 0.00

absolute foot below hori: 0, 0.00

-30.44

66.4178

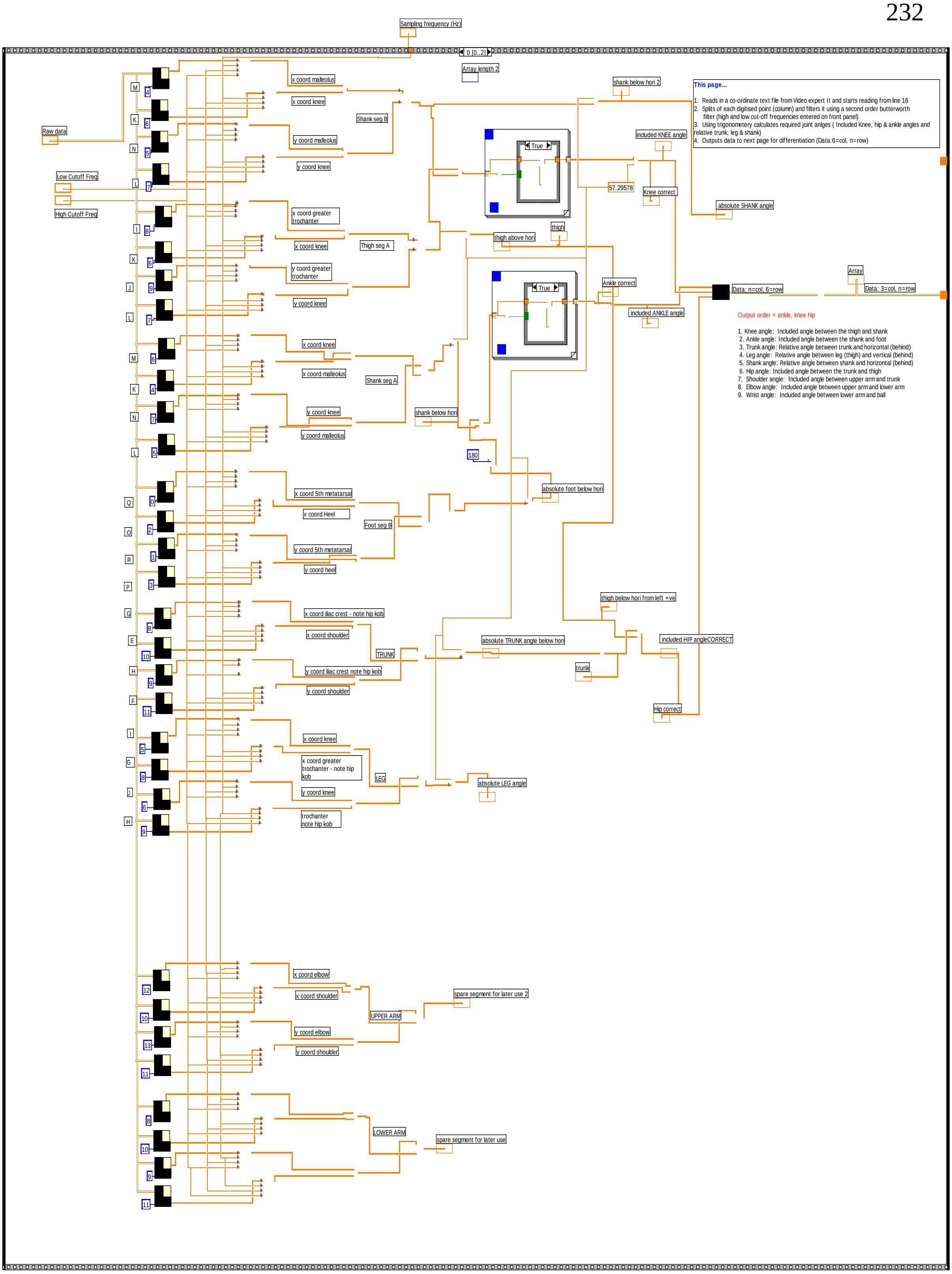
note currently no shoulder, elbow or wrist input yet

Angle conventions

1. Knee angle: Included angle between the thigh and shank
2. Ankle angle: Included angel between the shank and foot
3. Trunk angle: Relative angle between trunk and horizontal (behind)
4. Leg angle: Relative angle between leg (thigh) and vertical (behind)
5. Shank angle: Relative angle between shank and horizontal (behind)
6. Hip angle: Included angle between the trunk and thigh
7. Shoulder angle: Included angle between upper arm and trunk
8. Elbow angle: Included angle between upper arm and lower arm
9. Wrist angle: Included angle between lower arm and ball

Note in this file the person is standing the other way

Figure Q12. Front panel of the Labview programme for data analysis (changes of range of motion) from video analysis using SiliconCoach programme.



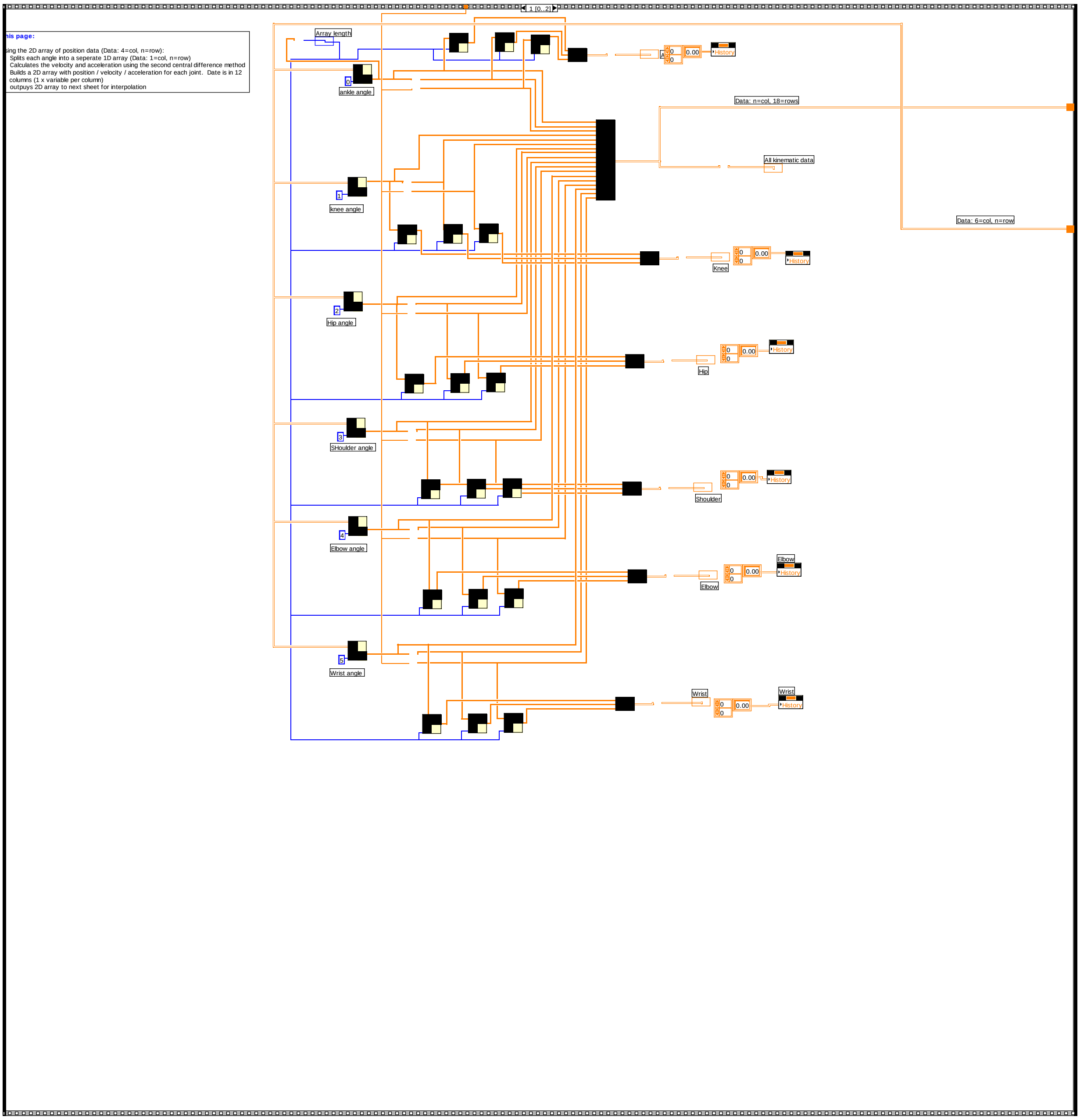
This page...

1. Reads in a co-ordinate text file from Video expert II and starts reading from line 16
2. Splits of each digitised point (column) and filters it using a second order butterworth filter (high and low cut-off frequencies entered on front panel)
3. Using trigonometery calculates required joint anlges (Included Knee, hip & ankle angles and relative trunk, leg & shank)
4. Outputs data to next page for differentiation (Data:6=col, n=row)

Output order = ankle, knee hip

1. Knee angle: Included angle between the thigh and shank
2. Ankle angle: Included angle between the shank and foot
3. Trunk angle: Relative angle between trunk and horizontal (behind)
4. Leg angle: Relative angle between leg (thigh) and vertical (behind)
5. Shank angle: Relative angle between shank and horizontal (behind)
6. Hip angle: Included angle between the trunk and thigh
7. Shoulder angle: Included angle between upper arm and trunk
8. Elbow angle: Included angle between upper arm and lower arm
9. Wrist angle: Included angle between lower arm and ball

Figure Q13. Diagram of the Labview programme for data analysis (changes of range of motion) from video analysis using SiliconCoach programme (page 0).



his page:

sing the 2D array of position data (Data: 4=col, n=row):
Splits each angle into a seperate 1D array (Data: 1=col, n=row)
Calculates the velocity and acceleration using the second central difference method
Builds a 2D array with position / velocity / acceleration for each joint. Date is in 12 columns (1 x variable per column)
outpuys 2D array to next sheet for interpolation

Figure Q14. Diagram of the Labview programme for data analysis (changes of range of motion) from video analysis using SiliconCoach programme (page 1).

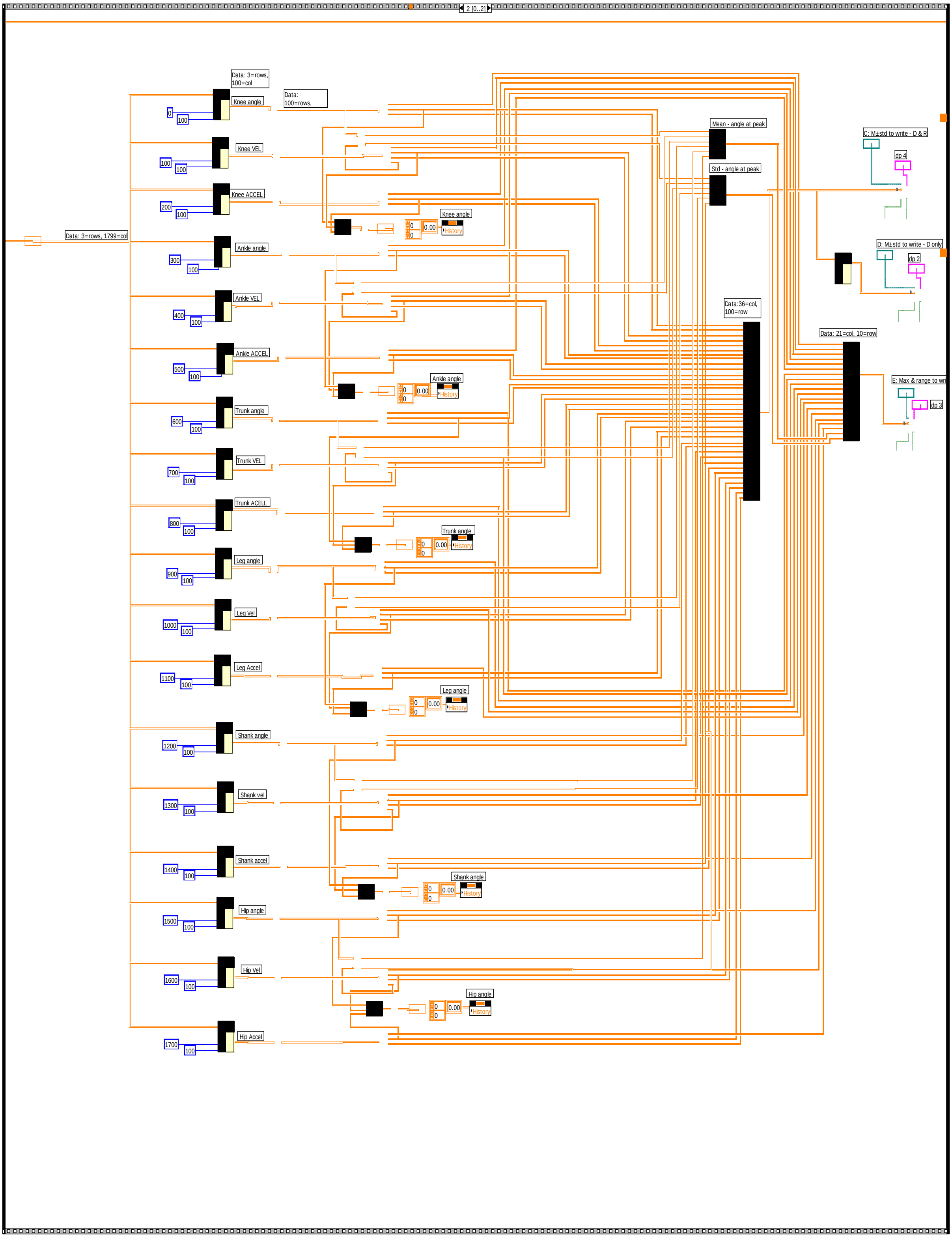


Figure Q15. Diagram of the Labview programme for data analysis (changes of range of motion) from video analysis using SiliconCoach programme (page 2).