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The effect of eccentric and concentric training on the size and strength of human skeletal muscle

Haydn J. N. McDonald
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**The Effect of Eccentric and Concentric Training on the
Size and Strength of Human Skeletal Muscle**

by

Haydn J N McDonald

A Thesis Submitted in Partial Fulfilment of the Requirements for the

Award of

Bachelor of Applied Science (Sports Science) Honours

at the School of Applied Science

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Western Australia

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USE OF THESIS

The Use of Thesis statement is not included in this version of the thesis.

ABSTRACT

The objective of this study was to determine if the high forces generated through eccentric contractions, and the subsequent damage sustained, contributes to greater growth and force increase in human skeletal muscle than other contraction types, and whether damage from eccentric exercise effects the increase in torque and muscle size expected after a progressive concentric strength training program.

20 healthy subjects were split into four groups which participated in 3 different training protocols, with one group serving as the control (C). Groups underwent either concentric training (CT), eccentric damage (ED), or a combination of the two protocols (DC) with the non-preferred biceps over a twelve week period. Isometric and concentric force at 50, 90 and 200°/sec was measured weekly with a Cybex isokinetic dynamometer. Upper arm girth was also measured pre and post training.

The CT group displayed the greatest increase in peak torque for both isometric and concentric contractions. The groups undergoing eccentric damage (ED & DC) showed a decrease in the ability to generate force in the weeks following damage, and showed only small torque increases over the twelve weeks with DC improving to a greater extent than ED at higher contraction velocities.

Eccentric damage appeared to attenuate the increases in peak torque displayed after concentric training. A hypertrophic response from damage may have resulted in a decrease in muscle strength per unit cross-sectional area, and the failure of DC to respond to training may be due to the inability to generate sufficient intramuscular tension required to elicit an adaptive response.

DECLARATION

"I certify that this thesis does not incorporate, without acknowledgment, any material previously submitted for a degree or diploma in any institution of higher education and that, to the best of my knowledge and belief, it does not contain any material previously published or written by another person except where due reference is made in the text".

ACKNOWLEDGMENTS

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CHAPTER ONE

INTRODUCTION

1.1 Background to the Study.

The optimum method of training required to elicit the desired physiological adaptation for a particular sport has long been a topic of interest for researchers in exercise physiology. In the case of maximising muscle growth and the development of strength, a continuum extends from the rehabilitation of atrophied muscle following injury or disease, to the huge relative strengths displayed by power lifters and the extreme muscular hypertrophy of the bodybuilder. The principles of stressing muscle fibres to promote adaptation are essentially the same across this range, though the magnitude of applied stress varies. The contraction types utilised to provide this stimulus for growth include isometric contractions, where force is produced though the muscle-tendon unit does not change length, and isotonic contractions, which include concentric (shortening of the muscle-tendon unit under tension), and eccentric contractions (lengthening of the muscle-tendon unit under tension). Of these types, the greatest force generation is achieved with eccentric contractions (Armstrong, 1984; McCully & Faulkner, 1985).

The nature of adaptation is highly specific to the demands imposed on muscle fibres during training, and it has been well established that high force contractions are needed to stimulate increases in the force producing capacity of muscle (Jones, Rutherford, & Parker, 1989). Increases in muscle size and

strength may occur in response to a number (or combination) of factors associated with high intramuscular forces, such as increased tension due to stretch (Goldberg, Etlinger, Goldspink, & Jablecki, 1975), and structural microdamage to muscle fibres (Ebbeling & Clarkson, 1989). Metabolic factors associated with activity may also compound the effects of exercise-induced damage (Newham, McPhail, Mills, & Edwards, 1983b). Though muscle undoubtedly responds to long term high force training by improving its ability to generate tension, the role of muscle damage in contributing to this process remains unclear. Strength training is more effective when accompanied by adequate recovery, usually in the form of rest days between training sessions, raising the suggestion that hypertrophy and increases in strength may represent an overcompensative response to muscle damage. If this is the case then progressive damaging exercise should result in increased strength compared to a training regimen where little or no damage is incurred.

The degree of muscle damage sustained from the high intramuscular forces associated with eccentric contractions makes eccentric training models attractive to researchers studying the process and effects of muscle damage and regeneration. Conversely, concentric contractions display lower force generating potential and do not result in the extent of damage present after eccentric training. Metabolic costs of exercise involving either eccentric and concentric contractions are also quite different. The degree of physical stress undergone by a muscle can be roughly assessed for each individual via the extent of damage sustained, which in turn can be measured from the release of intracellular proteins into the blood, most notably creatine kinase.

Comparisons of eccentric and concentric training regimens is difficult due to the very different nature of contractions. Ideally, to measure the effect of exercise induced muscle damage on muscle strength, it would be necessary to stress the fibres sufficiently to result in cellular disruption whilst not providing a simultaneous training effect with the induction of damage itself. This was impossible given the time restrictions over which the course of the current experiment had to proceed. This project attempts to address the area of uncertainty regarding the role of muscle damage in increasing strength by comparing the effect of concentric training on strength, with the added stimulus of an intermittent bout of exercise-induced damage during training. The study will look at how the dependant variables of strength and muscle growth are effected by the independent variables of eccentric damage and concentric training over a twelve week period. Four subject groups will consist of: 1. Eccentric damage (ED), 2. Concentric training (CT), 3. Eccentric damage and Concentric training combined (DC), and 4. Control (C) (no training and no damage).

1.2 Significance of the Study.

Strength training plays an important and ever increasing role in modern sport and rehabilitation. From the success dependant professional to the weekend recreational sports person, optimal muscle strength means improved chances of winning, realisation of potential, the greater likelihood of avoiding injury, and ultimately, greater enjoyment. For individuals at the other end of the continuum who are weak as a result of a pathological condition, or recovering following injury, improved strength may allow the execution of simple, but

otherwise impossible daily tasks. Improved knowledge of the most effective training modality necessary to elicit optimal strength gains would be of benefit not only to the sporting industry, but to exercise rehabilitation, and in the treatment of muscle disease.

1.3 Purpose of the Study.

The objective of this study is to determine if the greater forces generated through eccentric contractions, and the subsequent damage sustained, contributes to greater growth and functional development of human skeletal muscle. Muscle damage may or may not be an obligatory component of increased muscle strength, and this study aims to address this area of uncertainty.

1.4 Hypotheses.

1. The CT group will display increases in strength and muscle size when compared to the control group after the twelve week study period.
2. The ED group will display increases in strength and muscle size when compared to the control group after the twelve week study period.
3. Eccentric damage will produce larger plasma values of creatine kinase (CK) than the other groups.
4. There will be insignificant change in CK, strength, or muscle size for subjects in the control group over the twelve weeks comprising the data collection period.

1.5 Organisation of the Thesis.

Chapter One provides a discussion on the background, significance and purpose of the study, with a list of the major hypotheses being tested. Chapter Two reviews the related literature on the theory of training, muscle damage and adaptation. Chapter Three describes the theoretical framework of the study, and Chapter Four describes the design and methodology, including instruments of measurement, and procedures of data collection and analysis.

Results and data analysis are presented in Chapter Five, and the thesis concludes in Chapter Six by discussing the findings of the study, their relation to the literature, and their implications for future research.

CHAPTER TWO

LITERATURE REVIEW

2.1 The Theoretical Rationale of Training

The major objective of physical training is to stimulate biologic adaptations which will contribute to performance improvement in a specific task (McKardle, Katch & Katch, 1986). This stimulation entails "exposing the organism to a training load or work stress of sufficient intensity, duration and frequency to produce a noticeable or measurable training effect" (Astrand & Rodahl, 1986, p. 420). Increases in size, strength, and power of skeletal muscle requires the organism to be subjected to an overload, ie., to a resistance or stress greater than that which is normally encountered by the muscle. An improvement in performance over the course of training necessitates a corresponding increase in the intensity of the load required to stimulate adaptation.

The stimulus required to elicit optimal adaptation of muscle structure and function remains enigmatic, though almost certainly involves high intramuscular forces (Jones, Newham, Round, & Tolfree, 1986). Such high force generation necessitates maximal or near maximal contractions to provide the specific stimulus needed to increase the maximum force generating potential of the muscle. It is well known that due to the specific nature of adaptation progressive training of muscle involving sub-maximal

contractions, while promoting increased muscular endurance, does not generate significant improvements in maximum strength. Goldberg et al. (1975) indicate that passive stretch induces a hypertrophic response, and a subsequent increase in strength, suggesting force alone may be an adequate stimulus for adaptation (see section 2.2.2). Jones et al. (1986) identify damage due to high intramuscular forces as being a precursor to structural adaptation of muscle via the division of satellite cells and their incorporation into existing muscle fibres. Muscle damage involves microtrauma resulting in disruption of the structural organisation of the muscle fibre, potentially leading to muscle cell necrosis (see section 2.3). Damage has also been shown to occur with endurance training, where exercised muscles present with pathological changes resembling those induced through ischaemia (Hoppeler, 1986; Ebbeling & Clarkson, 1989) (see section 2.2.3). Though it is accepted that training intensity is more important than training volume in achieving strength gains, the role of metabolic depletion in contributing to an adaptive response remains unclear.

Increases in strength and size of muscle may be initiated through force generation alone (as with chronically stretched muscle), through metabolic changes accompanying high force contractions, or through the regeneration and overcompensation of muscle damaged due to high force contractions. The initiating mechanism of exercise-induced skeletal muscle damage is not well understood, but may relate to both metabolic and/or mechanical factors associated with activity.

2.2 The Stimulus for Adaptation.

2.2.1 Force Generation

During skeletal muscle contraction, fixed order motor unit recruitment occurs in accordance to the force generation requirements of the whole muscle (McDonagh & Davies, 1984; Noth, 1991). Recruitment follows Henneman, Somjen, & Carpenter's (1965) 'size principal' which states that preferential motor unit activation occurs in order of size, and subsequent force generating capacity, from small to large. Only very powerful contractions, and long duration submaximal contractions, provide the impetus for the full activation of high threshold motor units (McDonagh & Davies, 1984). High force resistance training, therefore, provides maximal stimulus for the structural adaptation of skeletal muscle. Long term progressive heavy resistance exercise increases lean body mass, as evidenced by body builders who display extraordinary muscle hypertrophy, attributable mainly to an increase in cross sectional area of individual muscle fibres (Tesch, 1988). Studies using cats have indicated that the number of muscle fibres may also increase (hyperplasia) in response to heavy resistance exercise in animals (Gonyea, 1981; Gonyea, Sale, Gonyea, & Mikesky, 1986), and that exercise induced hyperplasia may occur in human athletes relying on high levels of both muscular power and endurance (Tesch & Karlsson, 1985).

2.2.2 Stretch

Animal studies have shown that stretch of muscle *in vivo* may retard atrophy of denervated muscle and even induce muscular hypertrophy in normally innervated muscle (Sola & Martin, 1953; Sola, Christensen, & Martin, 1973). The effect of stretch is to increase the rate of protein synthesis as well as the number of sarcomeres in series (Goldspink 1992). Goldberg et al. (1975) indicate that the increased tension in the muscle as a result of passive stretch may be a crucial factor in inducing growth (and therefore strength gains) or retarding atrophy.

2.2.3 Metabolic Changes and Muscle Damage

Altered metabolic homeostasis resulting from long term submaximal exercise has been proposed to result in muscle damage and subsequent regeneration. The events thought to initiate muscle cell necrosis include ischaemia induced hypoxia resulting in altered ion concentrations, ATP deficiency, and accumulation of the products of muscle metabolism (Armstrong, 1984; Ebbeling & Clarkson, 1989).

Cell damage following exhaustive endurance exercise resembles that induced through ischaemia and pathological changes observed in the gastrocnemius muscle of marathon runners includes disruption of the contractile apparatus, mitochondria, sarcolemma and sarcotubular system (Hoppeler, 1986). Prolonged running may result in ischaemia which could

directly lead to damage, or contribute indirectly by delaying the clearance of waste products (Ebbeling & Clarkson, 1989). Running involves an eccentric contraction component as the body's centre of mass is decelerated on foot strike, and it has been suggested these repetitive eccentric contractions may cause muscle cell damage in endurance events (Evans, 1987). Newham et al. (1983b) posit that damage may be exacerbated by metabolic changes accompanying prolonged exercise.

2.2.4 Contraction type and muscle damage

Skeletal muscle relies on a high degree of plasticity in order to accomplish coordinated body movement, and as such is capable of three different modes of contraction. Isotonic contractions involve muscle in a dynamic state, and include both eccentric (negative) and concentric (positive) contractions. Eccentric contractions involve the forced lengthening of a contracting muscle, concentric contractions effect a decrease in muscle length, and isometric contractions involve tension development where there is no change in muscle length. The mechanical and energetic behaviour of actively lengthening muscle differs greatly to that of isometric and shortening actions, whereby exercise involving a predominance of high force eccentric contractions has been shown to result in muscle fibre injury (Abraham, 1977), the magnitude of which is greater than that produced by either isometric or concentric contractions (Friden, Sjoström, & Ekblom, 1981; Davies & White,

1981; Edwards, Mills & Newham, 1981a; Newham, Jones, & Edwards, 1983a; Jones, Newham & Torgan, 1989; McCully & Faulkner, 1985).

The metabolic cost of eccentric contractions is less than that of concentric (Newham et al., 1983b), though the maximum force produced is often greater, indicating damage may be more likely due to mechanical rather than metabolic factors. Many researchers have based their argument against a metabolic hypothesis on the evidence that active tension developed by muscles eccentrically requires less energy yet results in greater injury than muscles which contract concentrically. Electromyographic (EMG) activity is lower during negative work, which suggests fewer motor units are activated for a given load (Bigland-Ritchie & Woods, 1976) leading to higher tension development per cross-sectional area of active skeletal muscle fibres (Davies & White, 1981).

Eccentric contractions at long muscle lengths result in the greatest damage to muscle fibres (Newham, Jones, Ghosh, & Aurora, 1988). At long sarcomere lengths a fibre has high passive tension, and the reduced overlap zone between actin and myosin filaments allows only a small percentage of cross-bridges to form (Russell, Dix, Haller, & Jacobs-el, 1992). Cross-bridge cycling in this situation may result in breakage of titin filaments (which prevent the myosin filaments from moving too far from the Z-disks) due to unstable force production between adjacent sarcomeres (Russell et al. 1992; Armstrong, Warren, and Warren, 1991; Friden & Lieber, 1992), a phenomenon generally known as muscle 'creep' (Jones & Round, 1990).

High passive tension may also lead to sarcolemmal disruption (Armstrong et al. 1991), allowing an influx of calcium ions from the extracellular fluid, so increasing the calcium and initiating muscle cell autolysis. Much is still unknown about the mechanisms of muscle fibre injury, and more research is needed in order for us to fully understand this process.

2.3 Exercise-Induced Muscle Damage.

Skeletal muscle damage has been shown to occur as a result of intense or unaccustomed strenuous activity (Abraham, 1977; Friden, Sjostrom & Ekblom, 1981; Ebbeling & Clarkson, 1989). Damage is evident through morphological changes (Ebbeling and Clarkson, 1989; Friden et al. 1981, 1983; Jones et al., 1986), delayed-onset muscle soreness and pain (Armstrong, 1984; Friden et al. 1981; Newham et al., 1983b), performance decrements (Davies & White, 1981; McCully & Faulkner, 1985; Friden et al. 1981), and elevation of specific muscle proteins in the blood, including creatine kinase (Newham et al., 1983, 1986; Manfredi, Fielding, O'Rielly, Meredith, Lee & Evans, 1991; Clarkson, Nosaka & Braun, 1992; Jones et al. 1986). Destructive changes of fine muscle structure and function can be seen following sprint or distance running, and after resistance training (Friden & Lieber, 1992). Damage is temporary and repairable (Friden et al. 1981; Armstrong, 1984), and adaptation occurs such that all indicators of damage are greatly reduced following repeated exercise bouts (Ebbeling & Clarkson, 1989; Clarkson et al. 1992).

Muscle damage can be monitored by the measurement of intracellular components released into the blood, and include the proteins myoglobin, creatine kinase (CK), lactate dehydrogenase (LDH), and aspartate transaminase. CK and LDH have muscle-specific isoenzymic forms making them distinguishable from enzymes emanating from cardiac or other tissue (Jones & Round, 1990). CK is the most commonly used marker of muscle damage (Nosaka, Clarkson, & Apple, 1992), though the magnitude and time course of its efflux from the muscle cell into the plasma is highly variable between subjects, and can be influenced by such factors as the type of exercise, the exercise intensity, and the degree of specific training of the subjects (Clarkson, Byrnes, McCormick, Turcotte, & White, 1986; Newham et al., 1983a; Byrnes, Clarkson, & Katch, 1985). Though an increase in plasma CK is a good indicator that muscle damage has occurred, CK values do not always correlate well with the degree of damage or the muscle mass involved in the potentially damaging exercise (Nosaka et al., 1992; Manfredi et al., 1991). Plasma CK reaches peak values 1-7 days following high intensity or unaccustomed exercise (Clarkson et al., 1992; Jones et al., 1986).

It has been shown that one bout of damaging high force eccentric exercise can produce an adaptive effect such that less damage is produced when the same exercise is repeated up to several months later (Clarkson & Tremblay, 1988; Newham, Jones, & Clarkson, 1987; Clarkson et al., 1992). The exact mechanism responsible for protection from damage during repeated exercise bouts is still unknown, as are questions regarding the

volume, intensity, and type of exercise used in the initial bout relative to the time course of protection.

Skeletal muscle injury consequent to heavy resistance training and the high intra-muscular forces produced can involve primary or secondary sarcolemmal disruption, swelling or disruption of the sarcotubular system, distortion of the myofibrils' contractile components, cytoskeletal damage, and extracellular myofibre matrix abnormalities (Friden & Lieber, 1992).

Skeletal muscle possesses considerable powers of regeneration (Studitzky, 1964). Following the injury or death of a muscle fibre, the necrotic tissue is invaded by blood vessels. Mononuclear cells, including macrophages and T lymphocytes, move from the blood vessels and infiltrate the damaged tissue removing cellular debris and initiating regeneration (Jones & Round, 1990; Antonio & Gonyea, 1993).

2.4 Adaptation to Training.

2.4.1 Neural Adaptation

Strength training studies typically last 8-20 weeks, and early responses can be largely associated with neural adaptation (Sale, 1988) such as improved coordination (Rutherford & Jones, 1986), and increased activation of prime mover muscles (Moritani & deVries, 1979). The most rapid strength increases during a training program often develop within the first 2-8 weeks (Fleck & Kraemer, 1988). These strength increases have been shown to

occur without a corresponding increase in muscle size, and have been attributed to improved technique through neural adaptation, increased firing rates of motor units, recruitment of additional motor units in the muscle, and improved motor unit synchronisation (Rushall & Pyke, 1990). It has been suggested that morphological changes do not take place until after neural adaptation is complete (Fleck & Kraemer, 1988; Moritani & deVries, 1979). More research is needed, however, to answer questions regarding the time course of adaptive responses to high force strength training.

2.4.2 Strength

Muscle damage has been shown to result in increased protein breakdown and resynthesis in both human (Evans et al., 1986) and animal models (Wong & Booth, 1990a, 1990b). Wong and Booth (1990a & 1990b) identified increased protein turnover in rodents following both eccentric and concentric exercise protocols. Eccentric exercise resulted in a more prolonged increases in protein synthesis and greater muscle enlargement when compared to concentric contractions. Hypertrophy, however, has been shown to be inconsistent with improvements in muscle force (Kandarian & White, 1990), suggesting a decrease in muscle efficiency (force per cross-sectional area) following damaging eccentric exercise. Komi and Buskirk (1972) compared progressive eccentric and concentric biceps exercise in humans, and found an eccentric contraction protocol to elicit greater increases in muscle size, and in both isometric and dynamic force. Strength

improvement relative to increases in muscle size, however, was disproportionate, again implying a decrease in efficiency following adaptation from damage.

Whether concentric or eccentric contractions provide the greatest gains in either the size or strength of muscle in humans, and whether there is an interaction between both responses remains unclear. To my knowledge, previous studies have not utilised a combination of concentric training and eccentric damage when studying the functional adaptation of muscle to training. The current investigation employs a similar approach to that of Komi and Buskirk (1972) involving an eccentric training and a concentric training group, with the addition of a group undergoing a combination of both protocols in an effort to determine the effect of concurrent damage on adaptation to concentric training.

2.4.3 Hypertrophy

Skeletal muscle fibre types exist on a continuum from Type IIa (fast, glycolytic), through Type IIb (fast oxidative/glycolytic), to Type I (slow, oxidative). All muscle fibres are capable of undergoing hypertrophy, yet the extent of potential growth varies between fibre types (Goldspink, 1992). Type II glycolytic fibres are recruited during high force contractions, and when progressively overloaded undergo hypertrophy very readily. Type I oxidative fibres are fatigue resistant, but are incapable of the speed and power of contraction displayed by fast twitch fibres, and do not hypertrophy to the

same extent with high force training. Muscle fibres possess the potential for a great deal of plasticity between the broadly designated groups, and as such are able to adapt to imposed demands. Under normal circumstances, however, transformation of fibre types rarely occurs.

Muscle cross sectional area is proportional to the force generating potential of a muscle (Maughan, Watson & Weir, 1983), and a high percentage of fast twitch fibres is considered advantageous for success in events requiring a large degree of strength, power and speed. The great potential for growth of fast twitch fibres allows them to contribute up to 90% of the total area of a trained muscle, despite a fibre type composition within the normal range (Tesch, 1988).

Growth of skeletal muscle is a result of increased myofiber area due to an increase in the myofibril number. This involves a process by which myofibrils undergo longitudinal splitting into two or more daughter myofibrils (Goldspink, 1991). The myofibrillar mass becomes subdivided as it increases in volume, allowing the sarcoplasmic reticulum and transverse tubule system to remain in close juxtaposition with the actin and myosin filaments. Goldspink (1991, p.215) posits that the longitudinal splitting occurs due to a "built in mismatch between the actin and myosin lattice so that the actin filaments are slightly displaced as they run from the Z-disk (square lattice) to the A-band (hexagonal lattice)." During high force contractions, the oblique pull of actin filaments causes a mechanical stress which results in splitting of the myofibril at the centre of the Z-disk (Figure 2.1).

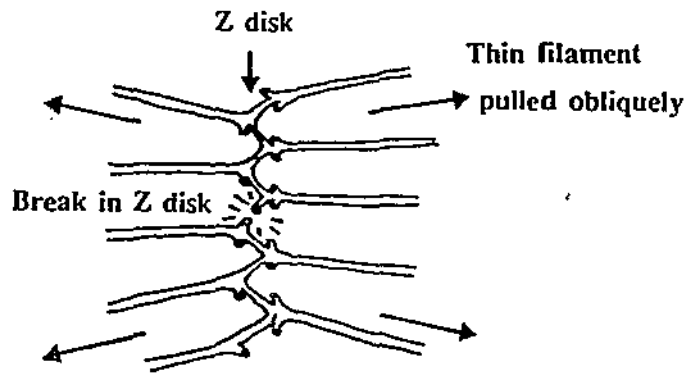


Figure 2.1: Mechanical stress causing splitting of the myofibril at the Z-disk.
(Reprinted from Jones & Round, 1990)

Recent studies using a CT scan to measure muscle density indicate a possible increase in myofilament packing. Jones and Round (1990) disagree, however, and suggest there is little evidence to support this hypothesis. MacDougall (1991) posits that with high resistance strength training, actin and myosin filaments are added to the periphery of existing myofibrils, thus creating larger myofibrils without altering filament packing density or cross-bridge spacing. Clearly, more research is needed in order to fully understand this process.

2.4.4 Hyperplasia

The relative contribution of hyperplasia to muscle enlargement following strength training remains controversial. Practical difficulties exist in accurately measuring muscle fibre numbers in histological cross-sections, and through individual fibre counting after nitric acid digestion. These problems are further compounded by the obvious ethical constraints when estimating fibre numbers of human subjects *in vivo*.

Almost one hundred years ago, Morpurgo (1897) (cited in MacDougall, 1991) ran dogs on a running wheel and concluded that muscle growth occurs exclusively through enlargement of existing fibres. A series of studies conducted during the 1970's, however, suggested that training-induced growth in animals may be the result of both hypertrophy of existing fibres as well as the addition of new fibres (MacDougall, 1991). MacDougall, Sale, Alway, & Sutton (1984), compared the biceps of elite body builders and untrained controls, concluding there was no significant hyperplastic response to the heavy resistance training undertaken by the bodybuilders over a six year period. Antonio and Gonyea (1993) challenge the reliability of MacDougall's findings by suggesting that muscle biopsies sample a very small portion of tissue which may not be representative of the whole muscle. In a review of the role of satellite cells in muscle growth, White and Esser (1989) consider skeletal muscle fibres to possess the appropriate mechanisms for both responses.

2.4.5 Satellite cells

Satellite cells are a type of non-functioning reserve cell that occur outside the plasma membrane of the muscle fibre, but within the basal lamina, and are thought to be derived from myoblasts which did not fuse to form myotubes and functional fibres during embryonic development (MacDougall, 1991). Mobilisation of satellite cells takes several hours following injury, and is thought to be activated and regulated by growth factors such as fibroblast growth factor (FGF) and insulin-like growth factor 1 (IGF-1). Following muscle damage the satellite cells within the muscle fibres become mitotically active, the extent of which seems to be dependant on the magnitude of the stimulus relative to the capacity of the fibre to respond (White & Esser, 1989). Adult muscles can repair injury or increase their mass by recruiting satellite cells as the source of new nuclei in much the same way as during embryonic development (Russell et al., 1992; Jones & Round, 1990; MacDougall, 1991). Where the extent of injury is great enough to cause muscle fibre death, the proliferating satellite cells mature into myoblasts, and then fuse to form new myotubes (Jones & Round, 1990). This process gives rise to a new multinucleated fibre within the existing, intact basal lamina (Antonio & Gonyea, 1993; White & Esser, 1989).

Repeated bouts of high force contractions may result in a continuing process of injury and regeneration, producing an overcompensation of protein synthesis and a net anabolic effect (Antonio & Gonyea, 1993). Relative to the magnitude of the stimulus, and subsequent fibre injury, satellite cell

proliferation may result in the "generation of new independent fibres [hyperplasia], the repair of injured myofibres, and/or the fusion of myotubes with existing fibres causing hypertrophy" (Antonio & Gonyea, 1993, p. 1341). The addition of new myonuclei to an existing fibre has the resultant effect of maintaining the nuclear-to-cytoplasmic ratio in steady-state hypertrophy (White & Esser, 1989; Antonio & Gonyea, 1993). Muscle fibres are long multinucleated cells, and it is presently unclear as to whether focal damage to a particular area of a fibre leads to degeneration of the whole fibre. It is feasible that damage may be contained and localised, with satellite cells replacing only the damaged portion (Jones & Round, 1990).

Cardiac muscle lacks satellite cells, yet responds hypertrophically to an adaptive stimulus. So while satellite cells clearly play an important role in muscle hypertrophy, and possibly hyperplasia, their involvement may not be obligatory (White & Esser, 1989). Repeated bouts of eccentric exercise have been shown to produce a hypertrophic response in animals, though this result is yet to be demonstrated in humans. Further research is needed to fully understand the adaptive processes of skeletal muscle following repetitive high force stimuli.

2.4.6 Connective Tissue

Large amounts of connective tissue exists within and around skeletal muscle and acts as a force conveying network. It provides a basic framework and supportive structure for contractile tissue and is arranged in and around muscle

to protect, strengthen, and bind fibres and bundles of fibres together. Individual skeletal muscle fibres are covered by a fine connective tissue sheath called the endomysium. The perimysium binds groups of fibres together into bundles called fasciculi, groups of which are covered by the epimysium, which in turn is continuous with a tendon, forming the whole muscle (Van De Graaff & Fox, 1988). The endomysium is intimately associated with the basal lamina of the muscle cell and is referred to as the series elastic component, thought to be damaged by eccentric contractions, particularly at long muscle lengths (Jones et al. 1989).

As muscle hypertrophies and increases in strength, connective tissue correspondingly undergoes a variety of morphological and biochemical changes. The maximal tensile strength and elastic storage capabilities of connective tissue is increased by physical training (Stone, 1991). More specifically, connective as well as contractile tissue adaptation is thought to be a response to the mechanical stress caused by the high forces associated with eccentric muscle actions (Stauber, 1989).

During resistance training, connective tissue is effectively stretched. Muscle damage and stretching of fibroblasts could promote the production of mitogens (substances that induce DNA, RNA, and protein synthesis) to generate changes in connective tissue strength by effecting fibril structure and number, matrix consistency, and matrix-fibre ratio (Stone, 1991). The mononuclear cell invasion of damaged tissue influences proteoglycans which, as well as playing a structural role, may also be important in regulating the myogenic process

following exercise induced damage (Stone, 1991; Ebbeling and Clarkson, 1989; Stauber, 1989).

The increased collagen synthesis accompanying work-induced hypertrophy may result in an increased connective tissue content of muscle (Jones & Round, 1990). MacDougall et al. (1984) determined that the relative proportion of collagen was similar in untrained, novice and elite bodybuilders, indicating an increased total collagen content accompanies hypertrophy. The increased connective tissue strength achieved through greater collagen content may be enhanced by changes in connective tissue attachments. Force is generally considered proportional to fibre cross-sectional area (CSA) due to longitudinal transmission of force through sarcomeres arranged in series. If connective tissue attachments were made between the tendons and intermediate sarcomeres, however, the force generated per unit CSA would be increased (Jones & Round, 1990) (Figure 2.2).

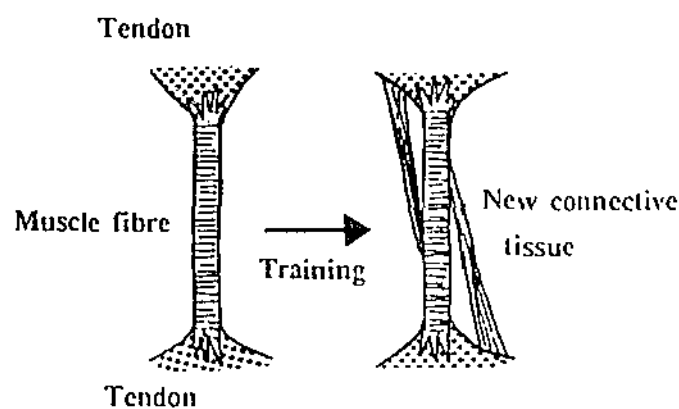


Figure 2.2: Changes in connective tissue attachments leading to an increase in force. (From Jones and Round, 1990).

2.5 Summary.

Improved strength through physical training requires a muscle or muscle group to be subjected to a stimulus, in the form of progressive overload, eliciting an adaptive response. The exact nature of this stimulus remains enigmatic, though it almost certainly involves high intramuscular force generation, and therefore necessitates maximal or near maximal contractions. Increases in strength and size of muscle may be initiated through force generation alone (as with chronically stretched muscle), through metabolic changes accompanying high force contractions, or through the regeneration and overcompensation of muscle damaged due to high force contractions. The initiating mechanism of exercise-induced skeletal muscle damage is not well understood, but may relate to both metabolic and/or mechanical factors associated with activity.

Eccentric contractions have been shown to result in the greatest injury to skeletal muscle fibres and can lead to disruption of the structural organisation of contractile units. Morphological changes are accompanied by a decrease in the muscle's ability to generate force immediately following damage, an increase in the concentration of plasma CK, and delayed onset muscle soreness. High force strength training results in neural adaptation, satellite cell proliferation (inducing hypertrophy and possibly hyperplasia), and the strengthening of associated connective tissue.

The role muscle damage plays in contributing to strength increases procured through high force training is as yet poorly defined. Adaptation and regeneration following damage from eccentric contractions affords a protective effect from damage for repeated exercise bouts in both animals and humans. Although progressive damaging bouts of eccentric contractions have produced a hypertrophic response in animals, the effect on human skeletal muscle clearly requires further investigation.

CHAPTER THREE

THEORETICAL FRAMEWORK

3.1 Theoretical Framework.

Muscle adaptation to a continued training stimulus involves increases in size and strength. The large force production inherent in eccentric muscle contraction results in proportionally greater micro-damage to the muscle fibre architecture than that experienced after concentric or isometric contractions (Friden et al., 1981; Davies & White, 1981; Edwards et al., 1981a; Newham et al., 1983a). Skeletal muscle undergoes a regeneration process following damage, after which the muscle displays greater resistance to potentially damaging exercise for several weeks (Clarkson et al., 1992). This disruption of muscle fibre structure through eccentric training may be a necessary component of overload, or strength development may occur independently of damage. This area of uncertainty has been addressed by examining the effect of progressive eccentric training relative to training regimens incurring little or no muscle fibre damage.

CHAPTER FOUR

METHODOLOGY

4.1 Design.

A 2 x 2 factorial design was used to investigate the independent variables of eccentric damage and concentric training, and their effect on muscle strength development and growth. A diagrammatical representation is shown in Table 4.1.

Table 4.1: 2 x 2 Factorial study design investigating the independent variables of eccentric damage and concentric training.

| POSITIVE TRAINING | NEGATIVE TRAINING | |
|-------------------|-------------------|---------|
| | ED | No ED |
| CT | ED & CT | CT |
| No CT | ED | CONTROL |

ED = Eccentric damage

CT = Concentric training

4.2 Subjects.

Subjects for this study consisted of 20 healthy males and females within the age range of 18 - 50 years who were not currently undertaking rigorous physical exercise. The protocol for the study was approved by the ethics committee of Edith Cowan University, and all subjects were told of the nature and risks of the procedures to be used, and informed consent was obtained

(Appendix A). Subjects were asked to refrain from making any major changes to diet or exercise habits over the course of the 12 week training study.

4.3 Instruments.

Cybex 6000 Isokinetic Dynamometer (Cybex Inc, N.Y.)

IBM microprocessor

Blood sampling equipment

4ml Vacutainer tubes and needles

heparinised 30 μ l capillary tubes

Reflotron spectrophotometer

Queen Elizabeth II Medical Centre Pathology Department

Lufkin constant tension tape (metal)

4.4 Reliability / Validity.

Calibration of all equipment was undertaken prior to each testing session. All measurement systems are computer software controlled, save the assessment of muscle growth (Lufkin Constant Tension Tape), where the technical error of measurement (TEM) was <5%. Interrator reliability was ensured through tester training sessions undertaken prior to the commencement of the study. Subject and corresponding equipment positioning was marked and recorded at the first testing and training sessions, and duplicated thereafter to maintain measurement stability. Subjects familiarised themselves with the testing equipment prior to the collection of data to minimise the effect of learning on test

results and subjects were exhorted to perform maximally throughout all testing and training sessions. Peak torque has been shown to be an accurate and highly reproducible variable when measured with modern computer controlled isokinetic dynamometers under consistent testing conditions (Kannus, 1994).

Plasma CK values were used to validate that muscle damage had occurred.

4.5 Data Collection.

4.5.1 Training.



Figure 4.1: Setup of equipment for both testing and training of the forearm flexors of the non-preferred arm.

Twenty-four subjects commenced training of the non-preferred forearm flexors for the twelve week training study, and were randomly assigned to four groups; 3 training groups, and 1 control group. Four subjects failed to complete the data collection period for various reasons leaving 20 subjects to provide data

for the investigation. Four groups of subjects were monitored throughout a twelve week period in which each group undertook the following training regimens:

1. Control (C) (n=4) - no training.

2. Eccentric Damage (ED) (n=5) - The application of force resulting in eccentric damage, was attained with a CYBEX 6000 Isokinetic Dynamometer (CYBEX Inc, N.Y.). Subjects were seated on a conventional 'preacher' bench to optimise isolation of the forearm flexors with the dynamometer axle positioned at the centre of rotation of the elbow. The lever arm was attached at the wrist with contractions made through approximately 140 degrees of motion (Figure 4.1). Subjects performed eccentric training of the forearm flexors of the non-preferred arm at three weekly intervals, allowing for full myofibre regeneration between exercise bouts. Eccentric training took place at the beginning of the study period (week 0), and at the start of the fourth (week 3), seventh (week 6), and tenth (week 9) weeks. 3.5 sets of 10 maximal voluntary contractions were performed at an angular velocity of 30°/sec. Passive recovery between repeats was performed at 90 deg/sec and recovery time between sets was of 1 minute duration. Subjects completed 35 contractions per 'damage' session, over a range of motion (ROM) of approximately 140°.

3. Concentric Training (CT) (n=6) - The biceps of the non-preferred arm was trained three times per week using the Cybex isokinetic dynamometer with subjects positioned as mentioned above. Subjects completed 6 sets of 10 maximal contractions at an angular velocity of 90 degrees /sec. Passive recovery

4. Eccentric Damage and Concentric Training (DC) (n=5) - Subjects followed the protocols of both the ED and CT groups. Weeks involving eccentric damage consisted of only 2 concentric training bouts, maintaining the same number of total training sessions as the CT group.

4.5.2 Testing.

4.5.2.1 Force.

Peak isometric torque and concentric torque at 50°, 90°, and 200°/sec was tested at the beginning of every week throughout the twelve week experiment. Testing was performed on the Cybex isokinetic dynamometer using the preacher bench and subject positioning as mentioned above (see Figure 4.1). All testing was performed before any training for that day to avoid undue fatigue, and the induction of damage took place after the completion of testing every third week. Subjects were exhorted to perform maximally during all testing and training sessions.

Peak isometric torque was established by performing a maximal voluntary contraction (MVC) at approximately 90 degrees of flexion. A minimum of 3 MVCs were performed, each at a 10 degree angle difference, to ensure the attainment of an optimal biomechanical angle and peak torque recording. Dynamic force was taken as the best of 4 maximal contractions for each of the test velocities. Forces produced by the muscle were digitised and recorded in 'real time' on an IBM PC before being transferred directly into a spreadsheet for later analysis.

Table 4.2: A diagrammatical representation of the timeline for the first of four 3 week testing / training blocks.

| One of four 3 week testing / training blocks | | | | | | |
|--|-----|---------|-------|-------|--|--|
| TRAIN | C | | | | | |
| TRAIN | ED | D | | | | |
| TRAIN | CT | T T T | T T T | T T T | | |
| TRAIN | DC | D T T | T T T | T T T | | |
| Week | | 0 | 1 | 2 | | |
| | | B B B B | | | | |
| TEST | ALL | S | S | S | | |

D = Eccentric damage.

T = Concentric training.

B = Blood sample for assay of plasma CK

S = Peak torque tested at 0°, 50°, 90°, & 200° /second.

4.5.2.2 Creatine Kinase.

The existence of damage was verified by measuring changes in intramuscular CK through analysis of serial venous blood. 25 ml samples were taken from the ante-cubital vein immediately prior to the induction of damage, and then 2, 4, and 6 days post damage. Samples were centrifuged and the plasma separated and frozen for later analysis, made courtesy of Queen Elizabeth II Medical Hospital Pathology Department.

2, 4, and 6 days post damage. Samples were centrifuged and the plasma separated and frozen for later analysis, made courtesy of Queen Elizabeth II Medical Hospital Pathology Department.

In order to minimise unnecessary discomfort to control subjects, whose CK values were highly unlikely to change, capillary blood from finger pricks was analysed with a Reflotron spectrophotometer to identify changes in CK.

4.6 Data Analysis.

Prior to data analysis missing data were managed by 1. deleting cases where >2 of 13 testing weeks were missed ($n=4$), and 2. replacing missing data with the mean of the weeks immediately preceeding and following the missing testing session (Tabachnick & Fidell, 1989).

Statistical analysis was performed using SPSS for Windows. Non-parametric tests were used due to the small number of subjects.

Variance in peak torque was analysed at week 1 and week 12 relative to pre-test values using the Wilcoxon matched-pairs signed rank test. The change in peak torque after 12 weeks was compared between the CT and C groups and between the DC and ED groups using the Mann-Whitney test for paired independant samples.

Variance in peak CK between damage and non-damage groups was analysed using a t-test for Equality of Means. Variance in upper arm girth from pre to post training was analysed by multiple ANOVA between the three training groups. The accepted level of significance was set at $p < 0.05$.

4.7 Limitations.

1. Selection of subjects restricted participants to people who were not currently undertaking rigorous physical activity, and so involved some degree of subjective evaluation on the part of both the researchers and the subject.
2. Volunteer subjects may not be representative of the population as a whole.
3. Central fatigue is a confounding factor, but of minimal importance (James, Sacco, & Jones, in press), but was uncontrollable save that all subjects were exhorted to perform maximally.

4.8 Assumptions.

1. Subjects will perform to the best of their ability during testing and training sessions.
2. Subjects will not make lifestyle changes likely to confound the results of the investigation, ie., major dietary or training adjustments.

CHAPTER FIVE

RESULTS

5.1 CK Responses.

Normal plasma CK values in humans are quite variable and range from 24-195 IU/l (Boehringer Mannheim). Means for non-damage groups (CT and C) remained in the normal range throughout the 12 week study period, though increases of 77% and 85% respectively were noted from pre-test values (Table 5.1, Figure 5.1a).

The eccentric damage group (ED) displayed significant increases ($p < 0.05$) in CK following the initial bout of damage (Figure 5.1b), with a mean increase of 750% from pre-test values (Table 5.1). Subsequent damage bouts failed to elicit a clinically significant rise in plasma CK, though values peaked outside the normal range, with the greatest being 207% of pre-test following the third damage bout.

Peak CK was significantly different from pre-test values ($p < 0.05$) for the damage / training combined (DC) group following the initial bout of damage (Figure 5.1b) with a mean increase of 690%. Further bouts of damaging exercise resulted in non-significant CK increases, the greatest being 248% of pre-test following the third damage bout, falling just outside the normal range (Table 5.1). The ED group displayed higher mean absolute CK values than the DC group after the initial bout of damage, with both groups exhibiting high standard errors indicative of the degree of inter-subject variability in CK response. ED, undergoing damage only, demonstrated

higher post damage CK values than the group simultaneously training with concentric contractions (DC), though the difference was not statistically significant (Figure 5.1b).

Table 5.1: Mean peak CK response to progressive damaging and non-damaging exercise ($p < 0.05^*$)

| Group | CK (lu/l) | | | | | |
|------------|-----------|-------|------|------|------|------------|
| | Pre-train | dam1 | dam2 | dam3 | dam4 | Post-train |
| C | 82 | 148 | 92 | 116 | 150 | 114 |
| <i>SEM</i> | 21 | 29 | 23 | 30 | 51 | 29 |
| CT | 107 | 189 | 104 | 183 | 126 | 94 |
| <i>SEM</i> | 29 | 64 | 14 | 39 | 28 | 19 |
| ED | 138 | 1036* | 177 | 286 | 257 | 210 |
| <i>SEM</i> | 13 | 370 | 20 | 63 | 48 | 29 |
| DC | 102 | 706* | 237 | 253 | 137 | 232 |
| <i>SEM</i> | 17 | 363 | 65 | 56 | 17 | 97 |

5.2 Isometric Strength.

Figure 5.2a shows isometric strength did not increase over the twelve weeks of data-collection for the Control group who did not train. The CT group, however, increased peak torque from week 1 and continued to improve over the 12 weeks demonstrating a 36% increase from pre to post training ($p > 0.05$) (Table 5.2a).

The ED group exhibited a decrease in force following the initial damage bout, which failed to recover to pre-training values until week 6 (Figure 5.2b). Peak isometric torque did not increase significantly after undergoing 4 bouts of eccentric damage over 12 weeks. The linear increase in force appears to be interrupted with further bouts of damage (ie., at weeks 3, 6, & 9), causing a downward trend in the graph, with force recovering to only 5.5% greater than the pre-training value.

Combined concentric training and eccentric damage (DC) also showed a decrease in force following the initial bout of damage, but recovered to pre-training force by week 3. Subsequent damage bouts appeared to interrupt recovery, as with ED, though a 17.5% increase in force was noted after 12 weeks of training.

Table 5.2: Mean peak isometric torque (Nm) for all four groups pre and post 12 weeks of training ($p < 0.05^*$)

| | GROUP | | | |
|---------------|-------|-----|------|------|
| | C | CT | ED | DC |
| Pre-training | 51.52 | 58 | 64.6 | 57.4 |
| <i>SEM</i> | 5.23 | 9.1 | 7.5 | 13.3 |
| Post-training | 53.12 | 73* | 67.8 | 63.4 |
| <i>SEM</i> | 5.67 | 7.4 | 7.2 | 12.3 |

5.3 Concentric Strength.

The control group showed no change in peak concentric torque at either 50° or 90°/sec over 12 weeks of data-collection, indicating regular strength testing had no effect on dynamic force at the slower contraction velocities (Figure 5.3a & 5.4a). Peak torque at 200°/sec, however displayed an increase from pre-training values of 16.7% after 12 weeks, probably due in part to improved motor learning (Figure 5.5a).

Concentric training produced marked increases in concentric torque over all testing velocities (Table 5.3). 55%, 30%, and 57% increases in peak torque were noted at 50°/sec (Figure 5.3a), 90°/sec (Figure 5.4a), and

200°/sec (Figure 5.5a) respectively, when compared to pre-training values, with the change at 50°/sec being the only statistically significant change. Adaptation to concentric training appeared to result in a greater increase in torque after approximately week 6, where there was an increase in the gradient of the % change in torque vs weeks graph (Figure 5.5a).

The ED group displayed a marked decrease in concentric force 1 week after the initial damaging exercise for all 3 concentric contraction velocities (Figures 5.3b, 5.4b, & 5.5b). Decrements in peak torque were more severe for the slower velocities of 50° and 90°/sec ($p < 0.05$) though improvements of approximately 20% existed at both speeds after the 12 week period. Peak torque at 200°/sec, however, showed no change and appeared to be effected to a greater extent by regular damaging exercise.

Concentric training and eccentric damage combined (DC) resulted in similar decrements in peak torque following the initially damaging exercise (Figures 5.3b, 5.4b, & 5.5b). Increases in peak torque over the 12 week training period were greater for progressively faster contraction speeds with 11%, 15%, and 35% increases being shown for 50°, 90°, & 200°/sec contractions.

Table 5.3: Mean peak torque at 50°, 90°, & 200°/sec for all four groups following 12 weeks of training ($p < 0.05^*$).

| 50°/sec | GROUP | | | |
|---------------|-------|-------|------|------|
| | C | CT | ED | DC |
| Pre-training | 27.03 | 43.3 | 45.8 | 45.4 |
| SEM | 3.7 | 6.39 | 5.5 | 9.24 |
| Post-training | 27.9 | 74.1* | 53.2 | 49.2 |
| SEM | 4.1 | 9.9 | 6.28 | 11.9 |

| | | | | |
|---------------|-------|-------|------|------|
| 90°/sec | | | | |
| Pre-training | 30.15 | 41 | 43.6 | 40.8 |
| SEM | 4.62 | 7.07 | 5.81 | 7.9 |
| Post-training | 30.77 | 59.04 | 50.4 | 46.4 |
| SEM | 5.14 | 4.84 | 6.73 | 8.82 |

| | | | | |
|---------------|-------|------|------|------|
| 200°/sec | | | | |
| Pre-training | 19.28 | 32.3 | 37.4 | 31.2 |
| SEM | 3.81 | 6.07 | 5.35 | 7.73 |
| Post-training | 22.75 | 65.9 | 37 | 37.8 |
| SEM | 4.96 | 5.69 | 4.59 | 6.5 |

5.4 Isometric vs Concentric Torque.

5.4.1 Effects of Damage

Peak isometric torque declined between 2% and 9% in all groups 1 week after the commencement of testing and training indicating some damage may have been incurred in all groups due to the unaccustomed exercise (Figure 5.6a & 5.6b). However the magnitude was greater for the ED and DC groups (8% and 9% respectively).

Initial testing and/or training caused no decrement in concentric torque for the two non-damage groups (CT & C). The control group showed little change in torque over the testing period, though noted changes were proportional to contraction velocity (Figure 5.6a), possibly due to learning and improved activation as mentioned above.

The concentric training group improved markedly in peak torque after 12 weeks with 30-57% increases being noted from pre-training values. There appeared to be little difference in adaptation between isometric and concentric torque, as shown in (Figure 5.6b).

Peak torque declined post damage (week 1) for both ED and DC, and was most notable for concentric torque, particularly at 50° and 90°/sec (Figure 5.6c & 5.6d). Concentric torque appeared to improve most at similar velocities after 12 weeks for ED (18-20%), though the magnitude of the change was far less than that of the CT group. ED displayed less improvement in isometric torque (5.5%) than concentric torque at

intermediate contraction speeds, though torque at 200°/sec showed no change (Figure 5.6c). Conversely, DC increased mostly in isometric torque and at 200°/sec, but to a lesser extent at intermediate velocities (Figure 5.6d).

5.5 Girths

Upper arm girth for all training and/or damage groups exhibited a significant increase of 3-4% from pre-test measurements ($p < 0.05$) (Figure 5.7) though no significant difference existed between the groups. Upper arm girth data was not obtained for the control group, who were expected to produce no change, and my interest was in the variance between training modalities.

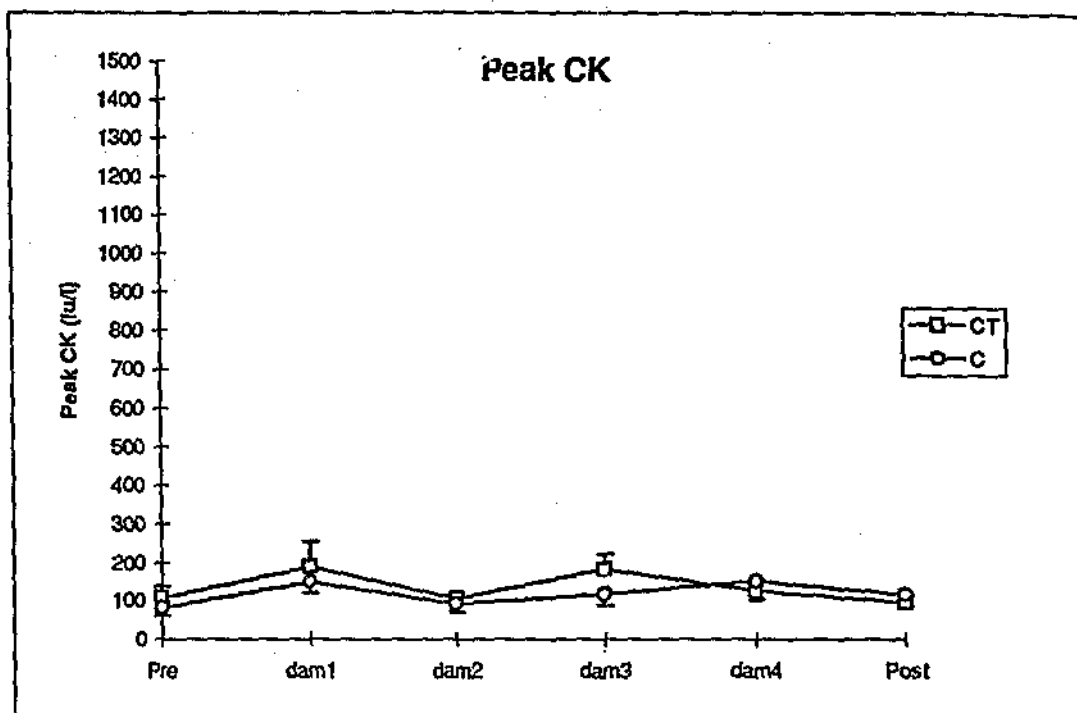


Figure 5.1(a): Mean peak CK for concentric training (CT) and control (C) groups for weeks corresponding to damage for the ED and DC groups (\pm SEM).

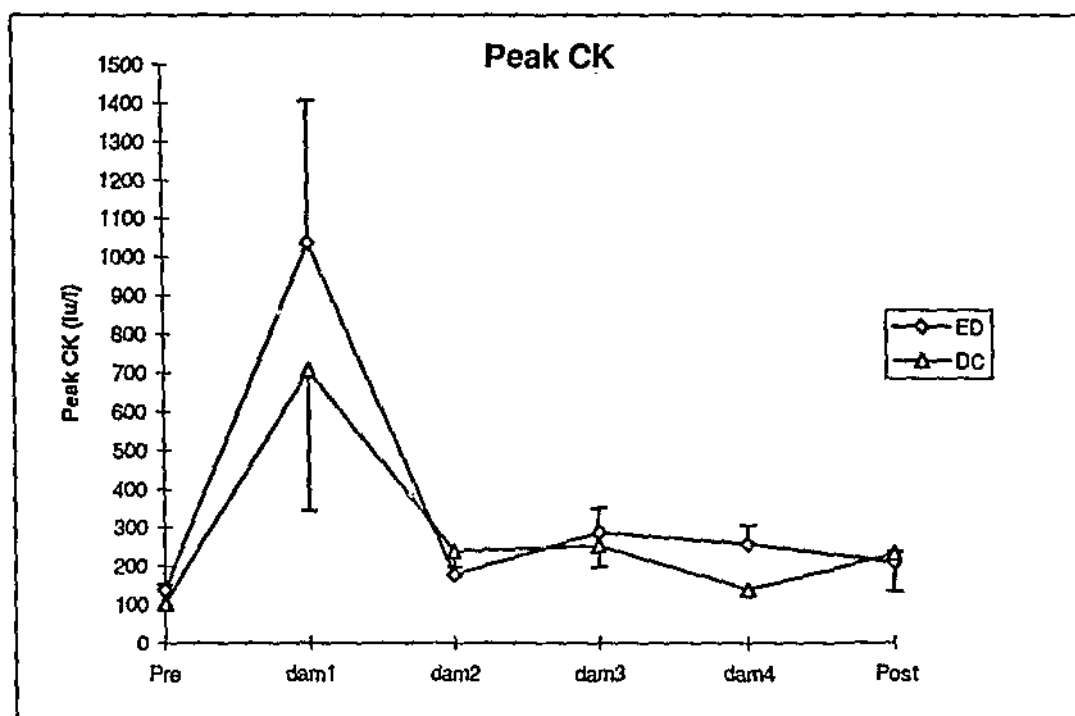


Figure 5.1(b): Mean peak CK for the eccentric damage (ED) and damage/training combined (DC) groups (\pm SEM).

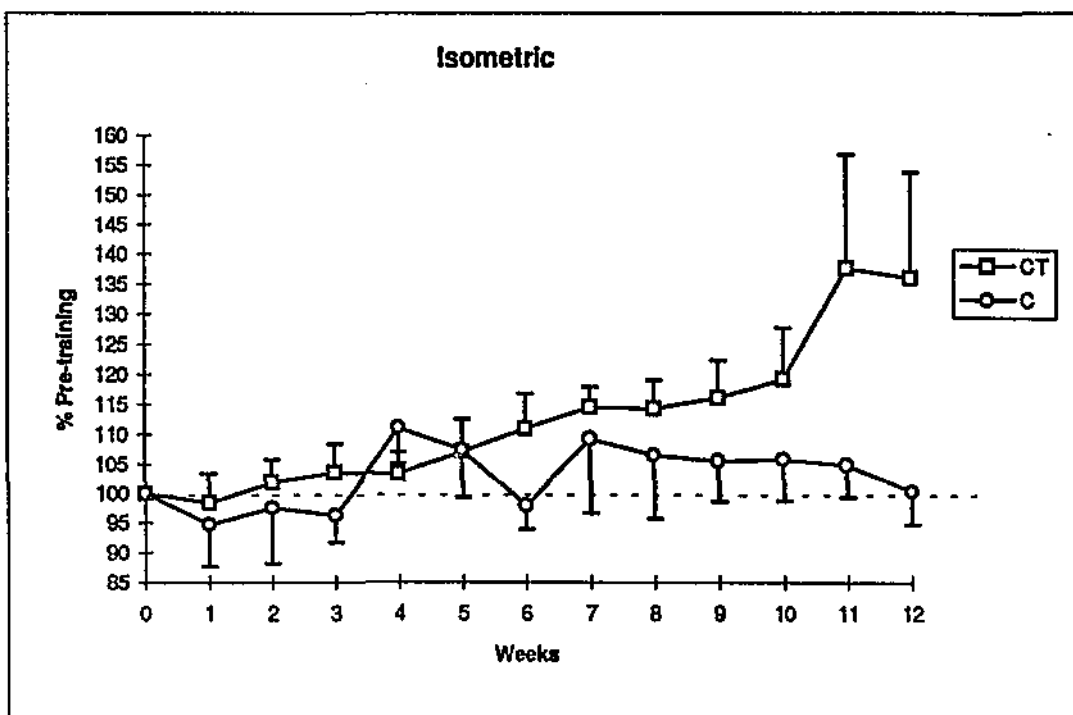


Figure 5.2(a): Normalised mean peak isometric torque for concentric training (CT) and control (C) groups (\pm SEM).

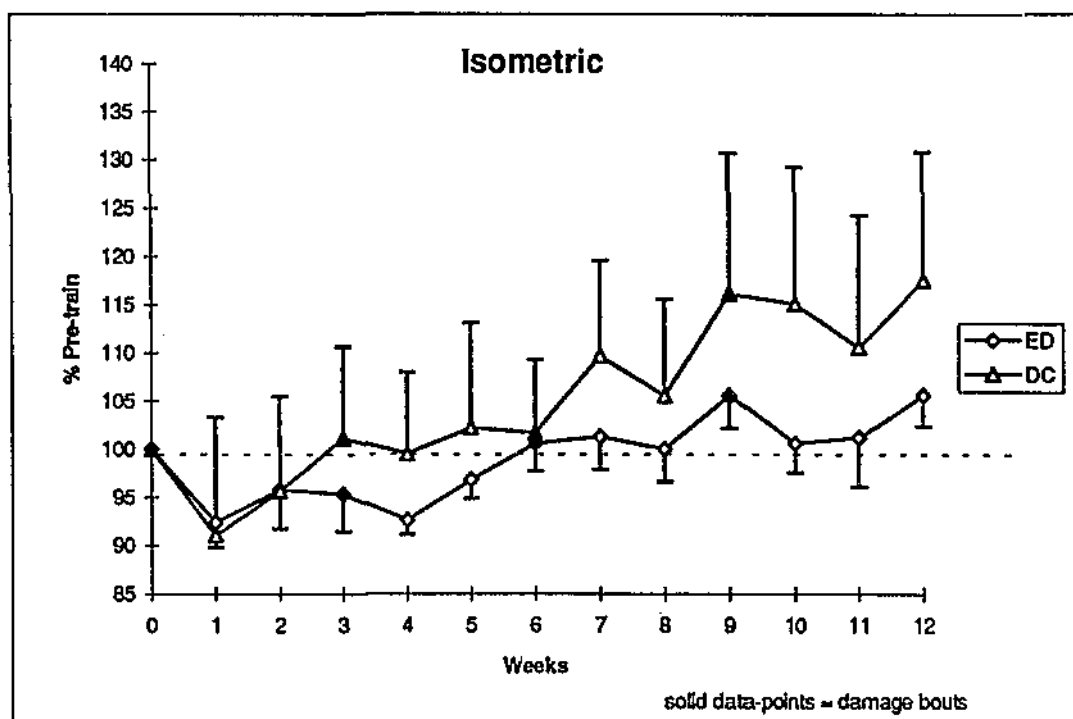


Figure 5.2(b): Normalised mean peak isometric torque for eccentric damage (ED) and training/damage combined (DC) groups (\pm SEM).

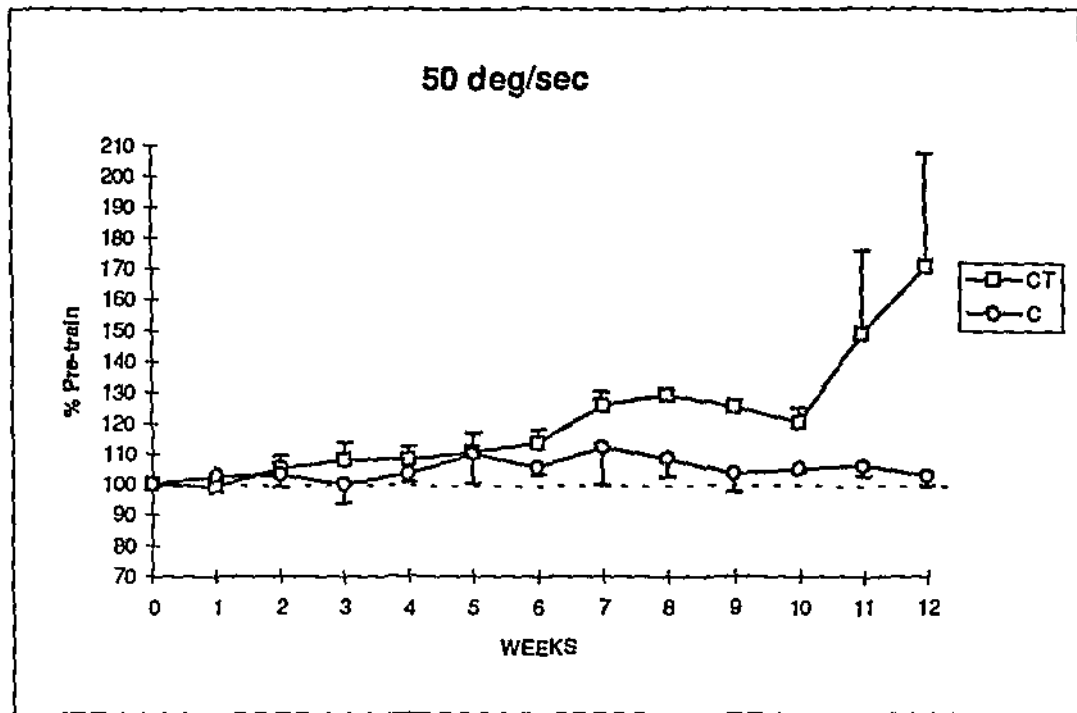


Figure 5.3(a): Normalised mean peak torque at 50 deg/sec for concentric training (CT) and control (C) groups (\pm SEM).

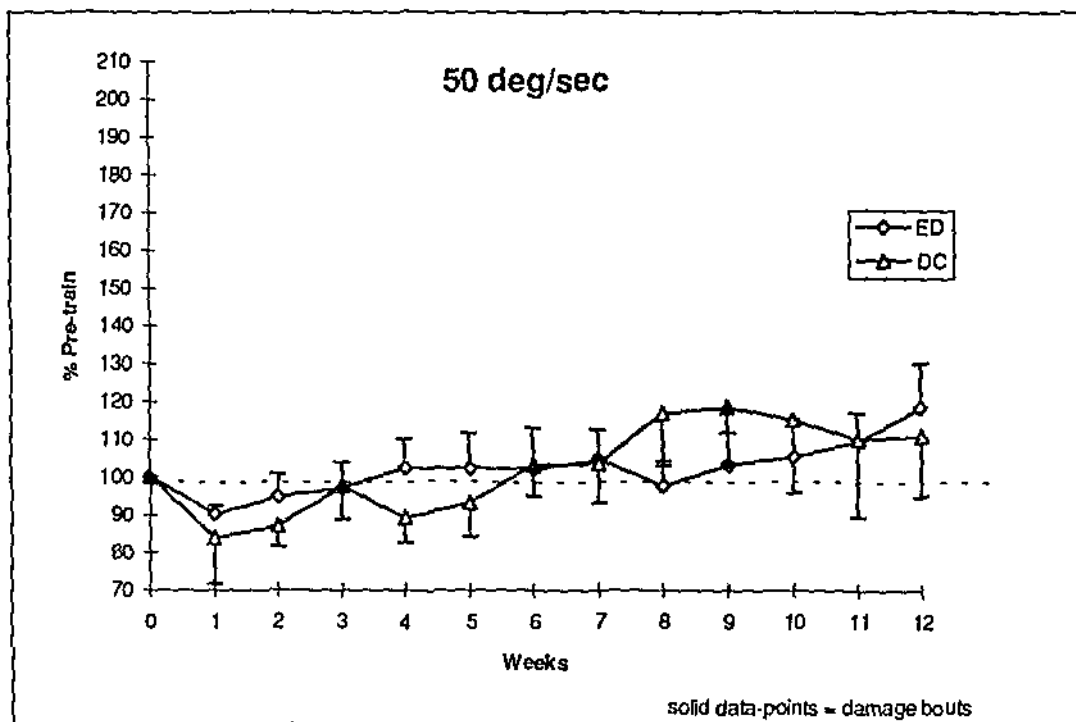


Figure 5.3(b): Normalised mean peak torque at 50 deg/sec for eccentric damage (ED) and training/damage combined (DC) groups (\pm SEM).

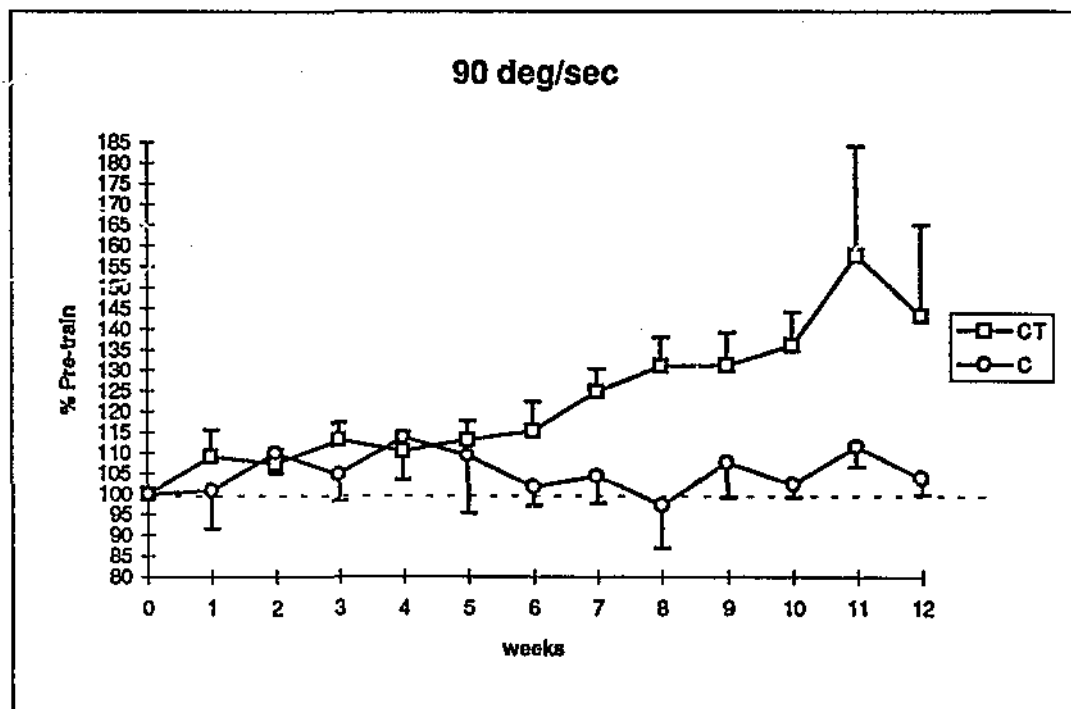


Figure 5.4(a): Normalised mean peak torque at 90 deg/sec for concentric training (CT) and control (C) groups (\pm SEM).

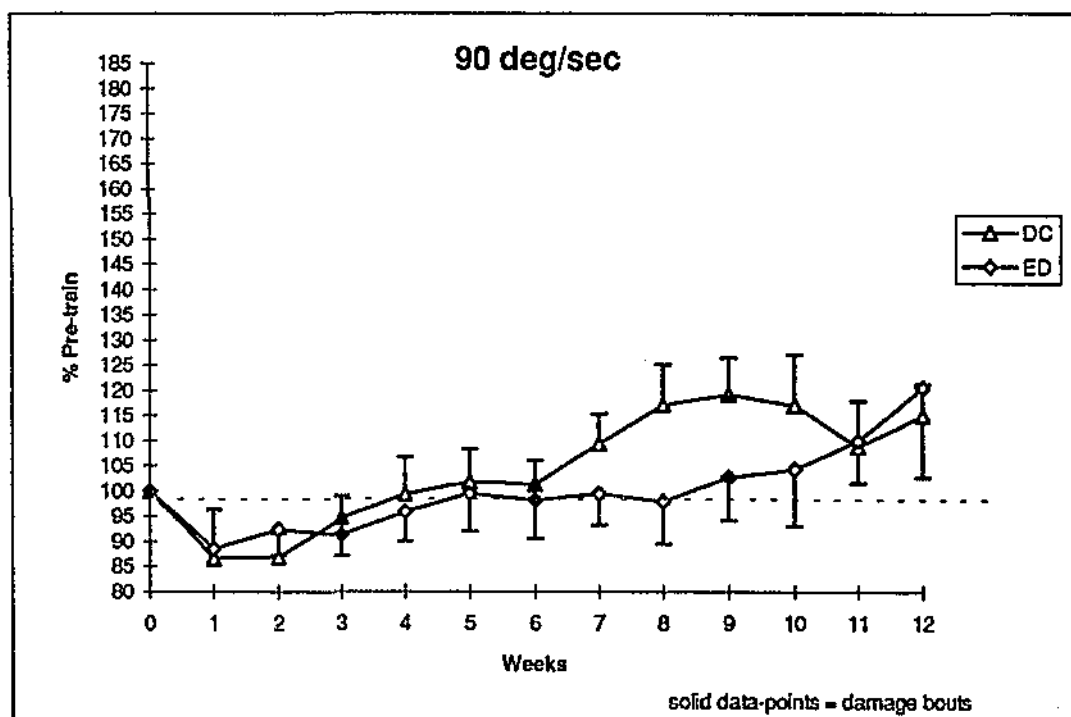


Figure 5.4(b): Normalised mean peak torque at 90 deg/sec for eccentric damage (ED) and training/damage combined (DC) groups (\pm SEM).

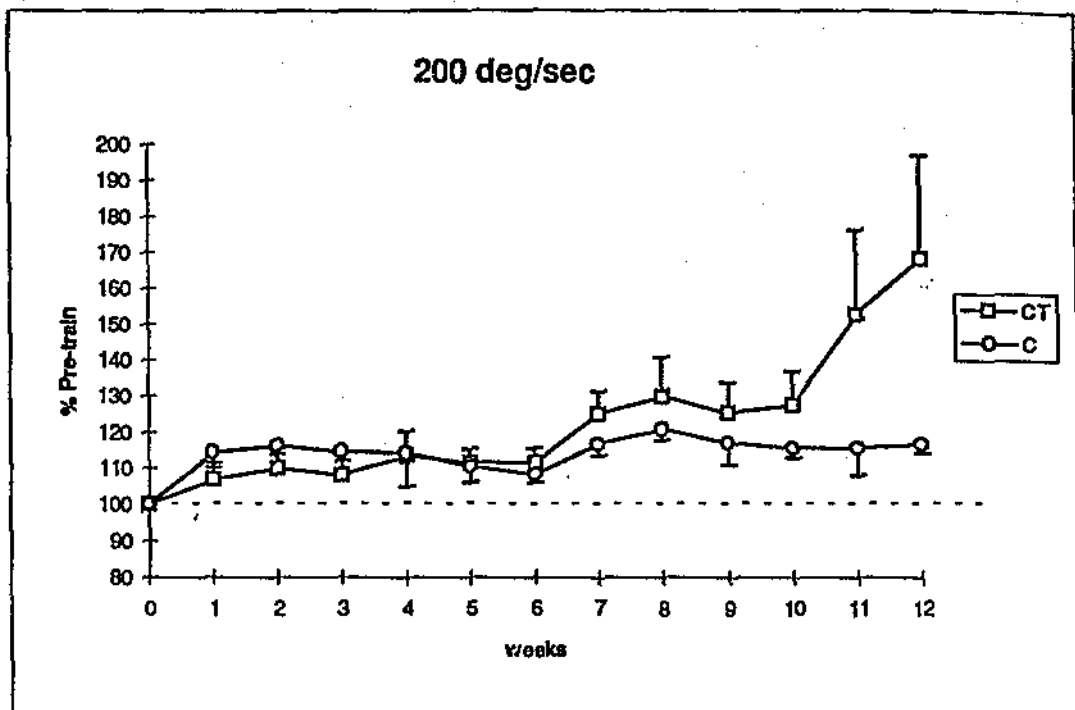


Figure 5.5(a): Normalised mean peak torque at 200 deg/sec for concentric training (CT) and control (C) groups (\pm SEM).

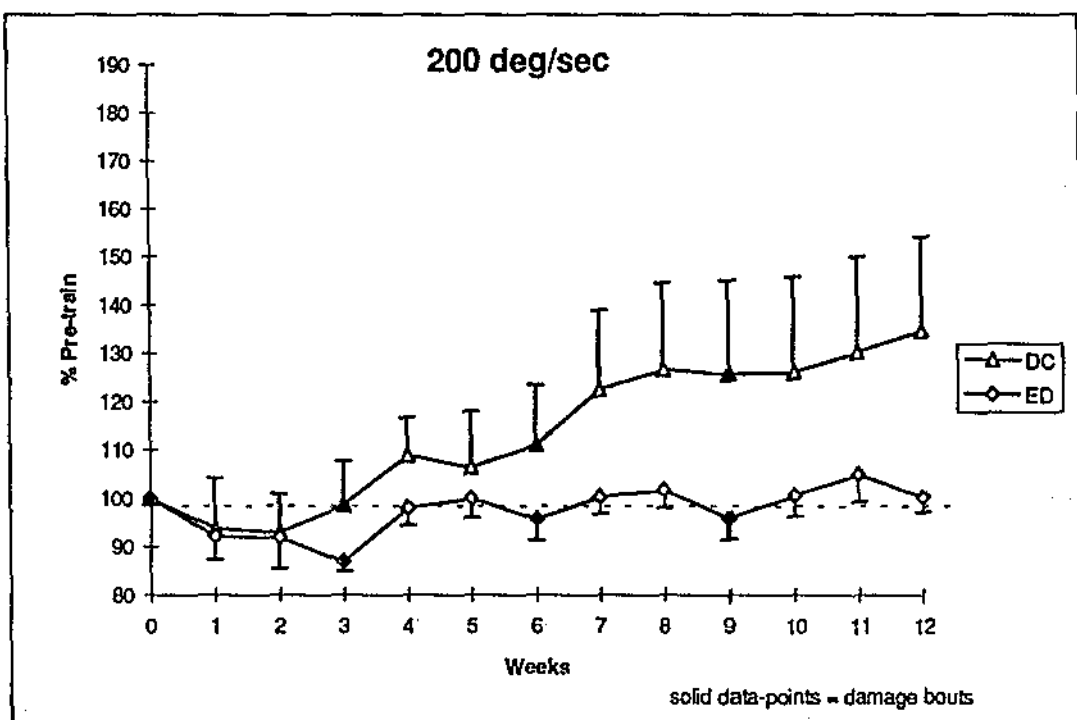


Figure 5.5(b): Normalised mean peak torque at 200 deg/sec for eccentric damage (ED) and training/damage combined (DC) groups (\pm SEM).

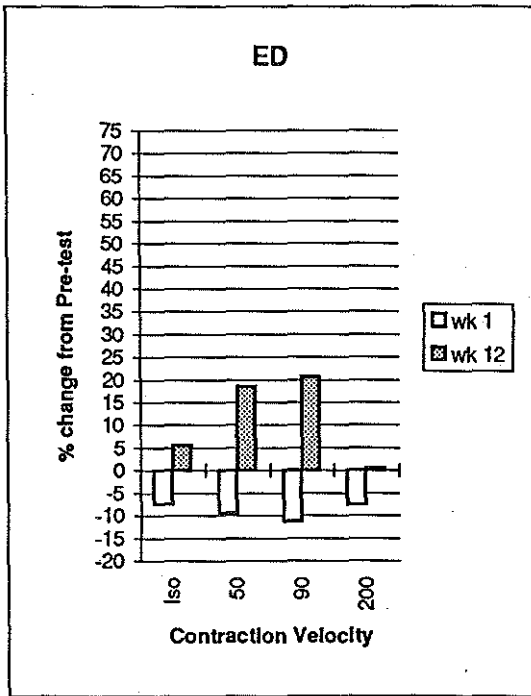


Figure 5.6(a): % change of pre-test torque after 1 and 12 weeks for the control group at each contraction velocity.

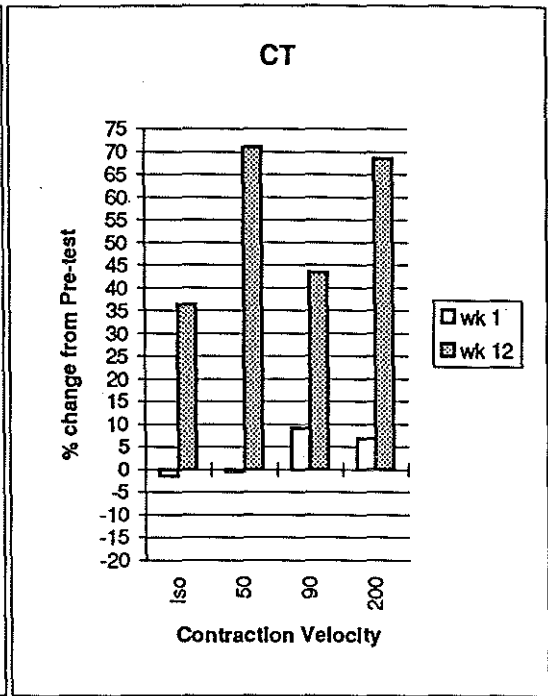


Figure 5.6(b): % change of pre-test torque after 1 and 12 weeks for the concentric training group at each contraction velocity.

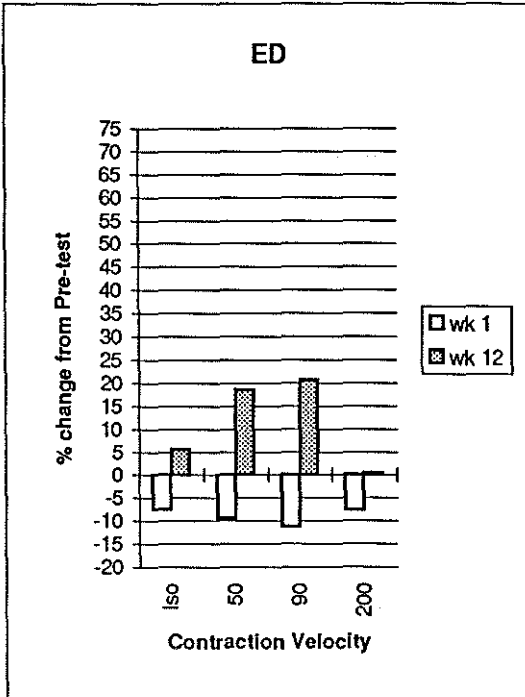


Figure 5.6(c): % change of pre-test torque after 1 and 12 weeks for the eccentric damage group at each contraction velocity.

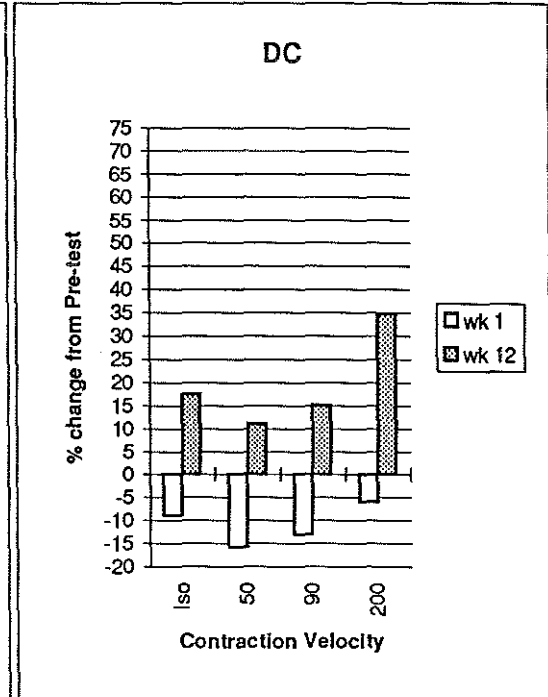


Figure 5.6(d): % change of pre-test torque after 1 and 12 weeks for the combined group at each contraction velocity.

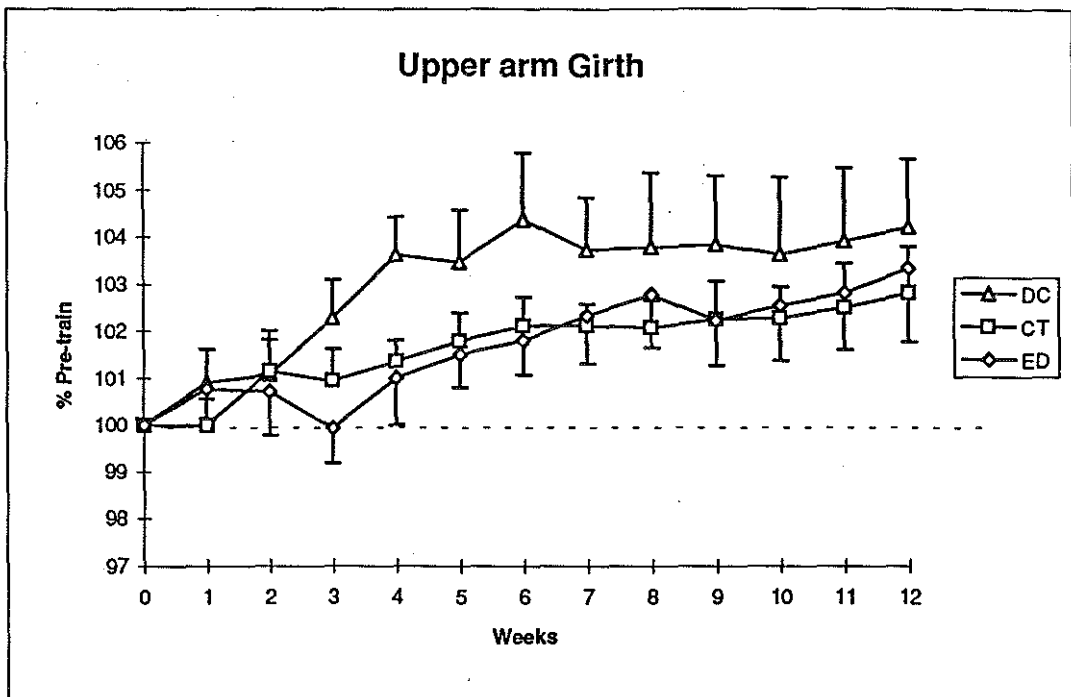


Figure 5.7: Normalised mean upper arm girths over twelve weeks of training for the eccentric damage (ED), concentric training (CT), and training/damage combined (DC) groups (\pm SEM).

CHAPTER SIX

DISCUSSION

The major objective of this study was to investigate the effect of progressive high force training on muscle strength, and to a lesser degree, muscle size. It is well established that muscle undergoes an adaptive response to high force training, though the nature of contractions required to elicit the greatest effect has remained enigmatic, along with questions regarding the role of muscle damage in contributing to the adaptive process. By comparing the response to concentric training, eccentric training, and a combination of the two protocols, the current investigation has aimed to provide some input into this area of uncertainty.

The control group experienced no change in peak torque for either isometric contractions or low velocity concentric contractions, though improvements were seen in the torque generated at the highest velocity (200°/sec). These findings are consistent with the literature and are probably due to learning and an increased ability to coordinate muscles involved in the movement, including those used to stabilise the body (Rutherford & Jones, 1986). Contractions at higher velocities require more skill in order reach peak torque before passing beyond the optimal muscle length. High velocity strength increases unaccompanied by strength increases in isometric and low velocity contractions have been identified by Sale (1988), who suggests repeated attempts to perform rapid contractions results in an increased firing

rate of motor units. Further, though subjects in this experiment familiarised themselves with the testing equipment prior to the commencement of data collection they may still have been in the steeply ascending part of the learning curve during actual testing. This suggestion is supported by the rapid increases in torque at 200°/sec even after only 1 week of testing (Figure 5.6a).

Our first hypothesis was that the CT group would increase peak torque for both isometric and concentric contractions, and increase muscle size over 12 weeks of training compared to the control. Findings of the current study supported this hypothesis, with the CT group displaying 36-70% increases in torque across the different contraction velocities. Concentric torque at 50°/sec showed the only statistically significant increase from pre-training values ($p < 0.05$), though the magnitude of change in force was consistent with that of previous findings (MacDougall, Ward, Sale, & Sutton, 1977; Dons, Bollerup, & Bonde-Petersen, & Hancke, 1979). Muscle size, as assessed by arm girth, increased in the CT group over 12 weeks of training ($p < 0.05$) probably due to longitudinal splitting of myofibrils (Goldspink, 1991) and the adding of myofillaments to the periphery of myofibres (MacDougall, 1991).

Results supported our second hypothesis that the ED group would display increases in peak torque compared to the control. Isometric torque and concentric torque at 200°/sec exhibited only marginal increases after 12 weeks, though concentric torque increased ~20% for the slower contraction velocities, consistent with results reported by Komi and Buskirk (1972).

Muscle size increased over the 12 weeks of data collection ($p < 0.05$), though improvements were disproportionate to changes in peak torque, a phenomenon previously reported (Komi & Buskirk, 1972; Jones et al. 1989).

Hypothesis three stated protocols involving damaging eccentric contractions (ED & DC) would produce larger plasma CK values than the non-damage groups ($p < 0.05$), supporting the findings of Clarkson et al. (1992) and Jones et al. (1986).

Hypothesis four stated there would be no change in CK or peak torque for the control group over 12 weeks of data collection. Though change in girth was not quantified, unaltered muscle strength suggests no reason for an increase in muscle size when considering the relationship between the two variables (Maughan et al. 1983).

Though the current investigation is chiefly concerned with contraction type and training, it is necessary to go to some lengths in establishing that damage has been induced in order to assess its effect on adaptation to concentric training. CK has been shown to be a good indicator that skeletal muscle damage has occurred (Newham et al., 1983a), but is inconsistent in determining the degree of damage sustained, or the muscle mass involved in the damaging exercise (Nosaka et al., 1992; Manfredi et al., 1991). The degree and time course of CK efflux for damaged groups in this study was comparable to that reported previously (Newham et al., 1987; Clarkson et al., 1986), as was the intersubject variability (Nosaka et al., 1992) which ranged from 91 - 2650 lu/l. Although the specific mechanism of exercise induced

enzyme efflux is unknown, it is generally assumed to reflect some form of membrane damage (Newham et al., 1987).

Muscle damage results in force decrements which can last for several weeks after an insult (Clarkson and Trembley, 1988; Newham et al., 1987). ED and DC displayed force decrements of 7-16% 1 week after damage, recovering to pre-test values over 3-6 weeks (Figures 5.2b, 5.3b, 5.4b, & 5.5b). Recovery to pre-test forces took longer in the ED group at the fastest contraction velocity, which is consistent with the findings of Friden et al. (1983) who found force recovered after damaging exercise more slowly at higher velocities of contraction. They reasoned that because type II fibres showed the greatest damage, and are preferentially recruited during maximal contractions, this effected rapid tension development to a greater extent.

Although direct quantification of damage is not possible with the data available from the current investigation, an indirect assessment is possible through CK efflux and post-damage force decrements. Friden et al. (1983) compared force decrement and morphological changes identified through biopsy samples, and found that 8-10% decreases in force accompanied marked broadening of myofibrillar Z-bands, with total disruption in places. Sarcomeres adjacent to the effected Z-bands were either disorganised or supercontracted. As the magnitude of damage and time taken to recover are very similar to the findings of this study, it seems reasonable to assume similar degrees of myofibre disruption.

The protective effect of damaging exercise to damage from further exercise is well substantiated by the findings of this study, both in terms of

diminished CK efflux (Figure 5.1b), and in a decrease in the magnitude of force decrement post damage (figure 5.2b - 5.5b). Earlier studies (Friden, Seger, Sjostrom, & Ekblom, 1983; Komi & Buskirk, 1972) assumed that the apparent force loss was due largely to delayed onset pain preventing subjects from fully activating their muscles. More recent investigations, however, have used electrical stimulation to ensure full motor unit activation, and shown force loss to be the result of changes in contractile elements (Newham et al., 1987). The present results display a notable difference in the time course of recovery of force generation after the first damaging exercise bout compared with the following 3 bouts, being slowest after bout 1. CK efflux shows a similar effect with the greatest response being demonstrated after the first damage bout (Figure 5.1b). It is possible that force loss and the release of CK are the consequences of replacement or removal of irreparably damaged fibres that may have been particularly susceptible to damage, perhaps those nearing the end of their natural cycle of cell turnover (Newham et al., 1987; Armstrong, 1984). Though eccentric damage provided some degree of protection against further insult, the ED group showed only marginal increases in peak torque after 4 damage bouts over 12 weeks. Clearly then, the resynthesis of muscle proteins associated with fibre regeneration was sufficient to recover to pre-damage levels but did not result in fibre hypertrophy as indicated by the torque output.

Concentric training resulted in greater increases in both isometric and concentric torque than the ED group (Figure 5.6b & 5.6 c). Isometric torque displayed 36% and 5.5% increases for CT and ED, and concentric torque for

the three contraction velocities showed an average increase of 60% for CT compared to 13% for ED. Though these findings concur with those reported by MacDougall et al. (1977) in terms of the magnitude of strength development following concentric contractions, they are inconsistent with those reported by Komi and Buskirk (1972) who found eccentric training to elicit greater gains in both isometric and dynamic torque when compared to a concentric training protocol. Komi & Buskirk (1972), however, employed a higher frequency and less intense protocol where subjects trained 4 times per week with only six maximal eccentric contractions per training session. This method may well have resulted in disruption of contractile units, though to a lesser extent than the present study, a notion supported by the relatively more rapid recovery of post damage torque present in Komi and Buskirk's results.

The exact mechanism of response to concentric training is not well known, though undoubtedly is initiated through some combination of tension development and associated metabolic and hormonal factors (Jones et al., 1989). Early responses can be largely attributed to improved neural activation from learning, where the correct sequence of muscle contractions is laid down as a motor pattern in the central nervous system (Jones et al., 1989; Rutherford & Jones, 1986; Moritani & deVries, 1979), a phenomenon which would explain the early response of torque for CT & C at the fastest measured contraction speed (Figure 5.5a).

From the response of the ED group it appears that eccentric damage attenuates the response to concentric training. The DC group showed a 17%

increase in isometric torque, whereas concentric torque appeared to improve most at the highest contraction velocities (Figure 5.6d), though the magnitude of force changes were considerably less than that of concentric training only. Results suggest severe damage does not aid concentric training in increasing peak torque, at least over the time frame of this investigation. The similar increase in arm girth of 3-4% from pre-training ($p < 0.05$) for all 3 training groups is perhaps surprising. One possible explanation may be that muscle damage may result in a fibre hypertrophy which is not reflected in increased force output. Thus there may be a reduction in muscle force per cross-sectional area as previously reported by Komi and Buskirk (1972). Muscle hypertrophy associated with a reduced force per cross-sectional area has also been demonstrated in a dystrophic (MDX) mouse strain (Sacco, Jones, Dick, & Vrbova, 1992). The inability to produce high intramuscular forces in the weeks after damage may, in effect, decrease the stimulus necessary for adaptation, and may provide a further explanation for the lack of improvement in the DC group.

The current study indicates a concentric training protocol may elicit the greatest gains in peak torque, at least in the short term. The importance of some degree of muscle damage in contributing to the initiation of adaptation is overwhelmingly supported in the literature, however, and cannot be discounted. The extent of damage sustained may be important, and it would be interesting to conduct a similar study incurring less damage, as evidenced through changes in plasma CK and post-damage torque decrements. A protocol involving a bout of damaging exercise prior to the commencement of

training for a DC and a CT group would provide further insight into the effect of concurrent eccentric and concentric training by affording the combined group with a protection effect from the potentially debilitating force loss early in training. Clearly, more research is needed to address unanswered questions regarding the importance of damage and/or high force production in eliciting maximal gains in muscle strength and hypertrophy.

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Appendix A Informed Consent Form

INFORMED CONSENT FORM

The aim of this research is to determine if the damage/repair process involved with rigorous physical exercise is necessary to stimulate the growth and development of muscle. The results of this investigation have the potential to effect the design of training programs used to promote increases in muscle size, strength, and power.

The study will be conducted over a twelve week period at the Australian Neuromuscular Research Institute, Queen Elizabeth II Medical Hospital. As a subject, you will be randomly assigned to one of four groups; three training, and one control group. Training will consist of either eccentric 'damage', where 10 sets of 12 maximal contractions will be performed at three weekly intervals causing micro-damage to the muscle, or 6 sets of 10 maximal contractions conducted three times/week for the twelve weeks. The third training group will use a combination of the two above methods. The biceps of your non-preferred arm will be trained during the investigation, and some localised discomfort may be experienced in the week following eccentric damage.

Blood samples will taken over 4 days at three weekly intervals (1 pre, and 3 post 'damage'), and will involve 30 microlitre whole blood samples being taken from a fingerprick, and analysed using a spectrophotometer (Reflitron).

All testing and training information is confidential and will only be used for the purpose of this study. Information will be kept under lock and key, with your data identifiable only through a number coding system held by the principal researchers. Data used for analysis will not include any names.

We ask that you refrain from making any major changes to diet or exercise habits throughout the twelve weeks comprising the study period. Participation in the study is voluntary and you may withdraw at any time, for any reason.

Any questions concerning the study can be directed to:

Haydn McDonald [REDACTED]
Principal Investigator.

Dr Colin James [REDACTED]
Exercise Physiologist, Human Movement Dept., Edith Cowan University.

I (the participant) have read the informed consent above, and any questions have been answered to my satisfaction. I agree to participate in this study realising that I may withdraw at any time.

I agree that the research data obtained from this study may be published, provided I am not identifiable.

Date Participant

Investigator 69