A 12 YEAR FOLLOW-UP OF ANKLE MUSCLE FUNCTION IN OLDER ADULTS

A 12 YEAR FOLLOW-UP STUDY OF ANKLE MUSCLE FUNCTION IN OLDER ADULTS

By

KAREN J. WINEGARD, B.P.E.

A Thesis

Submitted to the School of Graduate Studies

in Partial Fulfilment of the Requirements for the Degree Master of Science

McMaster University

MASTER OF SCIENCE (1995) (Kinesiology)	McMASTER UNIVERSITY Hamilton, Ontario
TITLE:	A 12 Year Follow-up Study of Ankle Muscle Function in Older Adults
AUTHOR:	Karen J. Winegard, B.P.E. (McMaster University)
SUPERVISOR:	Audrey L. Hicks, Ph. D.
SUPERVISORY COMMITTEE:	Anthony A. Vandervoort, Ph. D. Digby G. Sale, Ph. D. Alan J. McComas, M.D.
NUMBER OF PAGES:	x, 101

This thesis is dedicated to Ron, Erin, and Brandon,

without whom I could never have fulfilled the requirements of this thesis. Their never-ending encouragement, love and understanding enabled me to accomplish this life-long goal.

I would also like to dedicate this thesis to the memory of Joan Heimbecker,

who was tragically taken away from us on March 30th 1994,
without warning,
without goodbyes.

PREFACE

This thesis is presented in two chapters. Chapter I begins with a brief overview of aging theories, followed by a literature review of age-related changes in skeletal muscle. Chapter II presents the thesis research related to longitudinal muscle function changes in older adults after a 12 year follow-up period. Chapter II is presented in manuscript style suitable for publication.

ACKNOWLEDGEMENTS

Science is organized knowledge.
Wisdom is organized life.
--Immanual Kant

I would like to thank everyone who has made it possible for me to complete my thesis. I am grateful to my advisor, Dr. Audrey Hicks, for all the support and guidance she gave me throughout this endeavor. I am indebted to Dr. Tony Vandervoort, for giving me the opportunity to follow-up his Ph.D. thesis as part of my Master of Science degree. His assistance and availability allowed me to see this venture through to its completion. To the rest of my thesis committee, Drs. Digby Sale and Alan McComas, I would like to express my gratitude for their interest and knowledge in this research area.

I value the assistance of John Moroz, without whose technical knowledge, the testing would never have gone as smoothly. I appreciate Mary Cleland's calming support and administrative assistance during the past 2 years. Also, I will always cherish the enthusiasm and commitment of the Seniors who participated in this study.

I am grateful to the students in this program, who have made my experience as a graduate student a memorable one. Their unlimited support and friendship will always hold a special place in my heart.

To everyone else who assisted me along the way, thank-you for showing me that it is never too late to learn.

TABLE OF CONTENTS

APPENDICES

LIST OF TABLES

CHAPTER II: PAGE

LIST OF FIGURES

CHAPTER II: PAGE

CHAPTER I

REVIEW OF LITERATURE

1.1 INTRODUCTION

The study of the biological and physiological changes associated with aging has become an increasingly important and popular research area in recent years. Perhaps the main reason for the increased attention that aging is receiving is the growing proportion of people over 65 years of age, with 20% of the North American population predicted to be in that age category by the year 2025 (Booth et al., 1994). Along with this increase in the number of elderly persons, a higher percentage of people are living to be over 80 years of age (Spence, 1989).

The processes of aging begin at conception and the term is often associated with senescence, that period usually in the third trimester of adult life when the manifestations of aging become apparent (Baker & Martin, 1994). It is well established that aging is characterized by a general reduction in functional capacities and by structural changes in the body. What is less clear, however, is what is causing the various changes that are associated with aging.

This chapter begins with a brief overview of some of the theories of aging, followed by a review of the current realm of knowledge regarding age-related changes in skeletal muscle.

1.2 THEORIES OF AGING

Although many theories have been suggested to account for the manner in which aging comes about, at present there is no universally agreed upon hypothesis. Proposed thEeories include genetic programs, wear-and-tear theory, cellular senescence, cross-linkage theory, free radical theory, hormonal deficiencies, and the autoimmune theory (Cunningham & Brookbank, 1988; Timiras, 1988; Kenney, 1989; Spence, 1989; Cristofalo, 1990; Baker & Martin, 1994; Vandervoort, 1995). Two general conceptual approaches warrant consideration: first, aging is genetically programmed from development to death; and second, aging results from the gradual breakdown of cellular function (Vandervoort, 1995).

1.2.1 Genetic Factors

According to the genetic theories, the aging process is genetically controlled and programmed (Cristofalo, 1990). Aging is believed to be the result of one or more harmful genes within each organism becoming active late in life. These active genes lead to death of the organism by altering its physiology (Spence, 1989). As well, genetic factors may play a role in both the shortening and lengthening of life span, since increases in life expectancy have been found in the offspring of long-lived parents (Olshansky et al., 1990).

Alterations in genes may contribute to many age-associated diseases and disabilities. For example, the risk of developing late-onset Alzheimer's disease is increased due to the presence of certain alleles of the apolipoprotein E gene (Brousseau

et al., 1994). Another example involves osteoporosis, the Vitamin D receptor and bone density. Recent studies have genetically linked bone density to a Vitamin D receptor genotype (Morrison et al., 1994). Individuals with this low bone density BB genotype are more likely to show rapid aging of bone and subsequent osteoporotic bone (Baker & Martin, 1994).

1.2.2 Wear-and-Tear Theory

Another theory which promotes the idea that aging is a programmed process is the wear-and-tear theory. This theory proposes that each animal cell has a predetermined amount of metabolic energy available to it, and that the rate at which this energy is used determines the length of the animal's life (Spence, 1989). As well, this theory suggests that the accumulation of harmful metabolism by-products and faulty enzymes due to random errors, also contributes to changes associated with aging (Timiras, 1988).

1.2.3 Cellular Senescence

Important to the study of cellular senescence is the well-established notion that cells can divide only a limited number of times in cell culture (Baker & Martin, 1994). Recently, studies have shown that each time somatic cells divide, telomeric (end) regions of chromosomes shorten (Harley, 1991). This process eliminates sequences of DNA that are needed to stabilize chromosome structure; therefore, the loss of these sequences beyond a certain point could cause cellular senescence.

1.2.4 Cross-linkage Theory

The cross-linkage theory proposes that the structure of some proteins in cells is irreversibly altered by the formation of new cross-links. This leads to changes in protein function and causes the failure of the cells of tissues and organs. For example, the loss of elasticity in aging blood vessels has been associated with structural changes in the protein collagen (Spence, 1989). Collagen in tendons and connective tissue also becomes cross-linked by bonds between molecules and is less flexible with aging (Cunningham & Brookbank, 1983). As well, aging skin loses its elasticity due to the replacement of elastic fibres with collagen (Spence, 1989).

1.2.5 Free Radical Theory

Free radicals are believed to be key players in the age related changes seen at the tissue, cellular, and subcellular levels (Kenney, 1989). They occur normally as by-products of various metabolic processes involving interactions with oxygen. When free radicals react chemically with other substances, especially fats, they cause cell membranes to become more permeable to substances, and this can change the normal functioning of the cell (Spence, 1989).

Over time, the accumulation of free radicals in cells may lead to the changes commonly associated with aging. Left unchecked, their potential to cause cellular damage also increases. Antioxidants, which are compounds that prevent oxidation from occurring, may theoretically prolong the lifespan of a person by eliminating some of the

free radicals in their cells. However, the benefits of antioxidants such as Vitamin C and E, have yet to be proven (Kenney, 1989; Spence, 1989).

1.2.6 Hormonal Deficiencies

With aging, a decline is seen in the levels of circulating hormones. For example, the rapid loss of estrogen production following menopause leads to changes in body composition, bone loss, altered lipid metabolism, and increased risk of osteoporosis and cardiovascular disease (Meites, 1993). As well, the decrease in the level of growth hormone is followed by a significant loss of muscle mass and increased fat deposition (Corpas et al., 1993).

The prostate is also affected by reduced hormonal levels. Without testosterone, the cells of the prostate undergoes a process known as apoptosis, which causes the death of its cells (Williams & Smith, 1993). The active process of apoptotis has known inhibitory and stimulatory factors, external and internal to the cell. The role that apoptosis plays in the aging process is unknown. However, drug treatment of this active process, may prevent and treat a number of diseases such as cancer, AIDS, autoimmune diseases, and degenerative diseases of the central nervous system, and age-related cellular losses (Baker & Martin, 1994).

1.2.7 Autoimmune Theory

According to the autoimmune theory, the immune system's ability to discriminate between foreign proteins and the body's own proteins, decreases with aging. This results

in the formation of antibodies which attack the body's own proteins, causing the immune system to attack and destroy body cells (Spence, 1989).

There are two categories of autoimmune theories: those that suggest that with aging, new antigens may result from mutations that lead to altered RNA, DNA, and other proteins; and those that suggest aging is the result of an increase in autoimmune reactions caused by changes to the antibody molecules (Timiras, 1988; Spence, 1989).

Since it is apparent that there are multiple causes of aging, no one theory can adequately explain the aging process. Overall, aging theories suggest that aging is caused by damage to molecules, cells, and eventually tissues and organ systems from both endogenous and exogenous factors (Baker & Martin, 1994). Therefore, the process of aging is best interpreted when several of these theories are combined.

1.3 AGE-RELATED CHANGES IN HUMAN SKELETAL MUSCLE

Human movement ultimately depends on the conversion of centrally processed commands into force, and skeletal muscle is the vital effector organ of the motor system that is responsible for this action. The motor unit is the final common pathway of the central and peripheral motor commands, and it consists of a single motoneuron and all the muscle cells it innervates (Vandervoort, 1992). Therefore, maximum human strength can be thought of as a motor skill, reliant on an individual's motor units and his or her ability to activate groups of them together in the most efficient manner.

1.3.1 Aging And Muscle Strength

Common features of skeletal muscles in aged men and women are atrophy and weakness, and this area has been the topic of extensive research in the last two decades (Grimby & Saltin, 1983; Vandervoort & McComas, 1986; Aoyagi & Shephard, 1992; Vandervoort, 1992; Doherty et al., 1993a; Lexell, 1993; Rogers & Evans, 1993; Porter et al., 1995). The strength of a variety of limb muscles of young, middle-aged, and old adults has been studied. It has been well established that decreases in voluntary strength do not become apparent until after the age of around 60 years (Vandervoort, 1995). Healthy adults in their seventh and eighth decades have on average 20 to 40% less voluntary strength than young adults, and the very old show 50% or greater reductions (Porter et al., 1995). The size of the age effect varies from muscle to muscle, and sedentary populations tend to show greater strength loss in the lower limb muscles than upper (Vandervoort, 1992). Despite differences in muscle groups and study designs, similar age-related trends are seen in men and women.

The cause of the decline in muscle strength with aging remains unresolved. Particular attention has been paid to the following mechanisms: the reduction in muscle mass, volume and cross-sectional area; changes in the muscle morphology; changes in the nervous system and motor units; changes in the neuromuscular junction; and changes in muscle contractile properties and their effects on the performance of the neuromuscular system. Another contributing factor may be the decline in physical activity levels that is often associated with aging.

1.3.2 Reduction in Muscle Mass, Volume and Cross-sectional Area

Vandervoort & McComas (1986) attributed the decline in ankle muscle strength found in healthy older adults to a reduction in excitable muscle mass. Using the twitch interpolation technique (Belanger & McComas, 1981), they assessed descending motor drive by applying a brief percutaneous electrical shock to the motor nerve during a maximal voluntary contraction. The majority of the older adults, ranging from 60 to 100 years, were able to fully activate their ankle muscles, since a superimposed twitch stimulus did not significantly increase their volitional force. Because the elderly could generally achieve full motor unit activation, the decline in muscle strength with aging was associated with a reduction in muscle volume or mass. However, since this was an isometric, single joint task, it should be noted that central nervous system coordination could still be an important factor in dynamic strength manoeuvres which use multiple muscle groups (Sale, 1988).

The measurement of total creatinine excretion has provided indirect evidence that muscle mass is reduced with age. Tzankoff & Norris (1977) found that in their sample of 959 healthy persons (20-97 yrs), muscle mass was reduced by about 1/3 over the age of 50 years. A further reduction of 15% between the ages of 70 to 80 years was found when Grimby et al. (1982) used a whole-body counting of potassium technique to determine muscle mass.

Using radiological imaging techniques, muscle cross-sectional areas have been indirectly estimated. Young et al. (1984; 1985) used ultrasound scanning to illustrate that the total knee extensor cross-sectional area of older women (71-78 yrs) was 33% less

than young (20-29 yrs) women, and the cross-sectional area of older men (70-79 yrs) was 25% less than young (20-29 yrs) men.

Computed tomography (CT) scanning has shown age-associated reductions in cross-sectional areas for the quadriceps femoris (Klitgaard et al., 1990a; Overend et al., 1992), brachial biceps and triceps (Rice et al., 1989; 1990), and plantarflexor muscles (Rice et al., 1989; 1990). Klitgaard et al., (1990a) found that the cross-sectional area of quadriceps femoris and elbow flexors of elderly (68 $\pm$ 0.5 yrs) sedentary subjects was 24% and 20% lower, respectively, than young (28 $\pm$ 0.1 yrs) sedentary controls.

Along with the loss in muscle mass, Rice et al. (1989; 1990) and Overend et al. (1992) found increases in non-muscle tissue such as fat and connective tissue, within the boundaries of older muscles. Rice et al. (1989) reported reductions in cross-sectional areas for the plantarflexors (35%), biceps (36%), and triceps (28%) of aged (65-90 yrs) compared to young (25-38 yrs) men; along with a 81% increase in non-muscle tissue in the plantarflexor compartments, 27% in arm flexors, and 45% in arm extensors. Overend et al. (1992) reported reductions in cross-sectional area of 27% for the quadriceps and 18% for the hamstring muscles of elderly (65-77 yrs) compared to young (19-34 yrs) men, accompanied by increases in non-muscle tissue of 59% for the quadriceps and 127% for the hamstrings. CT scans revealed decreases in actual quadriceps and hamstring muscle masses, and increases in subcutaneous fat and non-muscle tissue in older men, despite no difference in overall thigh girth compared to young men. Therefore, because of the increases in fat and connective tissue found with

aging, the reduction in muscle contractile tissue may exceed the actual reduction in muscle volume and cross-sectional area (Porter et al., 1995).

In order to gain a better understanding of the mechanisms responsible for the age-related losses in muscle mass, direct measurement techniques such as whole muscle cross-sections obtained postmortem have been used. However, due to the technical and ethical constraints involved, these studies are very limited. Lexell et al. (1988) studied cross-sections of autopsied whole vastus lateralis muscle from 43 previously healthy men between 15 and 83 years of age, and found a 40% decrease in muscle cross-sectional area. They also found that the reduction in muscle area begins as early as 25 years, approximately 10% of the muscle area is lost by the age of 50, thereafter the loss accelerates, and by 80 years almost half of the muscle is wasted. Although these findings are limited to one limb muscle, we can assume that other human muscles could be affected in a similar way.

1.3.3 Changes in Muscle Morphology

Muscle fibre size

The vastus lateralis of the quadriceps muscle has been the muscle most often used in morphological studies of aging. Consistently, the overall conclusion is that Type II (fast-twitch) fibre size decreases with increasing age, while Type I (slow-twitch) fibre size remains relatively less affected (Grimby et al., 1982; Lexell et al., 1988). Grimby et al. (1982) found a 10-30% decrease in Type II fibre size in vastus lateralis muscle samples from subjects 78-97 years of age, compared to younger subjects drawn from

population studies. Lexell et al. (1988) found a 26% reduction in Type II fibre size between the ages of 20 and 80 yrs. Therefore, the decrease in size of Type II fibres with age accounts for part of the decrease in muscle size and strength that occurs with aging (Rogers & Evans, 1993).

Other studies have also shown that Type IIb muscle fibres may be more susceptible to the effects of aging compared to Type IIa fibres. Coggan et al., (1992) compared both kinds of Type II fibres in young (20-29 yrs) and old (60-69 yrs) men and women. The reductions for Type IIa and IIb fibres were 13% and 22%, respectively for males, and 24% and 30%, respectively for females.

Muscle fibre number

Whole muscle cross-section techniques by Lexell et al. (1983; 1986; 1988) have provided the most conclusive information on the loss of muscle fibres with aging. In their original study, they found that the number of fibres in whole medial vastus lateralis muscles was 25% fewer in older (70-73 yrs) men compared to young (19-37 yrs) men. Using larger sample sizes, Lexell et al. (1986; 1988) assessed the relationship between age and the total number of muscle fibres. They concluded that both Type I and Type II are lost to the same extent, that the loss of fibres begins at around 25 years of age, and that the total fibre number is reduced by 39% between the ages of 20 and 80 years.

Distribution of fibre types

Initially, studies on the effect of aging on fibre types stated that there is a shift toward a distribution with a higher percentage of Type I fibres and a lower percentage of Type II fibres. Larsson & Karlsson (1978) studied the quadriceps muscle of 50 men,

22-65 years of age, and found that subjects in the 20-29 year old group had 39% Type I muscle fibres, while the 60-65 year old subjects had 66% Type I fibres.

More recent studies, however, contradict these earlier findings (Grimby et al., 1982; Essen-Gustavsson & Borges (1986). Grimby et al. (1982) examined the right vastus lateralis muscles of 24 men and women (78-81 yrs), and found that the fibre distribution did not differ from that of younger subjects drawn from population studies. Essen-Gustavsson & Borges (1986) also found that no change occurred in the fibre composition of the vastus lateralis muscle from ages 20-70 years.

The majority of earlier studies used the muscle biopsy technique, which is limited to sampling only a small portion of a muscle at one time. To overcome this problem, Lexell and co-workers (1983; 1986; 1988) have excised whole vastus lateralis muscles at autopsy, to measure the fibre type distribution.

Lexell et al. (1983) reported no significant differences in the mean proportion of Type I fibres in the vastus lateralis muscle of young and old males. In 1986, Lexell et al. found that young men (15-34 yrs) had 49% Type I fibres, middle-aged men (49-56 yrs) had 52% Type I fibres, and the oldest men (71-83 yrs) had 51% Type I fibres.

However, fibre type properties are not static, and the histochemical profile of muscle fibres can change throughout life. Electrophoretic analysis of single muscle fibres (Klitgaard et al., 1990b) has shown that older subjects have a greater proportion of fibres with co-expression of myosin heavy chain Type I and IIa, and Type IIa and IIb. The authors suggest that this may reflect an ongoing transition process or "dynamic equilibrium" between the fibre populations, due to denervation and/or disuse.

1.3.4 Changes in the Nervous System and Motor Units

Fibre type arrangement

Extensive neuropathic changes are frequently observed in muscles of very old individuals. Neurogenic alterations such as denervation and reinnervation, could be due to impaired motor neurons in the spinal cord, or a degeneration of peripheral nerve axons and/or the myelin sheath. Repeated denervation and reinnervation results in fibre type grouping (Campbell el al. 1973; Lexell et al. 1983; Stalberg et al., 1989), and significant increases in fibre type grouping have been shown in studies of fibre type arrangement in muscles at various ages. In their study of men and women (20-70 yrs), Stalberg et al. (1989) revealed evidence of reinnervation, suggesting preceding denervation and subsequent loss of motor units. They found that the fibre density (mean fibre number per motor unit) of the 60-70 year olds had increased 14% and 20% for males and females, respectively.

Lexell et al. (1986) argued that denervation and reinnervation of fibres starts before the age of 50, and that it is probably caused by a continuous loss of motor neurons in the spinal cord. A later study by Lexell & Downham (1991) has confirmed these findings and concluded that the fibre type arrangement in young muscles up to 25 years may show segregation/ungrouping; the fibre type arrangement is random from 30-60 years; and an excess of enclosed fibres of both Type I and Type II fibres are found after the age of 60 years. Therefore, muscle fibre populations undergo continuous denervation and reinnervation with aging, due to a progressive loss of functioning motor units.

When the capacity of muscle fibres to reinnervate is depleted, such that muscle fibres are permanently denervated and thereby lost, fibres are replaced with fat and fibrous tissue. This demonstrates why a much smaller portion of contractile tissue is found in muscles of older individuals compared to muscles of younger individuals (Lexell et al., 1988; Rice et al., 1989; 1990).

Motor units

Based on neurophysiological studies, the estimated number of motor units has been found to decrease with age (Campbell et al., 1973; Doherty et al., 1993b; de Koning et al., 1988). Campbell et al. (1973) found no decline in the mean number of motor units in extensor digitorum brevis muscles between 3 and 58 years, however, beyond the age of 60, many subjects exhibited a loss of functioning motor units. The study by Doherty et al. (1993b) suggests that men and women in the seventh decade of life have approximately one-half the number of motor units of young adults (21-38 yrs) in the biceps brachii and brachialis muscles. de Koning et al. (1988) found that the decline in the number of motor units was particularly pronounced in the tibialis anterior muscle of subjects over the age of 60 years compared to younger subjects in their sample of healthy volunteers (21-76 yrs).

Studies using electromyography (EMG) have reported age-related changes in both duration and amplitude of motor unit action potentials (MUAPs) (Campbell et al., 1973; Stalberg et al., 1989; Doherty et al., 1993b). Greater amplitudes are characteristic of elderly compared to young subjects, indicating that an increase in the cross-sectional area of surviving motor units has occurred, due to adoption of denervated fibres or by

fibre hypertrophy, or by both mechanisms (Campbell et al., 1973). As well, since most surviving motoneurones innervate slow-twitch muscle fibres, larger durations of MUAP's are found in elderly subjects. Campbell et al. (1973) studied the extensor digitorum brevis muscle in elderly subjects (60-96 yrs), and found only a small increase in MUAP duration and no significant increase in the MUAP amplitude.

However, the amplitude of the MUAP detected in conventional EMG represents only a small portion of the muscle fibres in a motor unit. Using a more advanced macro EMG method provides a more representative estimate of motor unit size, since its amplitude is determined by more of the fibres in the motor unit. Stalberg eg al. (1989) found that the amplitudes of quadriceps muscles of 60-70 year olds, compared to 20-50 year olds, were 27% higher for males and 36% for females.

Doherty et al. (1993b) also found a 23% increase in single MUAP peak-to-peak amplitudes in 60-81 years olds, compared to 21-38 year olds, using the advanced method. These increases in amplitudes reflect the shift toward larger motor units with aging. Compared to the young subjects, the elderly subjects had a greater number of large force motor units, leading to larger motor unit twitch tensions. Therefore, specific tension (force development per motor unit) increases with age.

1.3.5 Changes in the Neuromuscular Junction

With aging, a number of changes in the neuromuscular apparatus have been shown to impair neuromuscular transmission and impulse propagation in older adults. In particular, deterioration of both the presynaptic and postsynaptic components of the

neuromuscular junction have been the subject of numerous animal and human studies (Smith & Rosenheimer, 1984).

Slower action potential conduction velocities have been found in the axons of senescent nervous systems (Campbell et al., 1973). As well, action potential propagation failure occurs more often in old than in young animals (Smith, 1979). Smith (1979) concluded that the capacity to maintain transmission is reduced at the neuromusclar junction of aged rats, which could be the underlying factor responsible for the deterioration of synaptic structures observed during senescence.

Biochemical studies at the neuromuscular junction have shown that the amount of acetylcholine (Ach) in the presynaptic terminals of the rat phrenic nerve is reduced in aged animals. As well, the availability of the substrate choline appears to decline with age (Smith, 1984).

The number of end plates per muscle fibre does not change appreciably with age, however, a downward trend is seen in the number of nerve terminals per end plate in fast-twitch muscle, accompanied by a slight increase in the number of terminals in slow-twitch muscle (Courtney & Steinbach, 1981). Wokke et al. (1990) studied morphological changes in the human end plate of intercostal muscles in subjects aged 4 to 77 years. They found that aged end plates became more complex mainly at the postsynaptic side; with increased length, area and branching of the postsynaptic membrane, and degeneration of junctional folds. At the presynaptic side, irregularly shaped nerve terminals were noted in the aged.

Throughout life, motor nerve terminals undergo a continual process of renewed growth (sprouting) and degeneration at the neuromuscular junction, constantly remodelling the structure of the end plate. With aging, this process may become unbalanced, resulting in significant changes in the architecture of the end plate (Smith & Rosenheimer, 1982). In the diaphragm muscle, the authors found 75.6% less terminal sprouting and 46.5% less degenerating terminals in older rats (28 months), compared to young rats (10 months).

Smith & Rosenheimer (1982) also found that the synaptic vesicles of older rats were less regularly shaped, increased in number, and decreased in size. In the diaphragm, 39% more vesicles were found in the terminal junctional region of older versus young rats, and the vesicle diameter and volume in older rats was 3% and 9%, respectively, smaller than in young rats.

With advancing age, therefore, the ability to maintain synaptic transmission declines, the amount of Ach decreases, and the balance between nerve terminal growth and degeneration becomes less stable.

1.3.6 Changes in Muscle Contractile Properties

Twitch Torques

Vandervoort & McComas (1986) reported that twitch torques declined beyond middle age for both genders. When the oldest group of men and women (80-100 yrs) was compared with the youngest (20-32 yrs), the mean losses in dorsiflexor and plantarflexor twitch torques amounted to 38 and 23%, respectively, for males and to 37%

in both cases for females. Klein et al. (1988) studied contractile properties of the triceps surae muscle, and found that the mean peak torque value of the evoked twitch from the older men (64-69 yrs) amounted to 73.6% of the mean value for the younger men (19-32 yrs). Vandervoort & Hayes (1989) found a 18% decrease in plantarflexor peak twitch torque of elderly (73-91 yrs) women compared to young (20-33 yrs) women.

Twitch potentiation

Post-activation potentiation (increased twitch tension) has been observed in skeletal muscle by eliciting a twitch immediately after a maximal voluntary contraction or a tetanic stimulation (Belanger & Quinlan, 1982; Vandervoort et al., 1983; Alway et al., 1987). Twitch potentiation has been shown to decline with increasing age in studies of lower limb muscles of young and old adults (Vandervoort & McComas, 1986; Petrella et al., 1989; Hicks et al., 1991). It has been argued that the difference between the age groups in terms of twitch potentiation could simply reflect the selective loss or decreased size of Type II fibres, since post-tetanic twitch potentiation has been shown to be larger in Type II muscles than Type I muscles (Vandervoort et al., 1983). One proposed mechanism underlying twitch potentiation is that fast myosin light chains become phosphorylated, which increases the sensitivity of the contractile element to activation by calcium (Houston et al., 1985). Perhaps the structure and function of the sarcoplasmic reticulum may change with age, thereby influencing the amount of calcium available to the contractile apparatus for potentiation (Petrella et al., 1989).

Contraction Time

Contractile characteristics are most frequently studied on the distal muscle groups, particularly the triceps surae group at the ankle. Vandervoort & McComas (1986) reported a significant increase in both the time-to-peak tension (TPT) and the half relaxation time (½ RT) in evoked twitches of the ankle muscles in old adults compared to young adults in their sample of 20-100 years olds. Similar results have been shown for individual muscles of the lower leg and foot (Petrella et al., 1989; Cupido et al., 1992). Petrella et al. (1989) reported a 35% increase in TPT and a 25% increase in ½ RT of the gastrocnemius muscle, while Cupido et al. (1992) found a 17% increase in TPT and a 20% increase in ½ RT of the tibialis anterior muscle of old subjects versus young subjects.

Only minor differences, however, have been shown for twitch contractile properties in the elbow flexors between young and old subjects (Doherty et al., 1993b; McDonagh et al., 1984). Doherty et al. (1993b) reported a small increase in TPT and a small decrease in ½ RT of the biceps brachii and brachialis muscles, and McDonagh et al. (1984) reported that TPT was similar in the elbow flexor muscle group of young and old adults. Histochemical studies have shown no preferential loss or atrophy of Type II fibres in the biceps brachii, yet preferential atrophy of Type II fibres in the lower limb muscles may exist (Grimby et al., 1982).

Slower contractions contribute to a reduced capacity for rapid production of force in protective reflexes. Vandervoort & Hayes (1989) found a 35% decline in rate of force

development of the ankle plantarflexor muscles of older adults (73-91 yrs) compared to young adults (20-33 yrs).

The slower contractile properties of elderly versus young adults may be due to a slowing of the calcium release and/or re-uptake mechanisms within the muscle (Klitgaard et al. 1989; Larsson & Salviati, 1989). McDonagh et al. (1984) suggested that a preferential atrophy of Type II fibres may lead to slower contraction times, or perhaps all fibre types become slower with aging.

Resistance to Fatigue

Research in the area of resistance to fatigue has been inconclusive in older adults, partly due to the variety of methods used to induce fatigue. For example, Narici et al. (1991) observed a decrease in fatiguability across ages in a study of the adductor pollicis in men (20-91 yrs), using a 30 Hz stimulation protocol. Conversely, Klein et al. (1988) reported no differences in fatiguability of the triceps surae muscle between young and old adults, using submaximal 20 Hz tetani, while Davies et al. (1986) found increased susceptibility to fatigue in the same muscle, using the same stimulation, in their aged sample.

Voluntary fatigue studies have been more consistant in showing that muscle fatiguability is not affected by the ageing process (Larsson & Karlsson, 1978; Hicks et al., 1992). Larsson & Karlsson (1978) observed no age-related changes in quadriceps muscular endurance in male subjects (22-65 yrs). During a 2-minute voluntary fatigue paradigm, Hicks et al (1992) found no evidence of excitability failure in the

brachioradialis, tibialis anterior, or thenar muscles of young (31.2 ± 4.9 yrs) or elderly (66.3 ± 3.7 yrs) subjects.

Therefore, fatigue-resistance of aged muscle may not be enhanced, even though a greater proportion of the muscle CSA is occupied by Type I fibres. This may be due to the lack of training, and consequently, the reduced functional capacity of aged muscle (Porter et al., 1995).

1.3.7 Decreased Physical Activity Levels

Disuse, i.e. a reduction in physical activity, has been associated with age-related changes in muscle strength. Some of the age-associated changes in muscle might be caused by reduced locomotor ability rather than being a primary effect of aging. In support of this, spontaneous activity has been shown to decline with aging. For example, the voluntary running of rats provided with running wheels declines dramatically throughout adulthood. Brown et al. (1992) found that the average distance in miles that female Long-Evans rats voluntarily ran per day was reduced by 79% from 6 to 26 months of age. Gulve et al. (1993) reported a similar decrease in voluntary running miles (68% decrease from 6-25 months) in female Long-Evans rats. Holloszy et al. (1991) recognized that atrophy is particularly marked in weight-bearing muscles, especially those with a high proportion of Type IIb fibres. These observations are consistent with the hypothesis that disuse contributes to some of the age-related changes in muscle (Cartee, 1994).

A more striking form of disuse atrophy occurs when already sedentary individuals become immobilized, due to enforced bedrest, acute or chronic illnesses, denervation, or wheelchair dependence, all of which are common in the elderly (Fiatarone & Evans, 1993). The effects of disuse may be the leading cause of muscle dysfunction and impaired mobility in the institutionalized elderly, in whom activity levels are reduced compared to free-living elderly or younger adults.

Physical Activity Patterns in the Elderly

Eight national surveys conducted in the United States and Canada between 1972 and 1983 were reviewed by Stephens et al. (1985) and Perusse et al. (1989), for patterns of leisure-time physical activity in the population. The authors reported that the proportion of the population defined as active declines with age. Furthermore, only 20% of the North American population exercises with an intensity recommended for cardiovascular benefits, while an additional 40% is active at a moderate level, perhaps sufficient to achieve some health benefits, and the remaining 40% is sedentary. The National Health Interview Survey of 1986 stated that only 10% of the adult population in the United States is habitually active, enough to induce beneficial physiological adaptations, but predicted that by 1990, that figure would rise to over 60% (Caspersen, 1986).

Studies on the effects of life-long physical training on aging human muscles have shown that people who have had an habitually active way of life have better muscle strength compared to sedentary people. Sipila et al. (1991) found that athletes (70-81 yrs) with a long-term history of training in both strength/speed and endurance have

superior muscle functioning compared to the average male population of the same age. Strength measures were significantly greater for the trained groups compared to the controls, in all muscles tested. One cross-sectional study of master athletes conducted by Kavanagh & Shephard (1990) proposed that lean tissue mass was well preserved into the seventh decade of life if subjects maintained a consistent level of physical activity. Life-long physical activity may not be able to change the normal aging process, however, it is more important to stress that regular exercise can keep remaining muscle fibres at their best function for as long as possible (Grimby, 1985).

Resistance training in the elderly

The decreased muscle strength of older adults is at least partly reversible by resistance training. Short-term weight training programs result in significant increases in dynamic strength and muscle hypertrophy in the elderly. For example, Frontera et al. (1988) conducted a 12-week strength training program in 12 older men (60-72 yrs). At the end of the training period, the authors reported an increase in knee extensor and flexor strength of 107.4% and 226.7%, respectively, and an increase in total muscle and quadriceps area of 11.4% and 9.3%, respectively. Brown et al. (1990) found a mean increase of 48% in elbow flexor 1 repetition maximum (1RM) values of 14 elderly males (60-70 yrs), after 12 weeks of dynamic elbow flexion training of one arm, compared to an increase of 12.7% in the control arm. As well, the mean cross-sectional area of the elbow flexors increased by 17.4% in the trained arm, but did not change in the control arm.

Most studies in this area have been carried out on males, and very little work has looked at the physiological responses of women to resistance training. Charette et al. (1991), however, conducted a training study of 27 healthy women (64-86 yrs), randomized to either a control or exercise group. After 12 weeks of weight-training, increases in muscle strength were significant in all muscle groups (28-115%), and the cross-sectional area of Type II muscle fibres also increased (20.1 $\pm$ 6.8%). The authors concluded that resistance training programs can be safely carried out by elderly women, such programs significantly increase muscle strength, and these gains can be attributed in part to muscle hypertrophy.

Fiatarone et al. (1990; 1994), have noted the potential of an appropriate training program to sustain or restore both muscle function and lean tissue even into the ninth and tenth decade of life. They studied the effects of strength training on very old, nursing home residents. The earlier study reported strength gains averaging 174 $\pm$ 31% in the quadriceps, and muscle cross-sectional area increases of 9.0 $\pm$ 4.5%, after 8 weeks of high intensity strength training in 10 frail male and female subjects (86-96 yrs). More recently, Fiatarone et al. (1994) trained a larger sample of 100 frail elderly subjects (72-98 yrs) for 10 weeks, and found that muscle strength increased by 113 $\pm$ 8%. The authors concluded that high intensity resistance exercise training is a practical and effective means of counteracting muscle weakness and physical frailty in elderly people.

Prolonged physical training can also significantly increase both muscle strength and cross-sectional area in the elderly. McCartney et al. (1995a; 1995b) conducted a 2

year resistance training study in 113 male and female subjects (60-80 yrs), randomized into control and training groups. Muscle strength was unchanged in the control subjects, but strength increases ranged from 32% for the leg press exercise, to 90% for the military press in the exercise group. Also, an increase of 8.7 $\pm$ 0.9% was found in cross sectional area of the knee extensors in the trained subjects, while no change was reported in the controls. Follow-up investigation of the de-training effect of 21 of the subjects, called back 5 months after completing the 2-year weight-training program, supported the hypothesis that decreased physical activity ultimately leads to a reduction in strength. For example, a loss of 15.7% in dynamic strength was found after 5 months without training. However, the residual strength was still 38.5% greater than the pre-training values. Therefore, despite a significant decline in strength within 5 months of de-training, the increases in dynamic strength attained as a result of regular weight-training are well preserved in the elderly. These findings are from the largest, and longest, prospective randomized weight-lifting training study in elderly men and women. After 2 years of twice-weekly training, dynamic strength was still increasing in the subjects. Consequently, the authors concluded that this is an appropriate form of exercise for elderly men and women, with the potential to extend the years of independent living.

1.3.8 Cross-sectional and Longitudinal Studies

Based on cross-sectional population studies, it has been well established that skeletal muscles of older adults are significantly weaker than those of younger adults.

For example, Vandervoort & McComas (1986) studied ankle muscle function in adults aged 20 to 100 years. When strength measures were expressed as a percentage of the youngest group of adults, they found that subjects in the 9th and 10th decades, had on average, 50% less strength than the youngest group. They also established that strength did not begin to decline until the 6th decade of life, at a rate of approximately 1.3% per year.

Other studies with a cross-sectional design have found age differences in strength to a more or lesser extent, depending on the muscle group under study (distal or proximal), and the type of muscle testing done (isometric, concentric or eccentric). Davies et al. (1986) investigated the isometric properties of triceps surae in elderly men and women (mean age 70 yrs), and found 38% and 28% differences, respectively, in ankle plantarflexion strength from young male and female adults (mean age 22 yrs). Petrella et al. (1989) reported a 43% decline in maximal voluntary contraction (MVC) strength in the plantarflexor muscles of elderly men (66.9 $\pm$ 5.3 yrs) compared to young men (25.7 $\pm$ 3.8 yrs). Sandler et al. (1991) found that plantarflexor strength declined by 46% in their large sample of older (>65 yrs) compared to young (25-34 yrs) women. Christ et al. (1992) reported a decline in dorsiflexor and plantarflexor strength of 42% and 45.1%, respectively, in their comparison of healthy women, 25-74 years of age.

Larsson & Karlsson (1978) found that maximal isometric and dynamic strength of the quadriceps increased up to 30 years of age, remained constant up to 50 years, and then decreased thereafter with increasing age, in their sample of 114 males (11-70 yrs). Cross-sectional studies have also compared quadriceps strength between young and old

men and women. Young et al. (1985) reported that the mean isometric strength of the quadriceps muscles of 12 men in their seventies was 39% less than that of 12 men in their twenties. When 25 older women (71-81 yrs) were compared to 25 young women (20-29), the quadriceps of the older women were found to be 35% weaker than the young women (Young et al. (1984).

McDonagh et al. (1984) compared the different effects of aging on arm and leg muscles in young (25.8 $\pm$ 6.1 yrs) and elderly (71.3 $\pm$ 3.7 yrs) men. Although the strength of both the elbow flexor and triceps surae muscle groups declined with aging, the reduction was greatest in the leg muscles (41% versus 20%). Narici et al. (1991) studied the effect of aging on voluntary strength of the adductor pollicis muscle in 70 healthy male subjects (20-91 yrs). After the age of 59 years, maximum isometric voluntary force declined significantly, dropping 57.6% by the eighth decade compared to the second decade.

Therefore, muscle strength appears to be relatively well preserved up through the 5th decade of life, after which time a loss of about 15% per decade is seen (Vandervoort & McComas, 1986). Although many cross-sectional studies have shown a decline in strength with aging, these studies are limited to describing changes at the population level, and cannot explain how a specific individual's muscle strength changes with age. As well, they are not suited for determining whether age-related strength declines are due to confounding secular or cohort effects.

Very few longitudinal studies have examined the loss of muscle strength with aging. Longitudinal studies have been done on quadriceps and handgrip muscle groups,

however the results have been somewhat controversial. Aniansson et al. (1986) conducted a 7 year follow-up study of quadriceps strength in 23 men (73-86 yrs), and reported a strength decline of 23% from 1975-1982, amounting to 3.2%/year. Greig et al. (1993) also conducted follow-up testing of quadriceps strength, in their sample of 14 men and women with a mean age of 81.5 years. However, they found that quadriceps strength was well-preserved over an 8 year period, with only a 0.3%/year decrease reported. They attributed the slow rate of decline in strength to the subjects' high levels of habitual physical activity levels.

Kallman et al. (1990) published results showing close agreement between their longitudinal study (9 year follow-up) of handgrip strength, and their cross-sectional analysis of strength in adult men, in the Baltimore Longitudinal Study on Aging. The 80-90 year old group had 37% less strength than the 30-39 year old group, representing a 4.1%/year decline in handgrip strength. The authors concluded that mean grip strengths were similar for each age group, whether they were calculated cross-sectionally, or after a 9 year period. Bassey & Harris (1993) originally tested 920 men and women over 65 years of age, and found a 2%/year decline in handgrip strength. When 420 survivors were retested 4 years later, they reported a 3%/year decrease in strength for males, and a 5%/year decline for females. Therefore, their cross-sectional study underestimated the loss of handgrip strength, especially for women.

To our knowledge, there have been no longitudinal studies reported on ankle muscle function in the elderly. Therefore, the purpose of the present investigation was to conduct follow-up testing of healthy Hamilton Seniors who underwent ankle muscle

function assessments in 1982 in Vandervoort & McComas's study (1986). This was done in order to determine the longitudinal rate of decline in muscle function of the surviving subjects over the past 12 years.

CHAPTER II

A 12 YEAR FOLLOW-UP STUDY OF ANKLE MUSCLE FUNCTION IN OLDER ADULTS

2.1 ABSTRACT

The purpose of this study was to conduct follow-up testing of healthy Hamilton Seniors who underwent assessments of ankle muscle function twelve years ago. The isometric strength and contractile characteristics of the dorsiflexors and plantarflexors were studied in 11 male and 11 female subjects, ranging from 73-97 yrs (mean age 84 $\pm$ 7.1 yrs). The same footplate apparatus was used as during the original testing. Results from the cross-sectional study completed in 1982-83 indicated that maximal voluntary isometric strength (MVC) decreased by 1.3% per year between the ages of 20 and 100 yrs (Vandervoort & McComas, J.A.P. 61:361-7, 1986). We found that, over the course of 12 years, MVC strength declined overall by 1.5% per year. Plantarflexor strength decreased from 89.1 $\pm$ 23.0 Nm to 67.0 $\pm$ 21.8 Nm in females, and from 122.3 $\pm$ 39.6 Nm to 85.3 $\pm$ 25.5 Nm in males ($p<0.01$). This loss was relatively less in the dorsiflexor muscles; strength decreased from 21.4 $\pm$ 2.8 Nm to 20.7 $\pm$ 3.2 Nm in females, and from 29.5 $\pm$ 5.5 Nm to 26.7 $\pm$ 9.3 Nm in males ($p>0.05$). There were no significant changes in evoked twitch torque in either muscle group, which may be due to the fact that passive tension significantly increased ($p<0.01$) over the past 12 years. We conclude from this longitudinal assessment of ankle muscle function that there

is a significant loss of voluntary strength only in the plantarflexors in older adults, and that increases in passive tension appear to prevent any corresponding declines in evoked twitch torques.

2.2 INTRODUCTION

Based on cross-sectional studies, it has been well established that voluntary strength declines with aging (Larsson & Karlsson, 1978; McDonagh et al., 1984; Young et al., 1984; 1985; Davies et al., 1986; Vandervoort & McComas, 1986; Petrella et al., 1989; Narici et al., 1991; Christ et al., 1992). For example, Vandervoort & McComas (1986) reported in their study of ankle muscle function in males and females aged 20 to 100 years, that subjects in the 9th and 10th decades had, on average, 50% less strength than the youngest group. They also established that strength did not begin to decline until the 6th decade of life, at a rate of approximately 1.3% per year.

Other cross-sectional studies have noted age differences in strength to a more or lesser extent (Doherty et al., 1993a), depending on the muscle group under study (proximal or distal), and the type of muscle testing done (isometric, concentric or eccentric). However, based on their cross-sectional design, these studies are limited to describing changes at the population level, and cannot explain how a specific individual's muscle strength changes with age. As well, they are not suited for determining whether these age-related strength declines are due to confounding secular or cohort effects.

The primary mechanism to explain the decreased strength of older versus young adults appears to be a reduced muscle mass, stemming from a loss of functioning motor units (Doherty et al. 1993b). Lexell et al. (1988) reported that while the total numbers of Type I (slow-twitch) and Type II (fast-twitch) muscle fibres are lower in older adults compared to young adults, atrophy of Type II muscle fibres is also characteristic of aging muscle. Studies by Hicks et al. (1991; 1992) have described the slowed speed of

contraction of the ankle dorsiflexor muscles in older adults, and the altered response to trains of tetanic stimulation. Furthermore, McCartney et al., (1995a; 1995b) and other investigators have shown that strength training programs can alter muscle function in Seniors, thereby reversing some of the aging effects (Vandervoort, 1992).

Longitudinal studies have examined the loss of muscle strength with aging in only the quadriceps and handgrip muscle groups, however, the results have been somewhat controversial (Aniansson et al., 1986; Greig et al., 1993; Kallman et al., 1990; Bassey & Harris, 1993). To our knowledge, there have been no similar studies reported on ankle muscle function. Therefore, the aim of this study was to conduct follow-up testing of healthy Hamilton Seniors who previously underwent ankle muscle function assessments in 1982; for the purpose of determining the longitudinal rate of decline over the past 12 years.

2.3 METHODS

2.3.1 Subjects

Based on the list of sixty-nine subjects (60-100 yrs) tested in the original 1982 investigation (Vandervoort & McComas, 1986), an attempt was made to locate and recruit the surviving subjects for retesting. Twenty-two subjects (11 males, 11 females, 73-97 yrs) participated in the present study (Table 1), and physical characteristics of each subject in 1982 and 1994 are outlined in Appendix A. The investigation carried the approval of the Ethics Committee at McMaster University, and each subject gave their written informed consent to participate (Appendix B).

Health status of the subjects over the past 12 years was discussed during an initial interview (Appendix C). Physical activity levels were then assessed using a physical activity questionnaire for the elderly from Voorrips et al. (1991), who adapted a previous questionnaire designed for young adults by Baecke et al. (1982). The questionnaire consisted of scores for household, sporting and leisure activities, altogether resulting in a total physical activity score for each subject (Appendix D).

2.3.2 Measurement of Muscle Contractile Properties

The same testing apparatus that was used in 1982 was also utilized in the present investigation. All measurements were conducted on the right ankle with the subjects seated in a vertically adjustable chair such that the leg was flexed 90° at the knee. Isometric torques produced about the ankle joint by the dorsiflexor and plantarflexor muscles were recorded using the leg holder and footplate first employed by Marsh et al. (1981) (Figure 1). The inclination of the foot could be altered by rotating the footplate about an axis that corresponded to that of the ankle joint. Torques acting on the footplate were sensed by strain gauges mounted on a rigid bar underneath the plate.

Adjustments to the footplate placed the muscle group under study at its optimal length for tension development, according to earlier investigations by Marsh et al. (1981) and Sale et al. (1982). The ankle was placed in 30° of plantarflexion for measurements on the dorsiflexor muscle group, and in 10° of dorsiflexion for testing the plantarflexor muscle group. Measurements of passive tension were recorded with the ankle in the neutral position (0°); 10 and 20° of dorsiflexion; and 10, 20, 30 and 40° of plantarflexion.

Plantarflexor range of motion about the ankle joint was measured by rotating the ankle joint in the dorsiflexion position, as far as possible, and recording the maximum angle in degrees.

2.3.3 Stimulating and Recording Apparatus

Prior to electrode placement, the skin was shaved, scraped, and cleansed with alcohol, and conducting gel was applied to the electrodes in order to minimize resistance. The stimulating electrodes consisted of a lead plate cathode (radius = 15 mm) and rubber anode (37 mm x 45 mm). For the dorsiflexor muscle group, the stimulating cathode was wrapped in gauze, dampened, and secured over the common peroneal nerve just distal to the proximal head of the fibula, while the anode was placed on the anterior aspect of the leg, approximately 50 mm distal to the patella. For the plantarflexor muscle group, the pair of stimulating electrodes were placed snugly against the skin in the popliteal fossa overlying the tibial nerve, with the cathode distal.

In order to record dorsiflexor muscle compound action potentials (M-Waves), a stigmatic recording electrode was placed on the skin over the tibialis anterior muscle belly, and a reference electrode was placed over the tendon of the same muscle at the level of the ankle. For recording plantarflexor M-Waves, the positions of the stigmatic and reference electrodes were over the soleus, just below the separation of the gastrocnemius muscle bellies, and over the Achilles tendon, respectively. Recording electrodes were disposable Ag/AgCl discs (3M, No. 2248), 4 mm in diameter. EMG signals from the recording electrodes were filtered into amplifiers with bandwidths of 20

Hz to 1.5 kHz, and were displayed on a computer monitor. The ground electrode was a strip of a silver foil placed between the stimulating anode and the stigmatic recording electrode.

A high-voltage stimulator (Devices System, Model 3072, Welwyn Garden City, Hertfordshire, UK) was used to deliver single 50 μsec rectangular pulses to the peroneal and tibial nerves. The stimulating voltage was gradually increased until there were no further increases in evoked twitch torque; this was the voltage that was subsequently used to evoke the maximum twitch. Data were streamed continuously to disc using a Dataq waveform scrolling board (WFS-200DC; Dataq Instruments, Akron, OH) configured in an IBM-compatible computer system.

2.3.4 Maximum Voluntary Strength

For analysis of maximal voluntary strength (MVC), subjects were given several attempts to achieve as large a torque as possible during either a dorsiflexion or plantarflexion movement. During the MVC, the same stimulus as that used to evoke the peak twitch was given to the appropriate motor nerve, to assess the extent of motor unit activation in the muscle group under study. Attempts at MVC were continued until either no interpolated twitch was present, or in the case of incomplete activation, the torque output became constant over several trials. Post-activation potentiation was examined by eliciting a twitch 3 seconds after a 5 second maximal voluntary contraction, at the same stimulus as above, similar to the method in Vandervoort & McComas (1986).

2.3.5 Data Analysis

Custom-designed Advanced CODAS software (Dataq Instruments) was used for analysis of all of the voluntary, evoked, and potentiated recordings: maximum voluntary strength (MVC), peak twitch torque (TT), time to peak torque (TPT), one-half relaxation time (½ RT), and peak-to-peak amplitude of the M-Wave. Contraction time (CT) for the twitch was calculated as the sum of TPT and ½ RT.

2.3.6 Statistical Analysis

All measurements taken in this study were compared to the 1982 values corresponding to the same subjects. The data were analyzed using a three factor (gender x leg muscle group x measurement time) mixed analysis of variance (ANOVA). Tukey A post hoc tests were conducted whenever significant main effects and interactions were found. Level of significance was set at $P<0.05$. Throughout the text, the data are presented as means $\pm$ the standard deviation (SD).

The reliability of each measurement technique was assessed by comparing values for 10 subjects on 2 different days. The mean reliability coefficient of 20 measurements was 0.905 ± 0.05, using the Intraclass Correlation Coefficient method (Appendix E). Several different reliability methods are also compared in Appendix E.

2.4 RESULTS

All of the subjects were generally in good health, and there were no incidences of any debilitating ankle injuries over the past 12 years. A few reported health problems such as hip replacement and bypass surgeries, heart attacks, high blood pressure, dizzy

spells, and arthritis in hands and legs. One subject could not have anything attached to the skin on her legs and thus could not be included in the 12 year comparison of isometric twitch properties and M-Wave characteristics. However, values were obtained on this subject for voluntary strength and plantarflexor range of motion.

Main effects were found for gender and time in height and weight changes over the past 12 years. Males were significantly taller and heavier than females, and height and weight declined significantly from 1982 to 1994 for both genders (Table 1). Height losses amounted to 2.5% for males and 3.0% for females, and weight decreased by 5.5% for males and 3.4% for females. In comparison, the cross-sectional study (Vandervoort & McComas, 1986) reported losses in height of 4.2% for males and 4.3% for females, and weight losses of 6.6% for males and 9.0% for females in the 80-100 yr group compared to the 70-79 yr group.

2.4.1 Physical Activity Levels

The physical activity questionnaire used for this study was divided into 3 parts, with scores for household, sport and leisure time activities. Total physical activity scores were related to the percent change in voluntary strength from 1982 to 1994 to determine whether a correlation existed between these variables. No significant relationships were found for the dorsiflexors (r = 0.40), or the plantarflexors (r = 0.20) (Figure 2).

2.4.2 Maximal Voluntary Strength

In both muscle groups, males were significantly stronger than females. There was an overall decline in voluntary strength over the past 12 years, for both genders and

muscle groups (Figure 3). Plantarflexor strength decreased from 122.3 ± 39.6 Nm to 85.3 ± 25.5 Nm in males, and from 89.1 ± 23.0 Nm to 67.0 ± 21.8 Nm in females ($p<0.01$). The loss was relatively less for the dorsiflexors; strength decreased from 29.5 ± 5.5 Nm to 26.7 ± 9.3 Nm in males, and from 21.4 ± 2.8 Nm to 20.7 ± 3.2 Nm in females ($p>0.05$). When strength measures were collapsed across both muscle groups, the rate of decline in voluntary strength was approximately 1.5% per year, which is close to the 1.3% per year predicted by Vandervoort & McComas (1986).

Voluntary strength, expressed as a ratio of body weight, decreased significantly ($p<0.01$) in the plantarflexors (26.6% decline for males, and 20.8% decline for females), while the dorsiflexor strength/mass ratios remained stable (5% decline for males, and 2.6% increase for women) over the past 12 years (Figure 4).

2.4.3 Motor Unit Activation

Interpolated stimulation during voluntary contractions indicated that all subjects were able to fully activate their dorsiflexor motor units, since there was no evidence of extra torque when a twitch was superimposed on the voluntary contractions. With respect to plantarflexor muscles, 9 out of 21 subjects (3 males, 6 females) could not achieve full activation, and the mean motor unit activation (MUA) in the 9 subjects was 83.4 ± 5.4%, ranging from 75.5% to 91.0% (Appendix A). MUA was calculated using a method first sited in Belanger & McComas (1981).

2.4.4 Ankle Muscle Stiffness

Passive tension increased significantly for both genders, in the dorsiflexor and plantarflexor testing positions (Figure 5). Dorsiflexor values increased from a mean value of 1.83 Nm to 3.13 Nm ($p<0.01$) from 1982 to 1994, and plantarflexor values increased from 2.49 Nm to 3.95 Nm ($p<0.01$). As well, there was an overall decline in plantarflexor range of motion about the ankle joint for males (19.4%) and females (7.0%), however, the decline was significant only for the male subjects (Table 1).

2.4.5 Isometric Twitch Torque

Resting twitch torques were significantly larger for the plantarflexors than for the dorsiflexors in both testing years. There were no significant changes in twitch torque from 1982 to 1994 for either muscle group, and the trend was similar for both genders (Figure 6). Dorsiflexor twitch torque increased slightly, while little change was observed in the plantarflexor muscles.

When peak twitch torque was expressed relative to maximum voluntary strength, the twitch/MVC ratio increased significantly ($p<0.01$) from 1982 to 1994 for both genders and muscle groups (Figure 7). As well, a main effect for gender was also found; females had significantly higher values than males, especially in the plantarflexor muscle group ($p<0.01$).

2.4.6 Post-activation Potentiation

Table 3 compares the mean values for resting and potentiated twitch values in 1982 and 1994. Dorsiflexor torques increased 28.6% for males and 38.1% for females

in 1982, and 30.2% for males and 38.2% for females in 1994; while plantarflexor values increased 29.3% for males and 3.1% for females in 1982, and 41.3% for males and 33.3% for females in 1994. No significant effects for time were found in the percentage change from resting to potentiated twitch torque for either muscle group, although there was huge variability between subjects in the plantarflexor group.

2.4.7 Contraction Time

From 1982 to 1994, time to peak torque decreased significantly for both genders and muscle groups, while half-relaxation times showed no significant change (Table 2). Contraction times (TPT + ½ RT) decreased from 1982 to 1994 across both genders and muscle groups (Table 2); the dorsiflexor decline amounted to 2.3% in males and 6.2% in females, whereas plantarflexors declined by 7.6% in males and 5.9% in females ($p<0.01$). As well, TPT, ½ RT and CT values were significantly higher for the plantarflexor muscle group than for the dorsiflexors.

2.4.8 M-Wave Amplitude

M-Wave amplitudes were significantly larger for plantarflexors than dorsiflexors for both genders. A gender by time by contraction type interaction revealed that plantarflexor M-waves increased significantly for female subjects from 1982 to 1994, while no other significant effects were shown (Figure 8).

2.5 DISCUSSION

This is the first report of a longitudinal assessment of ankle muscle function in very old adults (mean age 83.5 yrs). Since this study examined muscle changes over a 12 year period of aging, care should be taken when comparing these results with cross-sectional studies of aging, which tend to report differences between young and old subjects.

The subjects in the present investigation were originally selected based on health status, not on physical activity levels. All of the elderly subjects could walk independently, without aids, and most stated that daily walking was a part of their normal activity. After 12 years, all subjects could still walk independently (some with the use of a cane), and were involved in activities such as walking, gardening, and light household work.

Physical activity levels of the subjects were determined using a physical activity questionnaire for the elderly by Voorrips et al. (1991). To test the validity and reliability of their test, they administered the questionnaire to 60 free living, healthy adults (63-80 yrs), and found a mean physical activity score of 11.0 $\pm$ 4.6, with scores ranging from 2.5 to 21.7. In the present study of 22 subjects (73-97 yrs), the mean score was 10.8 $\pm$ 7.2, ranging from 4.7 to 24.6.

When physical activity scores were correlated with the percent change in voluntary strength from 1982 to 1994, no significant relationships were found for the dorsiflexors or the plantarflexors. However, physical activity assessment for the elderly population may present unique problems due to the diversity of activity types that are

appropriate for older persons (Dishman, 1989). Total energy expenditure for these individuals consists mainly of household activities of low intensity, which is difficult to quantify from verbal self-report alone.

1.5.1 Maximal Voluntary Strength

The longitudinal rate of decline in voluntary strength, calculated in this follow-up study of ankle muscle function in older adults, was comparable to the rate predicted in the cross-sectional investigation by Vandervoort & McComas (1986) (1.5% versus 1.3% per year, respectively). In 1982, voluntary strength losses amounted to 31.8% in the dorsiflexors and 36.6% in the plantarflexors, for the 80-100 yrs age group compared to the 60-69 yrs group. However, in the present study, the loss of voluntary strength from 1982 to 1994 was significantly less for the dorsiflexors than the plantarflexors (7% and 28%, respectively).

One possible explanation of the preferential loss of plantarflexor strength in older adults compared to dorsiflexor muscles may be that the gastrocnemius and the soleus muscles are more activity-dependent than the tibialis anterior muscle. The lower activity levels seen in older adults, may have accelerated the atrophy process of plantarflexor muscles. Or perhaps, muscles age at different rates, and the majority of the dorsiflexor strength loss had occurred prior to the first testing.

Christ et al. (1992) also reported dorsiflexor strength was maintained to a greater degree than that of the plantarflexors in their study of women aged 25-74 years. They credited the selective decline in the strength of the plantarflexors to the difference in the

size of the motor nerves innervating the two antagonistic ankle muscle groups. Type II fibres, which are more numerous in plantarflexor muscles, are innervated by larger motor neurons that tend to be affected before smaller ones during aging (Kanda et al., 1986); prompting the smaller motor neurons to reinnervate denervated Type II fibres. As a result, Type II fibres become metabolically more like Type I fibres (Close, 1972).

Based on human autopsy studies, the tibialis anterior and the soleus muscles have been found to consist of mostly Type I fibres (around 73% and 87%, respectively), whereas the gastrocnemius, the larger plantarflexor muscle, is made up of approximately equal proportions of Type I and Type II fibres (Johnson et al., 1973). This supports the reasoning by Christ et al. (1992) for the greater loss of strength in plantarflexor versus dorsiflexor muscles, since Type II fibres appear to be most affected by aging. However, Vandervoort et al. (1983) stated that the dorsiflexors are more representative of fast-twitch muscles than the plantarflexors, since the soleus muscle is likely the most powerful muscle during plantarflexion. With the knee bent at 90°, the soleus muscle may contribute over 70% to plantarflexor force, since flexion of the knee would put the gastrocnemius muscle at a less than optimum length for force production (Sales et al., 1982).

Changes in postural activity of leg muscles could also contribute to greater Type II fibre atrophy. Primarily Type I motor units are recruited at moderate force levels. Since slow-moving older adults rarely reach high force levels or produce rapid movements, it is likely that Type IIb motor units are rarely recruited (McDonagh et al., 1984). Winter et al. (1990) studied walking patterns of the healthy elderly compared

with young adults, and found that adaptations by the elderly lead towards a safer, more stable gait pattern. For example, the elderly used a shorter step length, an increased support stance period, decreased push-off power, and a more flat-footed landing; which required less power from their strength-reduced plantarflexors and dorsiflexors.

Several mechanisms have been proposed to explain the age-related decline in muscle strength: loss of excitable muscle mass; changes in muscle morphology; changes in the nervous system and motor units; as well as decreased physical activity levels. Lexell et al. (1988) concluded that the loss of muscle cross-sectional area (CSA) begins as early as 25 years, approximately 10% of CSA is lost by the age of 50, and almost 50% is lost by the age of 80 years. In the present study, there was a significant loss in body mass over the past 12 years, which could be attributed to a loss in muscle mass, and subsequently may have played an important role in the loss in muscle strength.

The loss in muscle mass with aging is due to both a decreased number and a decreased size of muscle fibres. Although fibre loss has been shown to occur in both Type I and Type II fibres to the same degree, decreases in size occur primarily in Type II fibres. Therefore, while the distribution of Type II fibres does not appear to be affected by aging, their size is significantly reduced (Grimby et al., 1982; Lexell et al., 1988).

With respect to the central nervous system, a progessive loss of motor neurons in the spinal cord (Lexell & Downham, 1991) leads to cycles of denervation and reinnervation within the motor unit population. Also, the number of motor units has been found to decline beyond the age of 60 yrs (Campbell et al., 1973; Doherty et al.,

1993b; de Koning et al., 1988), while the size of the remaining motor units increases with age. Vandervoort & McComas (1986) also credited the reduction in strength in part to muscle denervation, since estimates of functioning soleus motor units in five of the oldest subjects were reduced by 70%. However, de Koning et al. (1988) found that dorsiflexor strength was maintained in subjects (21-76 yrs), even though the number of motor units was considerably reduced.

Finally, reduced physical activity levels may also contribute to strength loss with aging, since the decreased muscle strength of older adults has been shown to be at least partly reversible with short-term (Frontera et al. 1988; Brown et al., 1990; Fiatarone et al., 1990; 1994) and long-term (McCartney et al., 1995a; 1995b) resistance training programs.

2.5.2 Motor Unit Activation

Vandervoort & McComas (1986) reported that losses of strength in older subjects were not entirely due to reduced drive from the motor cortex to spinal motor neurons, since most of their elderly subjects (60-100 yrs) were able to fully activate the plantarflexor and dorsiflexor muscles of the ankle joint. However, central activation of the plantarflexor muscles appears to be affected to a greater extent than the dorsiflexor muscles in older adults. In the present study, the retested subjects were all able to maximally activate their dorsiflexor muscles, which is in agreement with the study by van Schaik et al. (1994) that reported 96% MUA. With respect to plantarflexor muscles, full motor unit activation is not as easily achieved (Belanger & McComas, 1981; Belanger

& Quinlan, 1982). Forty-three percent (9 out of 21) of our subjects could not reach full activation, compared to 22% (14 out of 63) subjects (60-100 yrs) in the 1982 study. However, only small interpolated torques were produced relative to resting twitch torques, with calculated MUA's ranging from 75.5% to 91.9% in the 9 subjects. Unfortunately, there were no individual interpolated twitch values recorded in the 1982 study for comparison of MUA.

2.5.3 Ankle Muscle Stiffness

Two factors contributing to movement about the ankle joint are passive tension and range of motion. Passive resistive force of the dorsiflexors and plantarflexors increased from 1982 to 1994, while range of motion decreased. These findings have been confirmed in other studies of aging ankle muscles (Vandervoort et al., 1992). The increased muscle stiffness is thought to be due to the accumulation of connective tissue within the muscle (Lexell et al., 1988; Rice et al., 1989; Overend et al., 1992). Cross-sectional areas taken of the plantarflexors have shown that elderly limbs were of similar overall shape and girth as young adults, but elderly muscles were smaller with greater amounts of non-muscle tissue located within the muscle (Rice et al., 1989).

2.5.4 Isometric Twitch Torque

Resting isometric twitch torque values were similar when compared to 1982 values for both muscle groups. The lack of any decline in twitch torque was not unexpected, as other investigations from our laboratory have also reported similar twitch torques between young and old adults (Hicks et al., 1991; van Schaik et al., 1994). In

contrast, there are several reports of reduced twitch torques in the elderly (Campbell et al., 1973; Davies et. al., 1986; Vandervoort & McComas, 1986; Klein et al., 1988; Doherty et al., 1993b). Vandervoort & McComas (1986) reported reduced twitch torque values in elderly (60-100 yrs) compared to young adults (20-29 yrs). The greatest decline occurred in the 60-69 year old group, with smaller changes thereafter. In 1982, the mean age of the subjects that were retested for the present investigation was 71.5 years, which is older than the age when the greatest change occurred. Possibly, the majority of twitch torque loss had occurred prior to 1982, which may explain the lack of any significant changes found in our subject group.

There was a trend for dorsiflexor twitch torque to increase from 1982 to 1994 (p=0.07). Although this disputes cross-sectional findings (Vandervoort & McComas, 1986), increased dorsiflexor twitch torque in older versus younger adults agrees with a report by Cupido et al. (1992). Age-related increases in connective tissue within the muscle and increased muscle stiffness may lead to the maintenance of twitch torques in older adults. When a stiff muscle is twitched, there is less series elasticity, which may result in increased force production during the brief time that the twitch occurs.

2.5.5 Post-activation Potentiation

Following a maximal voluntary contraction, increased twitch tension has been observed in skeletal muscle (Belanger & Quinlan, 1982; Belanger et al., 1983; Vandervoort et al., 1983; Alway et al., 1987). Post-activation potentiation is thought to increase the state of readiness of muscle for movement. One proposed mechanism

underlying twitch potentiation is that fast myosin light chains become phosphorylated, which increases the sensitivity of the contractile element to activation by calcium (Houston et al., 1985).

With aging, the capacity for twitch potentiation has been shown to decrease (Vandervoort & McComas, 1986; Petrella et al. 1989; Hicks et al. 1991). In the present investigation, no significant differences were found in the percent of twitch potentiation from 1982 to 1994, for either muscle group, however, a great deal of variability was found in the plantarflexor muscles. Although the same timing was used for both testing years (a twitch delivered 3 seconds after a 5 second MVC), some discrepancy can occur if, for example, subjects held the MVC for a slightly longer or shorter time, which could have changed the timing of the post-activation twitch.

2.5.6 Contraction Time

It is well established that time to peak torque and half-relaxation times of the evoked twitches become prolonged with increasing age (Davies et al., 1986; Vandervoort & McComas, 1986; Klitgaard et al., 1989; Petrella et al., 1989; Cupido et al., 1992). However, when the results from the present investigation were compared to the 1982 values (Vandervoort & McComas, 1986), the trend was not in full agreement with previous findings. Over the past 12 years, time to peak torque and contraction times declined significantly, while half-relaxation times remained stable. Other studies on older adults conducted in our laboratory (Cupido et al., 1992; van Schaik et al., 1994) have reported TPT's and ½ RT's which were similar to the values that we found. For

example, our mean values for dorsiflexor TPT were 102.4 ± 15.3 msec for males and 90.1 ± 15.0 msec for females, which are comparable to van Schaik et al.'s (1994) times of 92.6 ± 8.3 msec for males and 90.5 ± 8.9 msec for females (60-80 yrs). Therefore, the manual methods used to calculate contraction times in 1982 may have overestimated the TPT's and ½ RT's published in 1986; in fact, the values reported by Vandervoort & McComas (1986) are consistently greater than most other investigations (Belanger et al., 1983; Davies et al., 1986; Klein et al., 1988; Petrella et al., 1989; Cupido et al., 1992). The computerized equipment used for the acquisition and analysis of evoked twitches in the present investigation is probably much more sensitive and reliable than the previous manual methods.

2.5.7 M-Wave Amplitude

Resting M-wave amplitudes increased significantly in the plantarflexor muscles of the female subjects in the present investigation. However, with increased age, smaller M-Wave amplitudes have been typically reported in older subjects (Vandervoort & McComas, 1986; Hicks et al., 1992; Cupido et al., 1992; Doherty et al., 1993b). M-Waves are commonly used as a measure of muscle excitability and neuromuscular propagation. Smaller M-Waves indicate reduced muscle membrane excitability and/or decreased excitable muscle mass with aging. Perhaps the larger M-Waves found in our female subjects may be related to a change in the subcutaneous fat in their legs after 12 years. The females may have less fat in their legs now than in 1982, which would allow the recording electrodes to pick up a stronger signal, since there would be less

subcutaneous resistance. This may allow for improved volume conduction of the action potential, and result in larger recorded M-Waves.

2.5.8 Summary

In summary, the rate of decline in overall voluntary strength that we calculated in this longitudinal study of ankle muscle function in older adults, was comparable to the rate predicted cross-sectionally in 1982. However, the decline in voluntary strength was statistically significant only in the plantarflexor muscle group. In the future, it would be of interest to examine the factors responsible for the apparent maintenance of twitch torque, and the role that passive tension may play in this.

The loss of muscle strength with aging has received increased attention in recent years, due to important links between muscle impairments and disability in the Seniors age group. Continued research in this area is needed to enhance our ability to promote independent living, and increase the quality of life in our expanding elderly population.

TABLE 1. Physical Characteristics (n=22, 11♂, 11♀)

Variable	Gender	1982	1994
Age (yrs)	M	73.5±7.5	85.5±7.5
	F	69.5±6.4	81.5±6.4
Height (cm)	M	174.0±7.5	169.7±6.9
	F	158.6±5.5	153.9±1.8
Weight (kg)	M	70.8±7.7	66.9±9.6
	F	56.3±9.1	54.4±12.0
PF ROM (degrees)	M	35.0±5.1	28.2±4.1
	F	28.5±7.0	26.5±7.0

Values are means ± SD.

TABLE 2. Resting Isometric Twitch Properties and M-Wave Amplitude (n=21, 11♂, 10♀)

Variable	Gender	1982	1994
Dorsiflexor:			
M-Wave (mV)	M	6.4± 1.3	6.8± 3.5
	F	8.1± 4.0	10.4± 4.5
TT (Nm)	M	2.8± 1.0	4.3± 1.5
	F	2.1± 0.8	3.4± 0.7
TPT (msec)	M	118.2±16.0	102.4±15.3
	F	110.0± 8.5	90.1±15.0
½ RT (msec)	M	117.3±21.5	127.6±23.8
	F	110.5±21.4	116.8±16.8
CT (msec)	M	235.5±34.7	230.0±33.4
	F	220.5±27.1	206.9±26.1
Plantarflexor:			
M-Wave (mV)	M	12.5± 4.0	9.3± 4.6
	F	10.6± 4.4	16.0± 4.5
TT (Nm)	M	12.3± 3.6	12.1± 5.2
	F	12.8± 3.5	12.6± 3.9
TPT (msec)	M	180.9±20.0	152.1±27.1
	F	178.0±17.5	160.4±23.5
½ RT (msec)	M	127.7±28.0	133.0±29.1
	F	136.0±20.1	135.2±34.5
CT (msec)	M	308.6±42.5	285.1±42.7
	F	314.0±34.5	295.6±48.0

Values are means ± SD.

TABLE 3. Twitch Potentiation
(n=21, 11♂, 10♀)

Variable	Gender	1982 Rest	1982 Pot.	1982 %↑	1994 Rest	1994 Pot.	1994 %↑
DF:							
TT (Nm)	M	2.8±1.0	3.6±1.4	28.6	4.3±1.5	5.6±2.4	30.2
	F	2.1±0.8	2.9±0.8	38.1	3.4±0.7	4.7±0.7	38.2
PF:							
TT (Nm)	M	12.3±3.6	15.9±6.6	29.3	12.1±5.2	17.1±6.5	41.3
	F	12.8±3.5	13.2±5.8	3.1	12.6±3.9	16.8±5.0	33.3

Values are means ± SD.

FIGURE LEGENDS

FIGURE 1. Footplate apparatus (Marsh et al., 1981).

FIGURE 2. Relationship between physical activity scores and the percent change in maximum voluntary strength from 1982 to 1994, for dorsiflexor *(above)* and plantarflexor *(below)* muscles, for 11 male *(filled squares)* and 10 female *(filled triangles)* subjects.

FIGURE 3. Maximum voluntary strength (MVC) values for dorsiflexor *(above)* and plantarflexor *(below)* muscles, for 11 male *(left)* and 11 female *(right)* subjects, for 1982 *(open bars)* and 1994 *(filled bars)*. Time by muscle group interaction is indicated by asterisks. Values are means ± SD.

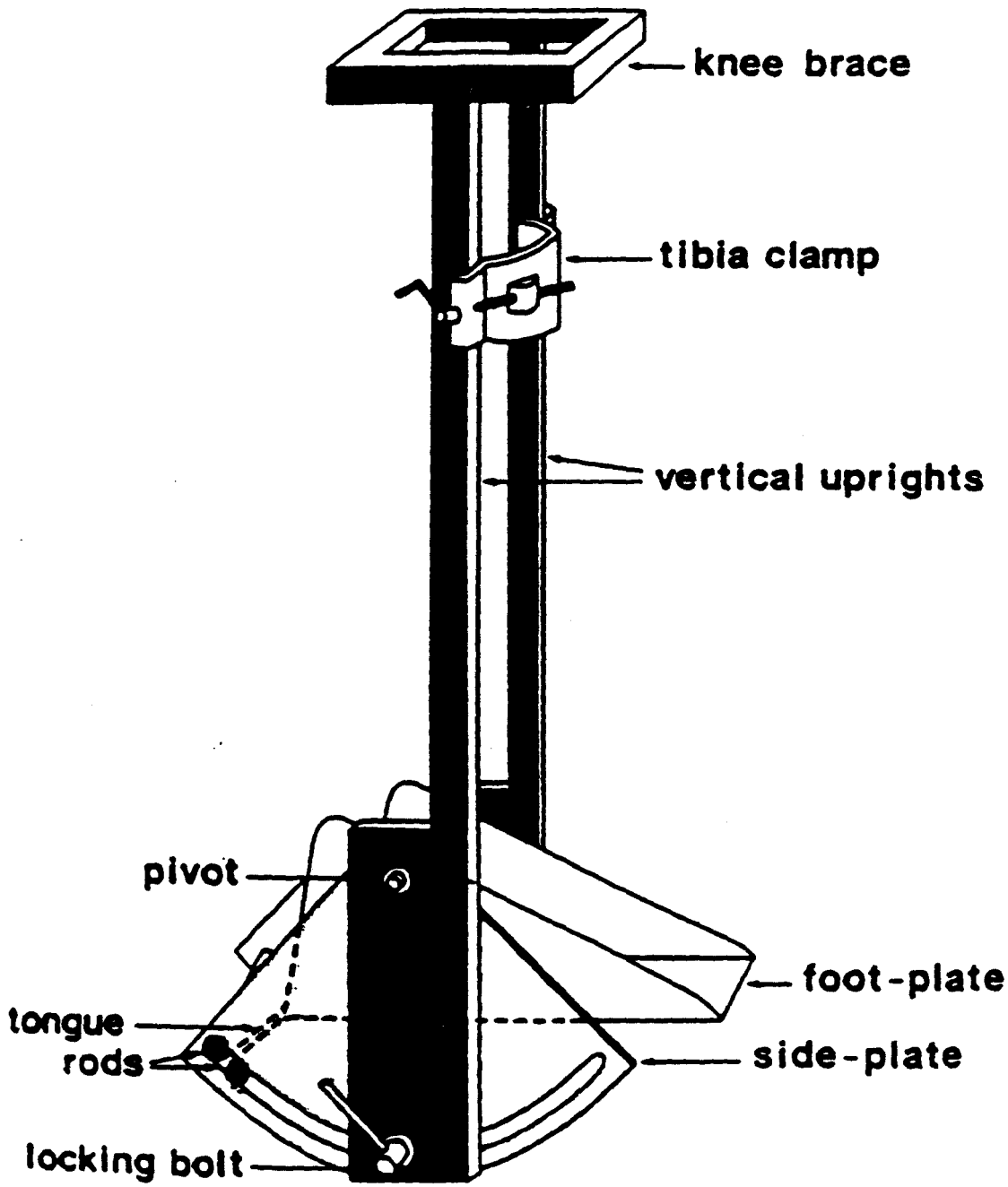
FIGURE 4. Maximum voluntary strength/mass ratios for dorsiflexor *(above)* and plantarflexor *(below)* muscles, for 11 male *(left)* and 11 female *(right)* subjects, for 1982 *(open bars)* and 1994 *(filled bars)*. Time by muscle group interaction is indicated by asterisks. Values are means ± SD.

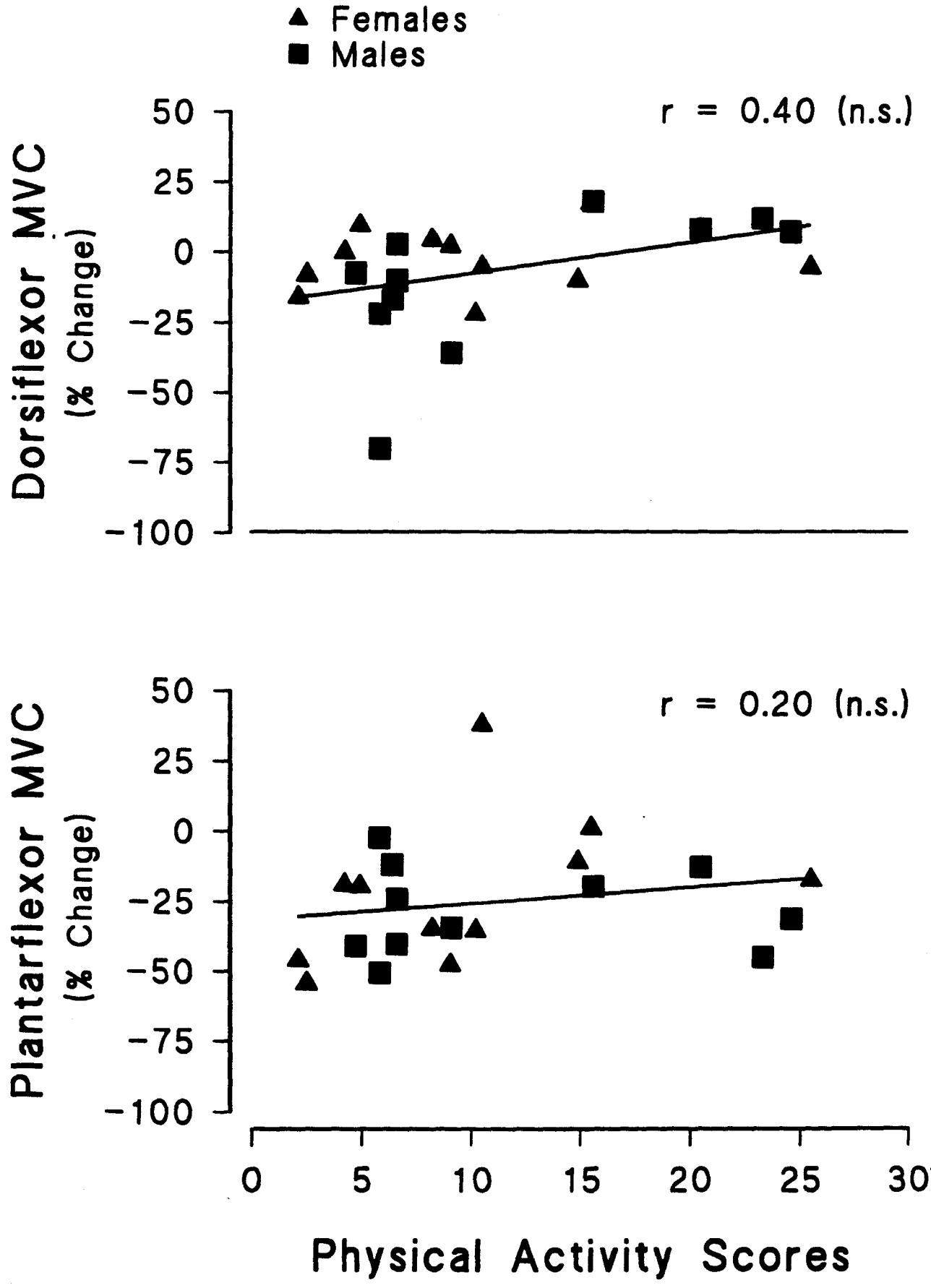
FIGURE 5. Passive tension values for dorsiflexor *(above)* and plantarflexor *(below)* muscles, for 11 male *(left)* and 10 female *(right)* subjects, for 1982 *(open bars)* and 1994 *(filled bars)*. A main effect for time was found for both genders and muscle groups. Values are means ± SD.

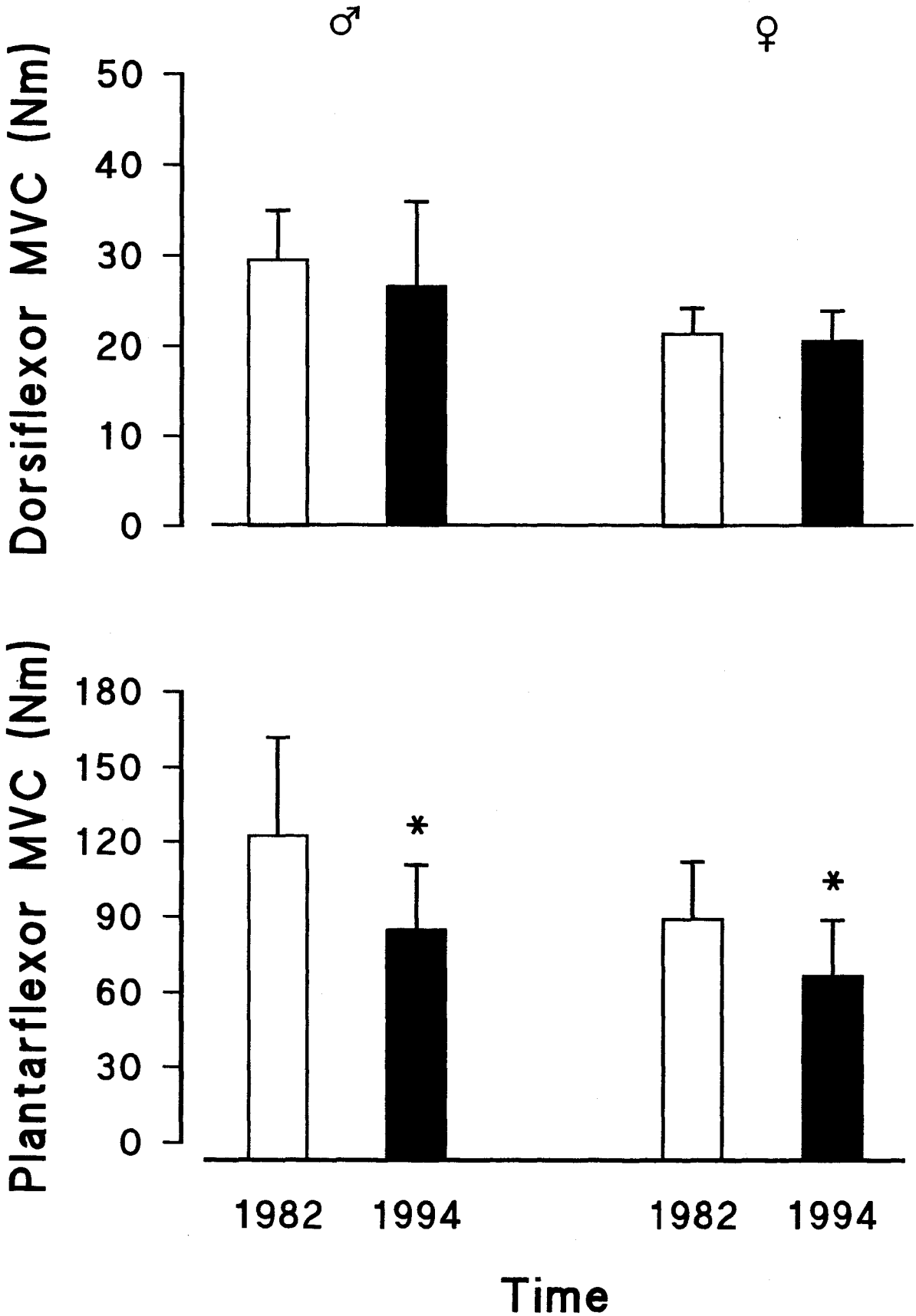
FIGURE 6. Peak twitch torque values for dorsiflexor *(above)* and plantarflexor *(below)* muscles, for 11 male *(left)* and 10 female *(right)* subjects, for 1982 *(open bars)* and 1994 *(filled bars)*. Values are means ± SD.

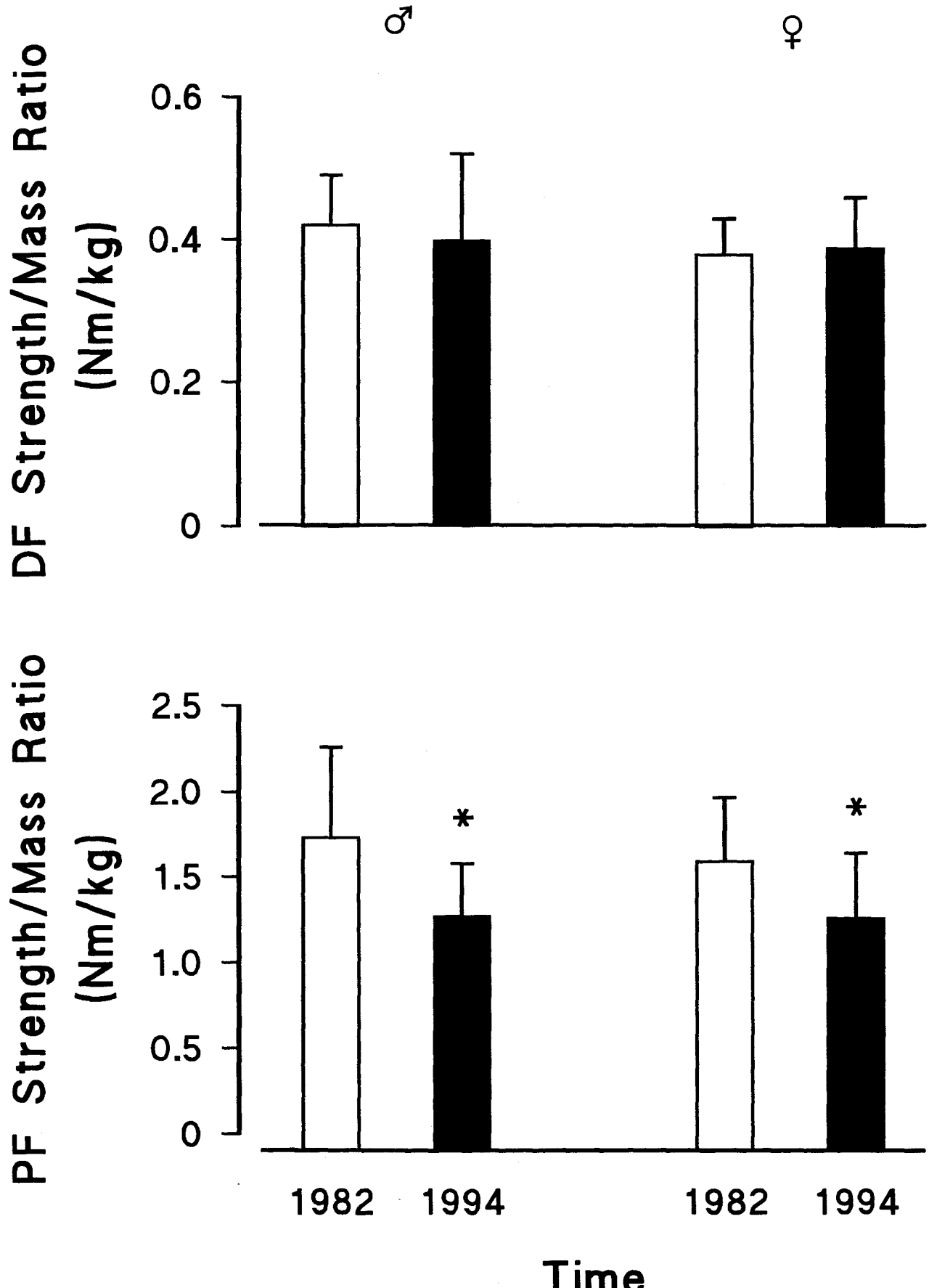
FIGURE 7. Peak twitch torque/maximum voluntary strength ratios for dorsiflexor *(above)* and plantarflexor *(below)* muscles, for 11 male *(left)* and 10 female *(right)* subjects, for 1982 *(open bars)* and 1994 *(filled bars)*. A main effect for time was found for both genders and muscle groups. Values are means ± SD.

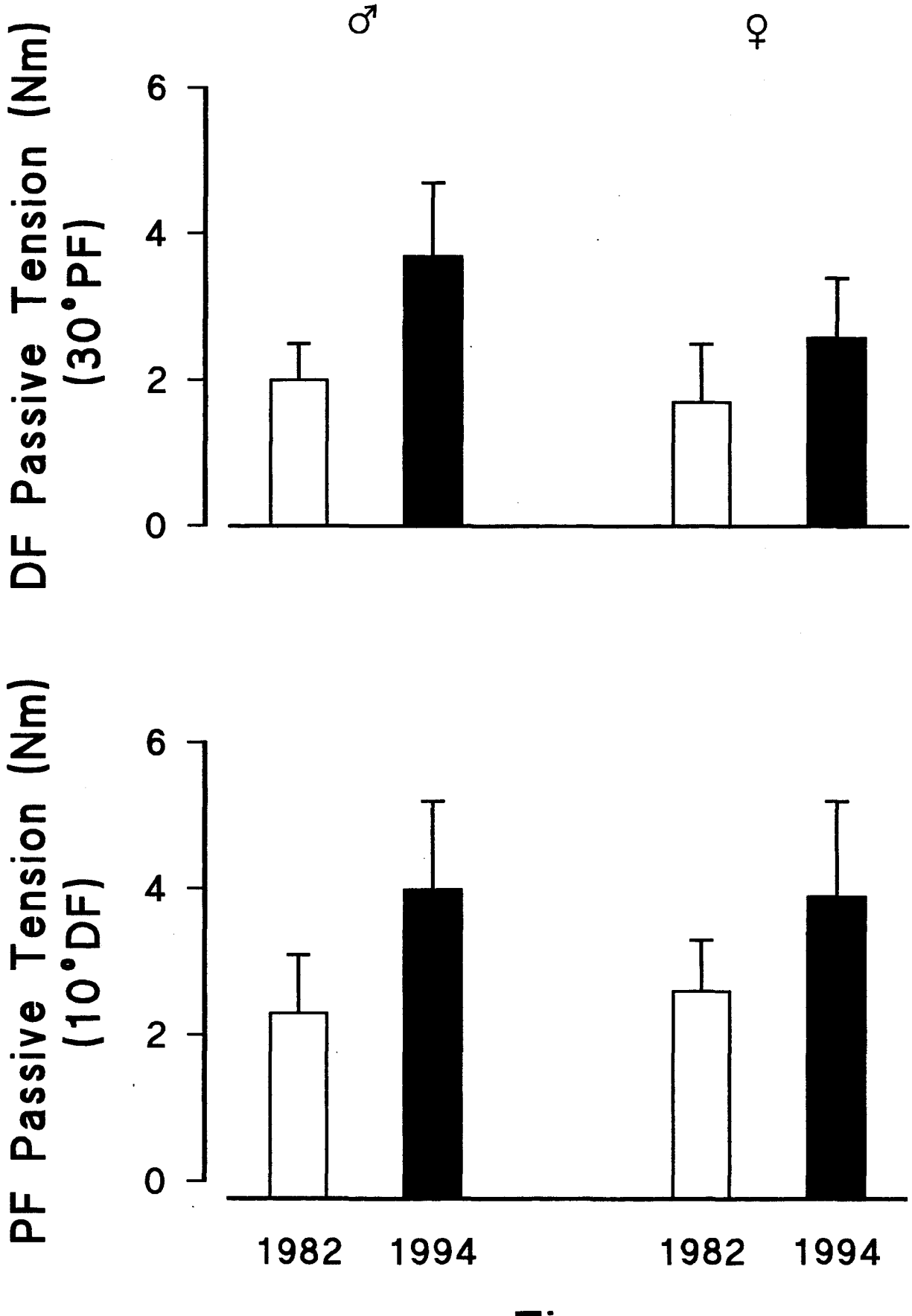
FIGURE 8. M-Wave amplitude values for dorsiflexor *(above)* and plantarflexor *(below)* muscles, for 11 male *(left)* and 10 female *(right)* subjects, for 1982 *(open bars)* and 1994 *(filled bars)*. Gender by time by muscle group interaction is indicated by asterisk. Values are means ± SD.

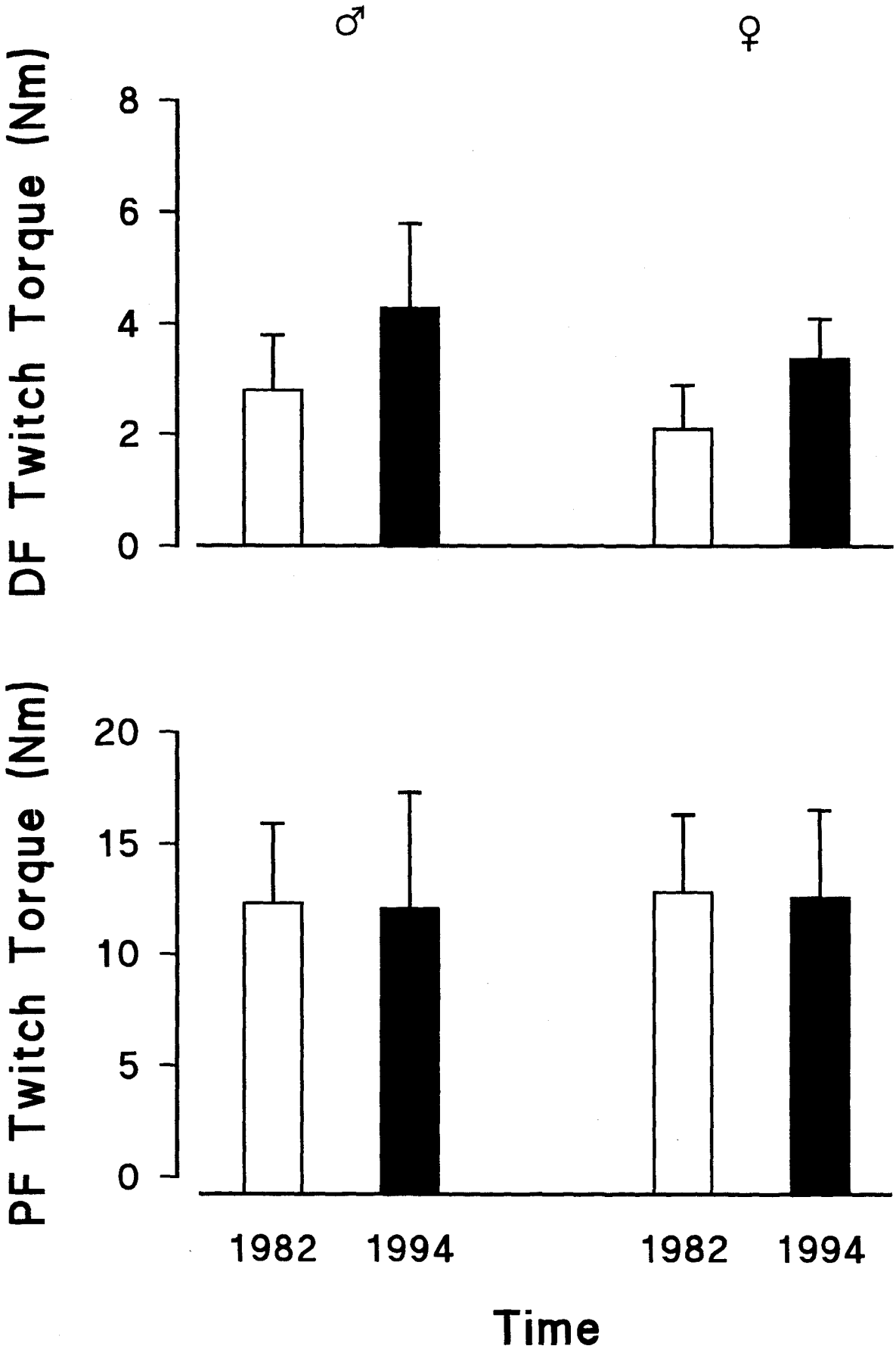

knee brace
tibia clamp
vertical uprights
pivot
foot-plate
tongue
rods
side-plate
locking bolt

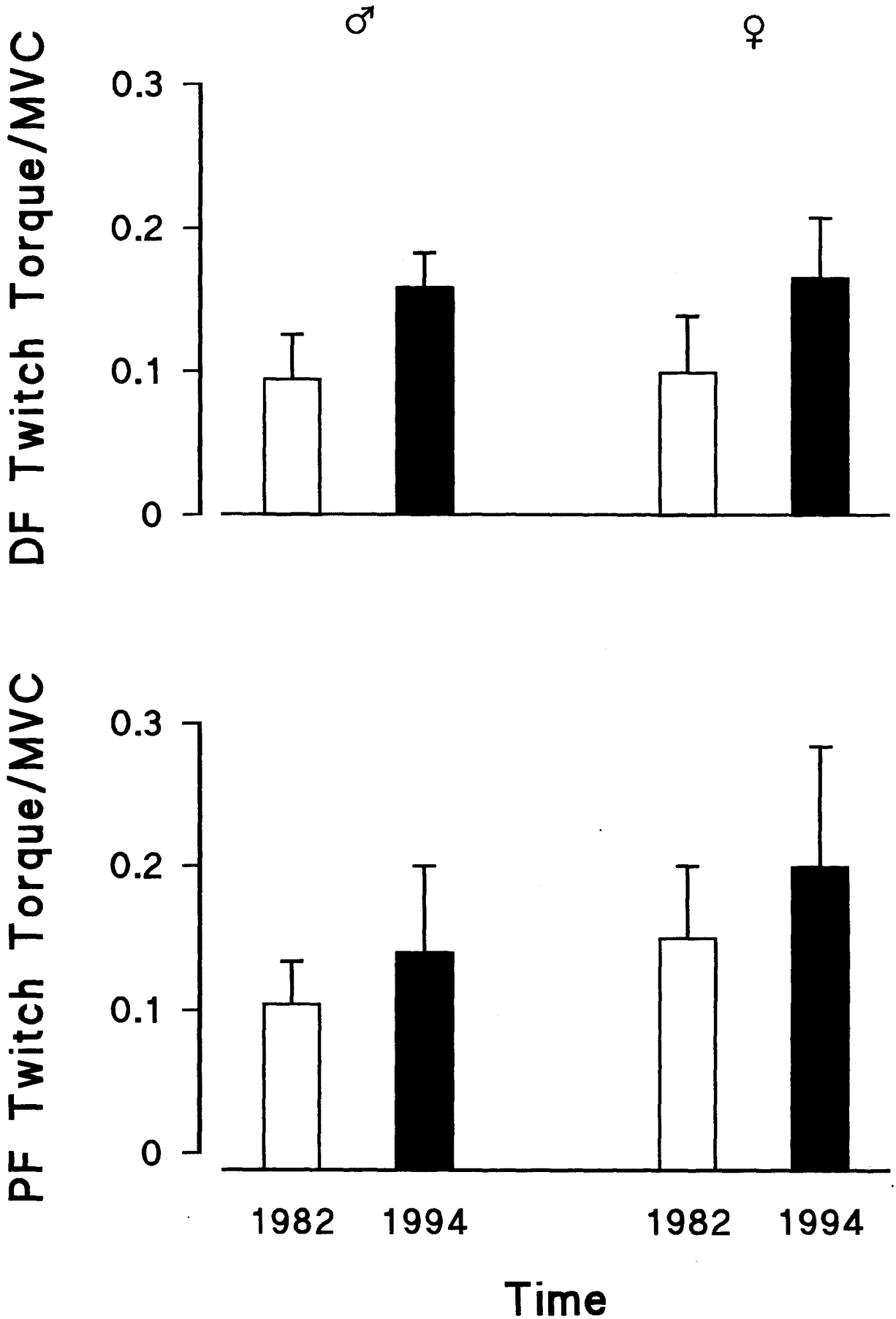

Females
Males
r = 0.40 (n.s.)
Dorsiflexor MVC
(% Change)
50
25
0
-25
-50
-75
-100
r = 0.20 (n.s.)
Plantarflexor MVC
(% Change)
50
25
0
-25
-50
-75
-100
0
5
10
15
20
25
30
Physical Activity Scores

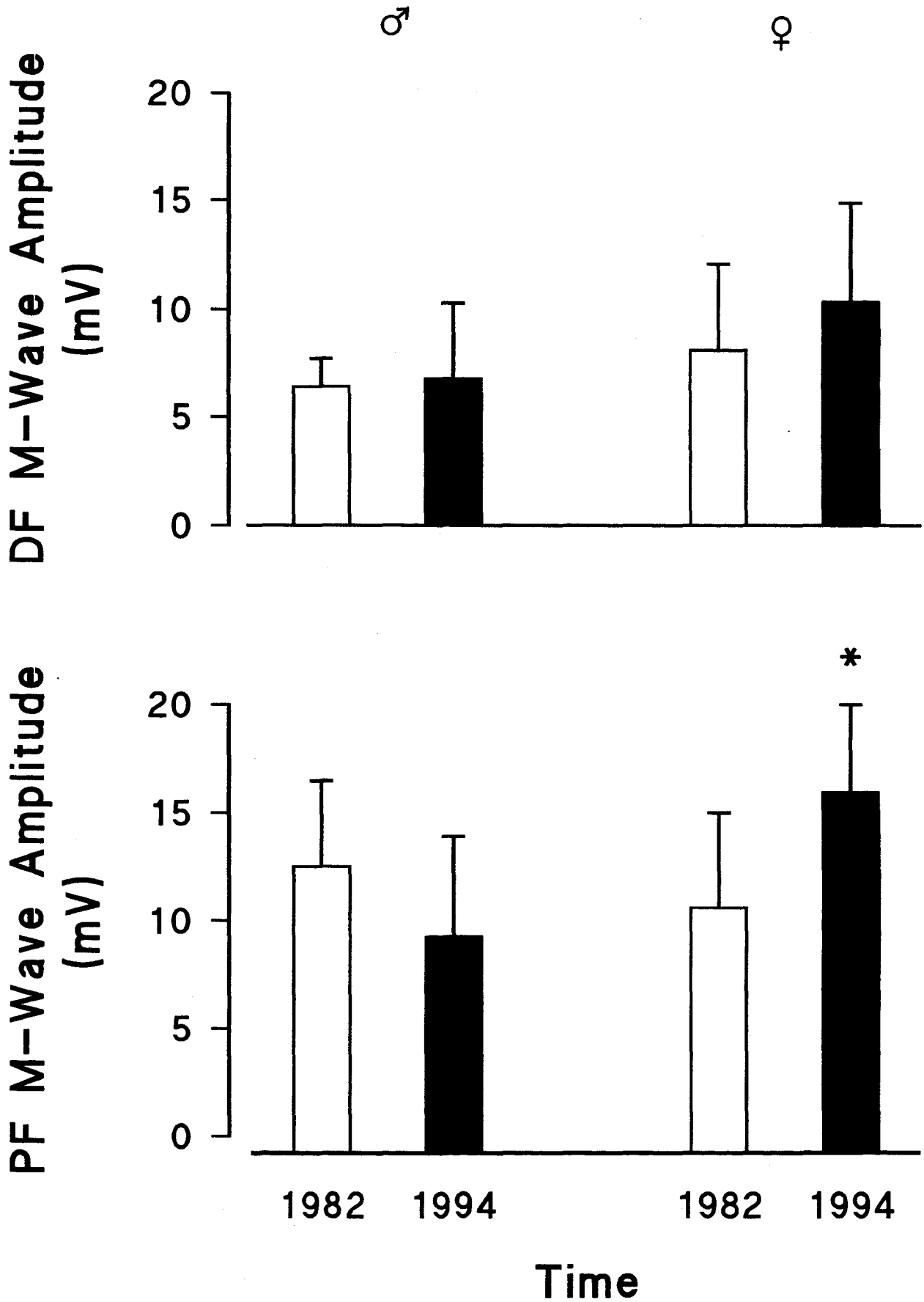

♂
♀
Dorsiflexor MVC (Nm)
50
40
30
20
10
0
Plantarflexor MVC (Nm)
180
150
120
90
60
30
0
*
*
1982
1994
1982
1994
Time


♂
♀
DF Strength/Mass Ratio (Nm/kg)
0.6
0.4
0.2
0
PF Strength/Mass Ratio (Nm/kg)
2.5
2.0
1.5
1.0
0.5
0
*
*
1982
1994
1982
1994
Time


♂
♀
DF Passive Tension (Nm) (30°PF)
6
4
2
0
PF Passive Tension (Nm) (10°DF)
6
4
2
0
1982
1994
1982
1994
Time


♂
♀
DF Twitch Torque (Nm)
8
6
4
2
0
PF Twitch Torque (Nm)
20
15
10
5
0
1982
1994
1982
1994
Time


♂
♀
DF Twitch Torque/MVC
0.3
0.2
0.1
0
PF Twitch Torque/MVC
0.3
0.2
0.1
0
1982
1994
1982
1994
Time


♂
♀
DF M-Wave Amplitude (mV)
20
15
10
5
0
PF M-Wave Amplitude (mV)
*
1982
1994
1982
1994
Time

REFERENCES

Alway, S.E., Hughson, R.L., Green, H.J., Patla, A.E., and Frank, J.S. Twitch potentiation after fatiguing exercise in man. *Eur J Appl Physiol.* 56:461-6, 1987.

Aniansson, A., Hedberg, M., Henning, G.B., and Grimby, G. Muscle morphology, enzymatic activity, and muscle strength in elderly men: a follow-up study. *Muscle & Nerve.* 9:585-91, 1986.

Aoyagi, Y., Shephard, R.J. Aging and muscle function. *Sports Med.* 14:376-96, 1992.

Baeke, J.A., Burema, J., and Frijters, J.E.R. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. *Am J Clin Nutr.* 36:936-42, 1982.

Baker, G.T., Martin, G.R. Biological aging and longevity: underlying mechanisms and potential intervention strategies. *J Aging Phys Act.* 2:304-28, 1994.

Bassey, E.J., Harris, U.J. Normal values for handgrip strength in 920 men and women aged over 65 year, and longitudinal changes over 4 years in 620 survivors. *Clin Sci.* 84:331-7, 1993.

Belanger, A.Y., McComas, A.J., and Elder, G.B.C. Physiological properties of two antagonistic human muscle groups. *Eur J Appl Physiol.* 51:381-93, 1983.

Belanger, A.Y., Quinlan, J. Muscle function studies in human plantar-flexor and dorsi-flexor muscles. *Can J Neurol Sci.* 9:358-9, 1982.

Belanger, A.Y., McComas, A.J. Extent of motor unit activation during effort. *J Appl Physiol Respirat Environ Exerc Physiol.* 51:1131-5, 1981.

Booth, F.W., Weeden, S.H., and Tseng, B.S. Effect of aging on human skeletal muscle and motor function. *Med Sci Sports Exerc.* 26(5):556-560, 1994.

Brousseau, T., Legrain, S., Berr, C., Gourlet, V., Vidal, I.O., and Amouyel, P. Confirmation of the E4 allele of the apolipoprotein E gene as a risk factor for late-onset Alzheimer's disease. *Neurology* 44, 342-4, 1994.

Brown, M., Ross, T.P., and Hollosky, J.O. Effects of ageing and exercise on soleus and extensor digitorum longus muscles of female rats. *Mech of Ageing & Develop.* 63:69-77, 1992.

Brown, A.B., McCartney, N., and Sale, D.G. Positive adaptations to weight-lifting training in the elderly. *J Appl Physiol.* 69:1725-33, 1990.

Campbell, M.J., McComas, A.J., and Petito, F. Physiological changes in ageing muscles. *J Neurol Neurosurg Psych.* 36:174-82, 1973.

Cartee, G.D. Aging skeletal muscle: response to exercise. In: *Exercise and Sport Sciences Reviews*, edited by J.O. Holloszy. St. Louis, Missouri: Williams & Wilkins, 22:91-120, 1994.

Caspersen, C.J., Christenson, G.M., and Pollard, R.A. Status of the 1990 physical fitness and exercise objectives-evidence from NHIS 1985. *Public Health Reports.* 101:587-93, 1986.

Charette, S.L., McEvoy, L., Pyka, G., Snow-Harter, C., Guido, D., Wiswell, R.A., and Marcus, R. Muscle hypertrophy response to resistance training in older women. *J Appl Physiol.* 70:1912-6, 1991.

Christ, C.B., Boileau, R.A., Slaughter, M.H., Stillman, R.J., Cameron, J.A., and Massey, B.H. Maximal voluntary isometric force production characteristics of six muscle groups in women aged 25 to 74 years. *Am J Human Biol.* 4:537-45, 1992.

Close, R.I. Dynamic properties of mammalian skeletal muscles. *Physiol Rev.* 52:129-86, 1972.

Coggan, A.R., Spina, R.J., King, D.S., Rogers, M.A., Brown, M., Nemeth, P.M., and Holloszy, J.O. Histochemical and enzymatic comparison of the gastrocnemius muscle of young and elderly men and women. *J Gerontol.* 47:B71-6, 1992.

Corpas, E., Harman, S.M., and Blackman, M.R. Human growth hormone and human aging. *Endocrine Reviews* 14(1):20-39, 1993.

Courtney, J., Steinbach, J.H. Age changes in neuromuscular junction morphology and acetylcholine receptor distribution on rat skeletal muscle fibres. *J Physiol.* 320:435-47, 1981.

Cristofalo, V.J. Overview of biological mechansim of aging. *Annual Review of Gerontology and Geriatrics 10:* 1-22, 1990.

Cunningham, W.R., Brookbank, J.W. *Gerontology: The Psychology, Biology, & Sociology of Aging.* New York: Harper & Row, 60-77, 1988.

Cupido, C.M., Hicks, A.L., and Martin, J. Neuromuscular fatigue during repetitive stimulation in elderly and young subjects. *Eur J Appl Physiol.* 65:567-72, 1992.

Davies, C.T.M., Thomas, D.O., and White, M.J. Mechanical properties of young and elderly human muscle. *Acta Med Scand Suppl.* 711:219-26, 1986.

de Koning, P., Wieneke, G.H., Van der Most van Spijk, D., Van Huffelen, A.C., Gispen, W.H., and Jennekens, F.G.I. Estimation of the number of motor units based on macro-EMG. *J Neurol Neurosurg & Psychiat.* 51:403-11, 1988.

Dishman, R.K. Determinants of physical activity and exercise for persons 65 years of age or older. In: *Physical Activity & Aging.* Champaign, IL: Human Kinetics Books, 140-62, 1989.

Doherty, T.J., Vandervoort, A.A., and Brown, W.F. Effects of ageing on the motor unit: a brief review. *Can J Appl Physiol.* 18:331-58, 1993a.

Doherty, T.J., Vandervoort, A.A., Taylor, A.W., and Brown, W.F. Effects of motor unit losses on strength in older men and women. *J Appl Physiol.* 74:868-74, 1993b.

Essen-Gustavsson, B., Borges, O. Histochemical and metabolic characteristics of human skeletal muscle in relation to age. *Acta Physiol Scand.* 126:107-14, 1986.

Fiatarone, M.A., O'Neil, E.F., Ryan, N.D., Clements, K.M., Solares, G.R., Nelson, M.E., Roberts, S.B., Kehayias, J.J., Lipsitz, L.A., and Evans, W.J. Exercise training and nutritional supplementation for physical frailty in very elderly people. *New Engl J Med.* 330:1769-75, 1994.

Fiatarone, M.A., Evans, W.J. The etiology and reversibility of muscle dysfunction in the aged. *J Gerontol.* 48:77-83, 1993.

Fiatarone, M.A., Marks, E.C., Ryan, N.D., Meredith, C.N., Lipsitz, L.A., and Evans, W.J. High-intensity strength training in nonagenarians: Effects on skeletal muscle. *JAMA.* 263:3029-34, 1990.

Frontera, W.R., Meredith, C.N., O'Reilly, K.P., Knuttgen, H.G., and Evans, W.J. Strength conditioning in older men: skeletal muscle hypertrophy and improved function. *J Appl Physiol.* 64:1038-44, 1988.

Greig, C.A., Botella, J., and Young, A. The quadriceps strength of healthy elderly people remeasured after eight years. *Muscle & Nerve.* 16:6-10, 1993.

Grimby, G., Saltin, B. The ageing muscle. *Clin Physiol.* 3:209-18, 1983.

Grimby, G., Danneskiold-Samsoe, B., Hvid, K., and Saltin, B. Morphology and enzymatic capacity in arm and leg muscles in 78-81 year old men and women. *Acta Physiol Scand.* 115:125-34, 1982.

Gulve, E.A., Rodnick, K.J., Henriksen, E.J., and Holloszy, J.O. Effects of wheel running on glucose transporter (GLUT4) concentration in skeletal muscle of young adult and old rats. *Mech Ageing & Develop.* 67:187-200, 1993.

Harley, C.B. Telomere loss: mitotic clock or genetic time bomb? *Mutation Research* 256:1271-82, 1991.

Hicks, A.L., Cupido, C.M., Martin, J., and Dent, J. Muscle excitation in elderly adults: the effects of training. *Muscle & Nerve.* 15:87-93, 1992.

Hicks, A.L., Cupido, C.M., Martin, J., and Dent, J. Twitch potentiation during fatiguing exercise in the elderly: the effects of training. *Eur J Appl Physiol.* 63:278-81, 1991.

Holloszy, J.O., Chen, M., Cartee, G.D., and Young, J.C. Skeletal muscle atrophy in old rats: differential changes in the three fiber types. *Mech Ageing & Develop.* 60:199-213, 1991.

Houston, M.E., Green, H.J., and Stull, J.T. Myosin light chain phosphorylation and isometric twitch potentiation in intact human muscle. *Pflugers Arch.* 403:348-52, 1985.

Johnson, M.A., Polgar, J., Weightman, D., and Appleton, D. Data on the distribution of fibre types in thirty-six human muscles: an autopsy study. *J Neurol Sci.* 18:111-29, 1973.

Kallman, D.A., Plato, C.C., and Tobin, J.D. The role of muscle loss in the age-related decline of grip strength: cross-sectional and longitudinal perspectives. *J Gerontol: Med Sci.* 45:M82-8, 1990.

Kanda, K., Hashizume, K., Nomoto, E., and Asaki, S. The effects of aging on physiological properties of fast and slow twitch units in the rat gastrocnemius. *Neurosci Res.* 3:242-6, 1986.

Kavanagh, T., Shephard, R.J. Can regular sports participation slow the aging process? Data on masters athletes. *Phys Sportsmed.* 18:94-104, 1990.

Kenney, R.A. *Physiology of Aging* (2nd ed.). Chicago: Year Book Medical Publishers,15-25, 1989.

Klein, C., Cunningham, D.A., Paterson, D.H., and Taylor, A.W. Fatigue and recovery contractile properties of young and elderly men. *Eur J Appl Physiol.* 57:684-90, 1988.

Klitgaard, H., Mantoni, M., Schiaffino, S., Ausoni, S., Gorza, L., Laurent-Winter, C., Schnohr, P., and Saltin, B. Function, morphology and protein expression of ageing skeletal muscle: a cross-sectional study of elderly men with different training backgrounds. *Acta Physiol Scand.* 140:41-54, 1990a.

Klitgaard, H., Zhou, M., Schiaffino, S., Betto, R., Salviati, G., and Saltin, B. Ageing alters the myosin heavy chain composition of single fibres from human skeletal muscle. *Acta Physiol Scand.* 140:55-62, 1990b.

Klitgaard, H., Marc, R., Brunet, A., Vandevalle, H., and Monod, H. Contractile properties of old rat muscles: effect of increased usage. *J Appl Physiol.* 67:1401-8, 1989.

Larsson, L., Salviati, G. Effects of age on calcium transport activity of sarcoplasmic reticulum in fast- and slow-twitch rat muscle fibres. *J Physiol.* 419:253-64, 1989.

Larsson, L., Karlsson, J. Isometric and dynamic endurance as a function of age and skeletal muscle characteristics. *Acta Physiol Scand.* 104:129-36, 1978.

Lexell, J. Ageing and human muscle: observations from Sweden. *Can J Appl Phys.* 18:2-18, 1993.

Lexell, J., Downham, D.Y. The occurrence of fibre-type grouping in healthy human muscle: a quantitative study of cross-sections of whole vastus lateralis from men between 15 and 83 years. *Acta Neuropathol.* 81:377-81, 1991.

Lexell, J., Taylor, C.C., and Sjostrom, M. What is the cause of ageing atrophy? *J Neurol Sci.* 84:275-94, 1988.

Lexell, J., Downham, D., and Sjostrom, M. Distribution of different fibre types in human skeletal muscles. *J Neurol Sci.* 72:211-22, 1986.

Lexell, J., Henriksson-Larsen, K., Winblad, B., and Sjostrom, M. Distribution of different fiber types in human skeletal muscles: effects of aging studied in whole muscle cross sections. *Muscle & Nerve* 6:588-95, 1983.

Marsh, E., Sale, D.G., McComas, A.J., and Quinlan, J. Influence of joint position on ankle dorsiflexion in humans. *J Appl Physiol: Respirat Environ Exerc Physiol.* 51:160-7, 1981.

McCartney, N., Hicks, A.L., Martin, J., and Webber, C.E. Long-term resistance training in the elderly: effects on dynamic strength, exercise capacity, muscle, and bone. *J Gerontol: Bio Sci.* 50A:B97-B104, 1995a.

McCartney, N., Hicks, A.L., Martin, J., and Webber, C.E. A two year trial of resistance training in the elderly: effects on strength, exercise capacity, muscle and bone. [Submitted for Publication] 1995b.

McDonagh, M.J.N., White, M.J., and Davies, C.T.M. Different effects of ageing on the mechanical properties of human arm and leg muscles. *Gerontology* 30:49-54, 1984.

McPherson, B.D. Sociocultural perspectives on aging and physical activity. *J Aging Phys Act.* 2:329-53, 1994.

Meites, J. Anti-aging interventions and their neuroendocrine aspects in mammals. *J Reprod & Fert.* 49:1-9, 1993.

Morrison, N.A., Q1, J.C., Takita, A., Kelly, P.J., Crofts, L., Nguyen, T.V., Sambrook, P.N., and Eisman, J.A. Prediction of bone density from vitamin D receptor alleles. *Nature* 367:284-9, 1994.

Narici, M.V., Bordini, M., and Cerretelli, P. Effects of aging on human adductor pollicis muscle function. *J Appl Physiol.* 71:1277-81, 1991.

Olshansky, S.J., Carnes, B.A., and Cassel, C. In search of Methuselah: estimating the upper limits to human longevity. *Science* 250: 634, 1990.

Overend, T.J., Cunningham, D.A., Paterson, D.H., and Lefcoe, M.S. Thigh composition in young and elderly men determined by computed tomography. *Clin Physiol.* 12:629-40, 1992.

Perusse, L., Tremblay, A., LeBlanc, C., and Bouchard, C. Genetic and environmental influences on levels of habitual activity and exercise participation. *Am J Epidem.* 129(5):1012-22, 1989.

Petrella, R.J., Cunningham, D.A., Vandervoort, A.A., and Paterson, D.H. Comparison of twitch potentiation in the gastrocnemius of young and elderly men. *Eur J Appl Physiol.* 58:395-9, 1989.

Porter, M.M., Vandervoort, A.A., and Lexell, J. Ageing of human muscle: structure, function and adaptability. *Scand J Med Sci Sport* 5:129-42, 1995.

Rice, C.L., Cunningham, D.A., Paterson, D.H., and Lefcoe, M.S. A comparison of anthropometry with computed tomography in limbs of young and aged men. *J Gerontol: Med Sci.* 45:M175-9, 1990.

Rice, C.L., Cunningham, D.A., Paterson, D.H., and Lefcoe, M.S. Arm and leg composition determined by computed tomography in young and elderly men. *Clin Physiol.* 9:207-20, 1989.

Rogers, M.A., Evans, W.J.. Changes in skeletal muscle with aging: effects of exercise training. In: *Exercise and Sport Sciences Reviews,* edited by J.O.Holloszy. St. Louis, Missouri: Williams & Wilkins, 65-102, 1993.

Runyon, R.P., Haber, A. *Fundamentals of Behavioral Statistics,* (7th ed.). New York: McGraw-Hill, 195-7, 1991.

Sale, D.G. Neural adaptations to resistance training. *Med Sci Sports Exerc.* 20(5): suppl. S135-45, 1988.

Sale, D., Quinlan, J., Marsh, E., McComas, A.J., and Belanger, A.Y. Influence of joint position on ankle plantarflexion in humans. *J Appl Physiol: Respirat Environ Exerc Physiol.* 52:1636-42, 1982.

Sandler, R.B., Burdett, R., Zaleskiewicz, M., Sprowls-Repcheck, C., and Harwell, M. Muscle strength as an indicator of the habitual level of physical activity. *Med Sci Sports Exerc.* 23(12): 1375-81, 1991.

Sipila, S., Viitasalo, J., Era, P., and Suominen, H. Muscle strength in male athletes aged 70-81 years and a population sample. *Eur J Appl Physiol.* 63:399-403, 1991.

Smith, D.O. Acetylcholine storage, release, and leakage at the neuromuscular junction of mature adult and aged rats. *J Physiol (London).* 347:161-76, 1984.

Smith, D.O., Rosenheimer, J.L. Aging at the neuromuscular junction. In: *Ageing & Cell Structure,* vol. 2, edited by J.E. Johnson Jr. New York: Plenum Press, 113-39, 1984.

Smith, D.O., Rosenheimer, J.L. Decreased sprouting and degeneration of nerve terminals of active muscles in aged rats. *J Neurophysiol.* 48:100-9, 1982.

Smith, D.O. Reduced capabilities of synaptic transmission in aged rats. *Exp Neurol.* 66:650-66, 1979.

Spence, A.P. *Biology of Human Aging.* Eaglewood Cliffs, N.J.: Prentice Hall, 13-26, 1989.

Stalberg, E., Borges, O., Ericsson, M., Essen-Gustavsson, B., Fawcett, P.R.W., Nordesjo, L.O., Nordgren, B., and Uhlin, R. The quadriceps femoris muscle in 20-70-year-old subjects: relationship between knee extension torque, electrophysiological parameters, and muscle fiber characteristics. *Muscle & Nerve.* 12:382-9, 1989.

Stephens, T., Jacobs, D.R., and White, C.C. A descriptive epidemiology of leisure-time physical activity. *Public Health Reports* 100(2):147-158, 1985.

Timiras, P.S. *Physiological Basis of Geriatrics.* New York: MacMillan Pub., 43-58, 1988.

Tzankoff, S.P., Norris, A.H. Effect of muscle mass decrease on age-related BMR changes. *J Appl Physiol: Respirat Environ Exerc Physiol.* 43:1001-6, 1977.

van Schaik, C.S., Hicks, A.L., and McCartney, N. An evaluation of the length-tension relationship in elderly human ankle dorsiflexors. *J Gerontol: Bio Sci.* 49:B121-7, 1994.

Vandervoort, A.A. Biological and physiological changes. [In Press] In: *Physiotherapy with Older People*, edited by B. Pickles, A. Compton, C. Cott, J. Simpson, and A.A. Vandervoort, 1995.

Vandervoort, A.A. Effects of ageing on human neuromuscular function: implications for exercise. *Can J Sport Sci.* 17:178-84, 1992.

Vandervoort, A.A., Chesworth, B.M., Cunningham, D.A., Paterson, D.H., Rechnitzer, P.A., and Koval, J.J. Age and sex effects on mobility of the human ankle. *J Gerontol: Med Sci.* 47:M17-21, 1992.

Vandervoort, A.A., Hayes, K.C. Plantarflexor muscle function in young and elderly women. *Eur J Appl Physiol.* 58:389-94, 1989.

Vandervoort, A.A., McComas, A.J. Contractile changes in opposing muscles of the human ankle joint with aging. *J Appl Physiol.* 61:361-7, 1986.

Vandervoort, A.A., Quinlan, J., and McComas, A.J. Twitch potentiation after voluntary contraction. *Exp Neurol.* 81:141-52, 1983.

Voorrips, L.E., Ravelli, A.C.J., Dongelmans, P.C.A., Deurenberg, P., and Van Staveren, W.A. A physical activity questionnaire for the elderly. *Med Sci Sports Exerc.* 23:974-9, 1991.

Williams, G.T., Smith, C.A. Molecular regulation of apoptosis: genetic controls on cell death. *Cell* 4: 777-9, 1993.

Winter, D.A., Patla, A.E., Frank, J.S., and Walt, S.E. Biomechanical walking pattern changes in the fit and healthy elderly. *Phys Ther.* 70:340-7, 1990.

Wokke, J.H.J., Jennekens, F.G.I., van den Oord, C.J.M., Veldman, H., Smit, L.M.E., and Leppink, G.J. Morphological changes in human end plate with age. *J Neurol Sci.* 95:291-310, 1990.

Young, A., Stokes, M., and Crowe, M. Size and strength of the quadriceps muscles of old and young women. *Eur J Clin Invest.* 14:282-7, 1984.

Young, A., Stokes, M., and Crowe, M. The size and strength of the quadriceps muscles of old and young men. *Clin Physiol.* 5:145-54, 1985.

APPENDIX A:

PHYSICAL CHARACTERISTICS AND DATA

Physical Characteristics: Age, Height, Weight, & Physical Activity Scores

		Age (yrs)		Height (cm)		Weight (kg)		PAS
Subject	Gender	1982	1994	1982	1994	1982	1994	1994
BC	M	75	87	182.0	176.5	87.8	81.8	6.6
RC	M	69	81	172.0	170.0	62.4	62.7	15.6
MC	F	62	74	156.0	152.4	54.6	50.0	25.5
LD	F	62	74	163.0	157.5	75.6	76.8	9.1
KD	M	73	85	180.0	177.8	71.8	70.5	6.6
MD	F	78	90	156.0	149.9	38.9	35.0	10.5
DK	F	69	82	157.0	147.3	57.3	50.0	2.5
HL	M	85	97	162.0	157.5	63.2	58.0	5.8
GL	M	79	91	166.0	162.6	67.8	54.5	4.7
TL	F	70	82	170.0	167.6	62.0	64.6	4.9
DL	F	82	94	155.0	147.3	49.3	40.0	2.1
RL	M	71	83	183.0	177.8	78.8	85.0	20.5
MM	M	74	86	180.0	174.0	68.5	57.3	5.8
DM	M	62	74	164.0	162.6	62.0	66.4	24.6
AM	F	70	82	160.0	154.9	61.2	57.3	10.2
CM	M	80	92	175.0	170.2	74.4	68.2	9.1
MM	M	61	73	179.0	172.7	69.3	63.6	23.3
AM	F	69	81	151.0	149.6	56.7	51.1	4.2
RP	M	80	92	171.0	165.1	72.8	68.2	6.4
AS	F	63	75	155.0	152.4	50.6	51.5	15.5
WS	F	73	85	165.0	158.8	54.3	54.6	8.2
VW	F	66	78	156.0	154.9	59.0	68.0	14.9

Passive Tension & Plantarflexor Range of Motion

		Age (yrs)		PT - DF (Nm)		PT - PF (Nm)		ROM	
Subject	Gender	1982	1994	1982	1994	1982	1994	1982	1994
BC	M	75	87	2.4	5.1	2.5	3.6	40	30
RC	M	69	81	1.7	3.2	2.5	4.7	31	25
MC	F	62	74	1.3	3.1	1.8	2.8	40	37
LD	F	62	74	1.8	3.7	2.9	5.4	31	30
KD	M	73	85	1.3	2.8	2.2	3.4	36	32
MD	F	78	90	0.8	2.3	2.9	4.4	25	20
DK	F	69	82	1.3	3.1	2.9	4.1	18	15
HL	M	85	97	1.7	3.0	1.4	2.8	38	30
GL	M	79	91	1.8	2.8	1.8	2.8	37	20
TL	F	70	82	1.5	2.4	1.8	6.0	23	20
DL	F	82	94	1.5	N/A	3.0	N/A	25	20
RL	M	71	83	1.7	4.5	2.2	4.8	40	28
MM	M	74	86	2.2	4.1	1.5	3.3	38	30
DM	M	62	74	1.7	2.8	3.6	5.7	30	30
AM	F	70	82	2.2	1.1	3.6	2.3	22	30
CM	M	80	92	3.1	5.2	2.9	5.1	25	25
MM	M	61	73	2.0	2.6	1.5	2.2	40	35
AM	F	69	81	3.4	3.3	2.5	2.8	29	25
RP	M	80	92	2.4	4.4	3.6	5.7	30	25
AS	F	63	75	0.9	1.6	1.8	2.6	40	35
WS	F	73	85	1.3	2.8	3.6	4.8	30	30
VW	F	66	78	2.0	2.4	2.5	3.7	30	30

Dorsiflexor Resting Twitch Properties & M-Wave Characteristics

		Age (yrs)		M-Wave (mV)		TT (Nm)		TPT (msec)		1/2 RT (msec)		CT (msec)	
Subject	Gender	1982	1994	1982	1994	1982	1994	1982	1994	1982	1994	1982	1994
BC	M	75	87	8.4	6.5	4.6	6.9	125	105	140	166	265	271
RC	M	69	81	5.2	5.5	2.2	5.2	110	95	125	130	235	225
MC	F	62	74	12.0	13.5	3.4	3.7	115	96	110	109	225	205
LD	F	62	74	4.0	13.5	1.9	1.9	125	78	120	128	245	206
KD	M	73	85	4.6	10.1	3.7	5.2	125	101	135	141	260	242
MD	F	78	90	5.6	7.3	1.7	3.6	100	83	90	101	190	184
DK	F	69	82	3.4	4.3	2.0	3.9	115	91	130	146	245	237
HL	M	85	97	5.4	5.9	2.1	4.3	100	93	75	96	175	189
GL	M	79	91	9.0	3.9	2.4	2.6	120	97	140	124	260	221
TL	F	70	82	4.0	8.4	2.9	4.1	115	124	145	140	260	264
DL	F	82	94	4.5	N/A	2.2	N/A	115	N/A	100	N/A	215	N/A
RL	M	71	83	6.6	7.9	2.9	4.5	115	102	110	103	225	205
MM	M	74	86	6.0	0.6	1.4	1.1	150	134	130	132	280	266
DM	M	62	74	6.7	11.5	2.0	5.0	90	86	90	104	180	190
AM	F	70	82	9.4	11.8	1.7	4.0	100	90	80	121	180	211
CM	M	80	92	5.8	5.5	4.0	4.2	130	127	115	142	245	269
MM	M	61	73	6.4	12.6	1.7	4.9	110	86	100	105	210	191
AM	F	69	81	12.0	11.8	2.6	3.6	105	69	120	106	225	175
RP	M	80	92	6.8	4.5	3.5	3.1	125	100	130	161	255	261
AS	F	63	75	15.0	18.3	2.9	3.4	115	98	120	98	235	196
WS	F	73	85	8.0	3.9	1.0	2.8	100	93	110	102	210	195
VW	F	66	78	8.0	11.5	1.2	3.3	110	79	80	117	190	196

Plantarflexor Resting Twitch Properties & M-Wave Characteristics

		Age (yrs)		M-Wave (mV)		TT (Nm)		TPT (msec)		1/2 RT (msec)		CT (msec)	
Subject	Gender	1982	1994	1982	1994	1982	1994	1982	1994	1982	1994	1982	1994
BC	M	75	87	16.0	5.5	16.5	12.7	200	182	140	119	340	301
RC	M	69	81	13.0	12.0	12.2	12.5	200	168	145	174	345	342
MC	F	62	74	8.5	17.8	10.0	11.8	175	160	145	124	320	284
LD	F	62	74	10.5	13.8	10.9	12.9	200	178	175	168	375	346
KD	M	73	85	16.0	13.3	17.2	15.1	180	168	115	128	295	290
MD	F	78	90	13.5	11.9	12.5	6.3	160	112	125	148	285	260
DK	F	69	82	11.5	12.1	13.2	12.3	185	162	145	173	330	335
HL	M	85	97	13.0	7.4	11.1	6.1	170	129	110	135	280	264
GL	M	79	91	14.5	8.0	11.4	12.5	200	137	150	173	350	310
TL	F	70	82	6.4	9.4	18.6	9.6	210	201	150	169	360	370
DL	F	82	94	7.8	N/A	12.5	N/A	185	N/A	135	N/A	220	N/A
RL	M	71	83	8.5	7.5	8.6	12.2	160	151	95	117	255	268
MM	M	74	86	5.0	2.2	5.4	1.3	175	141	185	114	360	255
DM	M	62	74	9.5	16.2	15.2	19.8	155	145	105	104	260	249
AM	F	70	82	12.0	18.2	11.2	14.2	150	167	120	126	270	293
CM	M	80	92	10.5	5.3	16.0	16.3	175	133	135	172	310	305
MM	M	61	73	19.5	16.3	10.4	16.7	160	112	90	89	250	201
AM	F	69	81	9.0	21.9	10.0	9.8	170	141	110	85	280	226
RP	M	80	92	12.0	9.0	11.3	8.2	215	207	135	138	350	345
AS	F	63	75	21.0	22.5	8.2	11.3	180	172	120	103	300	275
WS	F	73	85	6.0	13.4	17.9	19.7	175	149	120	89	295	238
VW	F	66	78	7.5	19.1	15.4	17.6	175	162	150	167	325	329

Voluntary Strength, Strength/Mass Ratio, & Twitch Torque/MVC Ratio

		DF MVC (Nm)		PF MVC (Nm)		DF MVC/Mass		PF MVC/Mass		DF TT/MVC		PF TT/MVC	
Subject	Gender	1982	1994	1982	1994	1982	1994	1982	1994	1982	1994	1982	1994
BC	M	38.6	39.7	177.0	106.0	0.44	0.49	2.02	1.30	0.12	0.17	0.09	0.12
RC	M	25.2	30.6	96.5	77.8	0.40	0.49	1.55	1.24	0.09	0.17	0.13	0.16
MC	F	22.9	21.7	89.4	74.0	0.42	0.43	1.64	1.48	0.15	0.17	0.11	0.16
LD	F	21.7	22.2	78.7	41.3	0.29	0.29	1.04	0.54	0.09	0.09	0.14	0.31
KD	M	37.2	33.6	164.5	125.2	0.52	0.48	2.29	1.78	0.10	0.15	0.10	0.12
MD	F	16.4	15.6	44.3	61.2	0.42	0.45	1.14	1.75	0.10	0.23	0.28	0.10
DK	F	24.0	22.1	93.0	42.6	0.42	0.44	1.62	0.85	0.08	0.18	0.14	0.29
HL	M	31.5	24.7	77.2	75.6	0.50	0.43	1.22	1.30	0.07	0.17	0.14	0.08
GL	M	18.3	17.0	94.7	56.2	0.27	0.31	1.40	1.03	0.13	0.15	0.12	0.22
TL	F	21.2	23.2	125.1	100.8	0.34	0.36	2.02	1.56	0.14	0.18	0.15	0.10
DL	F	18.3	15.4	93.0	50.4	0.37	0.39	1.89	1.26	N/A	N/A	N/A	N/A
RL	M	28.6	31.0	103.7	90.6	0.36	0.36	1.32	1.07	0.10	0.15	0.08	0.13
MM	M	26.9	8.1	100.1	49.6	0.39	0.14	1.46	0.87	0.05	0.14	0.05	0.03
DM	M	31.5	33.8	168.0	115.8	0.51	0.51	2.71	1.74	0.06	0.15	0.09	0.17
AM	F	24.0	18.8	70.1	45.4	0.39	0.33	1.15	0.79	0.07	0.21	0.16	0.31
CM	M	30.0	19.3	164.5	108.1	0.40	0.28	2.21	1.59	0.13	0.22	0.10	0.15
MM	M	29.7	33.3	128.7	71.0	0.43	0.52	1.86	1.17	0.06	0.15	0.08	0.24
AM	F	25.2	25.2	77.2	62.6	0.44	0.49	1.36	1.23	0.10	0.14	0.13	0.16
RP	M	27.5	23.0	70.1	61.9	0.38	0.34	0.96	0.91	0.13	0.13	0.16	0.13
AS	F	18.3	20.8	80.1	81.2	0.36	0.40	1.58	1.58	0.16	0.16	0.10	0.14
WS	F	22.6	23.6	114.4	74.8	0.42	0.43	2.11	1.37	0.04	0.12	0.16	0.26
VW	F	20.6	18.6	114.4	102.2	0.35	0.27	1.94	1.50	0.06	0.18	0.13	0.17

Dorsiflexor Potentiated Twitch Properties & M-Wave Characteristics

		Age (yrs)		M-Wave (mV)		TT (Nm)		TPT (msec)		1/2 RT (msec)		CT (msec)	
Subject	Gender	1982	1994	1982	1994	1982	1994	1982	1994	1982	1994	1982	1994
BC	M	75	87	8.6	7.4	6.4	9.9	120	100	110	126	230	226
RC	M	69	81	5.2	6.0	2.7	7.7	105	79	110	100	215	179
MC	F	62	74	12.5	15.0	4.3	4.9	105	82	80	88	185	170
LD	F	62	74	4.8	14.7	2.9	3.9	115	73	100	95	215	168
KD	M	73	85	5.0	11.5	5.0	7.4	115	100	110	105	225	205
MD	F	78	90	6.2	7.6	2.0	4.2	105	84	90	91	195	175
DK	F	69	82	3.4	4.6	2.3	5.9	115	93	90	102	205	195
HL	M	85	97	5.4	6.4	2.4	5.5	100	83	70	72	170	155
GL	M	79	91	9.0	3.9	2.4	4.1	115	95	120	98	235	193
TL	F	70	82	4.4	6.4	3.4	5.6	110	118	115	116	225	234
DL	F	82	94	4.8	N/A	3.6	N/A	100	N/A	70	N/A	170	N/A
RL	M	71	83	3.4	8.4	3.2	6.0	115	100	90	88	205	188
MM	M	74	86	6.0	0.9	1.6	1.0	120	112	140	121	260	233
DM	M	62	74	6.2	11.5	2.8	7.0	90	82	75	83	165	165
AM	F	70	82	10.0	12.3	2.9	4.6	70	83	65	86	135	169
CM	M	80	92	6.0	6.2	4.6	4.2	115	110	80	122	195	232
MM	M	61	73	6.1	14.0	3.2	5.8	120	69	95	90	215	159
AM	F	69	81	11.0	10.4	3.4	4.5	100	71	80	88	180	159
RP	M	80	92	6.8	5.4	4.7	3.1	105	96	95	102	200	198
AS	F	63	75	15.0	19.6	3.4	3.8	105	103	75	95	180	198
WS	F	73	85	8.0	3.9	1.9	4.5	110	96	80	84	190	180
VW	F	66	78	8.4	12.2	2.1	4.8	85	80	65	82	150	162

Plantarflexor Potentiated Twitch Properties & M-Wave Characteristics

		Age (yrs)		M-Wave (mV)		TT (Nm)		TPT (msec)		1/2 RT (msec)		CT (msec)	
Subject	**Gender**	**1982**	**1994**	**1982**	**1994**	**1982**	**1994**	**1982**	**1994**	**1982**	**1994**	**1982**	**1994**
BC	M	75	87	16.5	4.1	28.6	17.4	165	198	130	142	295	340
RC	M	69	81	13.0	12.8	12.9	17.7	195	151	135	182	330	333
MC	F	62	74	12.0	18.2	12.9	12.9	140	141	130	119	270	260
LD	F	62	74	13.5	15.7	12.5	13.1	190	180	160	165	350	345
KD	M	73	85	15.0	15.0	21.8	19.7	125	130	100	146	225	276
MD	F	78	90	10.5	13.6	12.5	17.4	145	115	105	134	250	249
DK	F	69	82	12.0	12.3	16.8	12.7	145	96	105	211	250	307
HL	M	85	97	13.0	7.3	12.2	16.1	155	91	100	134	255	225
GL	M	79	91	14.0	7.7	10.0	14.7	180	105	90	153	270	258
TL	F	70	82	6.4	9.4	19.3	22.8	170	185	130	204	300	389
DL	F	82	94	8.4	N/A	14.3	N/A	150	N/A	105	N/A	255	N/A
RL	M	71	83	8.0	7.1	12.7	15.0	140	156	85	96	225	252
MM	M	74	86	4.0	2.5	8.0	3.4	140	104	90	137	230	241
DM	M	62	74	10.0	16.3	20.4	25.4	125	104	110	137	235	241
AM	F	70	82	13.0	18.1	14.0	13.9	150	166	100	113	250	279
CM	M	80	92	13.0	5.8	23.6	23.6	125	112	125	154	250	266
MM	M	61	73	19.0	16.8	12.9	24.4	155	111	80	89	235	200
AM	F	69	81	9.0	22.6	8.9	12.4	160	87	110	132	270	219
RP	M	80	92	12.5	9.8	11.6	10.3	195	169	120	160	315	329
AS	F	63	75	21.5	24.4	9.1	14.9	150	161	95	105	245	266
WS	F	73	85	6.6	13.1	23.6	22.7	170	99	105	137	275	236
VW	F	66	78	6.5	19.9	2.7	25.4	140	139	130	149	270	288

% Motor Unit Activation - PF 1994 Values

Subject	Gender	Age	TT	Int. T.	% MUA
BC	M	87	12.70	2.12	83.3
LD	F	74	12.89	3.16	75.5
MD	F	90	6.27	1.18	81.2
DK	F	82	12.31	2.61	78.8
GL	M	91	12.45	2.40	80.7
AM	F	82	14.16	2.10	85.2
MM	M	73	16.02	2.72	83.0
WS	F	85	19.73	1.60	91.9
VW	F	78	17.58	1.55	91.2

FORMULA:

$$\% \text{ Motor Unit Activation} = \frac{\text{Resting TT} - \text{Int. TT}}{\text{Resting TT}} \times 100$$

APPENDIX B:

CONSENT FORM

CONSENT FORM

A 12 YEAR FOLLOW-UP STUDY OF MUSCLE FUNCTION IN THE ELDERLY

I _____, consent to take part in a research study under the direction of Dr. Audrey Hicks and Dr. Tony Vandervoort. The purpose of this study is to conduct follow-up testing of those subjects who underwent testing in 1982, to determine the rate of decline of ankle muscle function over the past twelve years. In as much as the results will have no direct benefit to me, I am aware that the results of this study will be made available to the scientific community, however, no reference will be made to my identity in any form whatsoever.

I am aware that the procedure will involve the taping of electrodes to my skin, and mild electrical shocks will be delivered to my lower leg. When this happens, there will be a little discomfort and my muscle will jerk and contract on its own. I understand that apart from this temporary discomfort, there is no long-lasting effects of the muscle stimulation. I will also be asked to make several muscular efforts on my own. Testing will require sitting in a chair with my leg strapped in a metal leg-boot frame. I will also be required to complete a physical activity questionnaire under the supervision of Karen Winegard.

I understand that it is my right to withdraw from the study at any time, whether or not the procedure is complete, even after signing this consent form.

_____	_____	_____
Name (Print)	Signature	Date
_____	_____	_____
Witness (Print)	Signature	Date

I have explained the nature of the study to the subject, and believe that he or she has understood it.

_____	_____	_____
Name (Print)	Signature	Date

APPENDIX C:

HEALTH QUESTIONNAIRE

HEALTH QUESTIONNAIRE

Study Title: A 12-year Follow-up Study of Ankle Muscle Function in Seniors.

Name of Subject: _____

1. Age: _____

2. Do you require a walking aid? no ___ yes ___

3. Past Medical History

I. Ankle Injuries

a) Do you suffer from a history of ankle injuries? yes ___ no ___

(i) Which ankle(s)? (R) ___ (L) ___

(ii) Types of injuries?

_____

_____

_____

b) Have you sought medical attention for any of the above ankle injuries? yes ___ no ___

(i) if yes, briefly describe treatment:

_____

_____

_____

(ii) if no, describe self-treatment:

_____

_____

c) Approximately how long ago was your last ankle injury? _____

(i) Which ankle(s)? (R) ___ (L) ___

II. Past Leg Injuries

Do you suffer from a history of injuries to your legs that required medical treatment or hospitalization?

(i) Which joint(s) _____ (R) ___ (L) ___

(ii) When? _____

_____

(iii) Type of injuries?

_____

_____

III. Past Surgeries to Legs

Have you ever undergone any major elective/emergency operative procedure to your lower limbs? yes ___ no ___

(i) When? _____

(ii) Specify surgery(ies): _____

IV. General Health

Have you, or do you currently suffer from...

a) Heart or lung problems? yes ___ no ___
Comment:

b) Stroke? yes ___ no ___
Comment:

c) Cancer? yes ___ no ___
Comment:

d) Parkinson's Disease? yes ___ no ___
Comment:

e) Arthritis or joint problems? yes ___ no ___
Comment:

f) Severe osteoporosis? yes ___ no ___
Comment:

g) Nerve injuries in the legs? yes ___ no ___
Comment:

h) Any congenital hip/knee/ankle/foot orthopaedic condition or disease? yes ___ no ___
Comment:

i) Foot deformities or podiatric conditions requiring orthotics or special footwear? yes ___ no ___
Comment:

j) Diabetes or circulatory problems? yes ___ no ___
Comment:

k) Blindness? yes ___ no ___
Comment:

l) Fainting or dizzy spells? yes ___ no ___
Comment:

Additional comments:_____
_____
_____

4. Medications

Are you currently taking any medication(s)? yes ___ no ___

List: _____
_____
_____

5. Do you regularly consume alcohol? yes ___ no ___
Number of drinks a day: _____

APPENDIX D:

PHYSICAL ACTIVITY QUESTIONNAIRE

PHYSICAL ACTIVITY QUESTIONNAIRE

Questionnaire, codes, and methods of calculation of scores on habitual physical activity in elderly people.

PART I: HOUSEHOLD ACTIVITIES

1) Do you do the light household work? (dusting, washing dishes repairing clothes, etc.)? ___
   0. Never (<once a month)
   1. Sometimes (only when partner or help is not available
   2. Mostly (sometimes assisted by partner or help)
   3. Always (alone or together with partner)

2) Do you do the heavy housework? (washing floors and windows, carrying trash disposal bags, etc.)? ___
   0. Never (<once a month)
   1. Sometimes (only when partner or help is not available)
   2. Mostly (sometimes assisted by partner or help)
   3. Always (alone or together with partner)

3. For how many people do you keep house? (including yourself; fill in "0" if you answered "never" in Q1 and Q2.) ___

4. How many rooms do you keep clean, including kitchen, bedroom, garage, cellar, bathroom, ceiling, etc.? ___
   0. Never do housekeeping
   1. 1-6 rooms
   2. 7-9 rooms
   3. 10 or more rooms

5. If any rooms, on how many floors? (fill in "0" if you answered "never" in Q4.) ___

6. Do you prepare warm meals yourself, or do you assist in preparing? ___
   0. Never
   1. Sometimes (once or twice a week)
   2. Mostly (3-5 times a week)
   3. Always (more than 5 times a week)

7. How many flights of stairs do you walk up per day? (one flight of stairs is 10 steps.) ___
   0. I never walk stairs
   1. 1-5
   2. 6-10
   3. More than 10

8. If you go somewhere in your hometown, what kind of transportation do you use? ___
   0. I never go out
   1. Car
   2. Public transportation
   3. Bicycle
   4. Walking

9. How often do you go out shopping? ___
   0. Never or less than once a week
   1. Once a week
   2. Twice to four times a week
   3. Every day

10. If you go out shopping, what kind of transportation do you use? ___
   0. I never go out shopping
   1. Car
   2. Public transportation
   3. Bicycle
   4. Walking

Household score = (Q1 + Q2 +...+ Q10)/10

PART II: SPORT ACTIVITIES

Do you play a sport?

Sport 1: name _____
intensity (code) _____
hours per week (code) _____
period of the year (code) _____

Sport 2: name _____
intensity (code) _____
hours per week (code) _____
period of the year (code) _____

$$\text{Sport score: } \sum_{i=1}^{2} (ia*ib*ic)$$

PART III: LEISURE TIME ACTIVITIES

Do you have any other physical activities?

Activity 1: name _____
intensity _____
hours per week _____
period of the year _____

Activity 2: name _____
intensity _____
hours per week _____
period of the year _____

Activity 3: name _____
intensity _____
hours per week _____
period of the year _____

Activity 4: name _____
intensity _____
hours per week _____
period of the year _____

Activity 5: name _____
intensity _____
hours per week _____
period of the year _____

Activity 6: name _____
intensity _____
hours per week _____
period of the year _____

Leisure time activity score: $\sum_{j=1}^{6} (ja*jb*jc*)$

QUESTIONNAIRE SCORE = HOUSEHOLD SCORE + SPORT SCORE + LEISURE TIME ACTIVITY SCORE.

CODES:

Intensity codes:

0:	lying, unloaded	code 0.028
1:	sitting, unloaded	code 0.146
2:	sitting, movements hand or arm	code 0.297
3:	sitting, body movements	code 0.703
4:	standing, unloaded	code 0.174
5:	standing, movements hand or arm	code 0.307
6:	standing, body movements, walking	code 0.890
7.	walking, movements arm or hands	code 1.368
8.	walking, body movements, cycling, swimming	code 1.890

Hours per week:

1:	< 1 hr/wk	code 0.5
2:	1-2 hrs/wk	code 1.5
3:	2-3 hrs/wk	code 2.5
4:	3-4 hrs/wk	code 3.5
5:	4-5 hrs/wk	code 4.5
6:	5-6 hrs/wk	code 5.5
7:	6-7 hrs/wk	code 6.5
8:	7-8 hrs/wk	code 7.5
9:	> 8 hrs/wk	code 8.5

Months a year:

1:	< 1 month/yr	code 0.04
2:	1-3 months/yr	code 0.17
3:	4-6 months/yr	code 0.42
4:	7-9 months/yr	code 0.67
5:	> 9 months/yr	code 0.92

APPENDIX E:

RELIABILITY DATA

Reliability Coefficients (n=10, 5 males, 5 females)

Variable	Intraclass Correlation Coefficients Dorsiflexor	Plantarflexor
MVC	0.98	0.97
Passive Tension	0.78	0.86
Resting:		
M-Wave Amplitude	0.97	0.93
Twitch Torque	0.94	0.94
Time to Peak Torque	0.87	0.89
1/2 Relaxation Time	0.87	0.80
Potentiated:		
M-Wave Amplitude	0.96	0.92
Twitch Torque	0.94	0.94
Time to Peak Torque	0.90	0.87
1/2 Relaxation Tlme	0.89	0.88
Mean	0.91	0.90
Standard Deviation	0.06	0.05

FORMULA: Intraclass Correlation Coefficient

$$\text{Reliability Coefficient} = \frac{\text{MS subjects - MS error}}{\text{MS subjects}}$$

$$\text{MS error} = \frac{\text{SS trial + SS error}}{\text{df trial + df error}}$$

MS = mean square
SS = sums of square
df = degrees of freedom

Reliability Data: Comparison of Methods

Variable	Mean Values			Method Error (%)	Pearson *r* *	Intraclass Correlation
	Trial 1	Trial 2	% Diff.			
MVC:						
DF	24.51	24.56	0.20	5.65	0.96	0.98
PF	82.61	80.63	2.40	8.96	0.93	0.97
Resting:						
DF M-Wave	11.28	11.75	4.17	9.09	0.94	0.97
PF M-Wave	15.15	15.51	2.38	12.30	0.86	0.93
DF TT	3.87	4.19	8.27	7.19	0.92	0.94
PF TT	14.39	14.60	1.46	7.94	0.89	0.94
Potentiated:						
DF M-Wave	12.24	12.15	0.74	10.80	0.92	0.96
PF M-Wave	16.00	16.21	1.31	12.87	0.84	0.92
DF TT	4.95	5.18	4.65	8.67	0.90	0.94
PF TT	18.36	17.53	4.52	10.21	0.90	0.94

* ***Pearson r:*** Runyon & Haber, 1991

FORMULAS:

Method Error:

$$\text{Reliability} = \frac{\sqrt{\text{MS error}}}{(\text{mean 1} + \text{mean 2}) / 2} \times 100$$

Intraclass Correlation Coefficient:

$$\text{Reliability} = \frac{\text{MS subjects} - \text{MS error}}{\text{MS subjects}}$$

$$\text{MS error} = \frac{\text{SS trial} + \text{SS error}}{\text{df trial} + \text{df error}}$$

MS = mean square
SS = sums of square
df = degrees of freedom

APPENDIX F:

ANALYSIS OF VARIANCE TABLES

Plantarflexor Range of Motion

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	184.09	1	184.09	3.03	0.094
Error	1213.82	20	60.69		
TIME	209.46	1	209.46	21.23	0.001
G x T	66.27	1	66.27	6.72	0.017
Error	197.27	20	9.86		

Height

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	2692.63	1	2692.63	32.85	0.001
Error	1639.25	20	81.96		
TIME	220.88	1	220.88	84.34	0.001
G x T	0.38	1	0.38	0.14	0.709
Error	52.38	20	2.62		

Weight

Source of Variation	Sums of Square	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	1999.36	1	1999.36	11.53	0.003
Error	3469.48	20	173.47		
TIME	90.80	1	90.80	5.80	0.024
G x T	10.98	1	10.98	0.70	0.417
Error	313.11	20	15.66		

Maximal Voluntary Contraction

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	5945.1	1	5945.1	6.81	0.016
Error	17467.5	20	873.4		
TIME	5399.3	1	5399.3	41.61	0.001
G x T	396.9	1	396.9	3.06	0.092
Error	2595.3	20	129.8		
CONTRACTION	96725.1	1	96725.1	173.20	0.001
G x C	1909.2	1	1909.2	3.42	0.076
Error	11169.4	20	558.5		
T x C	4249.2	1	4249.2	35.57	0.001
G x T x C	226.3	1	226.3	1.89	0.181
Error	2389.2	20	119.5		

Strength/Mass Ratio

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	0.048	1	0.048	0.288	0.603
Error	3.310	20	0.166		
TIME	0.874	1	0.874	25.902	0.001
G x T	0.034	1	0.034	1.008	0.329
Error	0.675	20	0.034		
CONTRACTION	25.049	1	25.049	214.916	0.001
G x C	0.015	1	0.015	0.129	0.721
Error	2.331	20	0.117		
T x C	0.800	1	0.800	24.661	0.001
G x T x C	0.014	1	0.014	0.432	0.525
Error	0.649	20	0.032		

Resting Twitch Torque

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	0.43	1	0.43	0.03	0.848
Error	316.36	19	16.65		
TIME	7.46	1	7.46	1.57	0.224
G x T	0.09	1	0.09	0.02	0.860
Error	90.30	19	4.75		
CONTRACTION	1808.46	1	1808.45	156.06	0.001
G x C	7.53	1	7.53	0.65	0.436
Error	220.17	19	11.59		
T x C	13.52	1	13.52	3.59	0.070
G x T x C	0.02	1	0.02	0.06	0.897
Error	71.49	19	3.76		

Resting M-Wave Amplitude

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	133.99	1	133.99	3.38	0.078
Error	752.83	19	39.62		
TIME	31.10	1	31.10	3.19	0.087
G x T	145.65	1	145.65	14.93	0.001
Error	185.33	19	9.75		
CONTRACTION	362.95	1	362.95	62.77	0.001
G x C	0.45	1	0.45	0.08	0.772
Error	109.85	19	5.78		
T x C	0.17	1	0.17	0.02	0.854
G x T x C	57.40	1	57.41	7.55	0.012
Error	144.48	19	7.60		

Peak Twitch Torque/MVC Ratio

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	0.018	1	0.018	9.70	0.006
Error	0.035	19	0.002		
TIME	0.062	1	0.062	29.06	0.001
G x T	0.000	1	0.000	0.14	0.713
Error	0.040	19	0.002		
CONTRACTION	0.008	1	0.008	2.78	0.108
G x C	0.012	1	0.012	4.22	0.051
Error	0.052	19	0.003		
T x C	0.003	1	0.003	1.02	0.327
G x T x C	0.000	1	0.000	0.07	0.779
Error	0.048	19	0.003		

Passive Tension

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	2.14	1	2.14	1.41	0.249
Error	28.87	19	1.52		
TIME	40.26	1	40.26	56.49	0.001
G x T	1.78	1	1.78	2.49	0.128
Error	13.54	19	0.71		
JOINT ANGLE	11.42	1	11.42	12.94	0.002
G x JA	3.46	1	3.47	3.92	0.060
Error	16.77	19	0.88		
T x JA	0.13	1	0.13	0.63	0.444
G x T x JA	0.15	1	0.15	0.70	0.417
Error	4.08	19	0.21		

Resting Time To Peak Torque

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	296.77	1	296.77	0.38	0.552
Error	14915.25	19	785.01		
TIME	8834.87	1	8834.87	64.42	0.001
G x T	66.29	1	66.29	0.48	0.502
Error	2605.75	19	137.14		
CONTRACTION	82340.24	1	82340.24	216.45	0.001
G x C	874.43	1	874.43	2.30	0.143
Error	7227.88	19	380.41		
T x C	149.61	1	149.61	1.51	0.232
G x T x C	306.92	1	306.92	3.10	0.091
Error	1878.38	19	98.86		

Resting 1/2 Relaxation Time

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	66.70	1	66.70	0.05	0.816
Error	28264.75	19	1487.62		
TIME	585.03	1	585.02	1.81	0.192
G x T	134.55	1	134.55	0.42	0.533
Error	6149.75	19	323.67		
CONTRACTION	4670.09	1	4670.09	9.34	0.001
G x C	1030.72	1	1030.72	2.06	0.164
Error	9502.63	19	500.14		
T x C	194.71	1	194.71	1.01	0.330
G x T x C	5.16	1	5.16	0.03	0.846
Error	3671.50	19	193.24		

Resting Contraction Time

Source of Variation	Sums of Square	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	645.60	1	645.60	0.16	0.693
Error	75853.00	19	3992.26		
TIME	4873.39	1	4873.39	9.37	0.006
G x T	10.48	1	10.48	0.02	0.859
Error	9883.50	19	520.18		
CONTRACTION	126230.20	1	126230.20	166.75	0.001
G x C	3806.78	1	3806.78	5.03	0.035
Error	14383.00	19	757.00		
T x C	685.54	1	685.54	3.68	0.067
G x T x C	232.45	1	232.45	1.25	0.277
Error	3537.00	19	186.16		

Potentiated Twitch Torque

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	26.76	1	26.76	0.77	0.395
Error	658.76	19	34.67		
TIME	97.88	1	97.88	7.60	0.012
G x T	6.08	1	6.08	0.47	0.507
Error	244.82	19	12.89		
CONTRACTION	2808.54	1	2808.54	148.63	0.001
G x C	2.11	1	2.11	0.11	0.738
Error	359.03	19	18.90		
T x C	1.06	1	1.06	0.10	0.752
G x T x C	9.28	1	9.28	0.86	0.369
Error	205.33	19	10.81		

Percent Change from Resting to Potentiated TT

Source of Variation	Sums of Square	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	72.61	1	72.61	0.095	0.755
Error	14508.11	19	763.58		
CONTRACTION	8.41	1	8.41	0.004	0.904
G x C	4254.94	1	4254.94	2.236	0.148
Error	36152.04	19	1902.74		
TIME	5266.11	1	5266.11	3.816	0.063
G x T	97.00	1	97.00	0.070	0.783
Error	26222.82	19	1380.15		
C x T	5860.31	1	5860.31	4.020	0.057
G x C x T	134.17	1	134.17	0.092	0.758
Error	27699.43	19	1457.87		

Potentiated M-Wave Amplitude

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	163.92	1	163.92	3.58	0.071
Error	869.29	19	45.75		
TIME	50.53	1	50.53	4.45	0.046
G x T	121.90	1	121.90	10.74	0.004
Error	215.68	19	11.35		
CONTRACTION	393.12	1	393.12	80.55	0.001
G x C	0.08	1	0.08	0.02	0.866
Error	92.73	19	4.88		
T x C	1.10	1	1.10	0.14	0.714
G x T x C	75.15	1	75.15	9.40	0.001
Error	151.92	19	8.00		

Potentiated Time to Peak Torque

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	41.33	1	41.33	0.06	0.801
Error	14008.88	19	737.31		
TIME	7344.79	1	7344.79	16.51	0.001
G x T	112.86	1	112.86	0.25	0.625
Error	8451.50	19	444.82		
CONTRACTION	43880.83	1	43880.83	69.26	0.001
G x C	642.08	1	642.08	1.01	0.328
Error	12037.75	19	633.57		
T x C	195.28	1	195.28	0.82	0.380
G x T x C	2.94	1	2.94	0.01	0.878
Error	4529.63	19	238.40		

Potentiated 1/2 Relaxation TIme

Source of Variation	Sums of Squares	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	27.34	1	27.34	0.03	0.846
Error	19478.56	19	1025.19		
TIME	6953.98	1	6953.98	36.82	0.001
G x T	24.72	1	24.72	0.13	0.720
Error	3588.06	19	188.85		
CONTRACTION	22823.36	1	22823.36	52.00	0.001
G x C	2352.31	1	2352.31	5.36	0.030
Error	8339.81	19	438.94		
T x C	3719.13	1	3719.13	13.34	0.002
G x T x C	155.10	1	155.10	0.56	0.471
Error	5297.69	19	278.83		

Potentiated Contraction Time

Source of Variation	Sums of Square	Degrees of Freedom	Mean Square	F Ratio	p Value
GENDER	77.92	1	77.92	0.03	0.843
Error	51875.00	19	2730.26		
TIME	0.65	1	0.65	0.10	0.925
G x T	340.15	1	340.15	0.51	0.492
Error	12767.00	19	671.95		
CONTRACTION	127942.80	1	127942.8	89.71	0.001
G x C	5038.40	1	5038.40	3.53	0.073
Error	27096.50	19	1426.13		
T x C	1948.57	1	1948.57	6.99	0.015
G x T x C	187.27	1	187.27	0.67	0.428
Error	5293.50	19	278.61		