T2 MAPPING OF MUSCLE ACTIVATION DURING SINGLE-LEG VERTICAL JUMPING EXERCISE

by

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To my loving family for their steadfast support

Table of Contents

List of Tables

List of Figures

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List of Acronyms and Abbreviations

1RM	one-repetition maximum
ACSM	American College of Sports Medicine
AL	*m. adductor longus*
ALI	Analytical Language for Imaging
AM	*m. adductor magnus*
ANOVA	analysis of variance
APAR	Average performance to activation ratio
ASCRS	Astronaut Strength, Conditioning and Rehabilitation Specialists
AT	anaerobic threshold
ATP	adenosine triphosphate
BF	*m. bíceps femoris*
BR	bed rest
BW	body weight
C	Control Group
CSA	cross-sectional area
DF	dorsiflexor muscles
DICOM	Digital Imaging and Communication in Medicine
DT	detraining
EDL	*m. extensor digitorum longus*
EHL	*m. extensor hallucis longus*
EKG	electrocardiogram
EMS	electro-myostimulation

EPI	echo planar imaging
ETL	echo train length
F	force
FDL	*m. flexor digitorum longus*
FHL	*m. flexor hallucis longus*
FOV	field of view
FTI	force/time integral
GL	*m. gastrocnemius lateralis*
GM	*m. gastrocnemius medialis*
Gr	*m. gracilis*
GT	general fitness training
h	height
HDT	head-down tilt
iEMG	integrated electromyography
J	Jumpers Group
LNP	lower-body negative pressure
m.	muscle
MAP	maximum aerobic power
MET	maximal exercise test
MHC	myosin heavy chain
MRI	Magnetic Resonance Imaging
MRS	Magnetic Resonance Spectroscopy
MVC	maximal voluntary contraction

nRM	n-repetition maximum
P	power
PCr	phosphocreatine
PF	plantar flexor muscles
PL	plyometrics
PL	*m. peroneus longus*
Post1	Post Exercise Bout 1 (body weight jumps)
Post2	Post Exercise Bout 2 (body weight + 33% jumps)
PPAR	Peak performance to activation ratio
QF	*m. quadriceps femoris*
RF	*m. rectus femoris*
RFD	rate of force development
ROI	region of interest
RT	resistance training
Sar	*m. sartorius*
SF	space flight
SM	*m. semimembranosus*
Sol	*m. soleus*
ST	strength training
ST	*m. semitendonosus*
T_1	spin-lattice relaxation time
T_2	spin-spin relaxation time
T_2+	T_2 elevation

TA	*m. tibialis anterior*
T_E	echo time
TP	*m. tibialis posterior*
T_R	repetition time
ULLS	unilateral lower limb suspension
USB	universal serial bus
v	velocity
VI	*m. vastus intermedialis*
VL	*m. vastus lateralis*
VM	*m. vastus medialis*
VSE	Vision for Space Exploration
W	power
ΔT_2	Change in T_2
ΔV	Change in muscle volume

T2 Mapping Of Muscle Activation During

Single-Leg Vertical Jumping Exercise

Abstract

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This study investigated using elevation of the spin-spin relaxation time (T_2) in magnetic resonance imaging (MRI) to map recruitment differences in the thigh and calf between two distinct populations during single-leg jumping. Twelve healthy subjects formed two groups based on jumping ability. Subjects took a maximal exercise test (MET) to determine aerobic fitness. Subjects performed 5x10 single-leg jumps at body weight (Post1) and at body weight+33% (Post2) on a force platform while wearing a weighted vest at Post2. Performance was determined as concentric jumping power normalized to the subject's maximum aerobic power. Spin echo MRI at Baseline, Post1 and Post2 determined muscle activation as the percentage of muscle pixels with elevated T_2 after exercise. A novel metric based on the performance/activation ratio highlighted recruitment efficiency differences between groups. Results suggest that recruitment efficiency throughout the lower limb (especially suppression of co-activating antagonists) was the dominant factor in enhancing jumping performance.

Introduction

Long-duration space travel is known to induce losses in muscle mass, strength, power, and endurance (Zamparo et al. 2002). Retaining astronaut muscular performance has always had mission-critical implications, but the unique demands of the proposed Vision for Space Exploration (VSE) missions, especially to Mars, require more specific attention to maintaining muscle power, i.e., the ability to simultaneously generate both force and velocity.

Muscle power is essential for the performance of functional tasks (Fatouros et al. 2000; Morissey et al. 1995), the maintenance of balance (Bruhn et al., 2004) and the prevention of injuries during falls by quickly stiffening muscles around vulnerable joint complexes (Alt et al. 1999). All three of these elements are critical to astronaut performance in a Martian VSE scenario where weight-bearing activity (albeit at 0.4*g*) must resume on an uninhabited world following an extremely long-duration period of microgravity. Additionally, these astronauts will likely experience neurovestibular symptoms, e.g., vertigo (Bacal et al. 2003), and decreased bone mineral density (Germain et al. 1995), making them even more prone to falls and even more susceptible to injuries from falls, respectively.

Resistance training (RT) programs have demonstrated partial success in reducing the loss of muscular performance incurred during real and simulated periods of microgravity. Adapted RT protocols will be used as in-flight countermeasures to maintain muscular performance of astronauts on long-duration missions (Tesch et al., 2004). However, there may be physical and logistical limitations on the effectiveness of in-flight training programs to maintain muscle power.

Meanwhile, surprisingly little *scientific* attention has been paid to the role of pre-conditioning as a countermeasure. The pre-flight fitness training of astronauts is a largely self-directed process assisted by astronaut strength, conditioning and rehabilitation specialists (ASCRS) who help astronauts plan, set and achieve their own fitness goals (Jennings & Bagian, 1996). The goals of pre-flight conditioning would seem to be the achievement of a high degree of overall fitness and the building of physiological reserves to offset losses in physical performance expected during the flight. Yet, since there has not been a controlled study of the effect of pre-flight RT on musculoskeletal losses seen after space flight, its efficacy as a pre-flight countermeasure remains undetermined.

Moreover, a published model of the interplay among changes in muscle size, strength and power under training or disuse conditions (Minetti, 2002; Zamparo et al., 2002) has shown that spaceflight de-conditions muscle in a unique manner, and that conventional strength training programs may not be the best strategy for pre-conditioning the lower limbs. Explosive resistance training, featuring a combination of strength and power movements and plyometrics, may be a better choice because of the added element of motor learning of complex functional tasks. Two studies have examined quadriceps performance after lengthy detraining that followed training periods where significant gains in both strength and hypertrophy occurred. Although detraining eliminated any significant gains from hypertrophy, the subjects still retained strength levels well above baseline (Hakkinen et al., 2000; Ivey et al., 2000).

At the time of this writing, published studies have tested neither the efficacy of pre-conditioning of the lower limbs prior to unloading nor investigated the use of Minetti model to design a training program to directly counteract the effects of unloading. The

author and his faculty advisers have proposed a longitudinal study to determine the efficacy of various types of pre-conditioning in enabling subjects to retain the benefits of training following an extended unloading event. The pre-conditioning programs to be investigated would include aerobic training, strength training and a combination of strength and explosive training. The unloading intervention would be unilateral lower limb suspension (ULLS), whereby the subjects would conduct all daily activities wearing a single raised platform shoe and walking with crutches (Ploutz-Snyder et al. 1995). Unloading from spaceflight microgravity conditions would be simulated locally in the non-walking leg.

One aspect of this proposed study would be to investigate whether neural adaptations to training, rather than myofibrillar adaptations, would enable subjects to retain superior performance in an exercise requiring functional power after unloading. Specific neural adaptations that might persist include the more efficient recruitment of agonist and synergist muscles, the more effective suppression of the proprioceptive co-activation of antagonist muscles and the ability to quickly stiffen joint complexes through optimal timing of pre-activation of synergist muscles (Chimera et al., 2004). The functional MRI technique of measuring the increase in the spin-spin relaxation time (T_2) in response to exercise provides a whole-muscle and non-invasive way to detect either type of neural adaptation (reviews: Bendahan et al., 2004; Patten et al., 2003).

A pilot study has also been proposed that would provide preliminary data to bolster the case for the feasibility of the longitudinal study. Since T_2 elevation with exercise has been studied only sparingly in the context of power production, it is necessary to characterize the technique in terms of activation versus performance (i.e.,

power production). Vertical jumping is a highly valid measure of lower limb explosive power (Markovic et al., 2003). Since ULLS would be the intended unloading technique, single-leg vertical jumping is an attractive candidate for the exercise protocol.

Several objectives of this pilot study are critical to the longitudinal study: 1) to map the T2 response of the lower limb muscles on an individual muscle basis for the jumping exercise selected, 2) to collect data at two levels of power production to determine if there is a corresponding scaling in the measured T_2 response, and 3) to determine whether populations of differing jumping ability demonstrate significant differences in muscle activation during maximal effort jumping. This paper contains the rationale, methods, results and conclusions of that pilot study.

Review of the Literature

This literature review explores two separate areas, both relevant to this pilot study. The first section examines neuromuscular adaptations to training and unloading, with specific emphasis on the Minetti model. Although not directly applied in the pilot study itself, which lacks both training and unloading interventions, this material justifies the objectives and methods of the proposed targeted pre-conditioning study from which the pilot study was derived. The second section explores the phenomenon of T_2 elevation with exercise and its applications to exercise physiology.

The Minetti model of adaptation to training and unloading: Any period of altered physical activity can result in changes in muscle size, strength, and power. Chronic application of an overload stimulus (i.e., training) will produce net gains in these quantities; whereas sufficient reduction in the stimulus intensity (detraining) or elimination of the load imposed on major muscle groups (e.g., space flight, bed rest, limb suspension or immobilization) for long periods will produce losses in these quantities. Minetti (Minetti, 2002) has derived a useful mathematical model of the quadriceps for describing the interplay among changes in muscle size (measured by cross sectional area, *CSA*), strength (measured by maximal isometric force, *F*), and maximal power (measured from a vertical jump test, *w*). These measurements (CSA, F, w) constitute the model input and are acquired before $(CSA_{pre}, F_{pre}, w_{pre})$ and after $(CSA_{post}, F_{post}, w_{post})$ an adaptation process (e.g., space flight, resistance training). From these measurements, the ratio changes of muscle CSA (CSA_{ch}), strength (F_{ch}), and maximal power (P_{ch}) are calculated as follows:

(1) $$CSA_{ch} = {CSA_{post}}/{CSA_{pre}}$$

(2) $$F_{ch} = {F_{post}}/{F_{pre}}$$

(3) $$w_{ch} = {w_{post}}/{w_{pre}}$$

The model produces two output parameters ($k_{a,ch}$, $k_{c,ch}$) which describe the level (local vs. central) at which physiological adaptations have taken place. The first parameter is defined as the ratio of muscle strength changes to muscle CSA changes, or

(4) $$k_{a,ch} = {F_{ch}}/{CSA_{ch}}$$

and the second is the ratio of muscle power changes to muscle strength changes, or

(5) $$k_{c,ch} = {w_{ch}}/{F_{ch}}$$

The first k-parameter, $k_{a,ch}$, has been used to determine whether local neuro-mechanical/neuro-muscular factors are involved in the training/detraining process. A $k_{a,ch}$ value of 1 indicates that all observed changes in strength are due to changes in CSA. If the value of $k_{a,ch}$ is not unity, then other local factors must have contributed to the observed changes in strength. These factors include non-hypertrophic muscle adaptations, as well as local neural factors. As examples of non-hypertrophic local adaptations following resistance training programs, muscle fibers have demonstrated altered myosin heavy chain (MHC) protein expression from type IIB toward type IIA (Adams et al. 1994, Staron et al. 1991), increases in pennation angle (Kawakami et al. 2002), partial reversal of suppressed Ca2+ kinetics (Hunter et al. 1999), altered aerobic and anaerobic enzyme activity (Costill et al. 1979, Fleck & Kraemer 2004, Green et al.

1999), and more favorable bioenergetic profiles in terms of increased resting [ATP], [PCr] and [Glycogen] (MacDougall et al. 1977). Fleck & Kraemer note that these myofibrillar adaptations depend on the subjects' initial training status, the muscle group examined, and the type of training conducted (Fleck & Kraemer 2004). The neural effects seen locally in individual muscles following resistance training include: a) decreased neural drive at submaximal loads, suggesting greater efficiency in motor unit recruitment (Hakkinen et al. 1985, Ploutz et al. 1994), b) increased neural drive at maximal loads, suggesting the learned ability to activate a greater percentage of motor units when needed (Hakkinen et al. 1985), c) synchronization of motor units (Felici et al. 2001), d) increased firing frequency of motor units, e) increased time of tonic activity (Grimby et al. 1981), f) expansion of the neuromuscular junction with concomitant increases in neurotransmitters and receptors (Deschenes et al. 2000) and g) disinhibition due to the learned overriding of protective reflexes (Fleck & Kraemer 2004). Unfortunately, Minetti's model (nor any other model yet published) cannot isolate the contributions of individual local factors; it merely describes whether their net aggregate effect is to further facilitate ($k_{a,ch}$>1) or inhibit ($k_{a,ch}$<1) the production of force for a given change in CSA. Of the items on this list, detection of neural drive at submaximal loads lends itself most readily to the functional MRI technique used in this pilot study.

The Minetti model also describes changes in peak power (w_p) observed after an adaptation with a second parameter, $k_{c,ch}$. Power is the product of force and velocity, but the takeoff velocity itself is a non-linear function of force (Minetti 2002). The useful force available to perform the jump is not necessarily the isometric force determined above, but its post/pre change ratio is assumed to be related to the change ratio of

isometric force by $k_{c,ch}$, such that $F_{use,ch} = k_{c,ch}F_{iso,ch}$. The model quantifies two non-linear relationships: one between change ratios in measured power to isometric force, another to change ratios in jump height ($h_{max,ch}$) to isometric force. Using either relationship, one may solve for $k_{c,ch}$. The significance of $k_{c,ch}$ is to account for changes in peak power that occur beyond the measured change in force production. When $k_{c,ch} \geq 1$, then central neural factors must be present to account for the discrepancy. Examples of central neural adaptation include a) multiple-joint coordination (Bawa 2002), b) the suppression of activation of antagonist muscles, (Hakkinen & Komi 1985), c) enhanced processing of proprioceptive afferents (Aagard 2003), and d) motor learning of a complex functional movement (Bawa 2002). Of the items on this list, detection of the suppression of antagonist muscle activity lends itself most readily to the functional MRI technique used in this pilot study. Again, the individual contributions of these various central neural factors cannot be isolated; the model only explains whether their net aggregate effect is to facilitate ($k_{c,ch} > 1$) or inhibit ($k_{c,ch} < 1$) the production of functional power given that a change in isometric force has occurred.

Summarizing, the Minetti model's two k-values do not necessarily indicate whether force or power have increased or decreased in magnitude: they merely indicate whether factors other than a change in quadriceps CSA have facilitated the production of force (or power) or whether they have inhibited it.

Application of the Minetti model: Zamparo (Zamparo et al. 2002) applied the Minetti model to published data in the literature from longitudinal studies where an adaptation event had taken place and where all three input parameters required for the model (CSA_{ch}, $F_{iso,ch}$ and w_{ch} (or $h_{max,ch}$)) were available, and produced an X-Y plot of the

output parameter values determined for each study. The positive adaptations included three types of exercise training: strength training (ST) (Hakkinen et al. 1998, Hakkinen et al. 2000, Hakkinen et al. 1981), plyometric and strength training (PL+ST) (Paavolainen et al. 1991) and general fitness training (GT) (DeVito et al. 1999). The negative adaptations included space flight (SF) (Antonutto 2002, Antonutto et al. 1998, Antonutto 1999), bed rest (BR) (Ferretti et al. 2001) and detraining from strength training (DT) (Hakkinen et al. 2000, Hakkinen et al. 1981). Zamparo's plot has been modified here by including only the studies measuring lower body function, and by adding results from additional studies of bed rest (Funato et al. 1997), strength training (Ferri et al. 2003, Gorostiaga et al. 1999, Kraemer et al. 2004) and combined plyometric and strength training (Kraemer et al. 2000, Thortenssen 1977). Also considered are additional cases of a sequential strength training program followed by detraining (ST+DT) as a single adaptation event (Ferri et al. 2003, Hakkinen et al. 2000, Hakkinen et al. 1981, Thortenssen 1977). These results are shown in Figure 1.

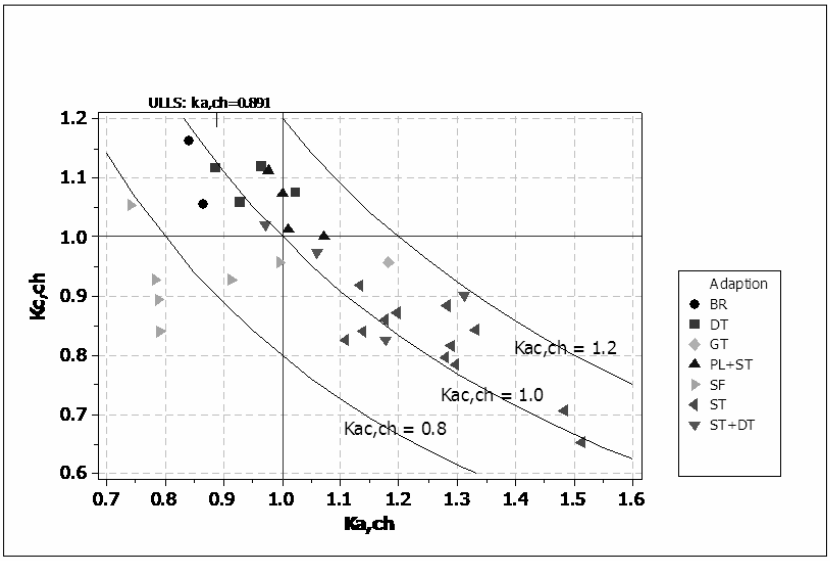


Figure 1 - Analysis of studies of adaptations to training using the Minetti model (GT = general training, PL = plyometrics, ST = strength training) and disuse (BR = bed rest, DT = detraining from ST, SF = space flight). The hyperbolae are contours of constant product of the two output parameters, kac,ch = ka,ch • kc,ch. No studies of muscle power adaptations to ULLS were available, so only the location of the ka,ch parameter for this intervention is indicated at the upper left.

* kc, ch. Modified from Zamparo et al. (145).

There are two trends of interest evident in the figure. First, conventional strength training falls exclusively within the lower right quadrant of the graph (higher $k_{a,ch}$ lower $k_{c,ch}$), indicating the facilitation of local factors and the inhibition of central factors. This is true even for programs where some lighter repetitions are performed in an "explosive manner (e.g., Hakkinen et al. 2000, Hakkinen et al. 1981). Second, only in programs where the explosive squat repetitions continue to include takeoff and landing (i.e., ballistic training) do we see values of $k_{c,ch} \geq 1$ and both local and central factors are facilitated. This has been explained by the large percentage of the work cycle spent decelerating in a light explosive exercise that avoids takeoff (Fleck & Kraemer 2004).

The net result is to inhibit the ability to generate power through the whole jumping motion due to activation of antagonists to stop the motion. Training programs with net gains in central factors ($k_{c,ch}$>1) are those which emphasize the velocity side of the power equation or the performance of the functional power movement itself (Kraemer et al. 2000, Paavolainen et al. 1991, Thortenssen 1977). There are other studies lacking CSA data (to compute $k_{a,ch}$) for which one could still calculate a $k_{c,ch}$>1 (Kraemer et al. 2003, Wilson et al. 1996).

An important metric is the distance of the various data points from the "origin" where both k-values are equal to 1. Analysis of the data from strength training, plyometrics plus strength training and general fitness training training programs as an aggregate shows a positive correlation (r=0.686, p=0.002) between program duration and the magnitude of the distance from the "origin" in the Minetti k-space up to 24 weeks. However, when a similar analysis is performed on the de-conditioning studies (space flight, de-training or bed rest) no such correlation exists (r=-0.046). This may be due to the relatively low statistical power of the space flight data, the much longer duration of these flights (54 weeks) and the fewer number of de-training and bed rest studies for which model-appropriate data are available.

Zamparo (Zamparo et al. 2002) has observed the difference between bed rest (lower $k_{a,ch}$ and higher $k_{c,ch}$) and space flight (lower $k_{a,ch}$ and lower $k_{c,ch}$). In the context of the Minetti model, bed rest appears to resemble de-training more than space flight. Even though all three input parameters (*CSA*, *F* and *w*) are certainly reduced in magnitude after a period of bed rest, the reduction in functional power is less than predicted by the reductions in muscle CSA and force. This suggests the central factors contributing to

jump performance have been retained better than local factors and muscle size after this type of unloading.

Meanwhile, the apparent "triple whammy" of space flight is elucidated by Figure 2. Space flight produces a loss of muscle mass, a further loss of strength beyond that predicted by the loss of mass, and a further loss of functional power even beyond that predicted by the loss of strength. The explanation offered by Zamparo for the difference between space flight and bed rest was due to differing effects on central neural factors (Zamparo et al. 2002), but one may also attribute part of it to the different effects of bed rest and space flight on the force-velocity curve. Bed rest studies consistently show greater percentage drops in isometric and slow velocity concentric strength than those seen at higher concentric velocities (Bamman & Caruso 2000, Berg et al. 1997, Dudley et al. 1989, Germain et al. 1995). Force-velocity effects on muscle groups from space flight are poorly studied, and no relevant data exist for the quadriceps (Adams 2003); however, one study of the plantar flexors after 175 days of space flight found higher percentage losses at higher concentric velocities than at isometric or slow concentric velocities (Koslovskaya et al. 1981). Assuming that similar results occur in the quadriceps, then the findings of the Minetti model make sense. At a given power, a disproportionate loss in isometric force will tend to elevate $k_{c,ch}$, all other factors being equal (Minetti 2002). Disproportionate losses in peak force at higher velocities would be expected to produce the opposite effect. To aid further analysis, Figure 2 shows only the central averages of the groups from in Figure 1. Figure 2 identifies the four quadrants of the Minetti k-value space and the location of the various adaptation events described. Note that the "origin" is actually the point $k_{a,ch} = k_{c,ch} = 1$.

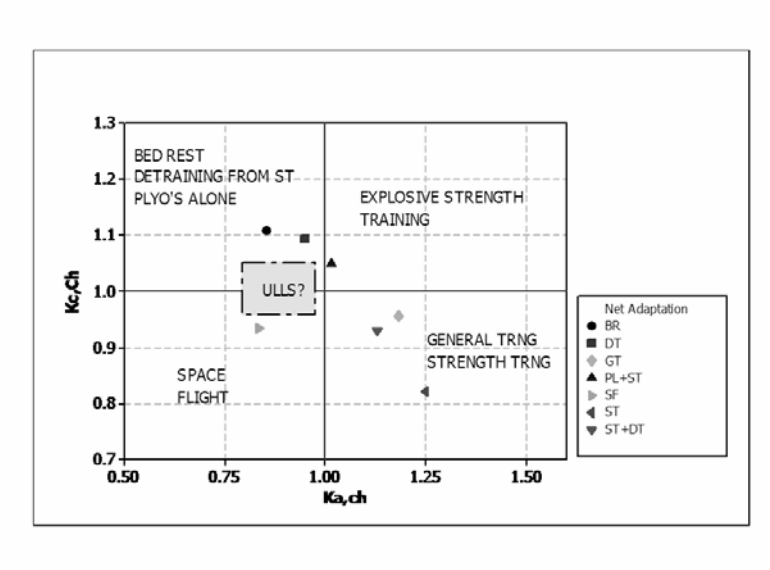


Figure 2 - Loci of central averages of adaptation events. Centroids are derived from Figure 1 and plotted within the four quadrants in the Minetti k-value space. An estimated location for the ULLS centroid in the parameter space is indicated by the box in the left half of the graph.

Figure 2 shows that strength training and de-training lie in opposite quadrants in Minetti *k*-space. Strength training facilitates local adaptations beyond hypertrophy that contribute to strength gains, but these gains come with the inhibition of central factors, such that not all of the newly-gained strength can be utilized to produce functional power. De-training tends to reverse both of these processes, as expected. Strength training plus de-training, treated as a single adaptation event, lies between strength training and de-training in the Minetti *k*-space, and the point averages of strength training, de-training and strength training plus de-training are nearly collinear. The fact that strength training plus de-training and strength training lie in the same quadrant indicates that strength training plus de-training is a net training adaptation (over the time studied), and it is testimony to the persistence of adaptations gained from strength training throughout periods of lengthy de-training. Strength training and bed rest appear also to be nearly

opposite adaptations in the context of this model. Unfortunately, there are no published studies of the effects of bed rest on resistance trained individuals that included the input data required for analysis with the Minetti model. As stated previously, studies of training prior to unloading are surprisingly scarce, so this area is currently poorly understood. Such a scenario would likely include both a de-training component as well as an unloading component from bed rest. It is reasonable to assume that the sequential events of strength training + (bed rest with de-training), treated as a single adaptation, would fall somewhere on the line connecting the strength training and bed rest averages. Its exact location would depend on the type of strength training program employed, the duration of strength training and the duration of bed rest/de-training. It appears that conventional strength training prior to bed rest is a valid pre-conditioning strategy. Strength training develops the appropriately targeted adaptations that enable subjects to retain strength and power in a potentially optimal manner based on the predictions of the Minetti model.

Targeted pre-conditioning: Extension of this reasoning led to the proposal of targeted pre-conditioning for space flight. Because of the unique location of space flight in Minetti *k*-space, an effect-opposing pre-conditioning program would optimally produce both $k_{a,ch}>1$ and $k_{c,ch}>1$. On a practical level, this means developing both local adaptations for increased strength and central adaptations for functional power to extend performance beyond that attributable to hypertrophy alone. The targeted training program should include training components that: 1) produce strength gains over a wide range of velocities to boost both $k_{a,ch}$ and $k_{c,ch}$, 2) increase strength more so than hypertrophy to boost $k_{a,ch}$, 3) increase the muscle power, rate of force development (RFD)

and functional performance to boost $k_{c,ch}$. Such a training program could be described as a combination of conventional and explosive resistance training with a goal of producing net gains in muscle strength and power simultaneously, without necessarily seeking to produce hypertrophy.

* * * * *

T2 elevation with exercise: Since Fleckenstein's original demonstration of acute elevation in both the spin-lattice relaxation time (T_1) and the spin-spin relaxation of time (T_2) of recently exercised human muscles (Fleckenstein 1988) this phenomenon has been utilized in a functional MRI technique that has received both considerable study and clinical usage (reviews: Bendahan et al. 2004, Meyer & Prior 2000, Patten et al. 2003). The particular elevation of T_2 with exercise has received the bulk of the attention, since the same amount of exercise produces greater changes in the signal amplitude of T_2-weighted scans than T_1-weighted scans (Fleckenstein 1988).

At present, T_2 elevation with exercise is recognized as a quantitative indicator of activity-induced patterns of muscle activation, as well as a tool for the diagnosis of muscle injuries and various myopathies (Patten et al. 2003). This review will explore the quantitative behavior of T_2 elevation after exercise, the underlying physiological mechanisms, the correlation of T_2 elevation with exercise intensity, and the practical applications of the phenomenon, especially regarding the study of training and unloading adaptations.

Acute vs. delayed-onset T2 elevation: Two distinct phases of T_2 elevation (acute and delayed-onset) have been observed following strenuous exercise. Acute T_2 elevation usually follows a somewhat consistent time course whereby it reaches a peak value

within 1-2 minutes following the cessation of exercise, and then decays back to baseline with a half-life of anywhere from 5 to 10 minutes (Kennan et al. 1995, Disler et al. 1995, Ploutz-Snyder et al. 1995) or a time constant of 0.114 min^{-1} (Archer et al. 1992). Conversely, the time course of delayed onset T_2 elevation is much more variable. Delayed-onset T_2 elevation begins anywhere from 36-48 hours after exercise and peaks at anywhere from 72-168 after exercise (Foley et al. 1999, Jayaraman et al. 2004, Mair et al. 1992, Prior et al. 2001). There appears to be no relationship between the magnitudes of acute versus delayed-onset T_2 elevation in the thigh (Prior et al. 2001). Acute T_2 elevation has become an accepted indicator of activation during exercise, whereas delayed onset T_2 elevation has become an accepted indicator of post-exercise micro-injury that is related to the phenomenon of delayed-onset muscle soreness (Yanigasawa et al. 2003a). Since this pilot study is exclusively concerned with acute T_2 elevation, the term "T_2 elevation" will refer only to the acute phenomenon from this point forward in this paper.

Quantitative behavior of T2 elevation: Resting muscle typically has a measured T_2 of 24-35 msec (Patten et al. 2003). Following intense exercise, the value can elevate as high as 38% above baseline (de Kerviler et al. 1991, Richardson et al. 1998) and the peak value occurs roughly 1 minute after the cessation of exercise (Kennan et al. 1995). Within a given muscle, the factors known to determine the amount of T_2 elevation include work rate (i.e., power) (Cheng et al. 1995, Fisher et al. 1990), the intensity of exercise relative to maximum voluntary contraction (MVC) (Adams et al. 1992), and intensity relative to maximum aerobic power (Reid et al. 2001). The number of muscular contractions at a given intensity level has a minimum threshold value which must be

crossed before an effect may be observed (Yue et al. 1994), but T_2 elevation will typically saturate as more contractions at this same intensity level occur (Fleckenstein et al. 1993, Yue et al. 1994). Only an increase in intensity will further increase muscle T_2 from this saturation value. Neither the total energy expenditure nor the duration of an exercise session determine the amount of observed T_2 elevation *per se* (Jenner et al. 1994). Several authors have concluded that a linear dependence exists between T_2 elevation and intensity of exercise (Adams et al. 1993, Fisher et al. 1990, Jenner et al. 1994); however, these studies appear to have under-sampled the full range of exercise intensity over which muscles may operate. When more thorough attention is paid to the extremes of the exercise intensity range, this relationship reveals itself to be sigmoidal in nature (Cheng et al. 1995, Fleckenstein 1988, Fleckenstein et al. 1993, Ogino et al. 2002) as shown in Figure 3.

This implies that for a given metabolic energy expenditure during exercise, a minimum "threshold intensity" must be achieved in order to detect any change in T_2. There is also a "saturation intensity" beyond which any additional changes in T_2 are minimal. However, since most exercise training and submaximal testing occurs within these two extremes, there exists a practical range of exercise intensity over which a linear dependence will be a valid approximation (Yue et al. 1994).

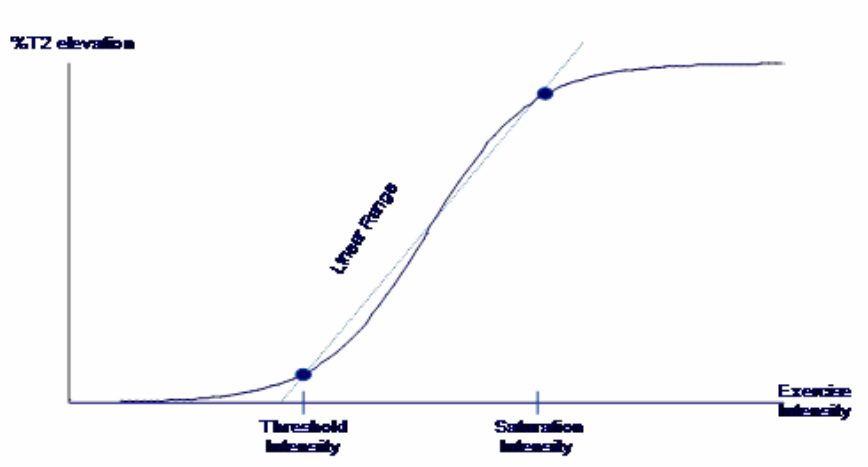


Figure 3 - Sigmoidal dependence of T2 elevation upon exercise intensity. The curve is unique to each subject and depends on training status. Most practical exercise testing and training occur within the approximately linear range between the threshold intensity value and the saturation value.

Another scenario for quantifying T_2 elevation is to fix the exercise intensity and vary the number of muscular contractions or repetitions performed at that intensity. One study determined the minimum number of contractions required to produce a statistically significant rise in T_2 in the elbow flexors at two intensities (Yue et al. 1994), expressed as 25% and 80% of the subject's one-repetition maximum (% 1RM) for an arm curl. The authors found that five repetitions were required to detect T_2 elevation at 25% 1RM, but only two were required at 80% 1RM. The same study also noted that no saturation behavior in T_2 elevation occurred at 25% 1RM up to 40 repetitions, but at 80% 1RM the T_2 elevation started saturating after 10 repetitions, i.e., additional repetitions beyond 10 produced less elevation in T_2. Sprint bicycling for 6 second bursts with 30 seconds rest between bursts produced a similar effect in all thigh muscles except *m. gracilis* (Akima et al. 2005). Collectively, these results also imply a plateau relation between number of repetitions (or total work) and T_2 elevation at a fixed intensity level of exercise. An echo planar imaging (EPI) study of *m. tibialis anterior* where the subjects dorsiflexed in the

magnet with a fixed load (35% 1RM) at contraction rates of 10/min, 20/min and 30/min produced a plateau behavior in T_2 over time (Jenner et al. 1991). In this case, the plateau value of T_2 changed according to the rate of contractions (i.e., power). In another study, calf raises produced greater elevation in T_2 going from body weight (BW) to 115% of BW than the increase from 50% BW to BW (Kinugasa et al. 2005). These results imply sigmoidal behavior for T_2 elevation versus number of contractions at a fixed level of intensity, as well as a direct dependence of the plateau value on the intensity level. This is depicted qualitatively in Figure 4.

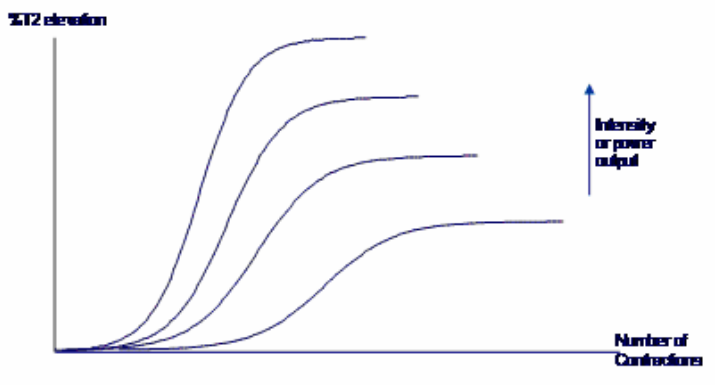


Figure 4 - Plateau behavior of T2 elevation versus the number of contractions at a given intensity. The family of curves shows that as intensity rises, the number of contractions required to produce a response and the number required to reach the plateau value both decrease.

One study used a dynamic single-shot echo-planar imaging technique to record T_2 measurements in the anterior calf while subjects dorsiflexed in the magnet at four intensity levels, 20%, 40%, 60% and 80% of 1RM (Disler et al. 1995). The resulting data resemble Figure 4. These authors are alone in the literature in concluding that total work did contribute to T_2 elevation, however, they did not vary the frequency of contractions in order to isolate work rate effects. The key consensus of the studies cited in this section is that the degree of T_2 elevation depends on the intensity (force) or work rate (power) of

exercise rather than the total caloric expenditure, the time spent exercising or the time under load. During sprint cycling, T2 elevation and power output are in fact well-correlated ($r > 0.75$, $p<0.0001$), particularly in the VM and VL (Akima et al. 2005).

The sensitivity of T_2 elevation is such that detectable response occurs after 5 contractions at 25% of 1RM and after only two contractions at 80% of 1RM (Yue et al. 1994). There is also a high correlation ($r \sim 0.9$) between force exerted and T_2 increase following resistance exercises (Adams et al. 1992, Fisher et al. 1990).

Axial spatial dependence: Since individual skeletal muscle fibers typically span the entire length of a muscle, one would expect little axial variation of T_2 elevation following exercise. Indeed, many studies have used a single axial slice at the belly of the target muscle to determine mean intramuscular T_2 (Price et al. 1995, Price et al. 1998, Yanagisawa et al. 2003a, Yanagisawa et al. 2003b, Yanagisawa et al. 2003c, Yanagisawa et al. 2003d) based on this operating assumption. There have been studies which have specifically investigated the axial dependence of T_2 elevation with exercise, but not always with the same outcome variables. In one study, isokinetic knee extensions produced T_2 elevation with no significant differences in the percentage of pixels with elevated T_2 values along seven axial slices of the QF (Akima et al. 1999). A resistance training intervention did not alter this observation. However, the same author later published a study where a similar exercise protocol induced a pattern of preferential mean T_2 elevation (a different outcome variable) in the distal slices of *m. rectus femoris*, while the three *vasti* QF muscles did not exhibit this behavior (Akima et al. 2003).

Transverse spatial dependence: The intra-slice spatial dependence of T_2 elevation is more controversial and it requires careful interpretation. Some researchers have used

the percentage of cross-sectional area that exhibits elevated T_2 (%CSA $_{T2+}$) as an outcome variable (Adams et al. 1993, Akima et al. 1999, Akima et al. 2000, Ploutz-Snyder et al. 1995a, Ploutz-Snyder et al. 1995b, Prior et al. 1999, Ray & Dudley 1998), although most others have used mean intramuscular T_2 (μ_{T2}). Furthermore, T_2 images pre- and post-exercise have been used to generate "T_2 maps" whereby elevated pixels, defined to be >1 SD above the baseline values of μ_{T2}, are identified in the post-exercise images as indicators of spatially localized regions containing activated muscle fibers. While one researcher has argued that these T_2 maps indicate cross-sectional muscle utilization patterns (Warfield et al. 2000), another researcher has convincingly argued that this technique is invalid in normal muscle tissue due to the heterogeneous contents of the tissue enclosed within an MRI voxel, the inherently random distribution and voluntary recruitment of motor units in the body, the similar magnitude of the variance in T_2 measurement and T_2 elevation, and the notable absence of a bimodal distribution of muscle pixels following submaximal exercise (Prior et al. 1999). This last point is critical, since if spatially localized T_2 mapping has validity, then preferential recruitment of "smaller motor units first, then larger ones" in performing submaximal tasks (Guyton & Hall, 10th ed.) should produce localized areas of T_2 elevation. This should in turn split the normal distribution of T_2 pixels into a bimodal distribution, which no study has demonstrated. Indeed, identical bouts of exercise can induce a wide variety of T_2 maps among healthy subjects (Prior et al. 1999), but the single-mode normal distribution of pixels remains. Even during electromyostimulation (EMS) which tends to recruit the same motor units with each contraction much more so that voluntary contractions, the T_2 maps among subjects are highly variable and quite sensitive to electrode placement

(Adams et al. 1993). The value of cross-sectional T_2 pixel-mapping of muscle seems to lie in the study of myopathologies or motor neuron disease, where localized areas of damaged or non-recruitable muscle may present themselves after exercise with no significant T_2 rise above baseline (Patten et al. 2003).

Physiological basis: Despite considerable investigation, the exact physiological mechanism for T_2 elevation with exercise remains elusive. This is largely due to the complexity of the phenomenon and its apparent dependence on a multiplicity of factors. The consensus of the scientific literature would attribute exercise-induced T_2 elevation to osmotically-driven shifts in intracellular water and the accumulation of osmotic metabolites and the by-products of anaerobic metabolism with some contribution from the aerobic metabolic pathway as well (Bendahan et al. 2004).

When edema alone is induced by lower-body negative pressure (LNP) it produces less T_2 elevation and a distinct shift from monoexponential to biexponential behavior in transverse relaxation (Ploutz-Snyder et al. 1997), an effect not seen after exercise. Conversely, head-down tilt (HDT) for 24 hours produced significant edema in neck muscles but no significant change in T_2 (Conley et al. 1996). These authors have concluded that while intracellular fluid shifts certainly contribute to increases in signal intensity of T_2-weighted images, they cannot fully explain the phenomenon (Fisher et al. 1990).

Several researchers report that pH is negatively correlated with T_2 during the positive phase of exercise-induced T_2 elevation (Cheng et al. 1995, Damon et al. 2002, de Keviler et al. 1991, Jehenson et al. 1993, Morvan et al. 1992, Weidman et al. 1991), but not necessarily so during the recovery phase (Cheng et al. 1995, Morvan et al. 1992).

Additionally, T_2 elevation precedes acidosis during incrementally graded exercise (Cheng et al. 1995). These observations suggest a contribution for acidosis in T_2 elevation, but that acidosis alone cannot fully explain the phenomenon.

Muscle glycogen concentration as determined by MRS does not correlate *per se* with T_2 elevation (Price et al. 1998). However, patients with pathologies in the glycogenolytic pathway display little or no T_2 elevation post-exercise (de Kerviler et al. 1991, Fleckenstein et al. 1991, Jehenson et al. 1993). This suggests that the products of glycogenolysis are more strongly related to T_2 elevation than the amount of stored muscle glycogen.

Lactate buildup may drive the osmotic fluid shifts that in turn drive T_2 elevation with intense exercise. The role of muscle glycogen appears to be more indirect, only to the extent that it is the reactant that produces lactate in anaerobic metabolism. No study could be found that correlated lactate concentration with T_2 elevation *per se*.

Conflicting data exist for the effect of metabolic phosphate concentrations. One MRI/MRS study reports a strong correlation between the ratio of inorganic phosphate to phosphocreatine (Pi/PCr) and T_2 elevation, with no such dependence reported for ATP (Weidman et al. 1991). This implies that the PCr metabolic pathway, which is utilized to a higher degree in more intense exercise than less intense exercise, has a key role in T_2 elevation. This is consistent with the previously cited observations of T_2 elevation scaling with exercise intensity. However, another MRS study reports no such correlation with PCr, but rather a correlation with glucose 6-phosphate (Price et al. 1998). These authors concluded that glucose transport into exercising muscle also plays a role. The

body of evidence would seem to suggest that the various metabolites have a synergistic effect that produces the water shifts that drive T_2 elevation.

Vascular occlusion has a positive reinforcing effect on T_2 elevation with exercise, yet occlusion alone produces only minimal changes in T_2 (Fisher et al. 1990). This concept may be exploited in clinical practice to detect occlusive disease in muscles that exhibit localized areas of excessive T_2 elevation following exercise (Yoshioka et al. 1995). One may therefore reason that occlusion elevates T_2 due to the buildup of metabolic byproducts accompanying decreased perfusion and the resulting osmotic fluid shifts that arise from this buildup.

Perfusion itself has been studied in conjunction with T_2 elevation. By occluding vascular flow during exercise and allowing intermittent periods of reperfusion, Archer demonstrated that reperfusion initially augments the T_2 elevation, but continuous reperfusion eventually drives the decrease in T_2 during recovery (Archer et al. 1992). However, Fleckenstein, noting a lack of T_2 elevation in McArdle's patients even under vascular occlusion, concluded that perfusion *per se* is not the primary factor in T_2 elevation with exercise (Fleckenstein et al. 1991).

Although PCr/ATP, Pi/ATP and (PCr + Pi)/ATP ratios correlate with the percentage of type II fibers, no such correlation exists for resting values of T_2 (Takahashi et al. 1994). However, Prior demonstrated that localized areas of greater type II (anaerobic) fiber density in a given muscle show greater T_2 elevation than areas with greater type I (aerobic) density (Prior et al. 2001).

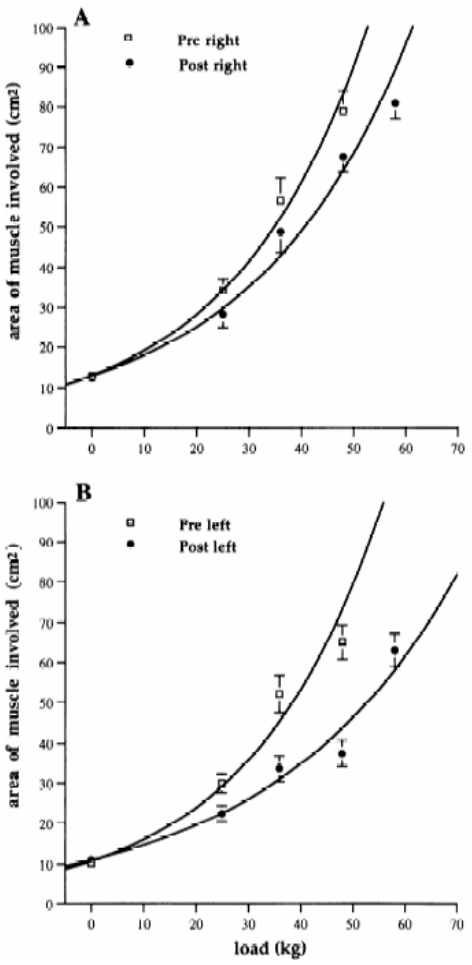
Despite the lack of a conclusive physiological explanation for T_2 elevation with exercise, the phenomenon remains widely accepted as a quantitative indicator of exertion, although the results often require careful interpretation (Patten et al. 2003).

Dependence on training status: Numerous studies have examined T_2 elevation among groups of individuals with widely varied training status. For traditional strength exercises, the intensity expressed as a percentage of the subject's *n*-repetition maximum (% of *n*RM) appears to be the key variable for producing T_2 elevation in isolated muscles (Adams et al. 1992). This is true both for cases where two muscles in the same individual are investigated or where the same muscle group is investigated across a population. For example, although the plantar flexors and dorsiflexors have a relative strength ratio of seven, when subjects exercised each muscle group at 25% of its 1RM, the measured T_2 increase in each muscle group was statistically the same (Price et al. 1995).

For more continuous exercises, the work rate expressed as a power appears to drive T_2 elevation, which is dependent on the subject's aerobic training status. A study of trained versus untrained cyclists found that at work rates of 50% and 90% of the subjects' maximum aerobic power, there were no significant differences in T_2, despite a 72% and 60% difference, respectively, in the absolute value of the power production between the two groups (Reid et al. 2001). When comparing work rates producing T_2 elevation across a population, the comparison must therefore be standardized relative to each subject's maximum aerobic power, and not absolute work rate (Bendahan et al. 2004). These results significantly impact the choice of methods used in both this pilot study and the proposed targeted pre-conditioning study. It is also possible to perform classification

analyses that identify the training class of specific individuals based on their location in a parameter space spanned by performance versus T_2 elevation (Le Rumeur et al. 1994).

Training adaptations and T2: The use of T_2 elevation in detecting adaptations to resistance training is best exemplified in two key studies. One study of the calf muscles noted strength gains early in a resistance training program accompanied by an increase in T_2, but not in muscle CSA. Based on this, the authors concluded that early strength gains in a resistance training program come primarily from increases in neural drive rather than hypertrophy (Akima et al. 1999). Another study of unilateral training in the quadriceps found that resistance training moved the T_2 elevation versus load curve of the trained limb down and to the right, so that less activation is seen post-training to lift the same load compared to the pre-training state (Ploutz et al. 1994). Refer to Figure 5. Even more interesting, the study found that the same effect (to a lesser degree) occurred in the untrained limb as well. This study documented the detection of an adaptation to resistance training that is purely resident in the central nervous system via functional MRI.

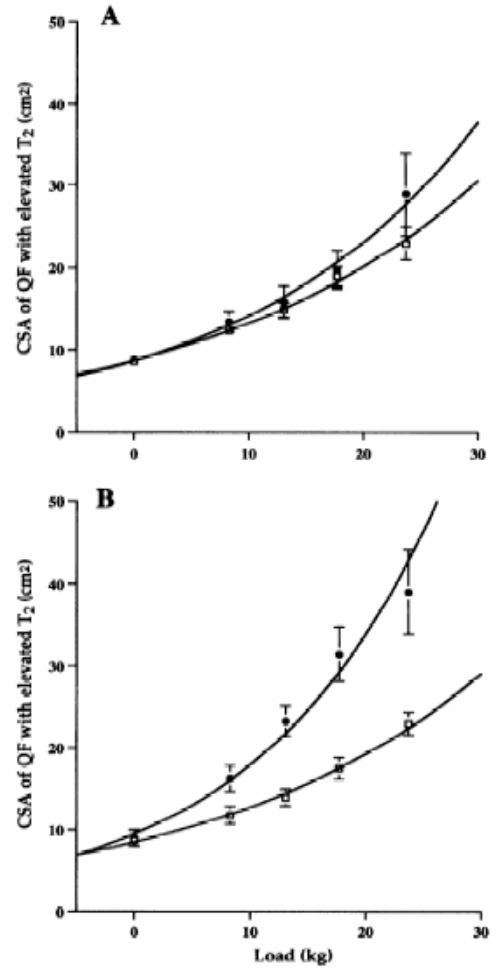


Taken from Ploutz LL, Tesch PA, Biro RL, Dudley GA. "Effect of resistance training on muscle use during exercise", J. Appl Physiol. 1994 Apr;76(4):1675-81

Figure 5 - Effect of resistance training on area of muscle showing elevated T2 after exercise. Average cross-sectional area (cm^2) of right untrained (A) and left trained (B) quadriceps femoris muscles (QF) showing MRI contrast shift, and thereby use, plotted as a function of load lifted during exercise pre- (□) and post-training (●).

Unloading interventions and T_2: The typical effect of unloading is not to change resting T_2 values, but rather to increase the amount of elevation seen during the performance of the same submaximal exercise performed both before and after the unloading event. Two bed rest studies have reached this conclusion (Akima et al. 2003, Conley et al. 1996), as have two studies involving ULLS (Ploutz-Snyder et al. 1995,

Ploutz-Snyder et al. 1996). Unloading therefore shifts the load to T2 elevation curve up and to the left as shown in Figure 6.



Taken from Ploutz-Snyder LL, Tesch PA, Crittenden DJ, Dudley GA. Effect of unweighting on skeletal muscle use during exercise. J Appl Physiol. 1995 Jul;79(1):168-75.

Figure 6 – Effect of unloading on area of muscle showing elevated T2 after exercise. Average cross-sectional area (CSA) of right weight-bearing (A) and left unweighted (B) quadriceps femoris muscle group (QF) with an elevated T2 and thereby suggested to have recently performed contractile activity, plotted as a function of absolute load lifted for 5 sets of 10 unilateral concentric repetitions before (○) and after (●) 5 wk of unweighting of left QF. Point 0 for load is pre-exercise. Values are means ± SE for group; n=7 subjects.

T_2 elevation versus iEMG for measuring activation: There is no direct measurement of neural activation in *in vivo* muscle. T_2 elevation and integrated electromyography (iEMG) are the two dominant methods for obtaining quantitative data that *relates* to muscle activation. These two methods offer different but complementary

means of evaluating neuromuscular activation. Surface iEMG directly measures the electrical activity of muscles in real time. But its use is limited to larger and more superficial muscles. Signal interpretation from iEMG can be difficult, as it may be confounded by skin cleaning, electrode placement, cross talk among muscles, and changes in the size of the muscle or fat layer over time when making longitudinal measurements.

Conversely, the challenge to the interpretation of elevated T_2 values is that the mechanism of the T_2 increase seems to be metabolic in origin, as we have seen. So in cases where muscle metabolism does not adapt (acute studies) T_2 can be used as a reliable indicator of muscle recruitment patterns. However, this may be complicated under conditions where muscle metabolism is likely to change (unloading, training, disease, etc). Therefore, longitudinal studies with either training or unloading interventions are advised to interpret data from both techniques. One author has found that EMG and T_2 elevation correlated ($r=0.99$) and scaled with intensity over the limited range of exercise intensity (5x10 reps at 40%, 60%, 80% and 100% of 10RM) that was studied (Adams et al. 1992).

Problem Statement

This main purpose of this study was to use T_2-relaxation time elevation to determine activation and recruitment (i.e., efferent neural drive) differences in the thigh and calf muscles of two distinct populations during single-leg jumping exercise. One population consisted of high performing jumpers, and the other consisted of otherwise healthy, but low-performing jumpers. Several hypotheses were quantitatively tested. For given muscles *m1* and *m2*:

H1) T2 elevation (ΔT2) will vary between specific leg muscles, e.g., $\Delta T2_{m1} \neq \Delta T2_{m2}$

H2) ΔT2 for primary agonist muscles will depend on the jumping power (P_{jump}) relative to aerobic fitness expressed as Maximum Aerobic Power (MAP), i.e., $\Delta T2_{m1} = f(P_{jump}/MAP)$

H3) ΔT2 will increase with an increase in the measured intensity of the jumping exercise (not necessarily linear), e.g., $If\ P_2 > P_1,\ then\ \Delta T2_{m1,P2} > \Delta T2_{m1,\ P1}$

H4) Within a given population there will be heterogeneity in the activation pattern of specific muscles in the leg, i.e., $\Delta T2_{m1}^{(J1)} \neq \Delta T2_{m1}^{(J2)}$

H5) High-performing jumpers (J) exhibit more efficient muscle recruitment than low performing jumpers (C) based on a metric (M) that rewards power production and penalizes higher T_2 elevation, i.e., $M_J > M_C$.

There were also two secondary objectives of the study, namely 1) to determine the effectiveness and feasibility of the intended exercise protocol and scan procedures for use in the proposed pre-conditioning study, as previously described, and 2) to demonstrate

and validate the data gathering and analysis methods of the proposed study, including the exercise protocol, the MRI scans, the jump performance measurement and the image processing techniques for analysis of MRI images.

Subjects and Methods

Initial screening: After the Institutional Review Board approved this study for human experimentation, twelve healthy volunteers, 21-48 yr old, were recruited from the University and the surrounding metro area. All subjects gave informed written consent before participating. Subjects completed both a medical history questionnaire to identify any risks that might preclude their participation and an activity questionnaire (Baecke et al., 1982) to quantify their relative levels of activity. Subjects were excluded based on recent or chronic joint injury in the legs, heart conditions, pregnancy, or current treatment with prescription medications. Three potential subjects were excluded for medical reasons.

Jump performance testing: The subjects were then screened into two experimental groups based on their performance in a two-leg jump and reach test conducted on a jump platform. A certified exercise physiologist explained the test procedure to each subject, who was then given 3-5 half-effort warm-up attempts to practice. Following two minutes of rest, the subject then performed 5 individual maximal-effort jumps separated by rest intervals of one minute between each jump. The best jump height of the five determined the raw score. This value was compared to published normative data (Payne et al., 2000) to obtain a percentile score based on the age and gender of the subject. The percentile score became the screening criterion. Subjects with a percentile score of 75 or higher were accepted into the accomplished jumpers (J) group while subjects with a percentile score of 50 or less were accepted into the control (C) group. Subjects falling in the 51st-74th percentile were not accepted for the study.

Maximal Exercise Test (MET): Each subject performed a maximal exercise test on a cycle ergometer under the close supervision of a certified exercise physiologist. The subjects executed the ACSM standard ramp protocol at a pedaling cadence of 60 rpm (ACSM, 1995). An initial warm-up period of two minutes at 25 Watts of pedaling power was followed by incremental increases of 5 Watts every ten seconds until fatigue prevented the subject from maintaining with the pedaling cadence at which point the test was terminated. During the entire test the subjects' oxygen uptake and carbon dioxide output were recorded. Heart rate and EKG were monitored for indications of distress, which would be cause to immediately terminate the test. This did not occur in any of the subjects. The key measurements from the MET were the subjects' power at anaerobic threshold (P_{AT}, Watts), maximum aerobic power ($P_{A,Max,}$ Watts) and maximal oxygen uptake ($V_{O2,\,max}$, mL/min).

Exercise protocol: Five sets of ten (5 × 10) single-leg vertical jumps (sometimes referred to as "hops") on a force platform constituted the exercise protocol for inducing T_2 elevation in the thigh and calf muscles. A certified exercise physiologist instructed each subject to maintain their hands on their hips, take off on their dominant leg and land on two legs. Landing criteria were generally observed, but were not always strictly enforced. Two levels of intensity were employed: at body weight (Post1) and at body weight + 33% (Post2). For the Post2 sets, the subject donned a weighted vest commonly used for hands-free training. Each subject was given a 60-90 second window to perform each set of ten jumps at their own pace. Rest periods were one minute between sets. Immediately following the completion of the last set of jumps, the subject was quickly assisted into the magnet (after expediently removing the vest at Post2) for the start of

MRI scanning. The force platform and the magnet were in adjacent rooms to ensure the subjects' safety. One hour separated the Post1 and Post2 exercise bouts. This is adequate time for acute T2 elevation to abate in the lower limbs (Kennan et al. 1995, Disler et al. 1995, Ploutz-Snyder et al. 1995). Appendix B explains the technical aspects of the force platform and Appendix D explains the rationale for this selection of exercise protocol.

Jumping performance measurements: The force platform (Kistler Quattro-Jump, Tonawanda, NY) recorded the subjects jumping weight, jump time in-flight and force production vs. time, *f(t)*, at a sampling rate of 500 Hz. From these measurements, the accompanying software computed the height (h_j, cm), instantaneous concentric power ($P_j(t)$, Watts), average concentric power ($P_{j,avg}$, Watts) and peak concentric power ($P_{j,peak}$, Watts) for each jump. From these, the peak (P_{peak}, Watts) and average (P_{avg}, Watts) jumping power for the entire exercise set were computed as

(6) $$P_{peak} = Max(P_{j,peak})$$

(7) $$P_{avg} = \frac{1}{N_{jumps}} \sum_{j=0}^{N_{jumps}-1} P_{j,avg}$$

The force-time integral (FTI) was computed during the concentric phase of each jump as

(8) $$FTI = \frac{1}{N_s} \sum_{t=0}^{N_s-1} f(t)\Delta t$$

where N_s is the number of samples collected during the jump and Δt is the sampling period, 20 msec. Landing forces were considered to be absorbed primarily by the bones and connective tissue with a small amount of eccentric energy expenditure in the quadriceps and plantar flexors. This eccentric power production was assumed to

contribute much less to the elevation of T_2 in all leg muscles than the concentric component at takeoff.

MR imaging techniques: Thirty (30) axial slice images each of the thigh muscles along the femoral length, from the greater trochanter to the adductor tubercle, and of the calf muscles along the entire tibial length were obtained using a 1.5 Tesla Siemens Symphony clinical MR scanner. T_2-weighted spin-echo sequences were 2,000/30,60 msec (repetition time / echo time 1, echo time 2). Each slice image represents 1 cm of axial thickness and a 500mm x 250mm field of view (FOV) on a 256 x 128 pixel grid. The gap between each slice was 1 cm. Appendix C contains the details of the MRI scan used for this study.

Image processing techniques: MRI image processing techniques were implemented using a combination of resident features and programmed macros in Optimas 6.5 (Media Cybernetics; Bothel, WA). Macros were written in the Analytical Language for Imaging (ALI), and are explained in Appendix A. For the thigh, the muscles of interest were *m. vastus lateralis* (VL), *m. vastus medialis* (VM), *m. vastus intermedius* (VI), *m. rectus femoris* (RF), *m. sartorious* (Sar), *m. adductor longus* (AL), *m. adductor magnus* (AM), *m. gracilis* (Gr), *m. semimembranosus* (SM), *m. semitendonosus* (ST) and *m. biceps femoris* (BF). Additionally, the *m. quadriceps femoris* (QF) muscle group (i.e., VL+VM+VI+RF) was considered as an aggregate for analysis. For the calf, the muscles of interest were *m. gastrocnemius lateralis* (GL), *m. gastrocnemius medialis* (GM), *m. soleus* (Sol), *m. flexor digitorum longus* (FDL), *m. flexor hallucis longus* (FHL), *m. tibialis posterior* (TP), *m. peroneus longus* (PL), *m. tibialis anterior* (TA), *m. extensor digitorum longus* (EDL), and *m. extensor hallucis*

longus (EHL). Additionally, the major plantar flexors (PF) (i.e., GL+GM+Sol) and dorsiflexors (DF) (i.e., TA+EDL+EHL) were analyzed as aggregate muscle groups. For each muscle or aggregate group, the region of interest (ROI) for T_2 analysis was drawn manually on three (larger muscles) or four (smaller muscles) adjacent slices located on the mid-belly of the muscle or group. Previous studies have shown that muscular T_2 is relatively insensitive to axial position, even after exercise (Akima et al. 2004; Akima et al. 1999; Richardson et al. 1998). Care was taken to exclude from the ROIs any visible non-muscle tissue, such as blood vessels, fat, bone or connective tissue. Any blood flow artifacts were also excluded from the T_2 ROI. Pixel T_2 values, $\hat{T}_2$, were estimated from the two spin echo images using

$$(9) \quad \hat{T}_2 = \frac{(T_{E2} - T_{E1})}{\ln(I_1 / I_2)}$$

where T_{E1} and T_{E2} are the two echo times and I1 and I2 are the pixel intensity values in each image. The output of this process is a T_2 map image, whereby each pixel in the image bears a gray scale value equal to its locally computed T_2 value in milliseconds.

Muscle volumes were computed by tracing the visible external boundary of each muscle as another ROI on a spatially calibrated image for each slice in which the target muscle appears over either the femoral or tibial length. An atlas of skeletal muscle on MRI images provided guidance for this process (Berquist, 1995). The muscle volume, *V*, was computed using the truncated cone formula between slices (Ross et al. 1996):

$$(10) \quad V = \sum_i A_i t + \frac{1}{3} \sum_i h(A_i + A_{i+1} + \sqrt{A_i A_{i+1}})$$

where $\boldsymbol{A_i}$ is the area in the *i*th slice, *t* is the slice thickness and *h* is the distance between slices. The mean muscular T_2 value (μ_{T_2}), the number of pixels with elevated T_2 ($N_{T_2,elev}$) and the total number of pixels in the ROI (N_{tot}) were computed for each muscle.

Performance to activation ratios: For this study two metrics were designed to detect output power with high recruitment efficiency in specific muscles. The metrics reward power production as measured by the force platform and incur a penalty for higher recruitment as measured by the percentage of elevated pixels within the muscle ROI of the T_2 image. The average performance to activation ratio (APAR) is defined as

$$APAR = \frac{P_{j,avg} / P_{A,\max}}{N_{T_2,elev} / N_{tot}} \tag{11}$$

and the peak performance to activation ratio (APAR) is defined as

$$PPAR = \frac{P_{j,peak} / P_{A,\max}}{N_{T_2,elev} / N_{tot}} \tag{12}$$

where the numerator of the APAR is the ratio of the average of all 50 peak jump power measurements to the subject's maximum aerobic power (MAP), the numerator of the PPAR is the ratio of the maximum of all 50 peak jump power measurements to the subject's MAP, and the denominator of both metrics is the fraction of pixels in the muscle ROI that have an elevated T_2 value. Jump power is referenced to MAP for consistency with a previous finding that increases in muscle T_2 vary with work rate relative to MAP, and not with absolute work rate (Reid et al. 1999). The choice of

percentage of elevated T_2 pixels rather than change in mean muscular T_2 is due to the fact that the former quantity showed no significant differences between groups at Baseline, whereas the latter quantity did show significant differences at Baseline in several muscles.

Statistical techniques: Conventional statistical analyses were employed to calculate mean values (μ), standard deviations (σ) and Pearson correlation coefficients (r). One-way analysis of variance (ANOVA) with repeated measures was used to compare differences between groups for individual muscles at Baseline, Post1 and Post2. Student's t-test was used to compare group differences in performance data and physical characteristics. Pair-wise t-test comparisons were made for individual muscles at Post1 and Post2 relative to Baseline values, and relative to each other.

Results

Subjects: Characteristics of the subjects are summarized in Table 1. The two groups did not have significant differences in age, height, weight, body mass index or Baecke activity score. Gender differences did exist, with the C Group being more heavily represented by females.

Table 1 - Subject characteristics

	J Group	C Group
Gender, female/total	2/6	4/6
Age, yr	28.7 ± 4.0	28.0 ± 1.8
Height, cm	180.7 ± 3.8	171 ± 3.1
Weight, kg	72.6 ± 5.0	84.4 ± 10.3
BMI, kg/m^2	21.7 ± 1.3	28.2 ± 2.9
Dominant leg, right/total	3/6	5/6
Baecke Activity Score	7.5 ± 0.7	6.7 ± 0.4

Values are means ± SE; n = 6. BMI = Body Mass Index

Jumping performance: A typical set of jump measurements is shown in Figure 7. Note that the peak power is defined as the peak concentric power, not landing (eccentric) power. Figure 8 summarizes the jumping performance data obtained for the J and C groups. The J Group outperformed the C Group in the two-leg jumping test that was used for screening (J: 53.1 ± 4.3 cm; C: 34.0 ± 2.5 cm; p=0.02) as expected.

As shown in Figure 9, J Group members demonstrated superior jumping performance at Post1 by all measurements, including peak jump height (J: 26.2 ±1.6 cm, C: 18.3 ± 1.2 cm, p=0.001), average jump height (J: 22.9 ± 1.3 cm, C: 15.7 ± 1.0 cm, p=0.006), peak jump power per body mass (J: 24.1 ± 1.5 W/kg, C: 16.5 ± 0.9 W/kg, p=0.021), average jump power per body mass (J: 11.5 ± 0.9 W/kg, C: 8.0 ± 0.5 W/kg, p=0.004), FTI per body mass (J: 0.25 ± 0.03 N-s/kg, C: 0.20 ± 0.01 N-s/kg, p=0.05) and peak force as a fraction of body weight (J: 0.91 ± 0.11; C: 0.50 ± 0.03, p=0.05).

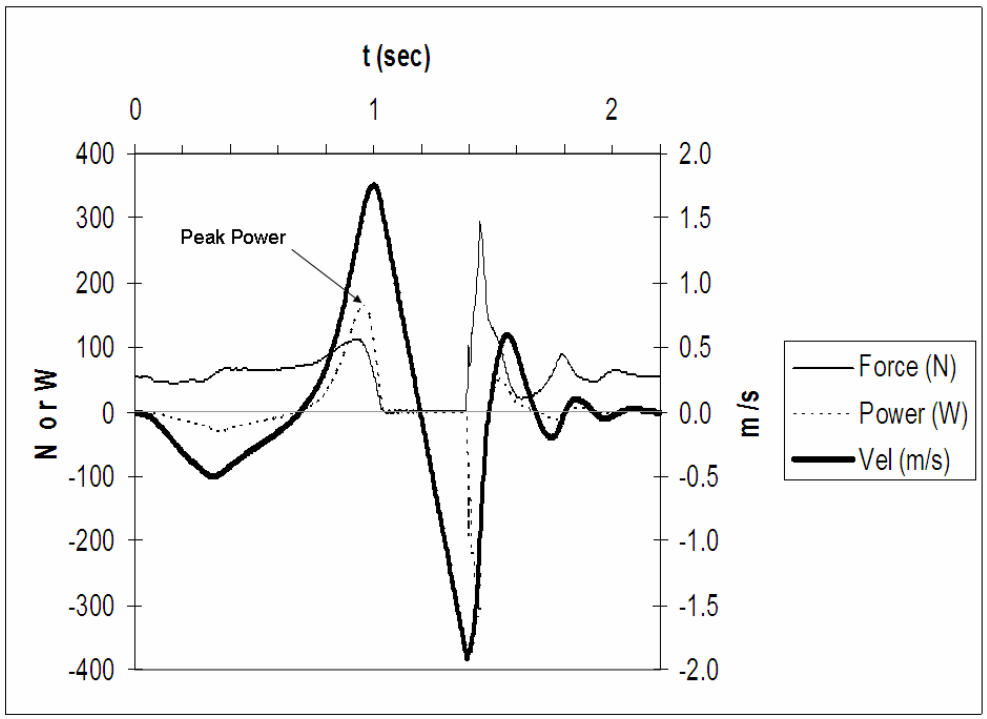


Figure 7 - Representative single-leg jump data from the force platform for a 56kg male from the J Group. Force, velocity and power traces are as indicated by the legend at the right.

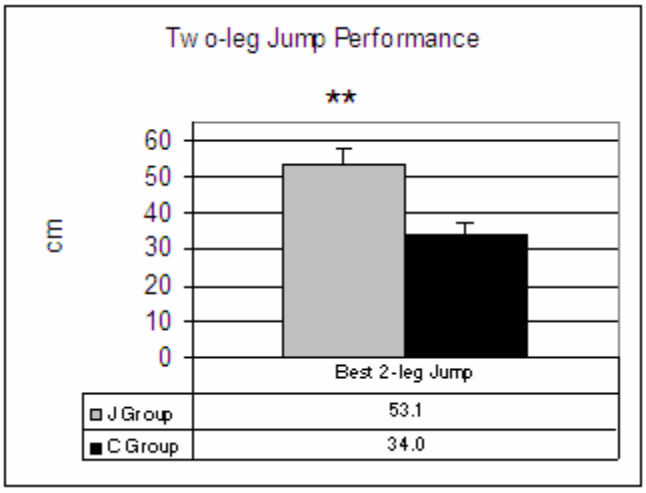


Figure 8 - Two-leg jump height (cm) from initial screening of subjects. (J>C: * = p<0.05, ** = p<0.01)

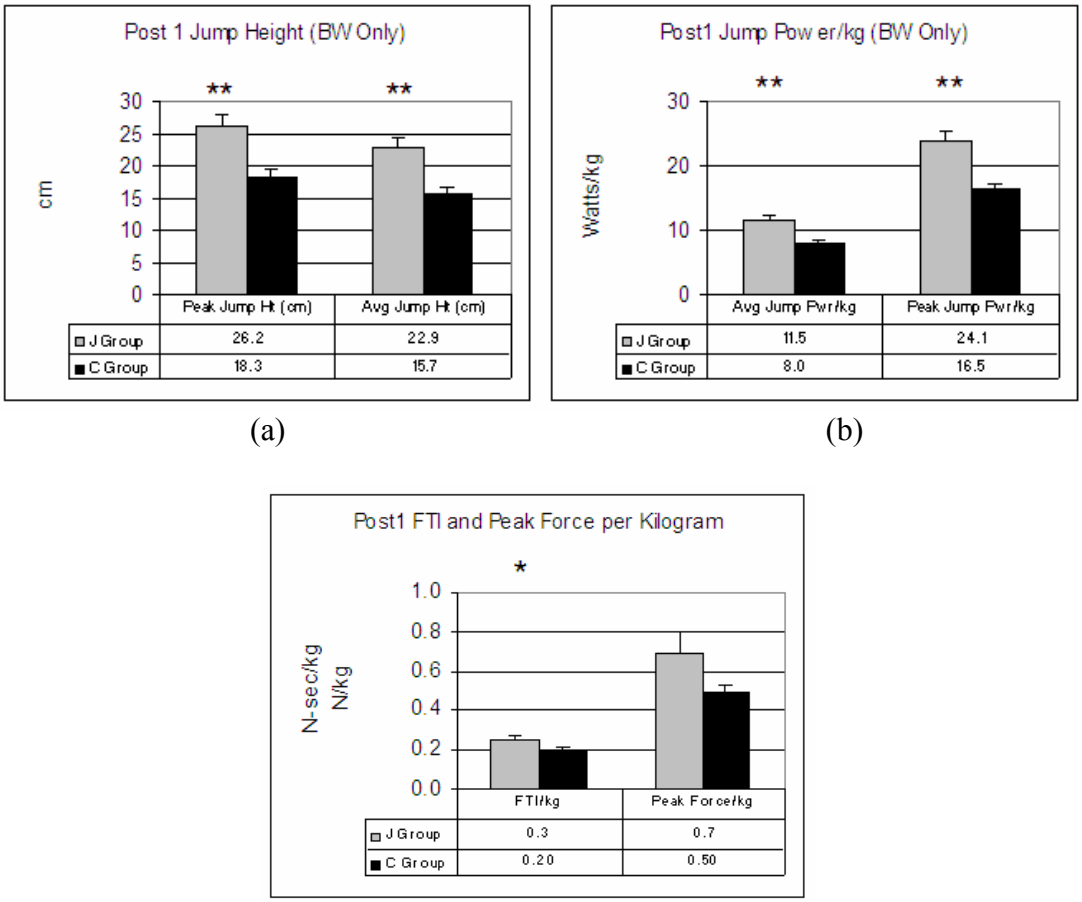


Figure 9 - Post1 (single-leg jumping at body weight) results: peak and average jump height (a), peak and average jump power per body mass (b) and force-time integral and peak force (c) . (J>C: * = $p<0.05$, ** = $p<0.01$).

As shown in Figure 10 at Post2, J Group still outperformed the C Group in peak jump height (J: 19.8 ± 0.9 cm, C: 15.6 ± 1.1 cm, p=0.033), average jump height (J: 16.9 ± 0.9 cm, C: 13.9 ± 1.1 cm, p=0.01), peak jump power per body mass (J: 22.8 ± 1.3 W/kg, C: 17.0 ± 0.6 W/kg, p=0.004), average jump power per body mass (J: 10.3 ± 0.7 W/kg, C: 7.8 ± 0.4 W/kg, p=0.02), FTI per body mass (J: 0.27 ± 0.01 N-s/kg, C: 0.21 ± 0.01 N-s/kg, p=0.04) and peak force as a fraction of body weight (J: 0.66 ± 0.06; C: 0.52 ± 0.02, p=0.12). In this case, the last measured difference fell short of statistical significance.

Jump height decreased significantly from Post1 to Post2 in the J Group (Avg: -6.0 cm, p=0.01; Peak: -6.4 cm, p=0.004). The jump height trended lower from Post1 to Post2 in the C Group (Avg: -1.8 cm, p=0.06; Peak: - 2.7 cm, p=0.24). Power production during jumping for both groups did not change significantly when the load increased from BW to BW+33%, both in terms of peak power/body mass (J: -1.3 W/kg, p=0.28, C: -0.2 W/kg, p=0.73) or average power/body mass (J: -1.2 W/kg, p=0.72, C: +0.5 W/kg, p=0.94).

Aerobic training status: As shown in Figure 11, the J Group demonstrated a greater degree of aerobic fitness than the C Group during the maximal exercise test (MET), as measured by anaerobic threshold/kg of body mass (J: 2.4 ± 0.5 W/kg, C: 1.75 ± 0.3 W/kg, p=0.13), maximum aerobic power/kg of body mass (J: 4.1 ± 0.3 W/kg, C: 2.98 ± 0.3 W/kg, p=0.02) and maximum oxygen uptake, $V_{O2,\ max}$ (J: 50.6 ± 5.0 mL/kg/min, C: 35.7 ± 3.6 mL/kg/min, p=0.02).

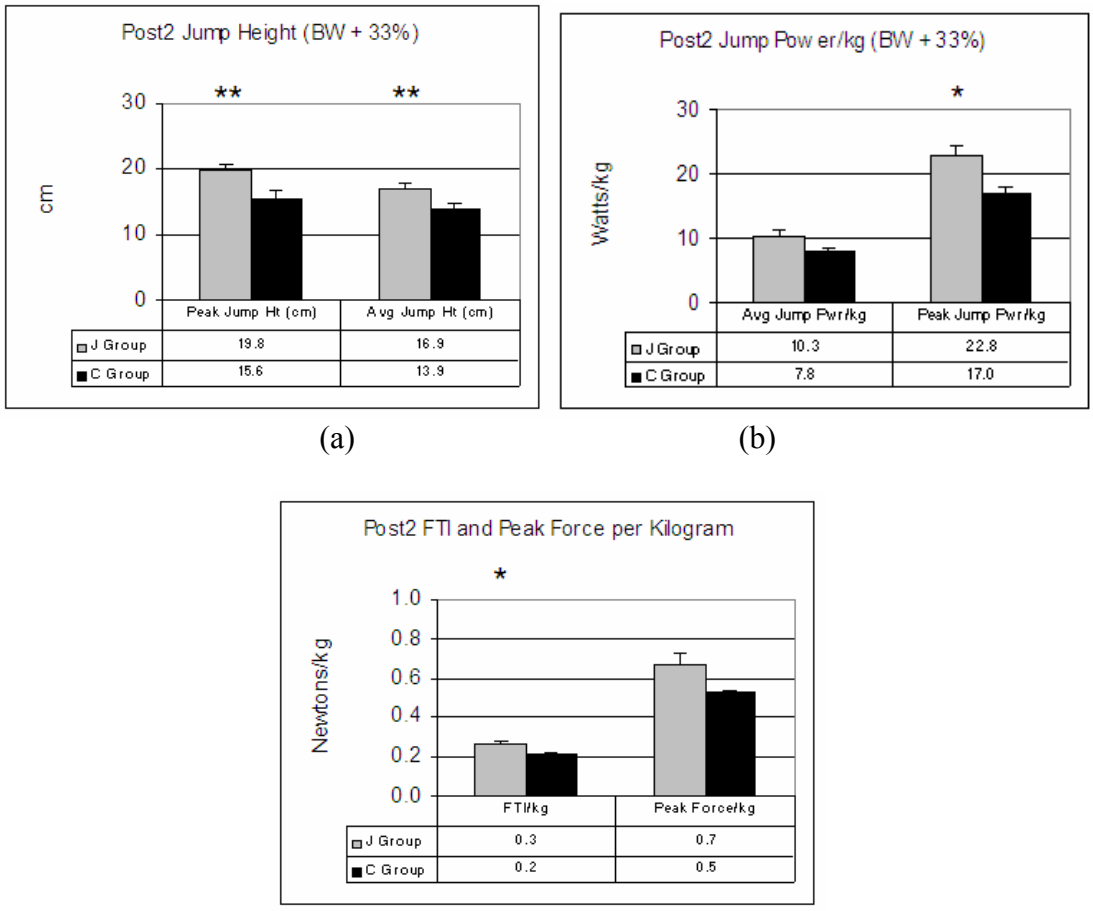


Figure 10 - Post2 (single-leg jumping at body weight + 33%) results: peak and average jump height (a), peak and average jump power per body mass (b), and force-time integral and peak force (c) . (J>C: * = p<0.05, ** = p<0.01).

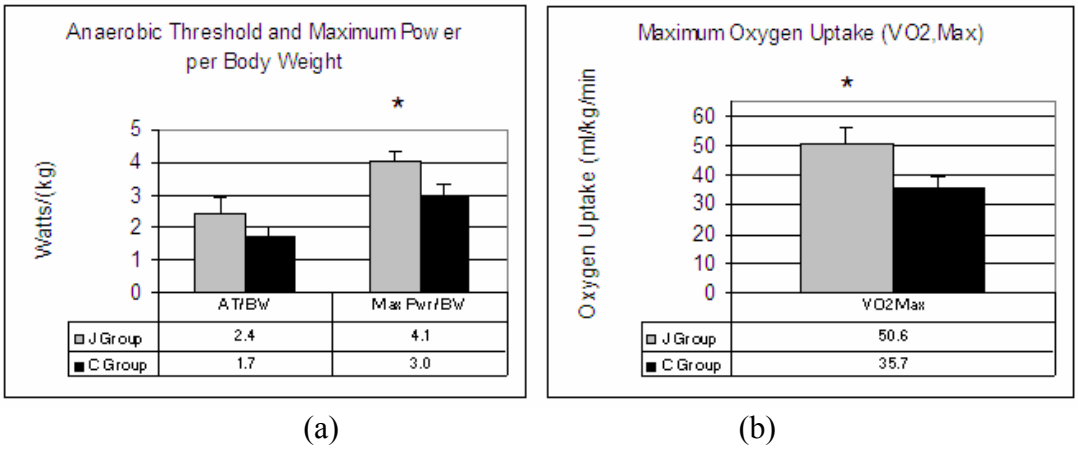
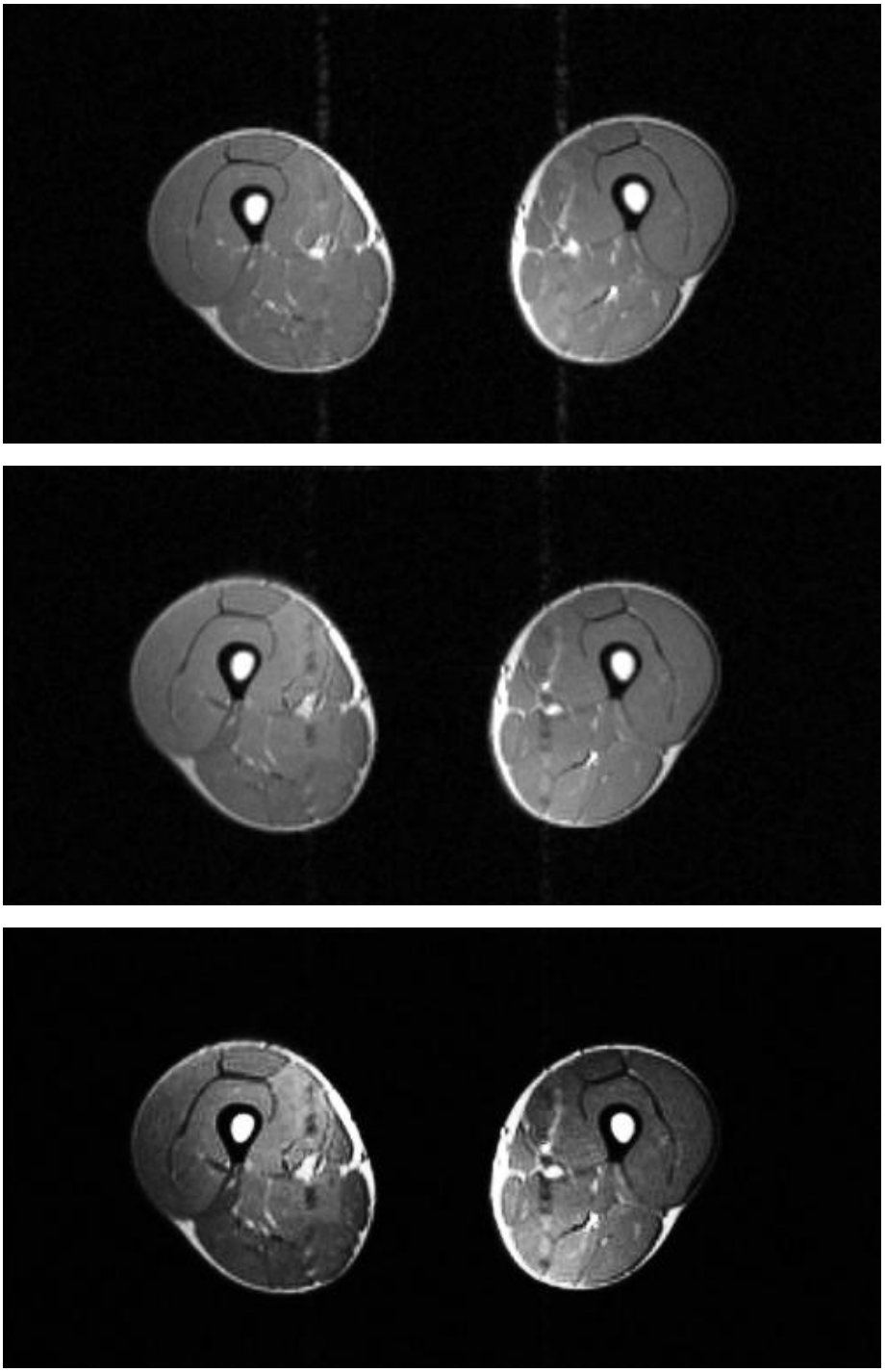


Figure 11 - Maximal exercise test results: Anaerobic threshold and maximum aerobic power per body mass (a), and maximum oxygen uptake (b). (J>C: * = p<0.05, ** = p<0.01).



(a)

(b)

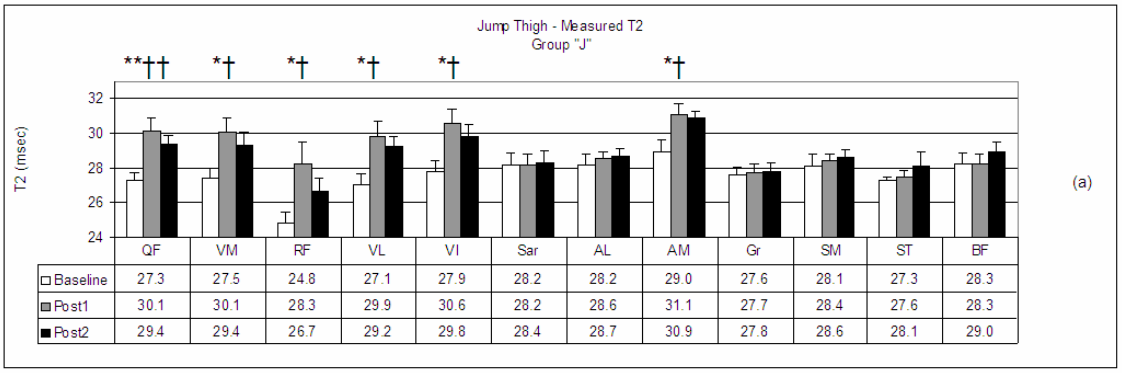
(c)

Figure 12 -Typical MR images of the thighs: Thighs at Baseline (a) and at Post1 without (b) and with (c) gray scale contrast enhancement to highlight activation differences among muscles. The jumping leg is at the left.

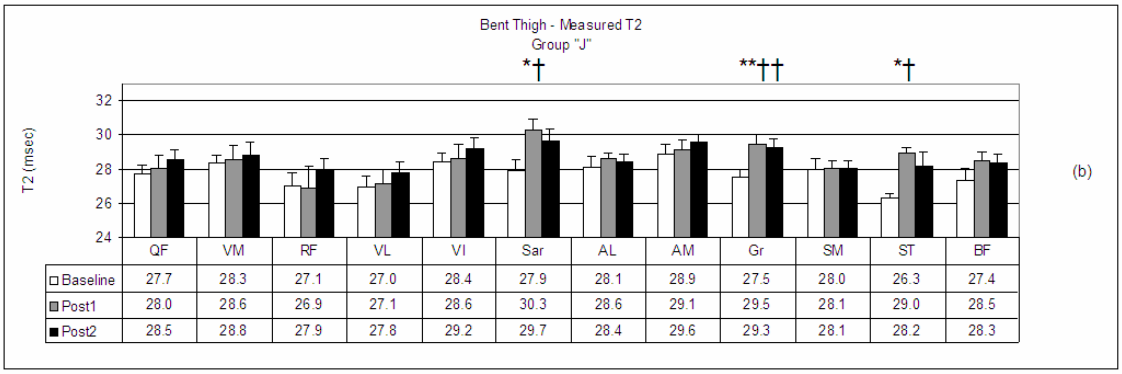
T_2 elevation with exercise: Representative T_2-weighted spin echo MR images of the thigh and calf at Baseline and at Post1 appear in Figure 12**.** Muscle activation with may be indicated by T_2 response to exercise in one three different ways: absolute T_2 values, percentage increase in T_2 or the percentage of pixels in the T_2 image that elevate post-exercise.

Measured T_2 elevation: Whole-muscle T_2 measurements at Baseline were measured within a range of 24 to 31 msec. Figure 13 and Figure 14 contain measured T_2 values for the J group thigh and calf, respectively, at Baseline, Post1 and Post2. Figure 15 and Figure 16 contain measured T_2 values for the C group thigh and calf, respectively, at Baseline, Post1 and Post2. The C group presented slightly higher Baseline T_2 values than the J Group in almost every muscle of both legs, although the difference was statistically significant ($p<0.05$) only in the Sar and Gr of each leg. After exercise, T_2 values exceeding 35 msec were measured in some cases. Both the J and C groups demonstrated significant changes in T_2 for specific muscles at both Post1 and Post2. In the thigh, significant T_2 changes ($p<0.05$) were seen primarily in the knee extensors and adductors of the jumping thigh (QF, VM, RF, VL, VI and AM) and the *sartorius* and knee flexors of the bent thigh (Sar, GR, and ST). However, C Group jumpers also showed significant T_2 increases ($p<0.05$) in the BF of both legs. In the calf, J Group jumpers exhibited no significant differences from Baseline at Post1 or Post2. However, nearly all jumping calf muscles trended toward higher T_2 in the J Group. Comparatively, C Group jumpers exhibited significant T_2 elevation ($p<0.05$) in the FDL, TP and PL muscles of the jumping calf, with an upward trend in nearly all of the remaining calf muscles of the jumping leg. There were no significant differences in the bent leg calf

muscles of either group. Furthermore, in no cases were the differences in T_2 measured at Post1 vs. Post2 significant in either thigh or calf of either group. There were no significant differences in pixel T_2 variances between times (Baseline, Post1, Post2) or groups (J, C).



	QF	VM	RF	VL	VI	Sar	AL	AM	Gr	SM	ST	BF
Baseline	27.3	27.5	24.8	27.1	27.9	28.2	28.2	29.0	27.6	28.1	27.3	28.3
Post1	30.1	30.1	28.3	29.9	30.6	28.2	28.6	31.1	27.7	28.4	27.6	28.3
Post2	29.4	29.4	26.7	29.2	29.8	28.4	28.7	30.9	27.8	28.6	28.1	29.0



	QF	VM	RF	VL	VI	Sar	AL	AM	Gr	SM	ST	BF
Baseline	27.7	28.3	27.1	27.0	28.4	27.9	28.1	28.9	27.5	28.0	26.3	27.4
Post1	28.0	28.6	26.9	27.1	28.6	30.3	28.6	29.1	29.5	28.1	29.0	28.5
Post2	28.5	28.8	27.9	27.8	29.2	29.7	28.4	29.6	29.3	28.1	28.2	28.3

Figure 13 - Measured T_2 values in the thigh muscles of the J Group at Baseline (white), Post1 (gray) Post2 (black) for the jump thigh (a) and bent thigh (b). (Post1>Baseline: * = $p<0.05$, ** = $p<0.01$; Post2>Baseline: † = $p<0.05$, †† = $p<0.01$; Post2>Post1: ‡ = $p<0.05$, ‡‡ = $p<0.01$)

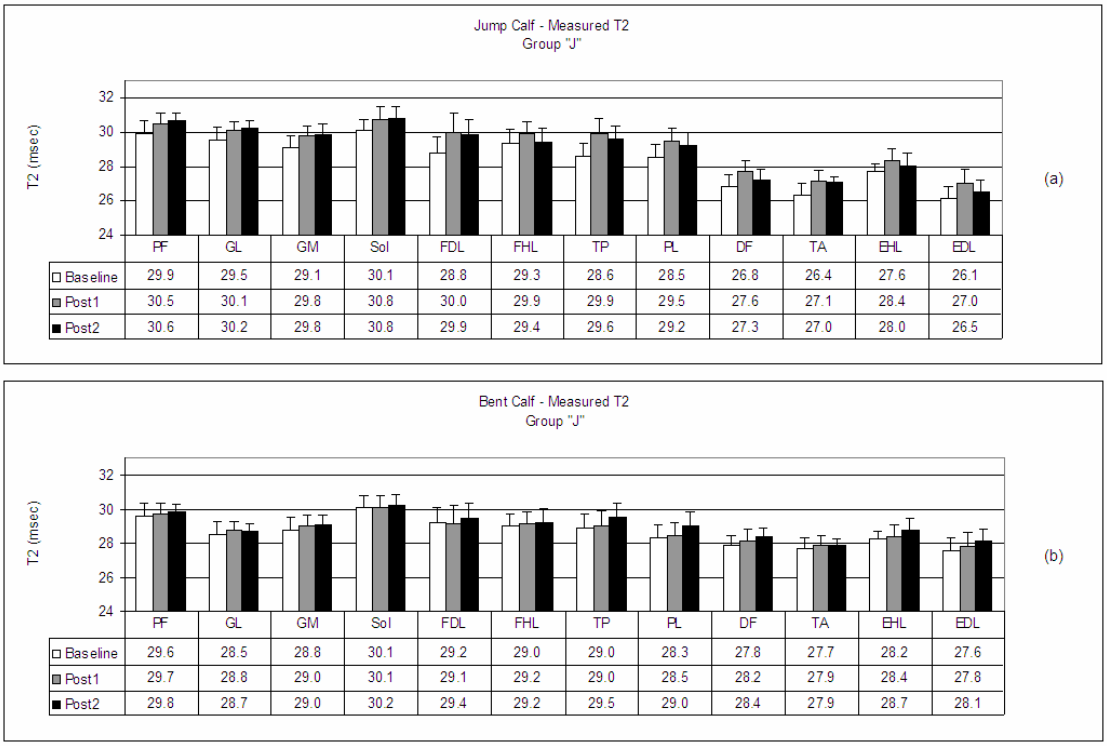
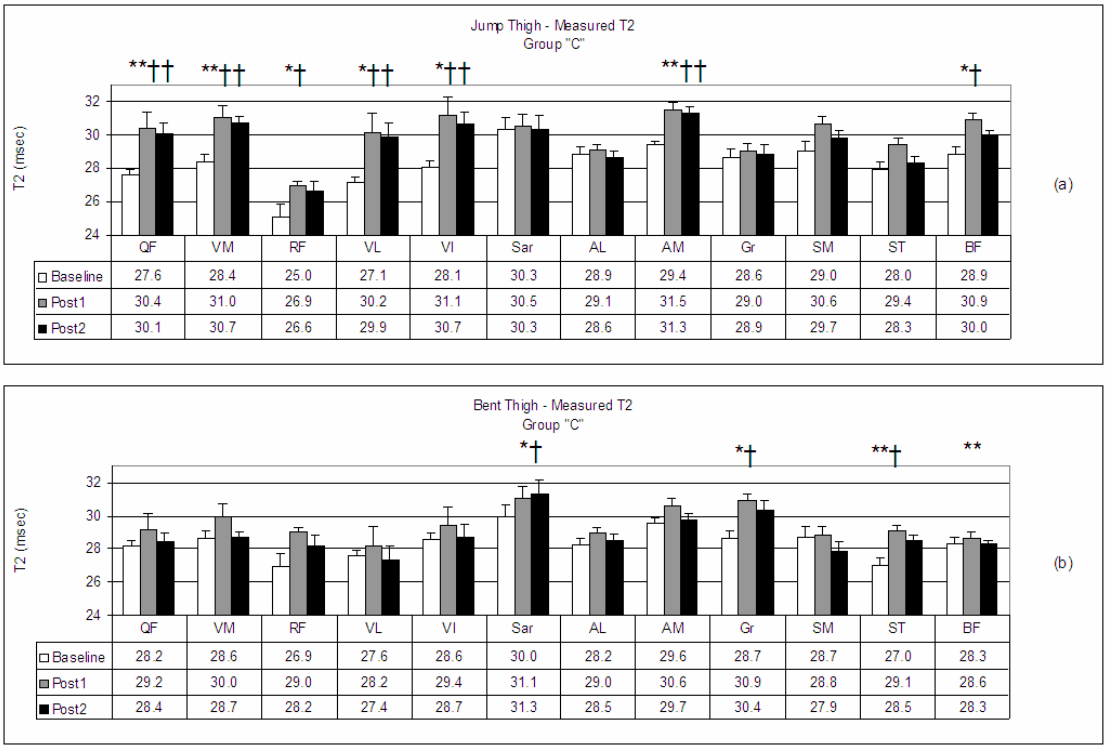


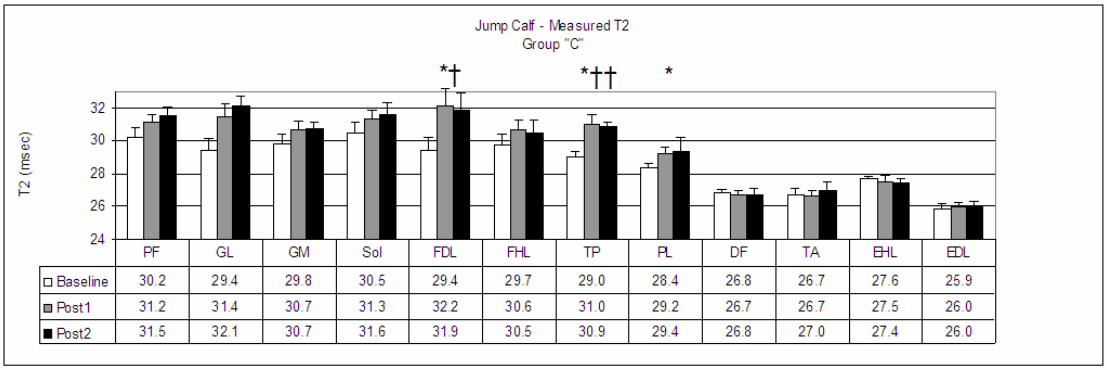
Figure 14 - Measured T_2 values in the calf muscles of the J Group at Baseline (white), Post1 (gray) Post2 (black) for the jump calf (a) and bent calf (b).
No significant differences were observed at Post1 or Post2 vs. Baseline.



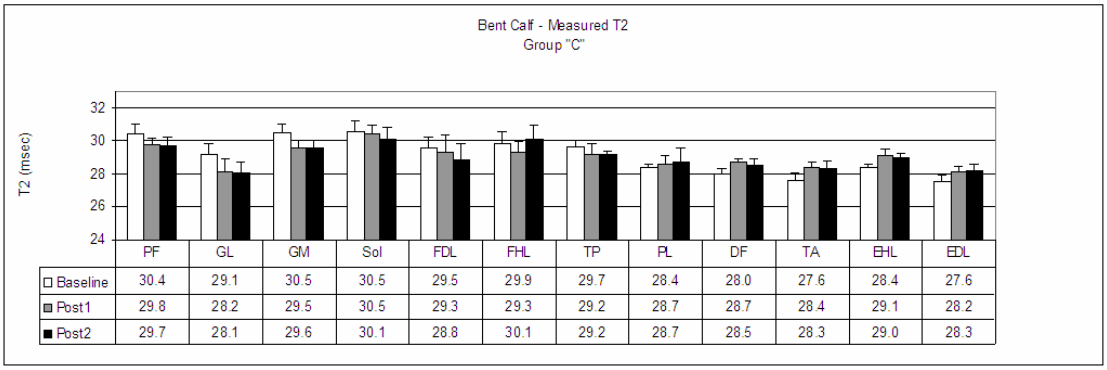
(a)	QF	VM	RF	VL	VI	Sar	AL	AM	Gr	SM	ST	BF
Baseline	27.6	28.4	25.0	27.1	28.1	30.3	28.9	29.4	28.6	29.0	28.0	28.9
Post1	30.4	31.0	26.9	30.2	31.1	30.5	29.1	31.5	29.0	30.6	29.4	30.9
Post2	30.1	30.7	26.6	29.9	30.7	30.3	28.6	31.3	28.9	29.7	28.3	30.0

(b)	QF	VM	RF	VL	VI	Sar	AL	AM	Gr	SM	ST	BF
Baseline	28.2	28.6	26.9	27.6	28.6	30.0	28.2	29.6	28.7	28.7	27.0	28.3
Post1	29.2	30.0	29.0	28.2	29.4	31.1	29.0	30.6	30.9	28.8	29.1	28.6
Post2	28.4	28.7	28.2	27.4	28.7	31.3	28.5	29.7	30.4	27.9	28.5	28.3

Figure 15 - Measured T_2 values in the thigh muscles of the C Group at Baseline (white), Post1 (gray) Post2 (black) for the jump thigh (a) and bent thigh (b). (Post1>Baseline: * = p<0.05, ** = p<0.01; Post2>Baseline: † = p<0.05, †† = p<0.01; Post2>Post1: ‡ = p<0.05, ‡‡ = p<0.01)



	PF	GL	GM	Sol	FDL	FHL	TP	PL	DF	TA	EHL	EDL
Baseline	30.2	29.4	29.8	30.5	29.4	29.7	29.0	28.4	26.8	26.7	27.6	25.9
Post1	31.2	31.4	30.7	31.3	32.2	30.6	31.0	29.2	26.7	26.7	27.5	26.0
Post2	31.5	32.1	30.7	31.6	31.9	30.5	30.9	29.4	26.8	27.0	27.4	26.0



	PF	GL	GM	Sol	FDL	FHL	TP	PL	DF	TA	EHL	EDL
Baseline	30.4	29.1	30.5	30.5	29.5	29.9	29.7	28.4	28.0	27.6	28.4	27.6
Post1	29.8	28.2	29.5	30.5	29.3	29.3	29.2	28.7	28.7	28.4	29.1	28.2
Post2	29.7	28.1	29.6	30.1	28.8	30.1	29.2	28.7	28.5	28.3	29.0	28.3

Figure 16 - Measured T_2 values in the calf muscles of the C Group at Baseline (white), Post1 (gray) Post2 (black) for the jump calf (a) and bent calf (b). (Post1>Baseline: * = $p<0.05$, ** = $p<0.01$; Post2>Baseline: † = $p<0.05$, †† = $p<0.01$; Post2>Post1: ‡ = $p<0.05$, ‡‡ = $p<0.01$)

Measured Delta T_2: Figure 17 and Figure 18 contain thigh and calf T_2 elevation (i.e., delta T_2) values, respectively, taken at Post1 and Post2 for the J Group. Figure 19 and Figure 20 contain similar thigh and calf values, respectively, for the C Group. No significant differences in delta T_2 at Post2 and Post1 were exhibited by either group, although most muscles actually showed more elevation of T_2 at Post1 than Post2. Even though these differences were not statistically significant, this is a notable and quite unexpected result given the design intent of the study. The highest delta T_2 values (between 1.8 and 3.5 msec) were observed in the knee extensor and adductors of the jumping leg and the ST muscles of the bent leg in both groups.

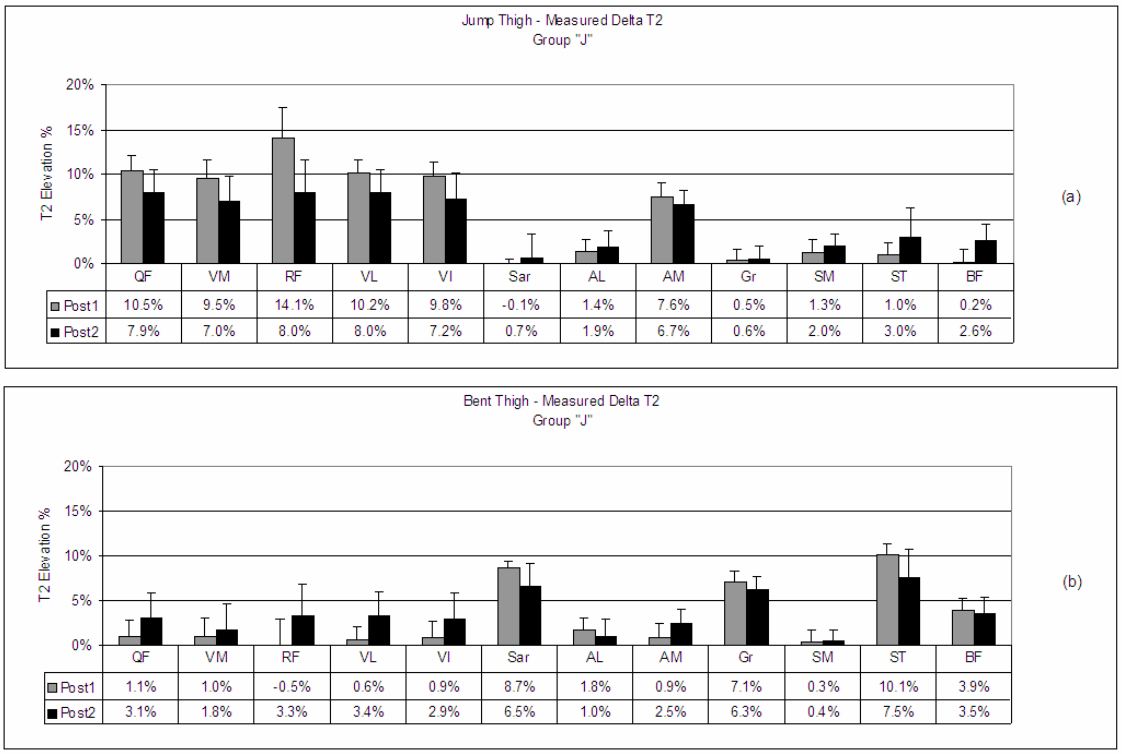


Figure 17 - Measured change in T_2 in the thigh muscles of the J Group from Baseline to Post1 (gray) and Baseline to Post2 (black) for the jump thigh (a) and bent thigh (b). No significant differences were observed, although measured delta T_2 values were actually higher in agonist muscles at Post1 than Post2.

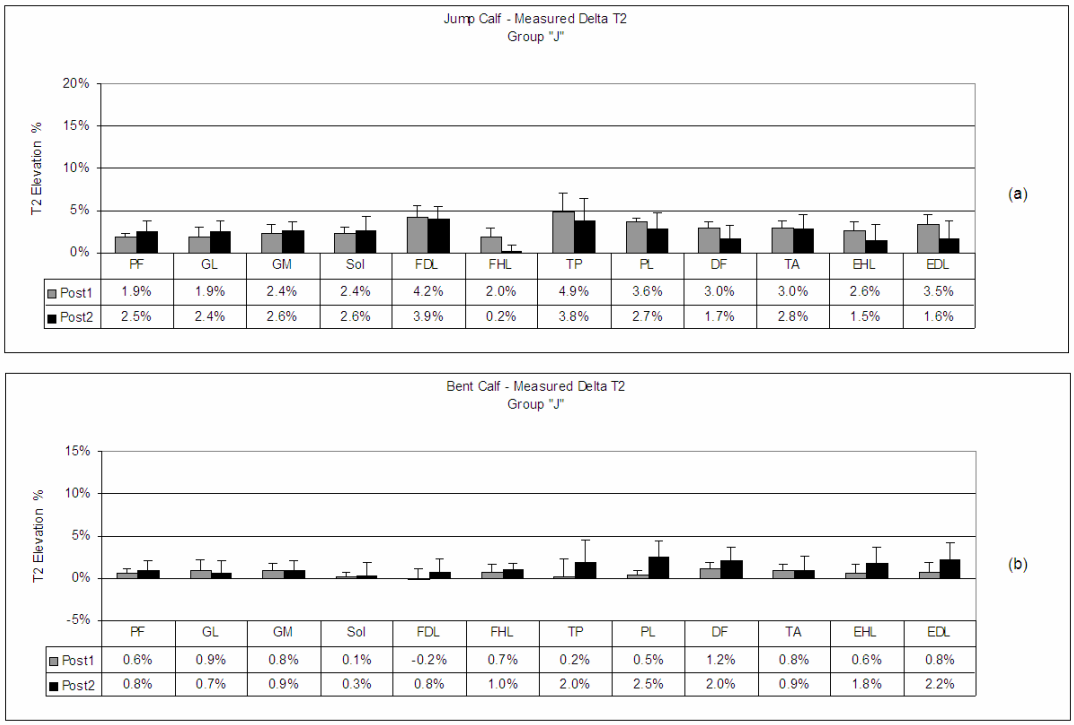


Figure 18 - Measured change in T_2 in the calf muscles of the J Group from Baseline to Post1 (gray) and Baseline to Post2 (black) for the jump calf (a) and calf thigh (b). No significant differences were found at Post1 vs. Post2.

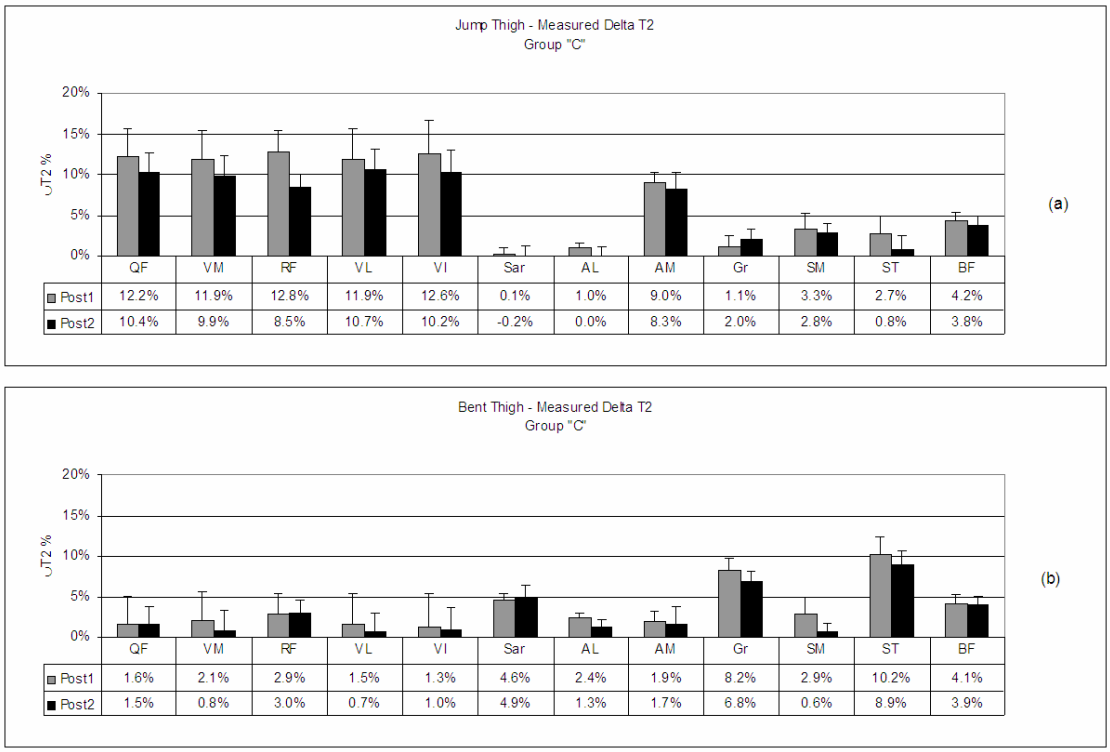


Figure 19 - Measured change in T2 in the thigh muscles of the C Group from Baseline to Post1 (gray) and Baseline to Post2 (black) in the jump thigh (a) and bent thigh (b). No significant differences were found at Post1 vs. Post2.

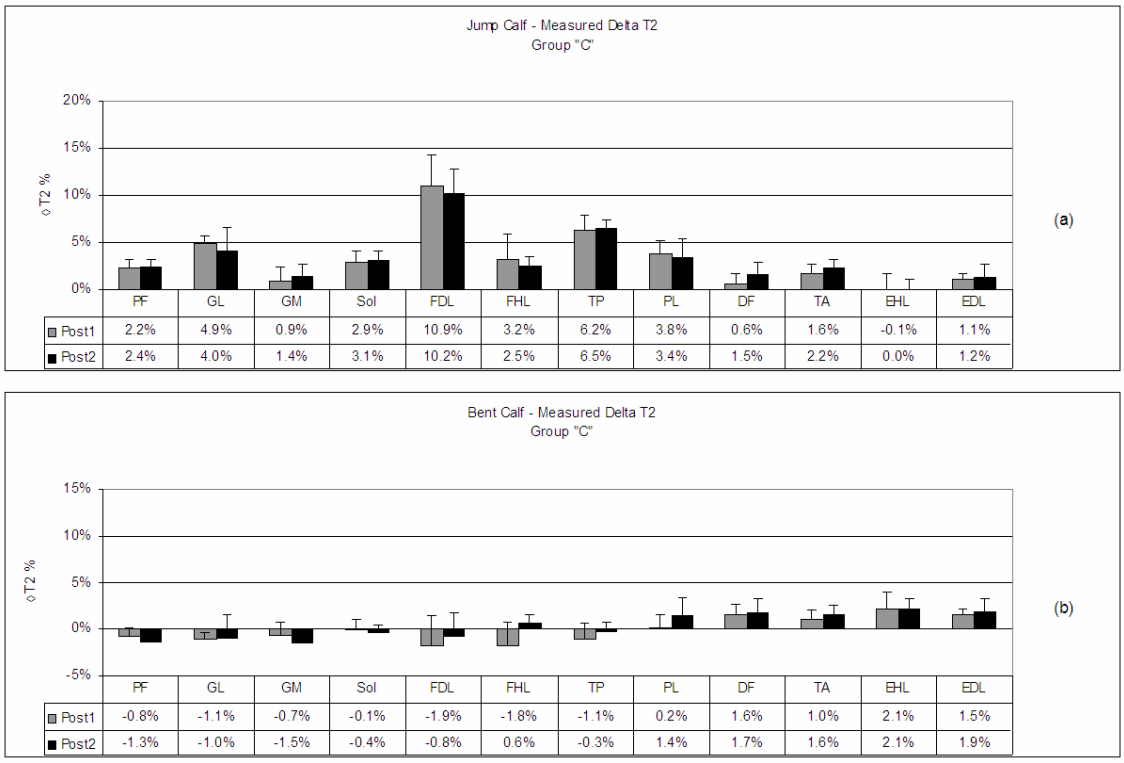
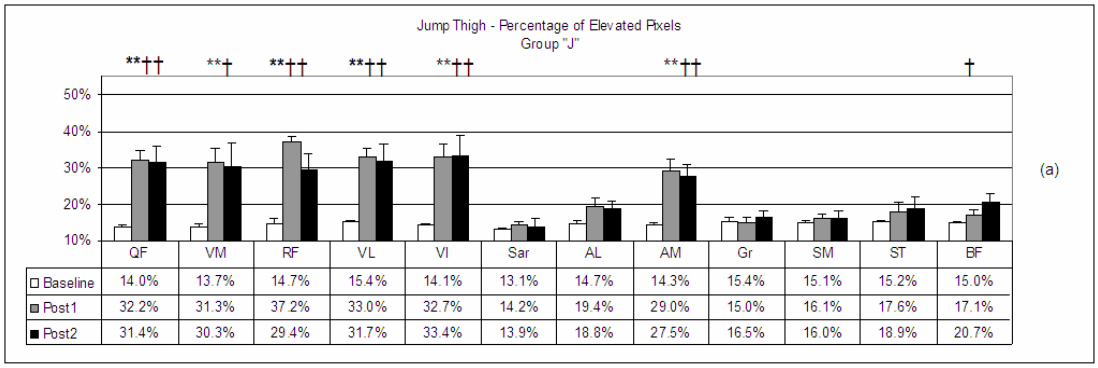


Figure 20 - Measured change in T2 in the calf muscles of the C Group from Baseline to Post1 (gray) and Baseline to Post2 (black) in the jump calf (a) and the bent calf (b). No significant differences were found at Post1 vs. Post2.

Percentage of pixels with elevated T_2 values: Figure 21 and Figure 22 contain thigh and calf values, respectively for the percentage of muscle pixels exhibiting elevated T_2 values in the J Group, and Figure 23 and Figure 24 contain similar thigh and calf results, respectively, for the C Group. Nearly all muscles of the thigh and calf contained between 13-17% elevated pixels at Baseline. For each muscle in both legs, the Baseline percentage of elevated pixels agreed within 2%, although even smaller differences were statistically significant ($p<0.05$) in the Sar, SM, TA and EDL of the jumping leg. The percentage of elevated pixels rose to over 40% in some cases for specific muscles following exercise. In the thigh, significant T_2 changes ($p<0.05$) were seen primarily in the knee extensors, adductors and knee flexors of the jumping thigh (QF, VM, RF, VL, VI, AM and BF) and the knee extensors, *sartorius* and knee flexors of the bent thigh (RF, VM, Sar, GR, ST and BF). In the calf, J Group jumpers exhibited significant differences in PF, GM, Sol, FDL, FHL, TP, PL , DF and EDL) from Baseline at Post1 or Post2. This is in contrast to the direct measurement of T_2 in these muscles where no significant differences were found. However, none of these differences in the percentage of elevated pixels exceeded a 9% increase from Baseline in the J Group at either time. All jumping calf muscles trended toward a small but higher percentage of elevated pixels in the J Group after exercise. Comparatively, C Group jumpers exhibited significant T_2 elevation ($p<0.05$) in the PF, GL, Sol, FDL, TP and PL muscles of the jumping calf, also with an upward trend in nearly all of the remaining calf muscles of the jumping leg. Measured differences in the FDL and TP were roughly 30% and 20%, respectively, at both Post 1 and Post2 in the C Group. Much of this effect was attributed to a very high level of T_2 response in one individual subject. The better agreement at Baseline between the J and C

groups for this metric made it a more robust choice for computing the performance to activation ratios.

Analysis of all jumpers as an aggregate group revealed that specific muscles displayed moderate correlations between jumping performance and activation. Activation of the QF, VM, VI, Sol, FDL and TP correlated negatively with average power production per kg of body weight (r=-0.59, r=-0.64, r=-0.58, r=-0.61, r=-0.67, r=-0.65, respectively, p<0.05) and activation of the RF correlated negatively with peak jump height (r=-0.62, p<0.05). Activation of the GM correlated positively with the force time integral ((r=+0.60, p<0.05).



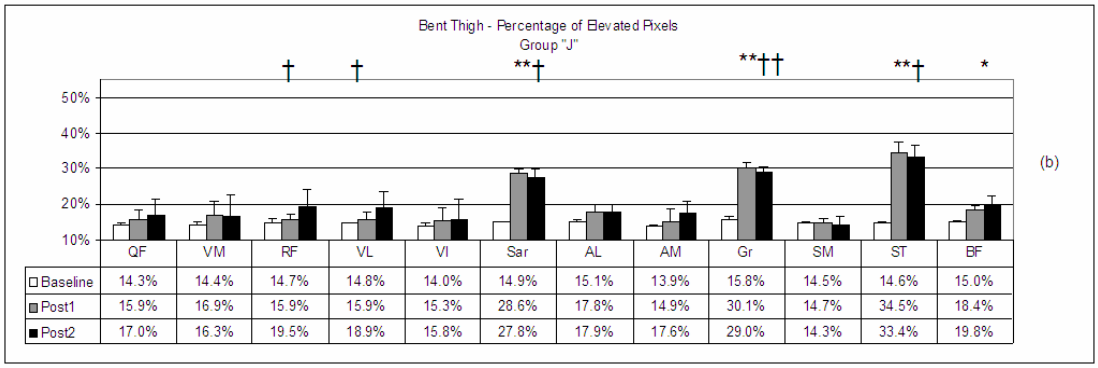
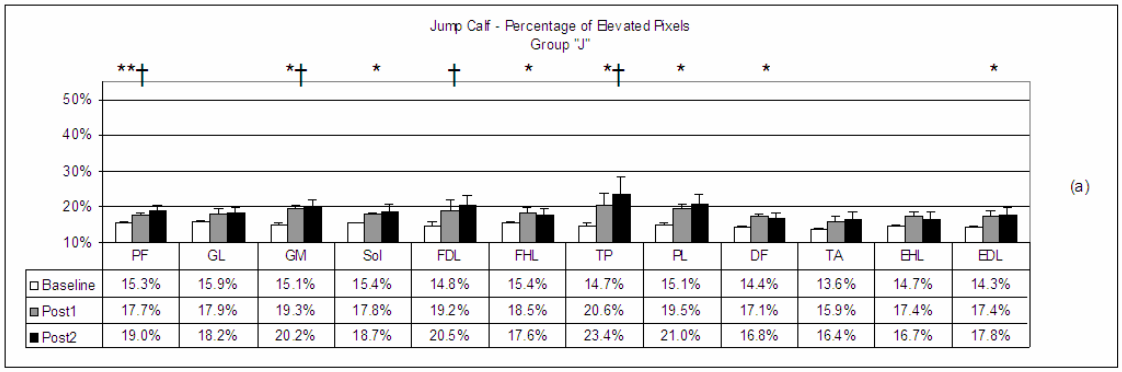
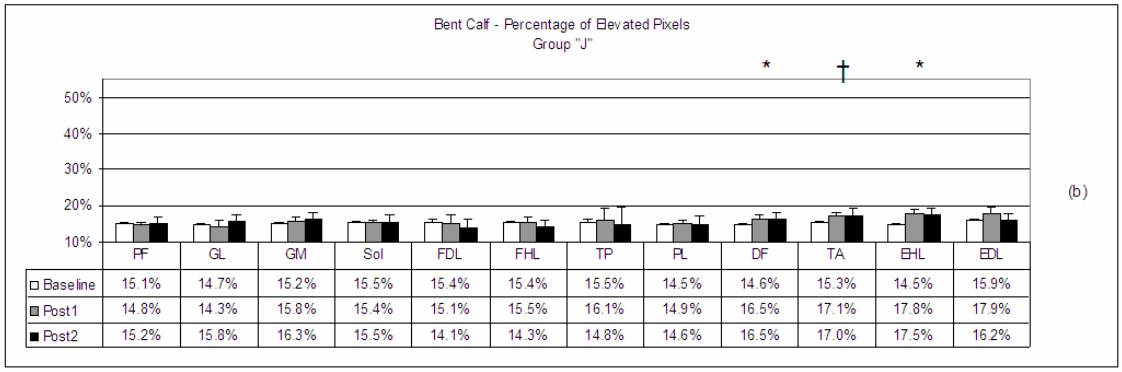


Figure 21 - Measured percentage of pixels exhibiting elevated T2 in the thigh muscles of the J Group at Baseline (white), Post1 (gray) and Post2 (black) for the jump thigh (a) and bent thigh (b). (Post1>Baseline: * = $p<0.05$, ** = $p<0.01$; Post2>Baseline: † = $p<0.05$, †† = $p<0.01$; Post2>Post1: ‡ = $p<0.05$, ‡‡ = $p<0.01$)



	PF	GL	GM	Sol	FDL	FHL	TP	PL	DF	TA	EHL	EDL
Baseline	15.3%	15.9%	15.1%	15.4%	14.8%	15.4%	14.7%	15.1%	14.4%	13.6%	14.7%	14.3%
Post1	17.7%	17.9%	19.3%	17.8%	19.2%	18.5%	20.6%	19.5%	17.1%	15.9%	17.4%	17.4%
Post2	19.0%	18.2%	20.2%	18.7%	20.5%	17.6%	23.4%	21.0%	16.8%	16.4%	16.7%	17.8%



	PF	GL	GM	Sol	FDL	FHL	TP	PL	DF	TA	EHL	EDL
Baseline	15.1%	14.7%	15.2%	15.5%	15.4%	15.4%	15.5%	14.5%	14.6%	15.3%	14.5%	15.9%
Post1	14.8%	14.3%	15.8%	15.4%	15.1%	15.5%	16.1%	14.9%	16.5%	17.1%	17.8%	17.9%
Post2	15.2%	15.8%	16.3%	15.5%	14.1%	14.3%	14.8%	14.6%	16.5%	17.0%	17.5%	16.2%

Figure 22 - Measured percentage of pixels exhibiting elevated T2 in the calf muscles of the J Group at Baseline (white), Post1 (gray) and Post2 (black) for the jump calf (a) and bent calf (b). (Post1>Baseline: * = $p<0.05$, ** = $p<0.01$; Post2>Baseline: † = $p<0.05$, †† = $p<0.01$; Post2>Post1: ‡ = $p<0.05$, ‡‡ = $p<0.01$)

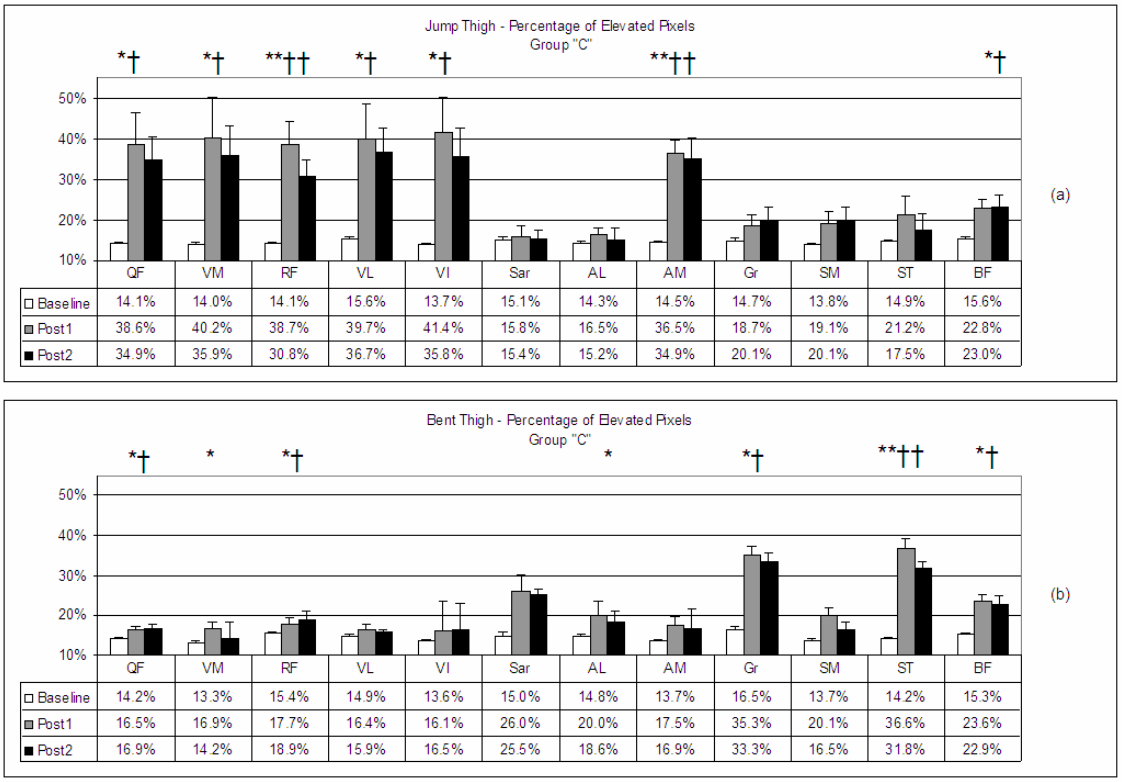
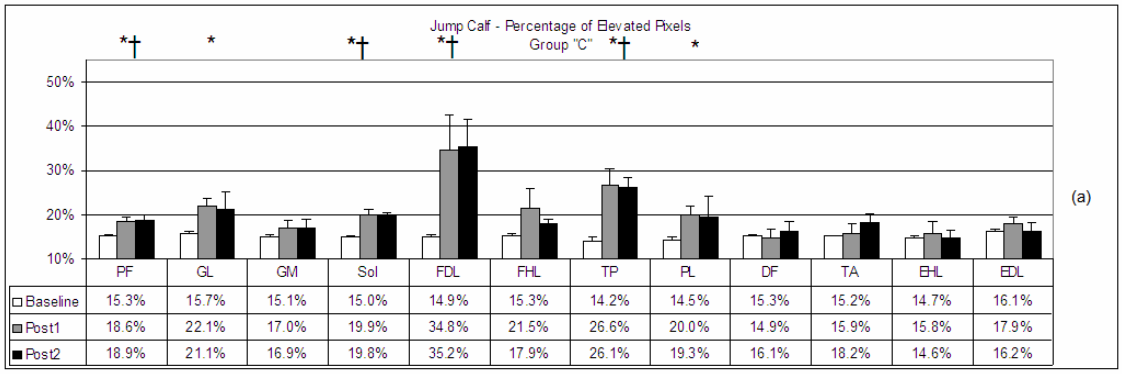
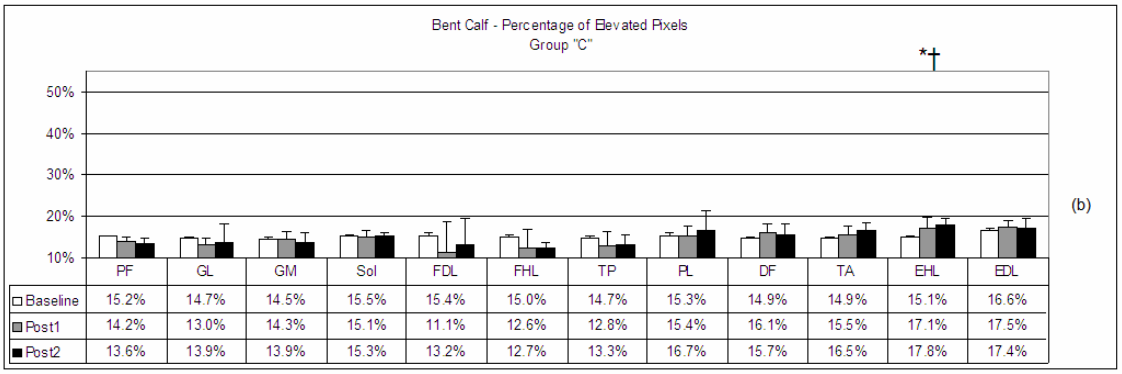


Figure 23 - Measured percentage of pixels exhibiting elevated T2 in the thigh muscles of the C Group at Baseline (white), Post1 (gray) and Post2 (black) for the jump thigh (a) and bent thigh (b). (Post1>Baseline: * = $p<0.05$, ** = $p<0.01$; Post2>Baseline: † = $p<0.05$, †† = $p<0.01$; Post2>Post1: ‡ = $p<0.05$, ‡‡ = $p<0.01$)



	PF	GL	GM	Sol	FDL	FHL	TP	PL	DF	TA	EHL	EDL
Baseline	15.3%	15.7%	15.1%	15.0%	14.9%	15.3%	14.2%	14.5%	15.3%	15.2%	14.7%	16.1%
Post1	18.6%	22.1%	17.0%	19.9%	34.8%	21.5%	26.6%	20.0%	14.9%	15.9%	15.8%	17.9%
Post2	18.9%	21.1%	16.9%	19.8%	35.2%	17.9%	26.1%	19.3%	16.1%	18.2%	14.6%	16.2%



	PF	GL	GM	Sol	FDL	FHL	TP	PL	DF	TA	EHL	EDL
Baseline	15.2%	14.7%	14.5%	15.5%	15.4%	15.0%	14.7%	15.3%	14.9%	14.9%	15.1%	16.6%
Post1	14.2%	13.0%	14.3%	15.1%	11.1%	12.6%	12.8%	15.4%	16.1%	15.5%	17.1%	17.5%
Post2	13.6%	13.9%	13.9%	15.3%	13.2%	12.7%	13.3%	16.7%	15.7%	16.5%	17.8%	17.4%

Figure 24 - Measured percentage of pixels exhibiting elevated T2 in the calf muscles of the C Group at Baseline (white), Post1 (gray) and Post2 (black) for the jump calf (a) and bent calf (b). (Post1>Baseline: * = p<0.05, ** = p<0.01; Post2>Baseline: † = p<0.05, †† = p<0.01; Post2>Post1: ‡ = p<0.05, ‡‡ = p<0.01)

Performance to Activation Ratios: Figure 25 and Figure 26 contain each jumping leg muscle's average performance to activation ratio (APAR) for both the J and C Groups at Post1 and Post2, respectively. Figure 27 and Figure 28 contain each jumping leg muscle's peak performance to activation ratio (PPAR) for both the J and C Groups at Post1 and Post2, respectively.

At Post1 differences in APAR scores between groups were significantly higher in the J Group (p<0.05) in the AM, Gr, BF, PF, GL, FDL, and TP. The J Group also trended (p<0.2) toward superior APAR scores in most all leg muscles, except for the RF, Sar, AL, GM, DF and EHL. APAR differences were highest in magnitude in the knee flexors (Gr, SM and BF) of the thigh and the GL, FDL and TP of the calf. Four muscle groups showed statistically higher PPAR scores (p<0.05) in the J Group at Post1 (AM, BF, FDL, and TP). The J Group also trended (p<0.2) toward superior Post1 PPAR scores in most all other leg muscles, except for the RF, Sar, AL, GM, DF and EHL. Differences were highest in magnitude in the Gr, ST and BF of the thigh and the GL, FDL and TP of the calf.

At Post2 the J Group APAR scores were significantly higher (p<0.05) in the Sar, Sol, FDL and FHL. For the FDL the difference was particularly significant (p=0.003). APAR scores for QF, VM, RF, VL, VI, AM, Gr, SM, BF, PF, TP and TA still trended higher (p<0.2) in the J Group. APAR differences were highest in magnitude in the Sar, Gr, and SM of the thigh and the FDL, TA and TP of the calf. PPAR scores were significantly higher (p<0.05) in the Sar, AM, SM, Sol, FDL, FHL and TP. Again, for the FDL the difference was particularly significant (p=0.002). The QF, VM, RF, VL, VI, Gr, BF, PF, GL, and TA still trended toward higher PPAR (p<0.2) in the J Group. The

highest differences in PPAR magnitude were seen in the Sar, Gr and SM of the thigh and in the FDL, TP and TA of the calf.

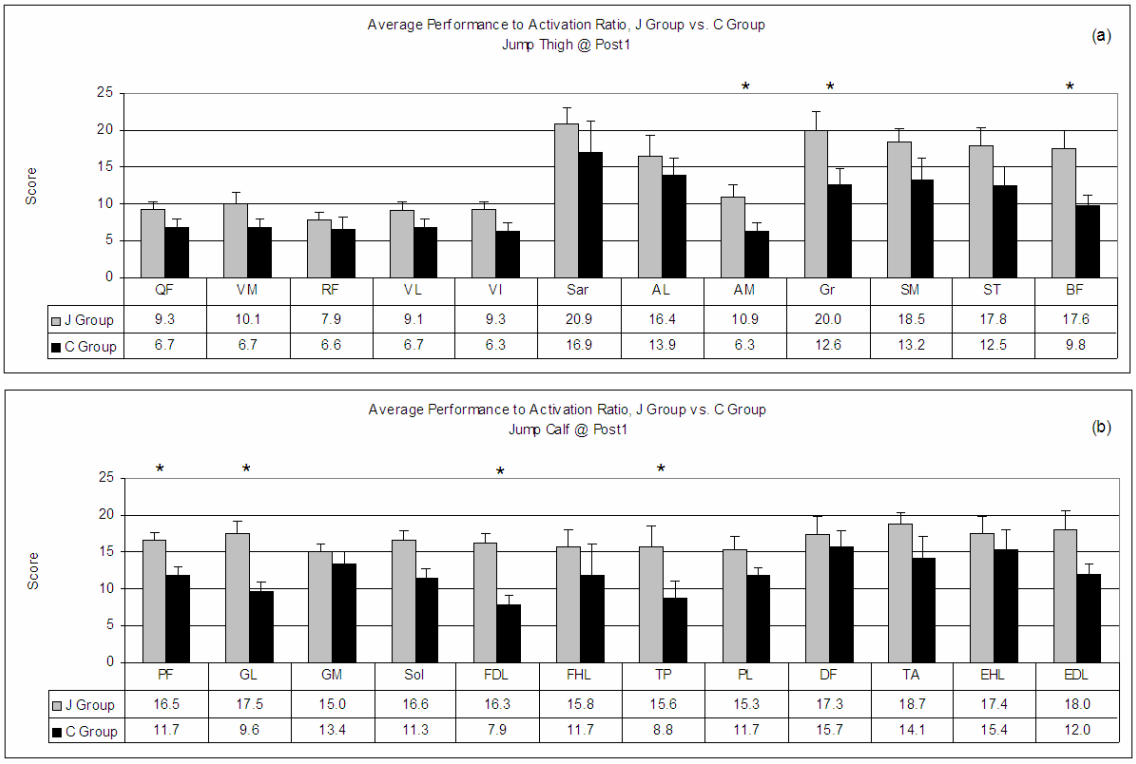


Figure 25 – Post1 average performance to activation ratio for J Group (gray) and C Group (black). (J>C: * = $p<0.05$, ** = $p<0.01$)

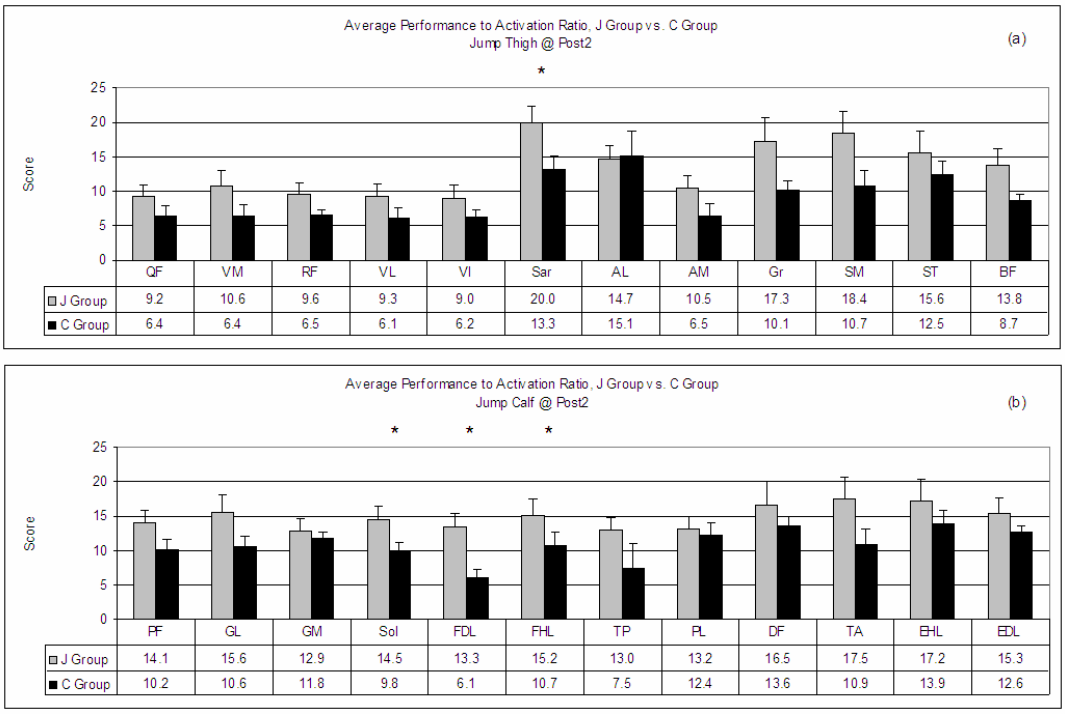


Figure 26 – Post2 average performance to activation ratio for J Group (gray) and C Group (black). (J>C: * = $p<0.05$, ** = $p<0.01$)

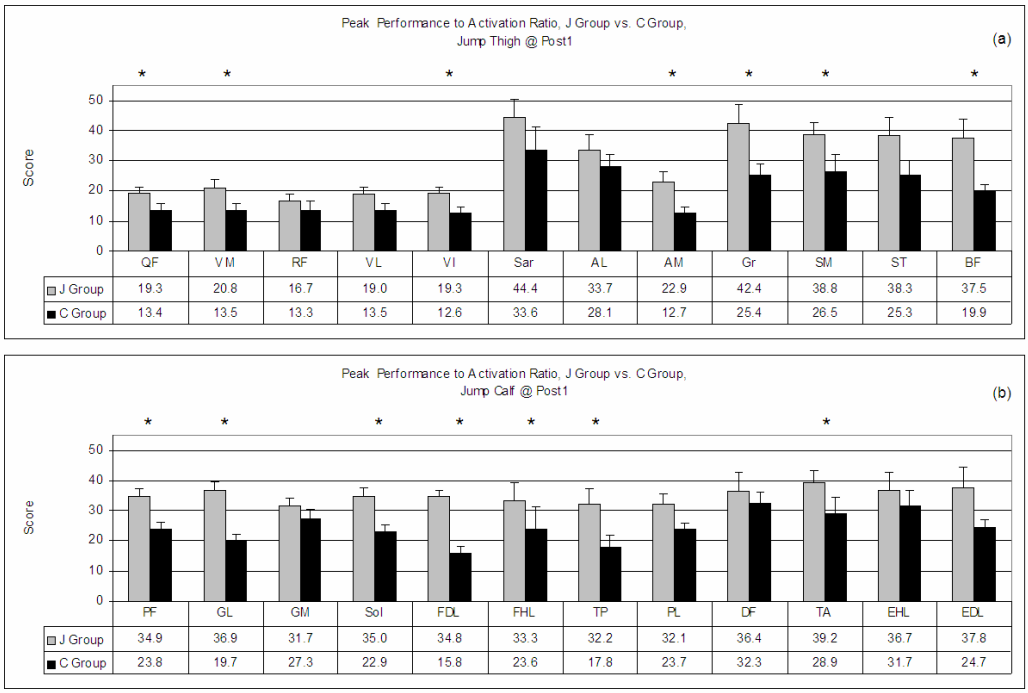


Figure 27 – Post1 peak performance to activation ratio for J Group (gray) and C Group (black). (J>C: * = $p<0.05$, ** = $p<0.01$)

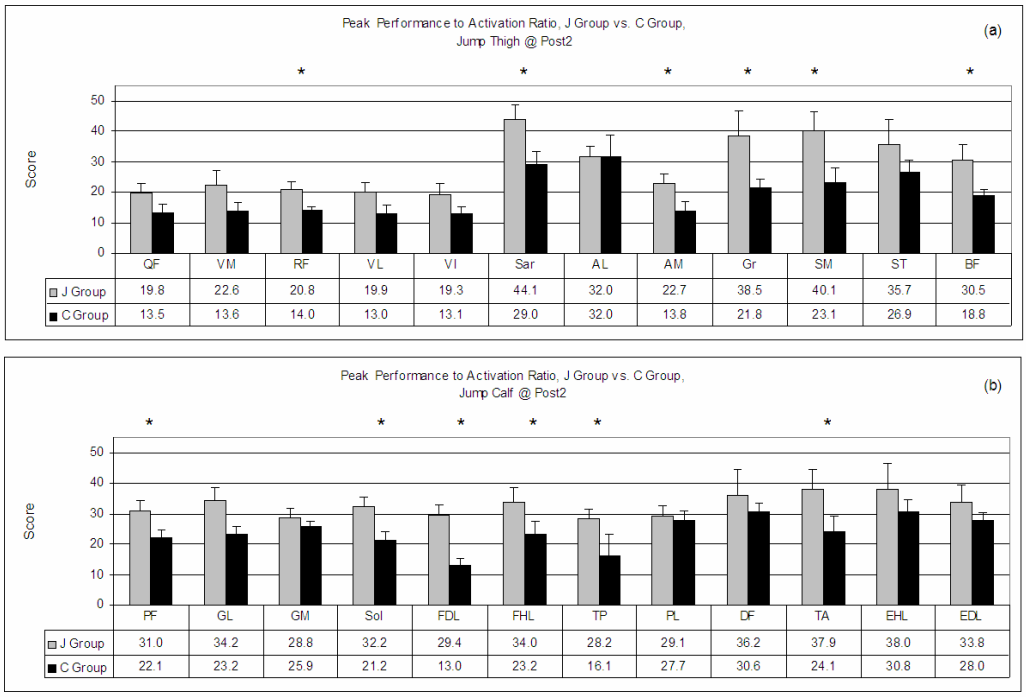
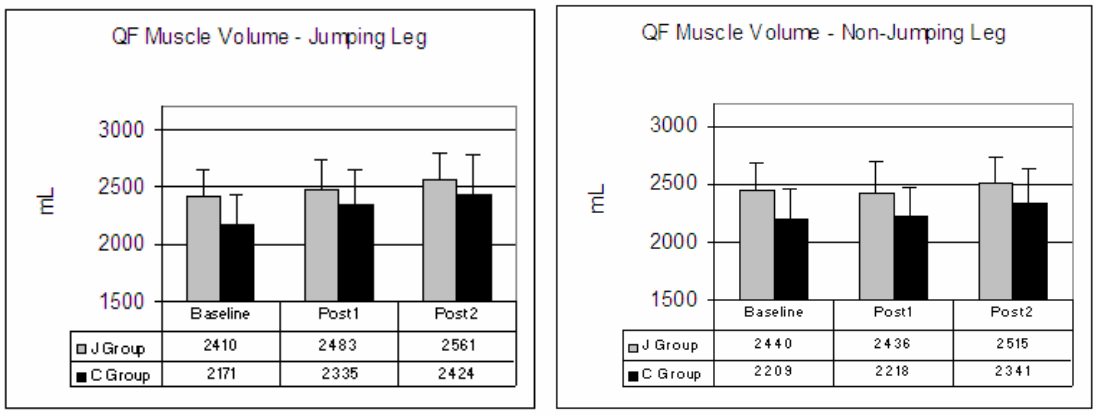


Figure 28 – Post2 peak performance to activation ratio for J Group (gray) and C Group (black) at Post2. (J>C: * = $p<0.05$, ** = $p<0.01$)

QF Muscle Volumes: Figure 29 contains the muscle volume data for both groups at Baseline, Post1 and Post2 for each leg. The J Group QF volume was higher at Baseline (2410 ± 245 mL vs. 2171 ± 249 mL) than C Group, but the difference was not statistically significant. QF volume at Post1 and Post2 trended higher versus Baseline in both groups (p>0.05), but the differences between the two groups remained non-significant at Post1 and Post2. QF activation and QF volume changes were only weakly correlated (Post1: r=0.41, p>0.05).



(a) (b)

Figure 29 - QF Muscle volumes for the J Group (gray) and C Group (black) at Baseline, Post1 and Post2 in the jumping leg (a) and bent leg (b). There were no significant differences at Post1 and Post2 vs. Baseline, no significant differences between Post1 vs. Post2, and no significant differences between groups at any time.

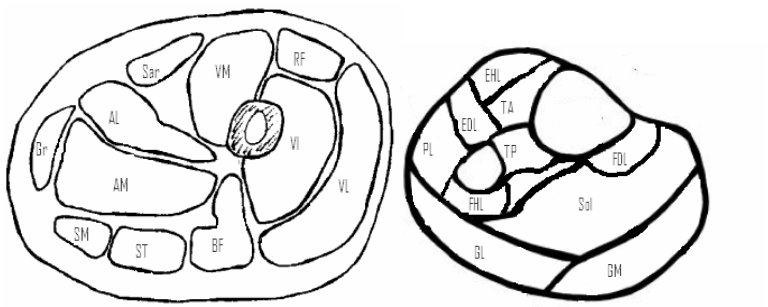
In summary, the results of this study indicate significant neuromuscular recruitment at both Post1 and Post2 in the primary agonist muscles (quadriceps, adductors, plantar flexors) as well as co-activating antagonists (hamstrings, dorsiflexors), as determined by T_2 elevation post-exercise. Better jumpers produced more jump power at lower absolute levels of T_2 elevation than their less able counterparts, even when the results were expressed with a performance to activation ration that accounted for the better jumpers' higher levels of aerobic conditioning. This implies greater recruitment

efficiency on the part of the better jumpers. This efficiency occurred in the agonist muscles, but even more so in the co-activating antagonist muscles. Neither jump power nor T_2 elevation changed significantly from Post1 to Post2 except in the hamstrings of the J Group. Quadriceps muscle volume increased at both Post1 and Post2, but this did not correlate with T_2 elevation.

Discussion

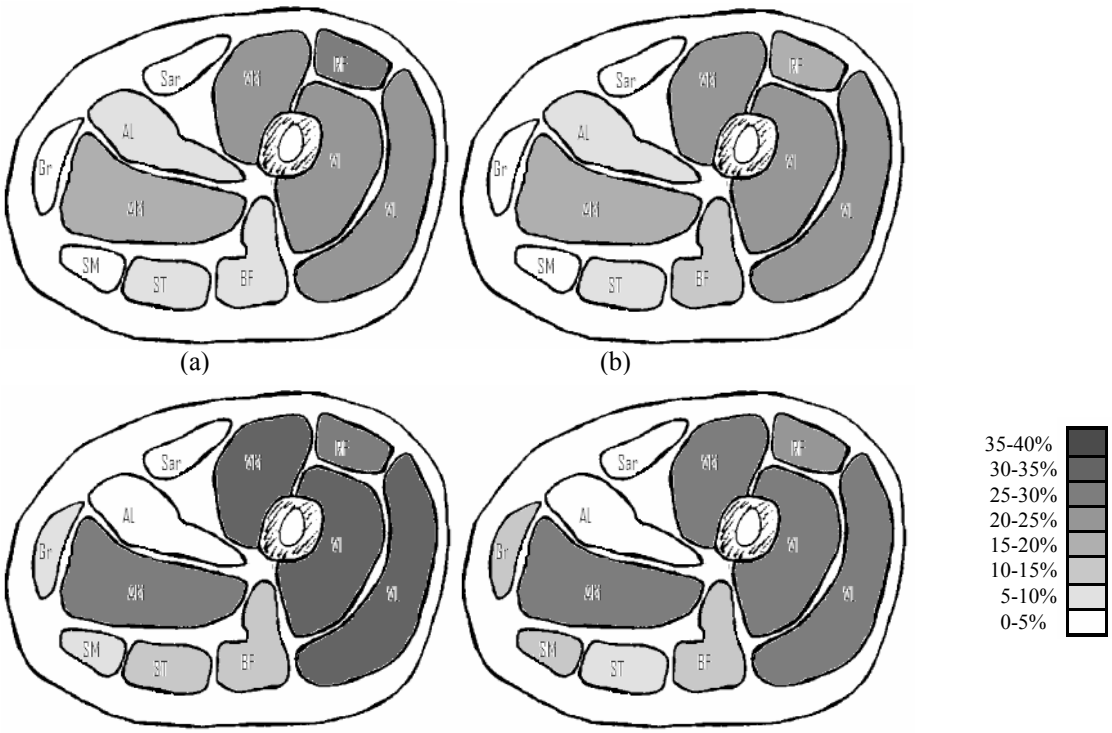
The present study represents the first T_2 mapping of muscle activation for single-leg vertical jumping exercise, and the first to use T_2 mapping to discriminate between activation patterns of two populations of jumpers with differing abilities. The primary finding was that better-performing jumpers demonstrated a higher degree of recruitment efficiency, allowing them to produce higher levels of jumping power at lower levels of muscle activation throughout all muscles of the lower limb. A significant component of this recruitment efficiency appeared to be the more effective suppression of co-contracting antagonist muscles, i.e., hamstrings in the thigh and dorsiflexors in the lower leg. The average and peak performance to activation ratios (APAR, PPAR) used in the present study suggest that this antagonist suppression played a more significant role in determining jump performance than did the higher recruitment of agonist muscles i.e., quadriceps, adductors and plantar flexors.

Recruitment maps: A useful tool for quickly discerning semi-quantitative recruitment differences in whole-leg musculature is a recruitment map (e.g., Kinugasa et al. 2003; Green & Wilson, 2000). By this method the gray scale coloring of an individual muscle on an axial slice template is made darker based on the rise in the percentage of pixels exhibiting T_2 elevation. Figure 30 contains the muscle templates with the individual muscles identified. Figure 31 illustrates the recruitment maps of the jumping thigh muscles of both groups after each exercise bout. Figure 32 illustrates the results for the jumping calf muscles of both groups after both bouts of jumping exercise.



(a) **(b)**

Figure 30 - Templates for recruitment maps of the thigh (a) and calf (b).



(a) (b)

(c) (d)

Figure 31 - Thigh muscle recruitment maps for the J Group at Post1 (a) and at Post2 (b) and for the C Group at Post1 (c) and at Post2 (d). Gray scale values represent percentage increases in elevated pixels from Baseline as indicated by the key at the far right.

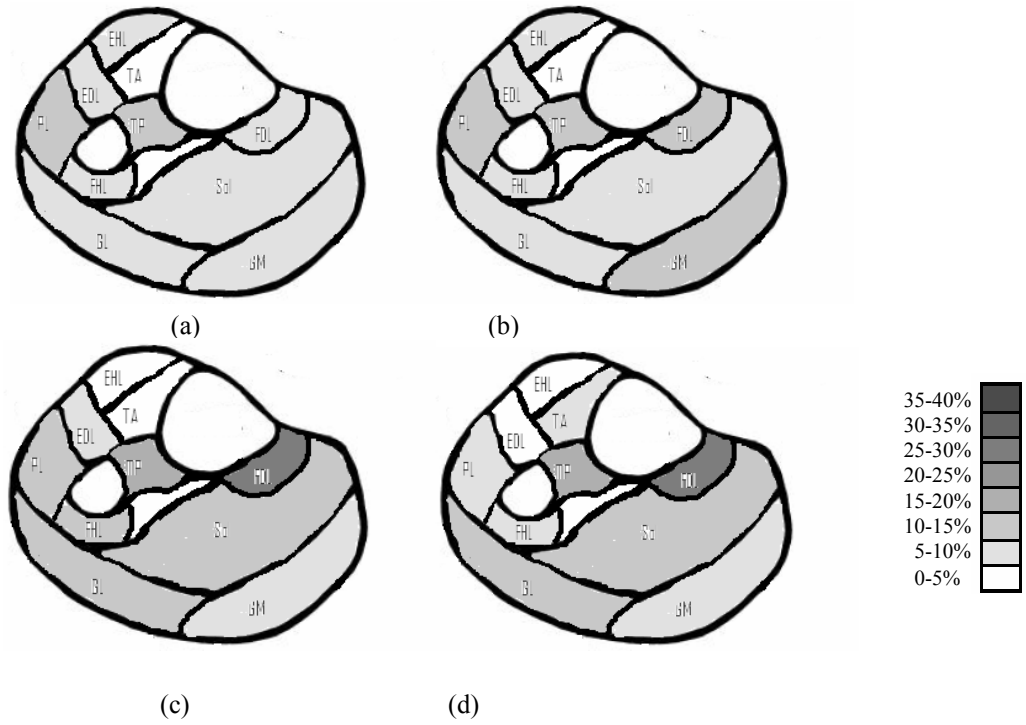


Figure 32 - Calf muscle recruitment maps for the J Group at Post1 (a) and at Post2 (b) and for the C Group at Post1 (c) and at Post (d). Gray scale values represent percentage increases in elevated pixels from Baseline as indicated by the key at the far right.

Using this tool, one may more easily identify recruitment differences. Despite producing less jumping power, the C Group presented higher recruitment levels in the three *vasti*, the AM and the hamstrings in the thigh, as well as the plantar flexors in the calf, most notably the FDL.

In a previous study, rowers of differing experience levels presented widely different activation maps at maximal exertion (Green & Wilson 2000). Like the present study, this is an expected result among subjects with varying levels of aptitude for a complex movement. The common feature was that more experienced rowers produced higher output power with generally lower levels of recruitment than the novice rowers.

However, even among a population of elite professional cyclists with similar performance there are significant differences in the recruitment of the individual lower limb muscles during both incremental and constant load pedaling exercise, as illustrated by both EMG and T_2 elevation (Hug et al. 2004). This occurred despite homogenously superior maximal power (range 438 to 516 Watts), aerobic fitness ($V_{O2,max}$ range: 67 to 82 ml kg^{-1} min^{-1}) and training behaviors among the subjects. The authors concluded that the nervous system has multiple ways of accomplishing a complex multi-joint task, even among elite performers.

A similar comparison (using T_2 elevation alone) among the six J Group jumpers of the present study is less meaningful because of two key non-homogeneities. First, the much higher variability in aerobic fitness ($V_{O2,max}$ range: 39 to 70 ml kg^{-1} min^{-1}) becomes a significant effect itself in determining T_2 elevation (Reid et al. 2001). Second, the high variability in jump power (range: 640 to 1130 Watts at body weight) diminishes the value of comparing activation of the same muscle among individuals of the same experimental

group. Despite this, an appreciation for recruitment variability among the J Group may be gleaned qualitatively by examining the most recruited thigh agonist muscles for each of the six jumpers as shown in Figure 33.

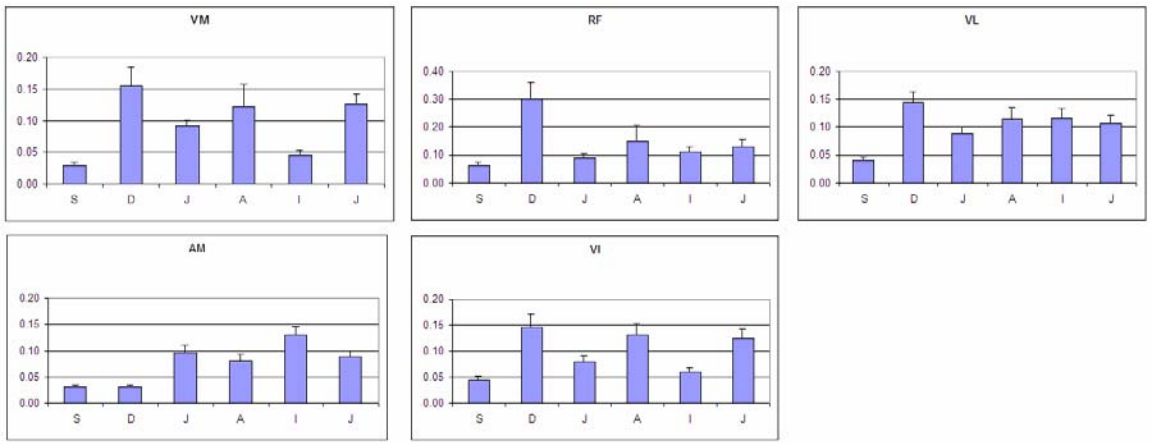


Figure 33 – Quadriceps and adductor T2 elevation by J Group subjects showing heterogeneity in recruitment at Post1.

At Post1, for example, each of the four quadriceps muscles was the most recruited muscle for at least one of the six jumpers in the J Group. This suggests that multiple neuromuscular strategies also exist for producing effective performance in the single-leg jumping exercise investigated in the present study.

Still another study examined the specificity of training relative to the exercise used to produce T_2 elevation (Le Rumeur et al. 1994). As measured at the same heart rate during cycling exercise of 165 beats/min, competitive triathletes recruit the *vasti* muscles more vigorously and produce superior output power (161% more) than untrained individuals. A third group of well-conditioned soccer players produced only 21% more output power than the untrained group, but recruited the three *vasti* significantly less to do so, implying better recruitment efficiency, which is consistent with the findings of the present study. In the case of the triathletes, the specificity of their cycling training

enabled them not only to develop better recruitment efficiency at sub-maximal levels of power output but also the ability to invoke more absolute recruitment when needed to produce dramatically superior maximal output. This suggests that for individuals that are highly trained in a motion that is specific to the testing procedure, the ability to recruit more motor units to produce superior levels of maximal output can develop. Training status specific to jumping was not controlled in the present study, but all subjects submitted a questionnaire about their exercise activity behaviors at screening and each received a numerical activity score (Baecke et al. 1982). There was no significant difference in reported activity between groups based on their Baecke scores (J: 7.5 ± 1.5, C: 6.7 ± 1.0), and none of the subjects in the study participated in jumping sports or training on a regular basis for more than 30 minutes per week. Recruiting "elite" J Group subjects only from a population that is highly trained in plyometrics, gymnastics and volleyball might have produced the same result.

Comparisons with iEMG: As noted earlier, T_2 elevation and iEMG are complementary methods for measuring muscle activation. The majority of activation studies to date have used iEMG because of its temporal component, its "direct" measure of neural drive and its ability to detect activation in both the takeoff and landing phases of the jump. T2 elevation retains its advantage of being able to detect activation in the entire musculature of the leg non-invasively, including muscles inaccessible to surface electrodes such as the VI and the TP.

In a previous study comparing one-legged and two-legged jumps, power production at the ankle joint increased due to a measured higher level of activation of the GM, a biarticulate muscle that can transport knee extensor power down to the ankle joint

(Van Soest et al. 1985). There was no T_2 elevation measurement for two-legged jumps in the current study, however results of the current study did not indicate a larger activation for the GM relative to its counterpart plantar flexor muscles. In fact the current study showed highest activation levels for the FDL and TP muscles of the calf, especially in the C Group. One possible explanation for this discrepancy is the difference in the subject pools that were recruited. The Van Soest study recruited only well-trained volleyball players, whose jumping technique, presumably, has been sharpened through training. The finding of relatively higher FDL and TP activation in single leg jumping in both groups of the current study is noteworthy and has not previously been reported, due to the limitations of surface iEMG to monitor these internal muscles.

Adaptations and recruitment: Although the present study did not include a training component, the results are consistent with most studies that have investigated the effect of training on recruitment as measured by both iEMG and T_2 elevation. Resistance training has resulted in improved recruitment efficiency (in this case more strength at the same level of activation) in both the trained and contra-lateral untrained limbs in a unilateral training study (Ploutz et al. 1994). Exactly the opposite effect was seen during a unilateral unloading intervention (Ploutz-Snyder, et al. 1995). Each of these studies trended in a manner consistent with the findings of the present study.

However, in another study of middle-aged and elderly subjects (Hakkinen et al. 1998) iEMG measured during squat jumps increased in agonists and decreased in antagonists following an explosive training program. The training program produced significant gains in jumping performance and maximal recruitment. At first glance these results appear to contradict the results of the present study, however the focus of the

iEMG study was training adaptations in individuals, and no explicit measure of recruitment efficiency was taken. In contrast, populations with different jumping ability levels were the focus of the present study, which included a specific metric to quantify recruitment efficiency. Also, the subjects in the iEMG study were capable of significantly higher jump height (and therefore power production) after training. No measure was made of muscle activation at the pre-training jump heights, which would be required in order to determine any gains in recruitment efficiency.

Plyometric training (i.e., a regimen of exercises featuring eccentric loading followed immediately by explosive concentric contraction) alters the activation strategies for vertical jumping so that increased preparatory adductor activity, increased adductor/abductor coactivation and decreased knee extensor activation (relative to knee extensor activation) all occur as measured by iEMG(Chimera et al. 2004). The J Group subjects of the current study were not plyometrically trained and did not display higher levels of adductor activation, but the relative decrease in knee flexor activation is a common finding.

Limitations: One of the primary limitations of the present study was that all muscle activation was assumed to be related to takeoff during the jump. Landing loads observed during drop jumps recruit the quadriceps and plantar flexor, as well as the antagonist hamstring and dorsiflexors to provide joint stiffness (Russell et al. 2007). Landing effects were somewhat mitigated by the fact that subjects in the present study were instructed to land on two legs, if possible. However, subjects were not actively coached away from landing on the takeoff leg alone if it appeared that they were more comfortable doing so. In both groups the VL and RF muscles of the quadriceps of the

bent leg displayed significant increase in the percentage of elevated pixels at Post2 only, while only the C Group displayed significant rises in mean muscular T_2 (p<0.05). Further investigations should isolate the effects of the landing in order to separate its effect on producing T_2 elevation in the muscles of interest.

The single leg jump, as performed in the present study, is not a common movement in the exercise repertoire of the training programs of any of the subjects based on their questionnaire responses. A motor learning effect likely played a part, but it was not controlled for in the present study. This effect could be counteracted by allowing subjects to gain familiarity with the jumping protocols before the screening and testing process begins.

The finding that exercise intensity (as measured by output power) did not scale with the additional 33% weight is a significant finding that prevented accomplishing all of the aims of this pilot study. Specifically, it was not possible to determine whether the amount of T_2 elevation scaled with power production because the subjects produced jumping power at Post2 that was not significantly greater than at Post1 (J: p=0.54, C: p=0.34). One possible explanation for this is that the subjects altered their jumping technique conservatively in order to compensate for the uncomfortable feeling of the extra loading of the weighted vest. This is supported by the observation of higher recruitment in the antagonist BF at Post2 vs. Post1 (p=0.03). This may be explained by previous findings that co-activation of the quadriceps and hamstrings is a strategy employed by the body to boost knee joint stiffness in order to prevent injury (Russell et al. 2007; Chimera et al. 2004).

The present study served as pilot study for a proposed targeted pre-conditioning study that includes limb suspension as an unloading technique and the single-leg jump as the exercise protocol for inducing T_2 elevation. Determining the relationship between T_2 elevation and intensity would be difficult to accomplish based on the findings of the present study, especially in an unloaded leg. A possible corrective action for this problem would be to scale jumping intensity at fractional value of the subjects' body weight (e.g., 50%) rather than increasing the load with additional weight. Reduction in body weight for jumping could be accomplished with a set of bungee cords and a jumping harness (Gollhofer & Kyrolainen, 1991). However, for drop jumps with positive (+200 Newtons) and negative (-495 Newtons) loading applied, subjects achieved both their maximum force production and takeoff velocities (i.e., power) under the bodyweight (BW) condition. iEMG levels were flat over the BW to BW+200 Newton range, but declined over the BW to BW-495 Newton range. Attempts to linearly scale the output power production of jumping exercise by varying loading or unloading versus body weight in a linear fashion appear to be invalid. Proposed targeted preconditioning studies may be better served by an alternative multi-joint, unilateral, power-oriented exercise, such as single-leg cycling on a calibrated ergometer.

A key piece of information that was not obtained in the present study was the time history of activation throughout the jumping exercise event. Timing, coordination and co-contraction of agonist, antagonist and synergist muscles all play key roles in determining jumping performance (de Ruiter et al. 2006; Chimera et al. 2004; St. Onge et al. 2004). Furthermore, the rate of force (or torque) development in agonist muscles at the start of contraction has a highly correlated linear relationship with jump performance

(de Ruiter et al. 2005). Unfortunately, this parameter (for individual muscles) was not obtainable using the methods of the present study. It is entirely possible that J Group jumpers in the current study were able to generate high bursts of activation only at the initiation of contraction and thereby retain a lower level of cumulative activation. Any such peaks in activation intensity would be undetectable with MRI methods alone.

As with any pilot study, the small number of subjects (N=6, each group) made it difficult to obtain enough statistical power to draw more definitive conclusions. Additionally, there were no controls for gender, age, or ethnic origin of the subjects. Inclusion in the two experimental groups was based on performance of a two-legged jump against normative data (Payne et al. 2000), whereas the exercise protocol utilized a single-leg jump. The results of the current study indicate a positive correlation between two-legged jump height and the average power produced during the exercise protocol (r=0.63, p<0.02) as expected. However, this imperfect correlation does create an additional source of variability.

Jumping articulations are not limited to the muscles of the thigh and calf. Indeed the gluteals, psoas and hip flexors play a significant role. A complete treatment of the present study should also include these key muscles, which were ignored in this pilot study in order to keep the MRI protocols as efficient as possible.

Implications: The present study has successfully demonstrated the T_2 mapping of muscle in a complex multi-joint movement requiring both power and strength and has identified muscle activation differences that exist between two populations of differing abilities. These differences are centered on recruitment efficiency, both in agonist muscles and in co-contracting antagonists.

This concept may be useful to the optimization of training strategies for sports (e.g., cycling, sprinting, and swimming) and to physical therapy and rehabilitation. It is noteworthy that similar differences in recruitment exist between healthy individuals and patients who are recovering from surgery to the anterior cruciate ligament (ACL). As measured by iEMG, ACL-deficient subjects have similar deficits in both recruitment efficiency of agonists and co-contraction of antagonists to less capable healthy jumpers, regardless of jumping proficiency before surgery (Doorenbosch, 2003). What is not clear is whether this effect occurs due to the injury, the subsequent unloading during recovery, or both. Further study is required to determine this.

These findings and reasoning provide insight for preconditioning prior to unloading, with possible direct benefits to manned space exploration. Such programs may be most effective when based on complex functional movements targeting the specific development of efficient recruitment. Again, further study is needed to determine the effectiveness of such training programs.

Conclusion

The present study investigated the use of acute post-exercise T_2 elevation to map recruitment differences in the thigh and calf muscles of two distinct populations (based on two-leg jumping ability relative to age and gender norms) during single-leg jumping exercise, both at body weight (Post1) and 133% of body weight (Post2). The results showed significantly higher peak and average jumping power among the better jumpers, as expected, including significant differences in jump height, force and power production at both Post1 and Post2. However, force and power production were not significantly different at Post1 versus Post2 in either group. The results generally showed that superior jumping performance at both Post1 and Post2 was accompanied by a lower degree of T_2 elevation in nearly all leg muscles. This was true even when T_2 response was normalized to the subjects' maximum aerobic power, thus implying a higher degree of recruitment efficiency throughout the lowers limbs of the better jumpers. Through analysis with the assistance of novel performance to activation ratio metrics, it was concluded that the better jumpers achieved their success more by successful suppression of antagonist and unproductive muscles than by higher recruitment levels in the agonist muscles themselves. Muscle volumes showed significant differences (from Baseline) at Post1 and Post2, but these measurements resulted in no significant correlations relative to T_2 elevation. The general and muscle-specific results of the study indicate that both central and localized neuromuscular factors play a role in single-leg jumping performance. The results also indicate that scaling the intensity of single-leg jumps by adding weight (beyond body weight) does not necessarily correspond to similar scaling of jumping power or T_2 response.

Appendix A – Image Processing Techniques

Media Cybernetics' Optimas image processing and analysis software provided the platform for defining the regions of interest (ROIs) within individual muscle groups in the MRI images, generating the T_2 maps and calculating intramuscular T2 values, percentages of elevated pixels and muscle volumes. Three Optimas macros were written in the proprietary Analytical Language for Imaging (ALI) that accompanies the product. ALI is a vector-based language that facilitates the development of custom image analysis solutions.

Because Optimas does not directly support the DICOM format in which images are stored on the Siemens Symphony scanner, it was necessary to convert all images from DICOM to 16-bit raw data files using a plug-in to the popular public domain program known as ImageJ developed by Wayne Rasband of the National Institute of Health (NIH).

Macro "MakeT2"

This macro takes a set of raw 16-bit integer MRI axial slice image pairs and generates a 32-bit floating point image for each pair that represents the map of T_2 values on a pixel by pixel basis using equation (4) in the Methods section. The user interface and representative images of the calf for macro "MakeT2" are shown in Figure 34.

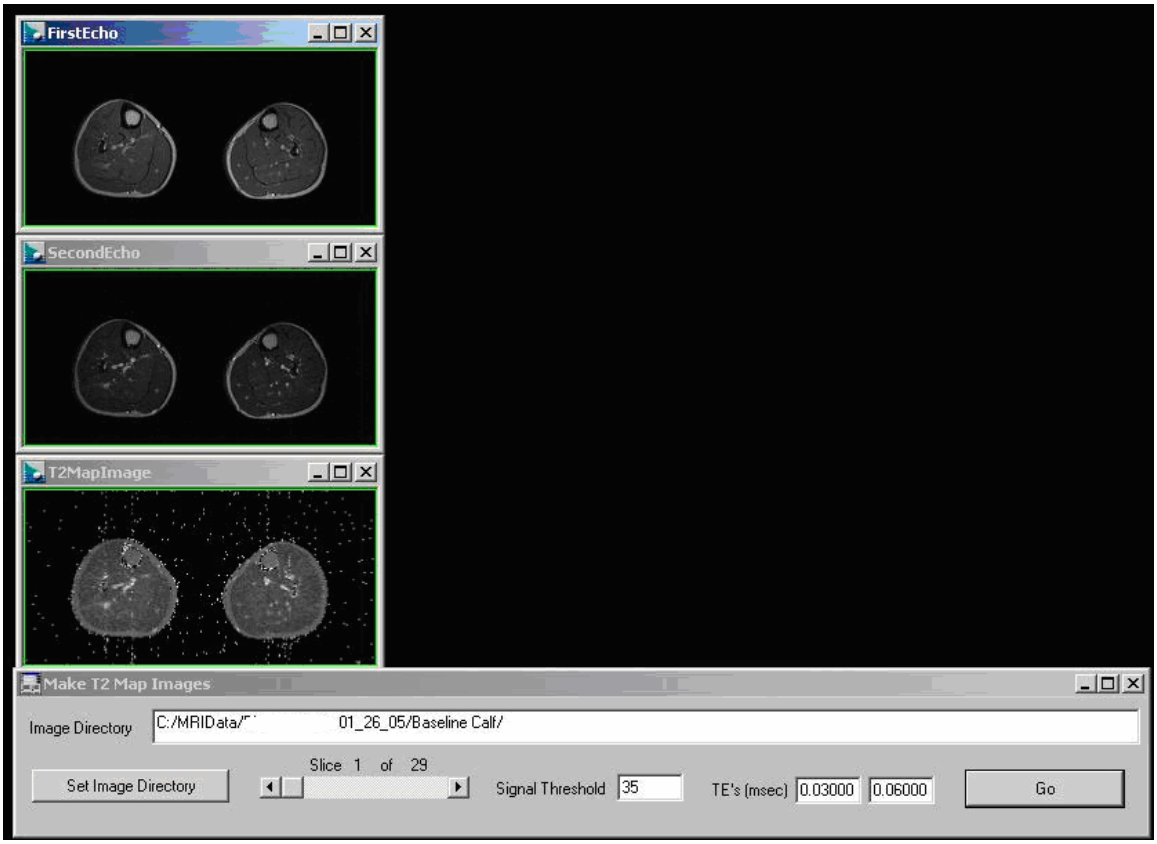


Figure 34 – User interface for Macro MakeT2 showing a single axial slice of a subject's calf. Pixel intensity images are taken at TE_1 = 30 msec and TE_2 = 60 msec. The resulting T2 map image assigns a gray value to the computed T2 value of each pixel in the image.

The user may select any image directory containing 16-bit raw images. The two echo times (TE_1 and TE_2) are specified in msec. The macro will zero out any pixel values lower than a user-specified threshold value. This feature eliminates the pixel noise in the background, improving the cosmetic appearance of the T_2 map images. The macro defaults to an image size of 256 x 128 pixels, but this can easily be modified within Optimas to accommodate any image size. The FOV is calibrated to be 50cm x 25cm by default, but this also may be adjusted by the user within Optimas.

Macro TraceMuscles

This macro allows the user to manually define ROIs for each muscle of interest on the intensity images. ROIs may be defined both for T_2 analysis and for anthropometric analysis (i.e., muscle cross-sectional areas and volumes). The user interface is shown in Figure 35.

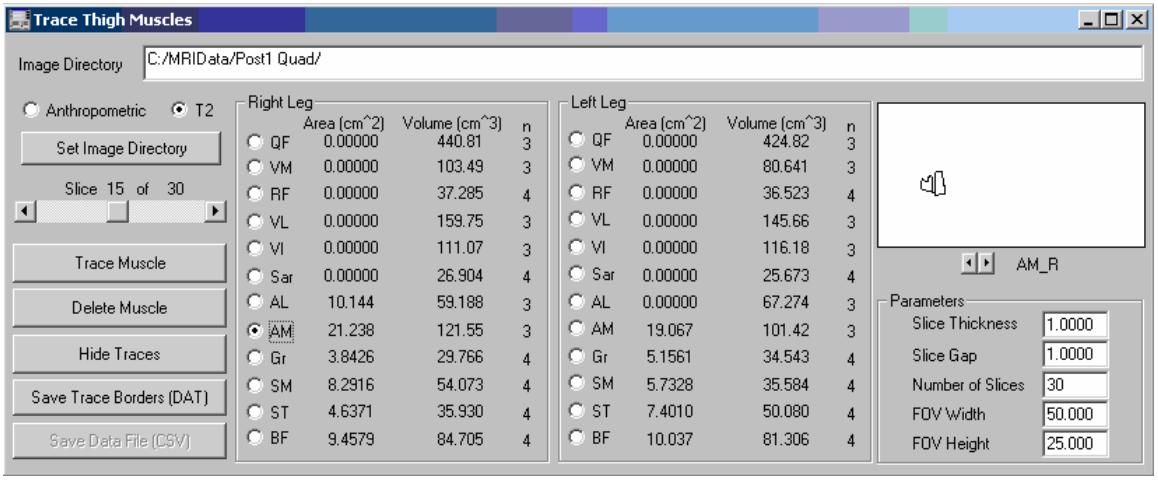


Figure 35 - User interface for Macro TraceMuscles

The user defines the directory where 16-bit raw image files reside. The images are assumed to be numbered sequentially with both echoes present. The macro counts the number of slices and loads them sequentially into the computer memory. The user selects either anthropometric analysis or T_2 analysis and scrolls to the slice of interest. Upon selecting a muscle group in the desired leg, the user clicks the "Trace Muscle" button and the macro enters the Optimas manual freehand ROI mode, indicated by a change in cursor. Each left click selects a point on the polygon that will represent the final ROI. There is no practical limit (except for memory) on the number of points comprised by an ROI. The macro affords the user the opportunity to correct an errant point (right click) or to abort an ROI (Escape key). Once the user double clicks to close

the polygon, the macro automatically computes the ROI area and updates the running muscle volume computation based on the formula given in (5) in the Methods section. The number of ROIs for a given muscle that have been specified by the user is recorded in the "n" column. Typical practice in the present study was to record three ROIs for larger muscle groups and four for smaller muscle groups for T_2 analysis.

Anthropometric and T_2 ROIs are specified individually so that the user may exclude intramuscular MRI artifacts and blood vessels from the T_2 calculations while retaining them for volumetric calculations. The slice thickness, slice gap and FOV dimensions default to those specified in the Methods section, but the user may change them if required. Figure 36 shows the results of tracing muscle ROIs in the dorsiflexors for subsequent T2 analysis. Note that the borders are not included to ensure only muscle tissue is selected.

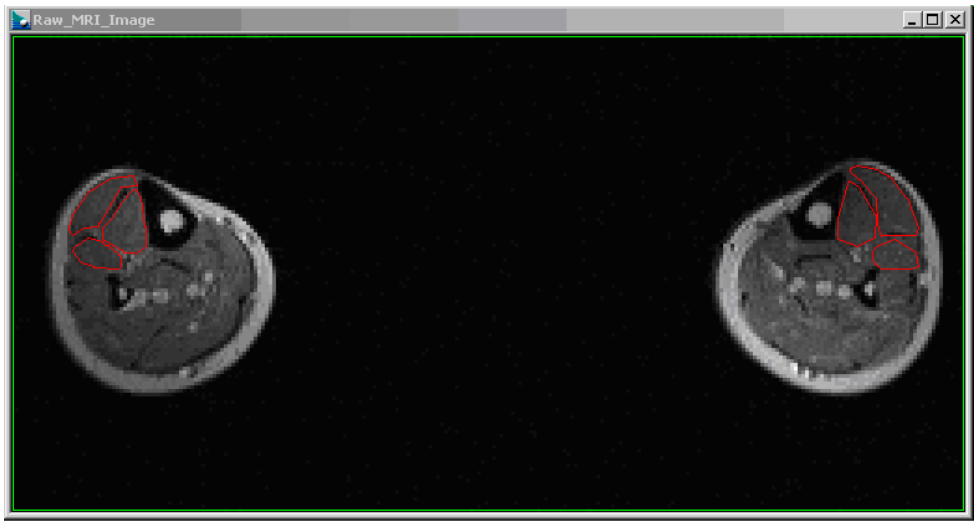


Figure 36 - Representative ROIs for the dorsiflexor muscles using Macro TraceMuscles.

Figure 37 shows the results of tracing muscle ROIs in the adductors for subsequent T2 analysis. Note that the MRI blood flow artifact is excluded from the ROI.

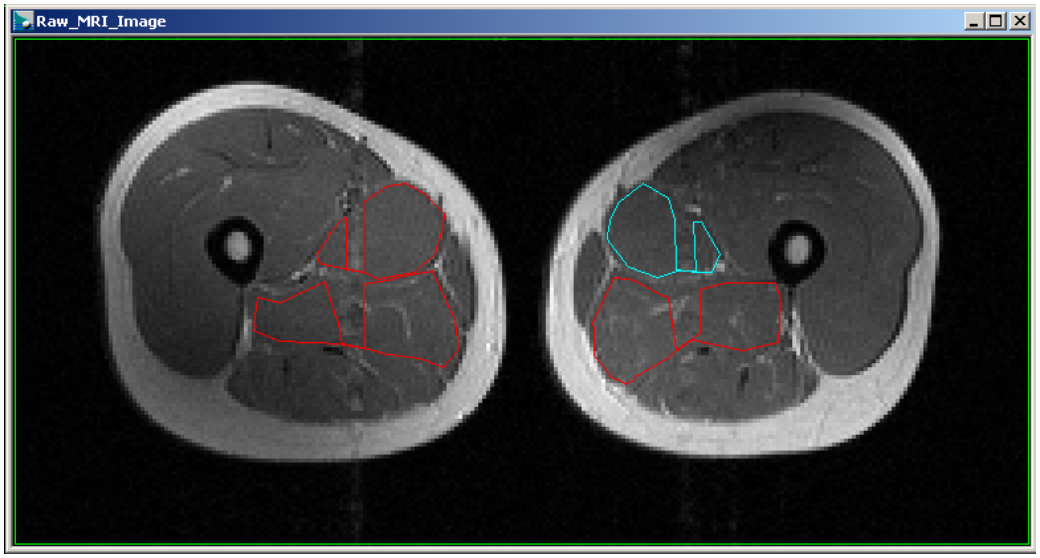


Figure 37 - Representative ROIs for the adductor muscles using Macro TraceMuscles

Figure 38 shows the user interface and a slice of the lower thigh indicating the ROI for the computation of muscle volume. Note in this case that the ROIs are drawn directly onto the muscle borders.

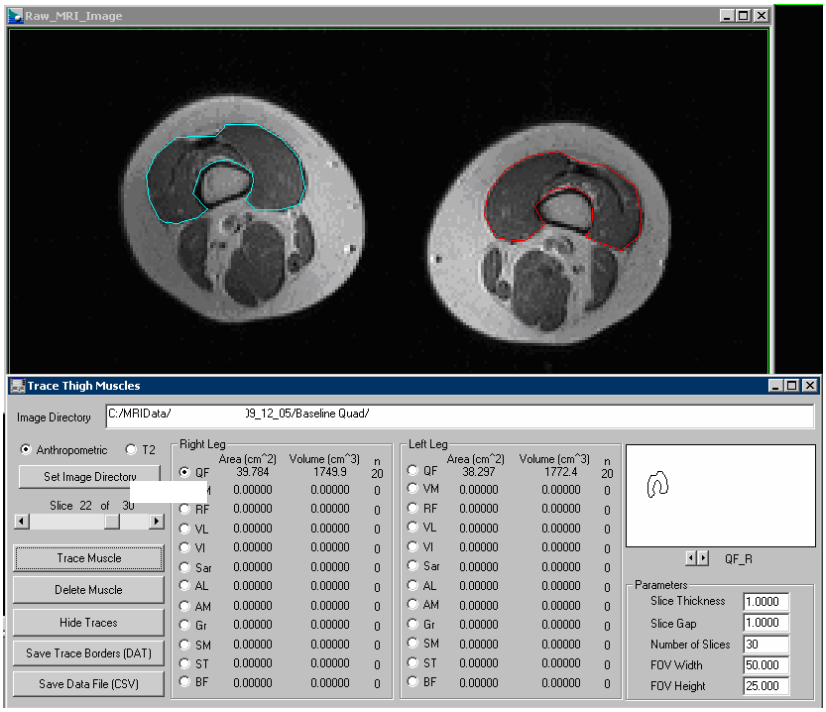


Figure 38 – Representative image and ROIs for the computation of quadriceps femoris volume.

Macro T2Map

This macro transfers the T_2 analysis ROI that were generated by the TraceMuscles macro onto the T2 map images made by macro MakeT2. The user interface and results for a Baseline thigh scan are shown in Figure 39. Figure 40 shows the same slice at Post1.

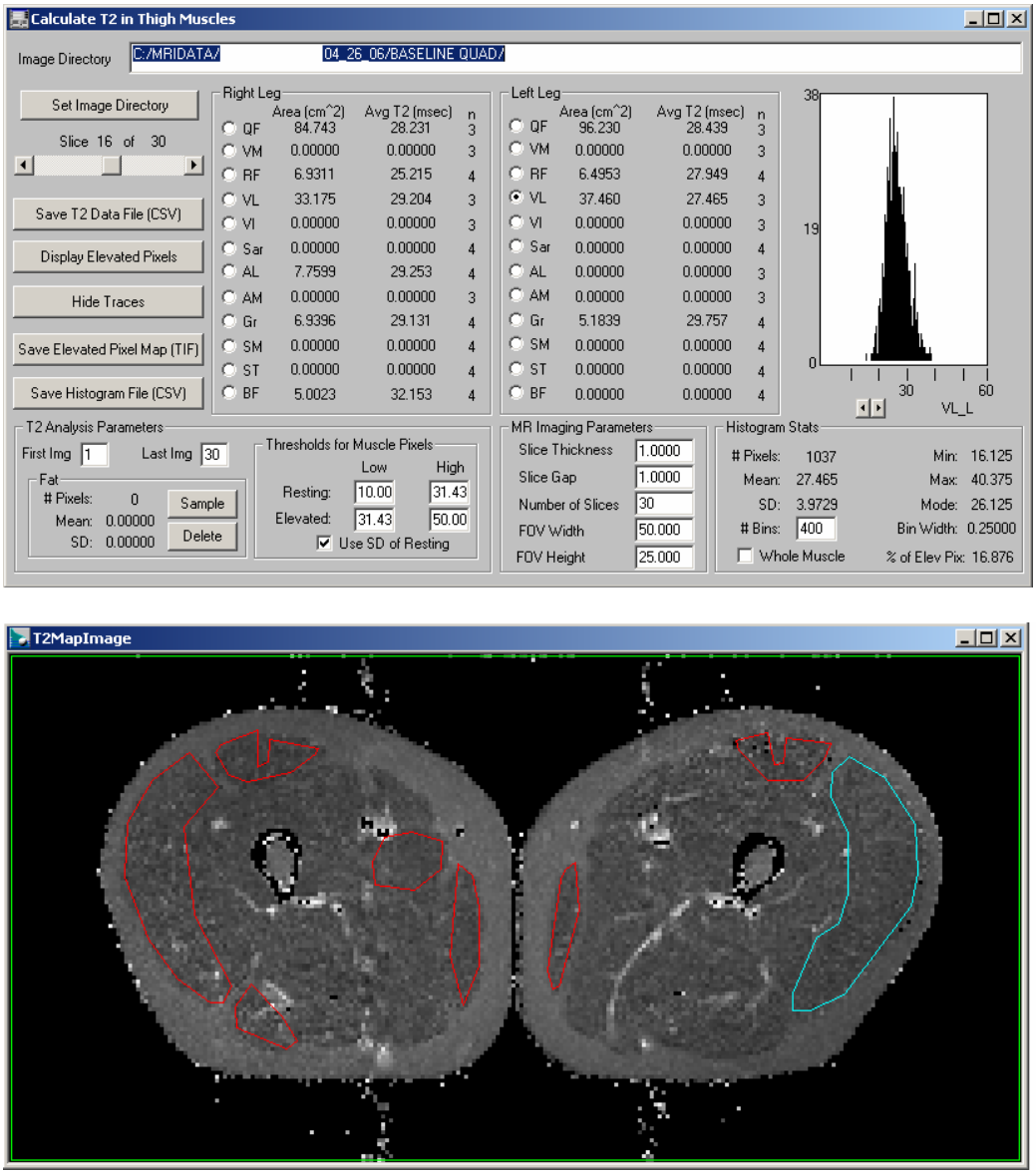


Figure 39 – User interface and ROIs for various muscles of the thigh at Baseline. The VL muscle at right is highlighted to indicate that it is the currently selected muscle.

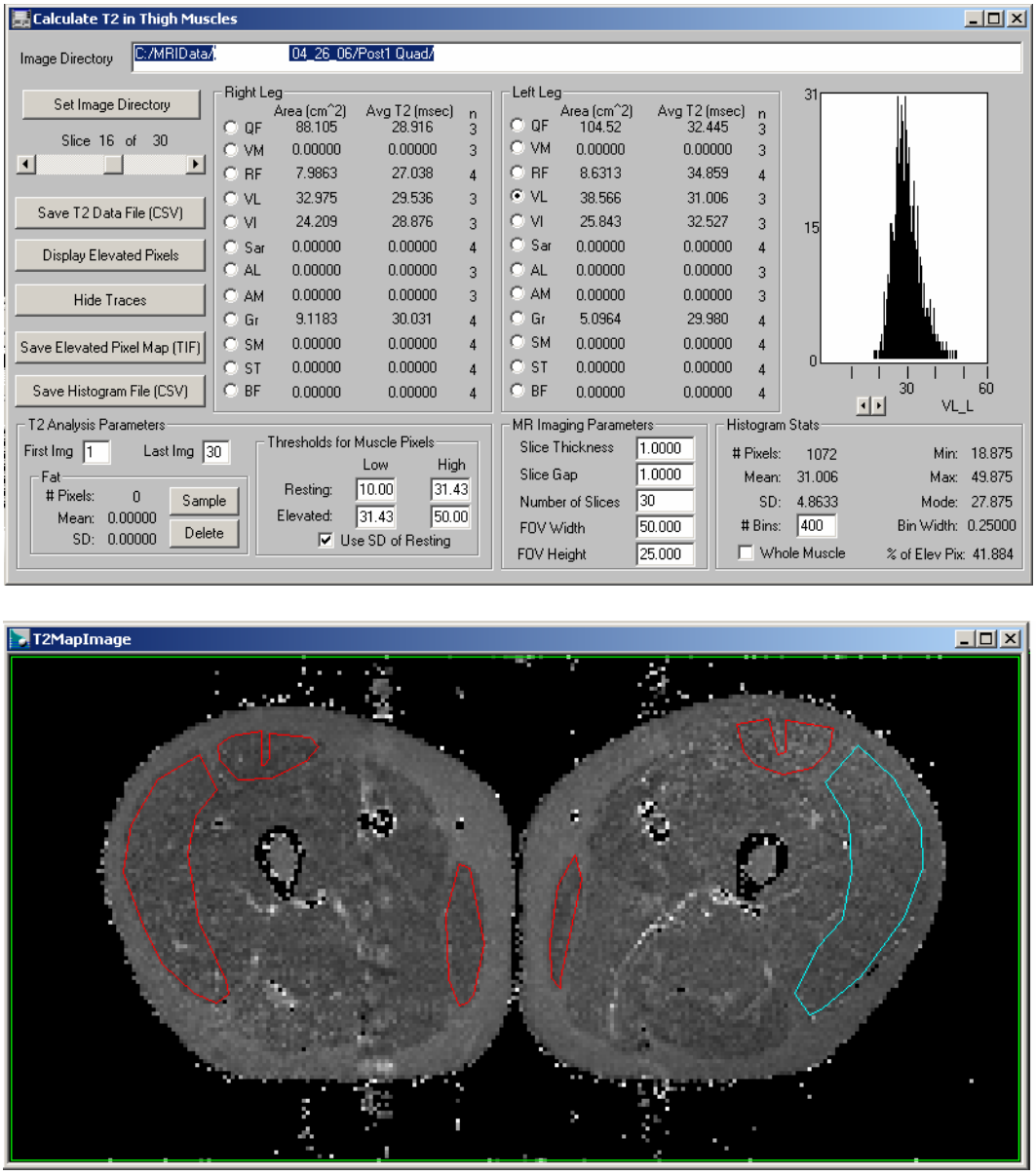


Figure 40 – User interface and ROIs for various muscles of the thigh at Post2. The VL muscle at right is highlighted to indicate that it is the currently selected muscle.

The macro automatically computes the average area and T_2 value for each muscle ROI in each slice. Additionally, the user may select individual ROIs for

further analysis. A T_2 pixel histogram indicates the relative distribution of T2 values within the ROI and the statistical properties for that distribution. By selecting "Whole Muscle" the same information is calculated over all slices in the muscle group, not just the slice being viewed. The thresholding for determining the percentage of elevated pixels is automatically determined from those image sets that are indicated as Baseline scans. It is essential that these scans be analyzed first, prior to the post-exercise scans. The default is to make the threshold one standard deviation above the Baseline mean, but the user may override this feature by unchecking the "Use SD of Resting Box" and entering values manually. Additional feature of this macro are the ability to sample an ROI of fat tissue for comparison with muscle and the ability to view the specific pixels in an ROI that have been classified as "elevated" such as in Figure 41.

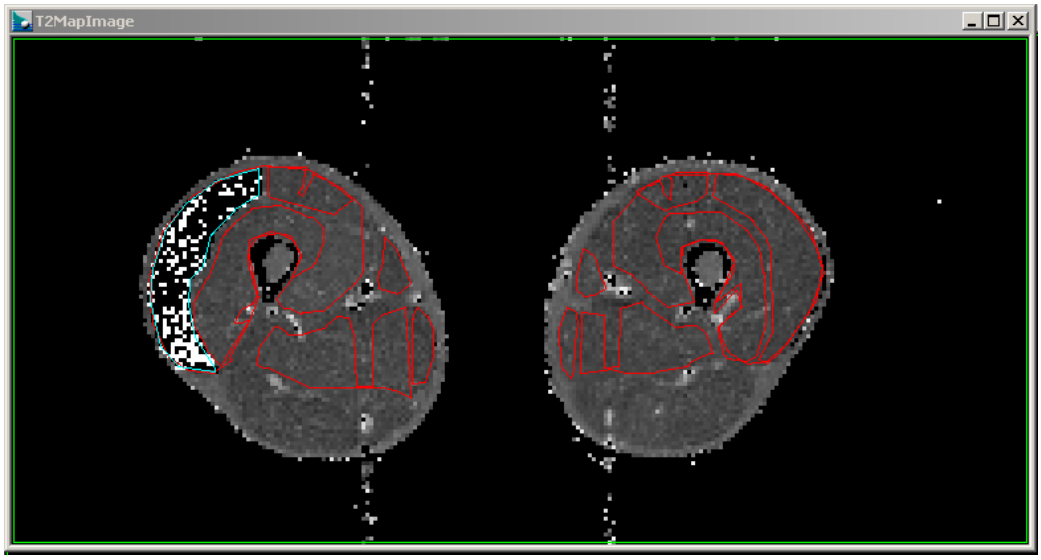


Figure 41 – Viewing of elevated pixels. Binary image within the ROI for the VL muscle indicates the pixels (white) which have been classified as elevated, i.e., greater than 1 SD above the Baseline resting whole-muscle T_2 value.

Appendix B – Jump Platform

The jump platform used for the present study is a Kistler QuattroJump with a 36”x36” contact surface as shown in Figure 42.

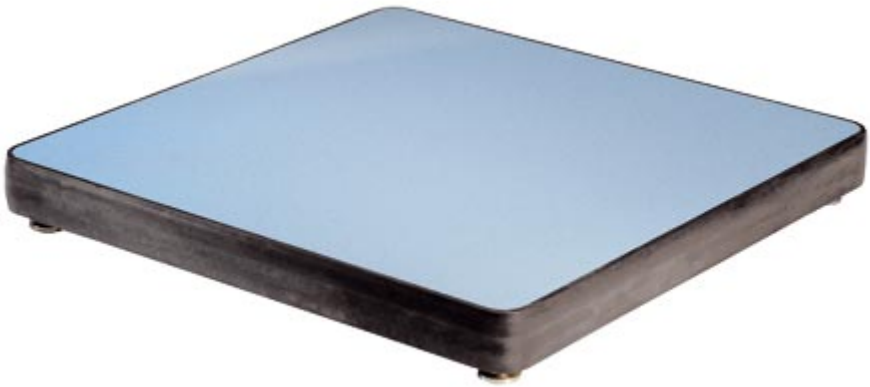


Figure 42 – Kistler QuattroJump force platform. Source: www.kistler.com

The platform is equipped with an internal controller that samples the force/time history at a rate of 500 Hz (default). The platform communicates with a laptop PC through an RS-232 serial interface. A USB to RS232 converter was required for the Dell Inspiron 5100 laptop used in the present study, as the laptop lacked a dedicated serial port. Kistler provides data collection and analysis software with the platform, making these processes rather straightforward. An export feature allows the user to further analyze the force/time data in spreadsheet (MS Excel). A typical data screen for one set of ten jumps is shown in Figure 43. Force history, velocity, average power and jump height are presented intuitively by the software. A separate input form screen (not shown) captured each subject’s height, weight, age and gender. A unique spreadsheet calculated quantities such as force-time integral and peak power for each subject.

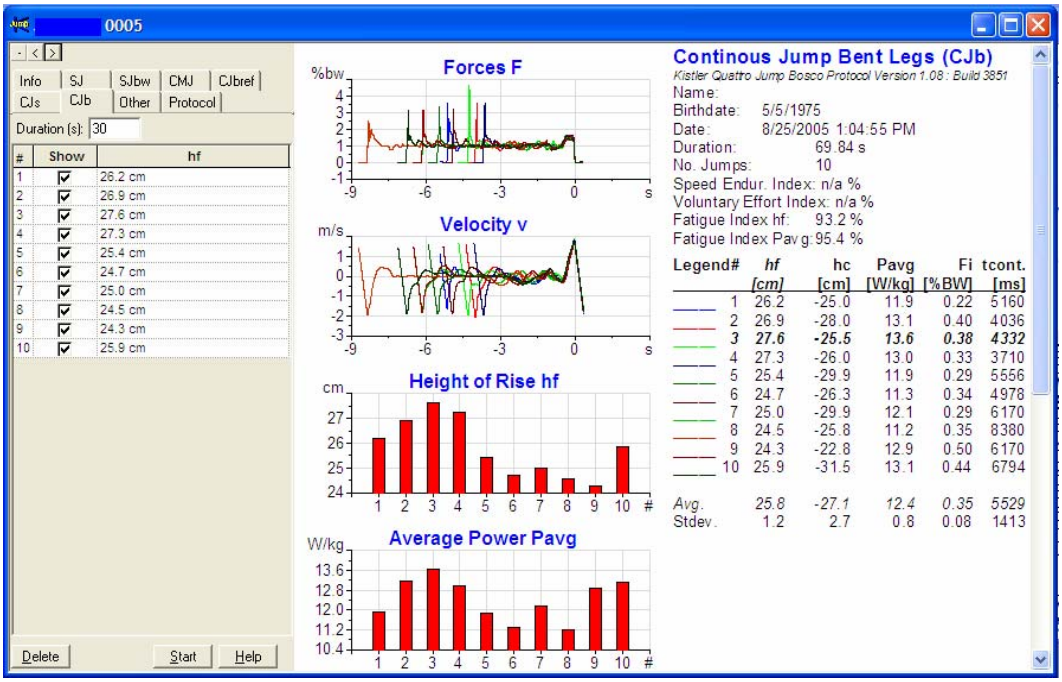


Figure 43 – Jump data screen capture for a typical subject.

When the subjects donned the weighted vest at Post2, the force and power per body weight calculations were scaled by a factor of 1.33 to account for the "dead weight penalty". The V-Max weighted vest is shown in Figure 44.

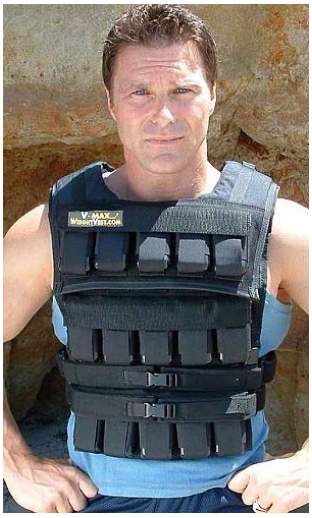


Figure 44 – V-Max weighted training vest. Source:V-max, www. weightvest.com

Appendix C – MRI Techniques

This study used a Turbo Spin Echo® with echoes at T_{E1}=30 msec and T_{E2} = 60 msec, an echo train length of ETL = 7, and a repetition time of T_R = 2000 sec. This scan was chosen as a standard clinical scan for producing T_2-weighted images and its similarity to scans used to produce post-exercise T_2 images in other studies. The scan diagram is show in Figure 45. The scan time was just under four minutes.

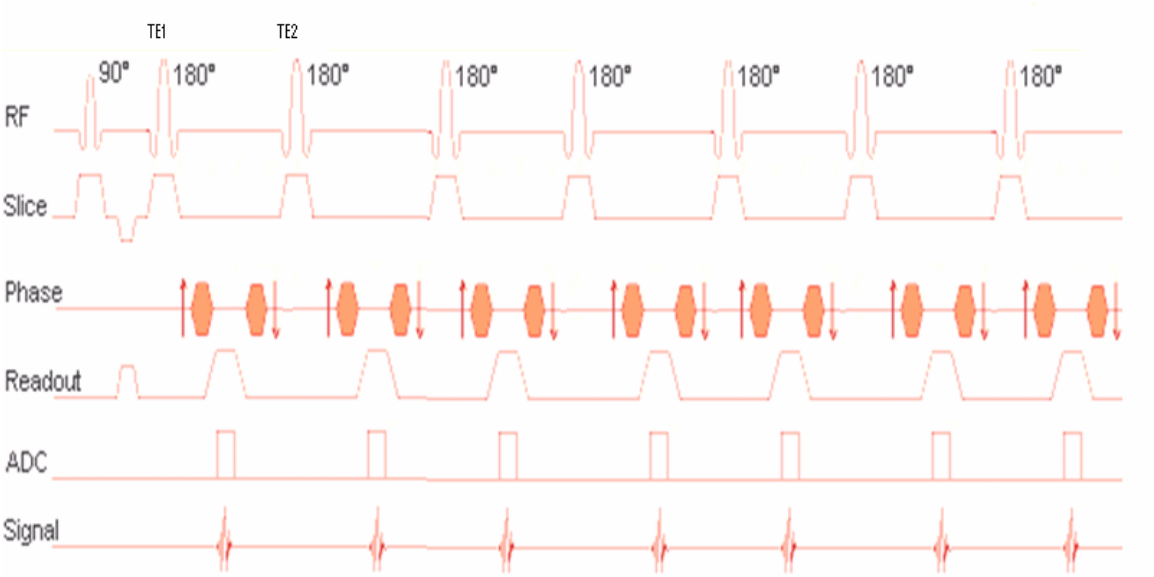


Figure 45 – Dual-echo Turbo Spin Echo MRI sequence, ETL=7.

Each slice image represents 1 cm of axial thickness and a 500mm x 250mm field of view (FOV) on a 256 x 128 pixel grid. The gap between each slice was 1 cm.

A saturation pulse technique was attempted during the developmental testing of the scans in order to eliminate the blood flow artifact. However, in order to successfully employ this technique it became necessary to forego the ability to measure quadriceps volume along the full length of the thigh. Therefore, the decision was made to simply work around the blood flow artifact when defining the ROI for any nearby muscles.

Appendix D – Preliminary Exercise Protocol/MRI Studies

Adequacy of T_2 return to baseline following rest: One of the key assumptions of this study was that the acute T2 elevation had a sufficiently short half-life to decay within a 1 hour period. This effect was tested in one preliminary subject (who did not participate in the clinical study) by taking quadriceps scans at Baseline, Post1, Post2 and a follow-up scan after an additional rest period of 1 hour to check for Return to Baseline (RTB). For this particular subject, the Post2 load was BW+50%. Although most measured T_2 differences between Baseline and RTB are statistically significant (p<0.05), the results in Figure 46 show that the T_2 values at Baseline and RTB agree within ±5%,.

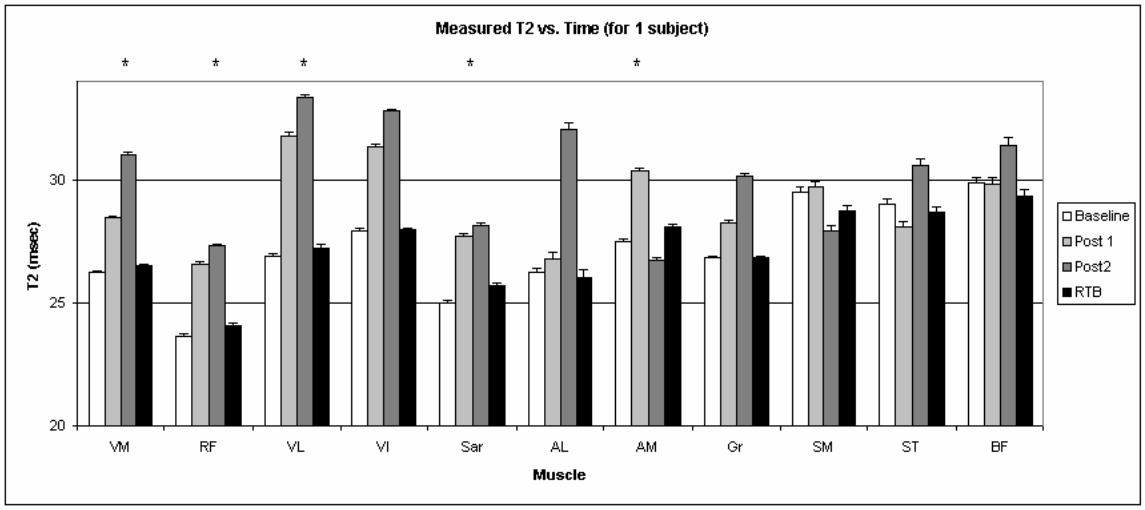


Figure 46 – Measured T2 values at Baseline, Post1, Post2 and RTB for one subject. RTB followed Post 2 by one hour. Post2 load was body weight plus 50%. (RTB > Baseline: *, p<0.05)

Repeatability of T_2 measurements: Intra-subject variability is illustrated in Figure 47 where Baseline T2 values are recorded for the same subject on two dates, three months apart. All measured T_2 values agree within 5%, except AL (-6%) and SM (+6%); however, the measured T_2 differences are statistically significant (p<0.05) in most muscles.

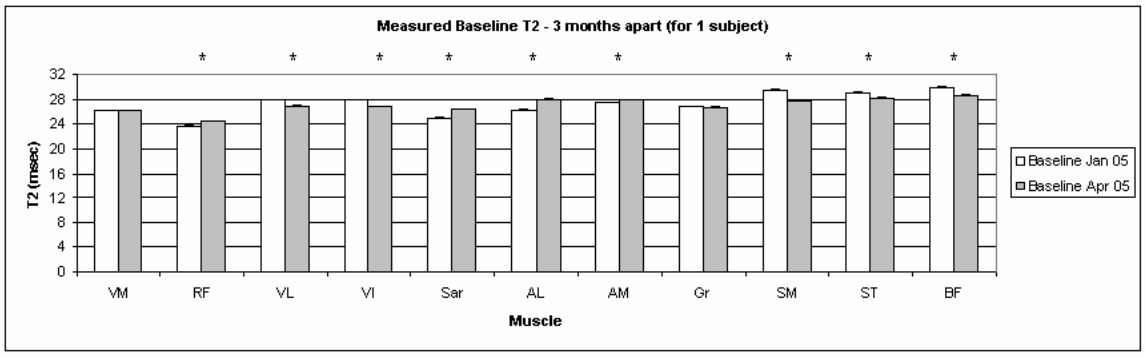


Figure 47 – Measured T2 values at Baseline on two separate dates for one subject. Although most differences are statistically significant, all values agree within 5%. (*: $p<0.05$)

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