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The Effects of Selected Exercise Intensities on Skeletal Muscle Protein Degradation

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463288

Thesis

Submitted to the School of Human Kinetics
in fulfillment of the requirements
for the degree of Master of Science in Kinanthropology
University of Ottawa, 1994



Ed McNeely, Ottawa, Canada 1994



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ABSTRACT

The purpose of this study was to examine the effects of selected exercise intensities on muscle protein degradation. On two occasions Eight male trained subjects performed a bench press exercise: on one occasion each set consisted of 10 repetitions at 70% of 1RM; on the other occasion each set consisted of 5 repetitions at 85% of 1RM. Venous blood samples were drawn preceding, immediately following and 24 hours after each exercise sessions and analyzed for 3-methylhistidine and tyrosine.

The results showed a significant decrease in the concentration of 3-methylhistidine immediately following exercise. There was a significant decrease in tyrosine following exercise at 85% 1RM when compared to baseline. There was not a significant difference found between the 70 and 85% of 1RM protocols for either 3-methylhistidine or tyrosine. The lack of difference between 70 and 85% of 1RM may have been due to similar motor unit recruitment patterns for the two intensities.

The exact mechanisms responsible for decreased protein degradation following strength training exercise are presently unclear but may be the result of mechanical tension placed on the muscle fibre.

Keywords: 3-methylhistidine, Tyrosine, Protein degradation, strength training

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INTRODUCTION

INTRODUCTION

Strength trainers have developed a series of training systems that are designed to produce adaptations in either or both the structural or control components of contraction. Schmidtbleicher (1985) has divided strength training methods into two categories which promote (A) Maximal strength methods, which are characterized by short term explosive maximal contractions against high loads (90%-100% 1 RM) or supramaximal loads (150% 1RM or above). These methods are designed to bring about neural adaptations. (B) Submaximal contraction repetition methods, characterized by several sets of repetitions with submaximal loads (60%-80% 1RM). These latter methods are most common in bodybuilding and are intended to produce muscular hypertrophy.

Moritani and DeVries (1979) have shown that the adaptations during the first four to six weeks of a new strength training program are predominantly neural in nature. Their work also demonstrates that as hypertrophy becomes the predominant source of strength gains there is a detraining of the neural adaptations. Therefore it is necessary to continually train for both neural and hypertrophic adaptations if maximal strength levels are to be achieved.

It has been recommended that training methods that result in hypertrophy of the muscle should be performed before methods that focus on the enhancement of motor unit activation (Poliquin, 1988; Stone, O'Bryant, Garhammer, 1981). Stone et al. (1981) have developed a model of strength training that is composed of four stages, a hypertrophy stage, a strength

stage, a strength-power stage and a peaking or maintenance stage. Poliquin (1988) has suggested that a two week period of hypertrophy methods should be alternated with a two week period of neural oriented training (see Strength Training Methods Ch.2).

These authors agree that the most effective means of producing muscular hypertrophy through strength training is to perform a high volume of work. There is some disagreement as to how the high volume is to be achieved. Reed, Ablack and McNeely have proposed that the high volume of work should be the result of a greater number of sets (10-15) of low repetitions (3-6) to keep lactate accumulation low and therefor eliminate it as a source of fatigue. The other authors have suggested that the high work volume should come from performing many repetitions (8-12) and fewer sets (3-6).

According to MacDougall (1986) there is a belief among lay people involved in strength training that strength training results in a breakdown of muscle proteins. The repair process following this breakdown is presumed to be responsible for increases in muscle cell size and whole muscle strength. This hypothesis has gained popularity in the scientific community as well. Pivarnik, Hickson and Wolinsky (1989) and Hickson and Hinkelman (1985) state that chronic, exercise induced skeletal muscle protein catabolism would be an advantage to the athlete training for tissue strength and size gains.

Protein adaptations in muscle are determined by the pattern and frequency of usage (Booth, Nicholason, and Watson 1982). The result of a protein adaptation is that subsequent work at the same absolute intensity causes less disruption of the systems homeostasis (Booth and Watson 1985). The protein adaptation can be the result of increased synthesis decreased degradation or a combination of both.

The use of 3-methylhistidine or tyrosine in the serum or urine has been proposed as an indicator of skeletal muscle breakdown (Young and Munro 1978; Davis, Karl, Tegtmeyer, Osborne, Klahr, and Harter, 1985). Several researchers have attempted to demonstrate that strength training results in an increased release of 3-methylhistidine (Hickson and Hinkelman

1985; Dohm, Williams, Kasperek, and vanRij 1982, Pivarnik, Hickson, and Wolinsky 1989). Discrepancies and lack of control in the application of the strength training stimulus have lead to mixed results. However, There is still a belief among practitioners that exercise exerts a catabolic effect upon muscle tissue before an anabolic adaptation occurs.

Rationale

Strength training with high work volumes in trained and untrained subjects results in hypertrophy of individual muscle fibres and the whole muscle. High volume strength training is usually accomplished by using a lower work intensity. This sometimes leads to decreases in maximal strength even though the muscle hypertrophies (Stone and O'Bryant, 1987). If a high volume of work was accomplished with a high intensity, both hypertrophy and increased strength may occur. If the exercise response cycle that leads to a muscular adaptation to strength training consists of alterations in the rates degradation or synthesis of proteins, by measuring changes in degradation in skeletal muscle some insight should be gained into the training response of this tissue.

3-methylhistidine has been proposed as an indicator of skeletal muscle degradation (Young and Munro, 1978; Viru, 1987; Long, Dillard, Bodzin, Geiger, and Blakemore, 1988). Thus, if 3-methylhistidine were measured after different training systems were used the effects of these systems on the degradation of skeletal muscle could be determined.

Research Hypothesis

1. Strength training exercise will result in an increase in the serum concentration of 3-methylhistidine (3-mh) and tyrosine.
2. The high intensity (85% 1RM) protocol will produce a greater change in 3-mh and tyrosine than the low intensity (70% 1RM) protocol.

Statement of the Problem

The purpose of this study is to compare the effects of two resistance training sessions, one at 70% and one at 85% of 1RM, on the degradation of skeletal muscle proteins, in trained male subjects aged 20-35 years, as measured by changes in serum concentrations of 3-methylhistidine and tyrosine.

Definitions and Abbreviations

| | |
|-----|--|
| 85% | A resistance that is 85% of the weight that the subject can bench press for only one repetition. |
| 70% | A resistance that is 70% of the weight that the subject can bench press for only one repetition. |

1 RM

One maximal repetition. This refers to the maximum amount of weight that can be lifted successfully one time through a maximum range of movement.

3-mh

3-methylhistidine

Fatigue

The point at which the subject cannot complete a repetition of the bench press without assistance while keeping their head, buttocks, and shoulders in contact with the bench and their feet flat on the floor.

Hypertrophy

An increase in the cross sectional area of a whole muscle or muscle fiber

Neural Adaptation

An adaptation that is characterised by improved activation of motor units through increased frequency of firing, synchronization of firing, increased recruitment, inhibition of inhibitory responses, or some combination of these responses.

Strength

The maximum amount of force a muscle is capable of generating under a given set of circumstances.

II

REVIEW OF LITERATURE

Introduction

The purpose of this section is to review the literature related to the adaptations to resistance training, methods of resistance training, protein degradation following exercise as measured by 3-methylhistidine, and to present a possible model for the protein degradation cycle.

Adaptations to Resistance Training

When a program of resistance training is undertaken the result is an increase in muscular strength. This increase in strength is an adaptation to heavier than normal loads placed upon the body (Fischer and Jensen 1990). These adaptations can be divided into two groups, neural adaptation and increased muscle mass.

Neural Adaptations

The strength increases seen during the first four to six weeks of strength training are predominantly the result of neural adaptations (Moritani and DeVries 1979). These adaptations usually consist of improved motor unit activation, through recruitment of more motor units or more frequent activation of motor units, and greater synchronization of the firing patterns of the motor units (Sale, 1992).

Untrained individuals or those not accustomed to resistance training are often not able

to fully activate all their motor units (Sale, MacDougall, Upton, and Comas, 1983). The use of EMG has allowed scientists to investigate the level of motor unit activation during exercise. Following strength training there is a higher EMG than what was seen before training and the EMG increases correlate with the increases in strength (Moritani and DeVries 1979; Hakkinen and Komi, 1983; Hakkinen, Komi, and Alen, 1985). The above authors feel that this increased EMG represents a change in the facilitatory and inhibitory neural pathways which act at various levels in the nervous system. These changes, which are not completely understood, permit the trained individual to recruit more motor units or fire the motor units at a higher rate (Sale 1992). The theory of improved motor unit activation is the most accepted theory of neural adaptation to strength training, but it is not universally accepted. Rutherford and Jones (1986) using twitch superimposition to measure the level of muscle activation found that all their untrained subjects were able to fully activate their quadriceps during the performance of a maximal isometric contraction. They were not able to measure the rate of motor unit firing using this technique. They suggest that the adaptations seen during the first four to six weeks of training are due to improved coordination and the establishment of new neural pathways in the central nervous system. Rutherford and Jones (1986) go on to suggest that the neural adaptations occur because the muscle is not capable of generating sufficient force to act as a stimulus for hypertrophy until a certain degree of coordination has been established. This is a plausible explanation since the rate of neural adaptation decreases throughout the strength training process as the rate of hypertrophy increases (Moritani and DeVries 1979; Hakkinen and Komi, 1983; Hakkinen, Komi, and Alen, 1985).

Changes in Muscle Mass (Cross Sectional Area)

Even though neural adaptations are the first adaptations that are evoked by a strength training regime cross sectional area is ultimately the limiting factor in the expression of muscular strength (Sale, 1992). The process of muscular hypertrophy and atrophy are affected by the pattern of muscular activity. Increased work can result in hypertrophy while disuse leads to atrophy. These processes are fundamental biological adaptations that offer selective advantages. Atrophy insures that an inactive organism does not have to maintain structures that are metabolically active but physiologically unnecessary. Hypertrophy can help an organism to improve physical capacity (Goldberg, Etlinger, Goldspink and Jablecki, 1975). Exercise induced muscle growth has been attributed to an increase in the size of the muscle fibres. The initial response of the muscle is to increase the fibre size and decrease the extracellular space. This results in an increased cross sectional area of the muscle fibre without an increase in the cross sectional area of the whole muscle (Goldspink, 1992). The increase in fibre area is directly related to an increased myofibril area and myofibril number (MacDougall, 1992). The increased number of myofibrils is thought to be the result of longitudinal splitting of a myofibril into two or more daughter myofibrils (Goldspink, 1992). When a myofibril reaches a critical size and strength, forceful contractions cause a tearing of the connective tissue in the Z-discs. This is transmitted down the length of the myofibril resulting in the production of the daughter myofibrils (Goldspink, 1970).

In humans the cross sectional area of both fibre types has been found to increase with heavy resistance training (MacDougall, 1986). There is greater hypertrophy associated with the type II fibres than the type I fibres. In a study over a period of six months there was a mean increase in fibre size of 33% in type II fibres and an increase of 27% in type I fibres (MacDougall et al., 1979).

There is some debate about whether hypertrophy or hyperplasia is responsible for the

increases in cross sectional area of a whole muscle. Gonyea (1983), one of the main proponents of the hyperplasia hypothesis, has suggested that there is a size limit beyond which gains in cross sectional area become counterproductive. Gonyea feels that because the capillary bed is located outside the muscle fibre an increase in muscle size also increases the diffusion distance. In studies using cats there is hypertrophy in all three fibre types (Gonyea, and Bonde-Petersen, 1978). When the exercise intensity is sufficient to stimulate hypertrophy in FG fibres an increase in fibre number also occurs (Gonyea, 1980). Not all investigators have been able to find fibre splitting. Gollnick, Timson, Moore and Reidy (1981) found increases in muscular size but no increases in fibre number in rats who had undergone surgical ablation. MacDougall (1986) does not believe that hyperplasia occurs to any great extent in humans. He cites a study in which there was no significant difference in the number of muscle fibres in bodybuilders of different experience. Studies using biopsy samples involving humans have observed hypertrophy of fibres but do not mention increased fibre number (Costill et al., 1979; Prince, Hikida and Hagerman, 1976).

The processes of hypertrophy and hyperplasia seem to be capable of occurring at the same time. The mechanisms by which these adaptations occur are not completely understood, as definitive studies are yet to be completed, but the result is an increase in the protein content of the muscle.

Strength Training Methods

Strength training has been practiced since the time of the early civilizations (Atha, 1981). Strength training has evolved to become an integral part of the development of many athletes. Scientists have devoted much time and resources in the pursuit of the optimal methods and systems of strength development.

Schmidtbleicher (1985) has devised a classification of strength training methods. His classification consists of four major categories of strength training. Maximal strength methods,

Sub maximal repetition contraction methods, reactive methods, and strength endurance methods, and is based upon a review of the scientific literature and current strength training practices.

Maximal strength methods are designed to increase maximal force and power output predominantly through neural adaptations. These methods call for short term explosive contractions against very high resistance (90-100% of 1RM).

Sub maximal repetition contraction methods are designed to produce strength gains through muscular hypertrophy. These are the methods that are commonly practiced among bodybuilders and are characterized by many repetitions with as relatively low resistance (60-80% of 1RM).

Reactive methods are designed to increase strength during high speed movements. Plyometrics and explosive movements with a low resistance (30-50% of 1RM) make up this category.

Strength endurance training methods are designed to increase the anaerobic capacity of a muscle. These methods should reflect the energy demands of the sport that is being trained. They generally consist of very high volumes with low resistance (20-60% 1RM).

Stone and O'Bryant (1987) have a five stage periodized model (see table 1).

Table 1. Periodized Model of Strength Training (adapted from Stone and O'Bryant, 1987)

| Phase | Hypertrophy | Strength | Strength and Power | Peaking | Active Rest |
|-----------------|-------------|----------|--------------------|-----------|-------------|
| Sets | 3-10 | 3-5 | 3-5 | 1-3 | ----- |
| Repetitions | 8-12 | 4-6 | 2-3 | 1-3 | ----- |
| Days/week | 3-4 | 3-5 | 3-5 | 1-5 | ----- |
| Times/day | 1-3 | 1-3 | 1-2 | 1 | ----- |
| Intensity cycle | 2-3/1 | 2-4/1 | 2-3/1 | ----- | ----- |
| Intensity | low | high | high | very high | ----- |
| Volume | high | moderate | low | very low | ----- |

This model offers a training program that follows an increased intensity decreased volume pattern through each of the phases. According to the research that the authors have performed on this model it yields greater strength and power increases than other methods.

Schmidtbleicher and Poliquin have a slightly different view on the planning process of strength training. They recommend alternating between stages of two weeks in duration that emphasize adaptation through the volume of training (accumulation) and then through the intensity of training (intensification). There is an inverse relationship between the volume and intensity of training throughout each of their phases. (Poliquin, 1988).

Table 2. Alternating Accumulation and Intensification Phases for Strength Development (Poliquin, 1988).

| Weeks | 1-2 | 3-4 | 5-6 | 7-8 | 9-10 | 11-12 |
|---------------------|------------|------------|------------|------------|-------------|--------------|
| | A | I | A | I | A | I |
| Repetitions | 10-12 | 4-6 | 8-10 | 3-5 | 5-7 | 2-3 |
| Sets | 3 | 5 | 4 | 5 | 4 | 6 |
| Intensity(%) | 70-75 | 82-88 | 75-78 | 85-90 | 80-85 | 90-95 |
| Volume | 30-36 | 20-30 | 32-40 | 15-25 | 20-28 | 12-18 |

It has been accepted amongst strength training practitioners (Poliquin, 1988) and some scientists (Stone ,O'Bryant, and Garhammer ,1981; Reed, Ablack, and McNeely, 1992, Schmidtbleicher 1985) that high volume training with moderate low resistance results in an increase in muscle cross sectional area and lean body mass. It is also accepted that high intensity exercise leads to increased strength (Stone and O'Bryant, 1987; Poliquin, 1988; Atha, 1981; Schmidtbleicher, 1985).

Tesch (1992) points out that most of the evidence for the use of high volume moderate resistance is empirical and is derived from the programs that bodybuilders use. It is not known if bodybuilders show greater muscle mass than competitive weightlifters or powerlifters who tend to train with much higher resistance. Tesch (1992) points out that there is very little if any data available comparing the effects of different combinations of volume and intensity on the muscles hypertrophic response.

There is evidence that the use of 8-12 repetitions per set does indeed produce muscular hypertrophy. Moritani and DeVries (1979) used two sets of 10 repetitions of elbow flexion, at 66% of maximum, three times a week for 8 weeks. They found significant increases

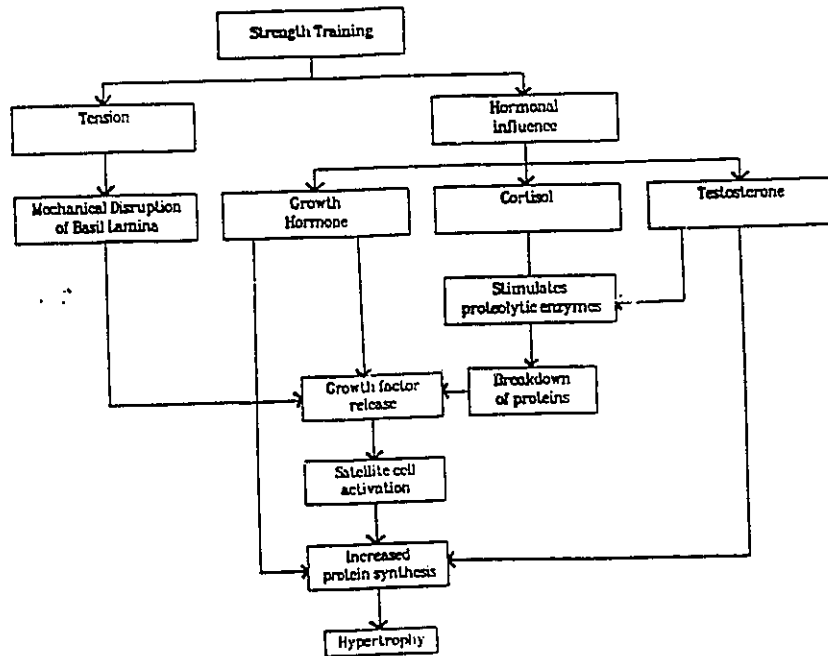
in the muscle girth of the exercised arm following the training period. Luthi, Howald, Claassen, Rosler, Vock and Hoppeler (1986) found an increase of 8.4% in the vastus lateralis following six weeks of training using sets of 8-10 repetitions. Unfortunately the training methodology is only vaguely described so no comparison of intensity or total training volume can be made with other studies. Using one set of 20 repetitions at 50% 1RM and one set of 12 repetitions at 80% 1RM, three times per week for seven weeks, Dons, Bollerup, Bonde-Petersen, and Hancke (1979) found no significant increase in cross sectional area for either experimental group when compared to the controls but there was a tendency towards an increase in gross muscle cross section as measured by ultrasonic methods. The low total volume of work in each training session probably accounts for the lack of results in the 80% protocol. Though the work volume in the 50% protocol is comparable to some other papers reviewed here, the intensity probably was not sufficient to stimulate hypertrophy. MacDougall (1986) used 3-4 sets of 6-8 maximal repetitions for six months. Using biopsy methods he found significant increases in the cross sectional areas of both fast twitch and slow twitch fibres. The intensity that corresponds with 6-8 maximal repetitions is about 79-83% 1RM (Poliquin, 1988). This suggests that a higher intensity of exercise than that which is recommended in the models of Poliquin and Schmidtbleicher or Stone and O'Bryant can produce significant levels of hypertrophy. Dudley, Tesch, Miller, and Buchanan, (1991) found that increased 3 RM strength in the leg press was highly correlated with the increase in resistance used during training and not the volume. They also found that those displaying the greatest increase in resistance and 3 RM also displayed the greatest hypertrophic effect. From this it has been suggested that the intensity of the exercise may be as or more important than the volume of training. MacDougall (1986) states that the main determinant of whether increases in strength and size occur is the intensity of loading on the muscle. In his review on strengthening muscle Atha (1981) points out that 5-6 contractions at 90% of maximal strength will prove effective in increasing muscle size and strength.

It seems that there are two positions formed around the issue of which methods should be used to produce muscular hypertrophy.

- A. high intensity, 80-95% 1RM, and lower volume, 3-6 repetitions for 3-5 sets
- B. lower intensity, 70-75% 1RM, higher volumes, 8-12 repetitions for 3-10 sets,

The Stimulus for Muscular Hypertrophy

Figure 1 represents a model of two possible mechanisms responsible for creating muscular hypertrophy.



The exact mechanisms that are responsible for larger muscle fibres following a program of resistance training are unknown (MacDougall, 1986). There are several ways in which proteins can accumulate during muscular growth. Either the rate of protein synthesis can be increased or the rate of protein degradation can be decreased or both can occur simultaneously.

Two mechanisms may be responsible for the increase in muscle size following strength training. The first is a tension mediated increase in protein synthesis and the second is a hormonal increase in the rate of protein synthesis. These two mechanisms are capable of working independently of each other but would probably function better when their effects are combined.

The tension in the muscle resulting from passive stretch or muscular contraction as the primary stimulus for hypertrophy has been proposed by Goldberg, Etlinger, Goldspink, and Jablecki (1975). This theory has received support from Sola, Christensen, and Martin (1973) who found that adding weight to an innervated anterior latissimus dorsi of a chicken produced greater hypertrophy than stretching a denervated anterior latissimus dorsi. Both of these studies have shown that mechanisms exist that link stretch to increased protein synthesis. This increase in protein synthesis is capable of occurring in the absence of anabolic or catabolic hormones. This is considered to be physiologically advantageous to the organism because the organism can induce selective hypertrophy in overworked muscles without inducing overall developmental growth (Goldberg, Etlinger, Goldspink, and Jablecki, 1975). The activation of the satellite cell is one possible mechanism responsible for this tension induced hypertrophy. The satellite cell is located in a slight depression outside the muscle fibre plasma membrane but within the basal lamina (White and Esser, 1989). The satellite cell contributes new myonuclei to an existing fibre so that the nuclear/cytoplasmic ratio may be maintained (White and Esser, 1989; Appell, Forsberg, and Hollman, 1988). Muscle trauma results in the activation of satellite cells (Bischoff, 1989) but is not necessary for the satellite cell to become

active (Schultz, 1989; Appell, Forsberg, and Hollman, 1988). It has been proposed that stretch or tension may result in a mechanical distortion of the basal lamina allowing the satellite cell to be activated (White and Esser, 1989). The satellite cell is induced into the cell cycle by various growth factors and particularly fibroblast growth factor (Bischkoff, 1989). Fibroblast growth factor is stored in the fibres extracellular matrix and is released as a result of muscle trauma (Yamada, Buffinger, Dimario, and Strohman, 1989). The growth factors produced by the muscles and liver (Deschenes, Kraemer, Maresh, and Crivello, 1991) are probably sufficient to stimulate some hypertrophy through satellite cell activation but once in the cell cycle the satellite cell responds like other muscle cells to hormonal influences (White and Esser, 1989). Vandenburg (1992) has suggested a mechanism whereby mechanical deformation of the cell may alter protein degradation. One such mechanism hypothesizes that mechanical forces may alter the growth factor receptor sites on the muscle cell so as to increase their effectiveness at producing second messengers, such as Na, K, and ATP-ase, that help regulate either protein synthesis or degradation.

A second possible mechanism is a hormone mediated change in the rate of protein synthesis, degradation, or both. These changes may be mediated through actions of hormones such as testosterone, cortisol and growth hormone which are seen to change during strength exercise (Kraemer, 1992). These hormones are capable of acting independently or in conjunction with other hormones and growth factors to elicit hypertrophy. This review is not intended to be an exhaustive review of endocrine physiology rather is meant to give an overview of some of the factors involved in producing muscular hypertrophy.

Hypothalamic gonadotropin releasing hormone stimulates the secretion of luteinizing hormone (LH) and follicle stimulating hormone (FSH) which in turn stimulates the production of testosterone in the Leydig cells of the testes. Testosterone acts as an anabolic hormone through a direct interaction with a cytoplasmic receptor leading to migration of the hormone and receptor to the nucleus resulting in RNA synthesis and muscle protein increases

(Kraemer, 1988).

Cortisol is an adrenal cortex hormone whose production is stimulated by the release of adrenocorticotrophic hormone (ACTH). Cortisol inhibits protein synthesis, plays a role in the conversion of amino acids to carbohydrates and increases protein degradation (Kraemer, 1992).

Growth Hormone (GH) is a peptide hormone synthesized in the anterior pituitary. Growth hormone may bind to the plasma membrane of muscle tissue and have a direct anabolic effect but most of the anabolic effects of GH are probably indirect and are mediated through growth factors (Deschenes, Kraemer, Maresh, and Crivello, 1991).

Accute increases in growth hormone concentration result from strength training. There is no evidence that resistance training results in increased resting levels of GH (Deschenes, Kraemer, Maresh, and Crivello, 1991). Vanhelder, Radoimski, and Goode (1984) found increased GH throughout an exercise period at 85% of 7RM but found no increases during a session with 28% of 7RM. Their results indicate that there is an intensity factor involved in GH response to exercise. The intensity necessary for increasing GH during resistance exercise seems to be less than 85% of 1RM. Kraemer et al. (1991) and Kraemer et al. (1992) found increased GH to exercise sessions that were conduted at 65% of 1RM. They also found that training with 10RM induced a greater increase in GH than training at 5RM. Kraemer et al. (1990) investigated the effects of six different strength training protocols on GH and growth factor responses. They found that the 10 RM protocols elicited the greatest GH increases. There was no pattern to growth factor response and it did not track growth hormone response.

The actions of testosterone and cortisol are closely related. Testosterone is thought to act as an antagonist to cortisol's catabolic properties. It has been suggested that testosterone does this by reducing the binding of the glucocorticoids to cytoplasmic receptors (Waterlow, Garlick, and Millward, 1978). A ratio of testosterone/cortisol has been used as an indicator of the catabolic activity within the tissues (Vervoorn, Quist, Vermulst, Erich, deVries, and

Thijssen, 1990).

Kraemer et al. (1990) investigated anabolic hormone and growth factor responses to six different strength training sessions. They found that serum testosterone increased as a result of each of the exercise sessions. They also found that testosterone increases may occur in rebound fashion late in the recovery period. Testosterone seemed to decrease during the early recovery and then climb after 90-120 minutes of recovery. Kraemer et al. (1991) examined the effects of two strength exercise protocols on anabolic hormone and growth factor responses in men and women. They used 5 RM and 10 RM resistances for 3-5 sets. They found significant increases in testosterone for male subjects for both exercise protocols. Women did not show increases in testosterone in either protocol. Hakkinen, Pakarinen, Alen, Kauhanen, and Komi (1988) found increased testosterone and cortisol during the second of two Olympic weightlifting training sessions performed in one day. Earlier in the day cortisol and testosterone decreased in response to the training session. The decreased hormonal concentrations were explainable by diurnal variations that are greater during the morning than the afternoon. Guezennec, Leger, Lhoste, Aymonod, and Pesqueres (1986) did not find any increase in testosterone to bench pressing at 70% of 1RM but they did find a significant increase in cortisol. Hakkinen, Pakarinen, Alen, and Komi (1985) found significant correlations between strength increases and the testosterone/cortisol ratio. A high ratio occurred during periods of increased strength and a low ratio was found during periods of detraining. This work demonstrates the importance of these hormones in the development of strength and hypertrophy.

There may be a mechanism whereby proteolytic enzymes are stimulated by testosterone and cortisol. Cortisol exerts a catabolic effect through two mechanisms. Cortisol induces muscle wasting by suppressing protein and DNA synthesis (Waterlow, Garlick, and Millward, 1978). Cortisol also increases the activity of enzymes involved in the autolysis of myofibrillar proteins (Kraemer, 1992; Mayer, Shafir, Kaiser, Milholland, and Rosen, 1976;

Mayer, Amin, and Shafir, 1974). Testosterone is usually thought of as an anabolic hormone but it may have a dual function. Testosterone has been found to increase the activity of lysosomal hydrolases in mouse skeletal muscle while the muscle is undergoing hypertrophy (Koenig, Goldstone, and Lu, 1980).

Proteolytic enzymes are responsible for the degradation of the various protein pools in the body. The lysosome contains a wide range of peptide hydrolases which are capable of degrading most, if not all, cellular proteins (Waterlow, Garlick, Millward, 1978). Several lysosomal and cytoplasmic proteases have been found to be specific to myofibrillar proteins. These proteases include cathepsin B and D, Ca²⁺ activated protease, and an unnamed alkaline protease that is bound to the myofibril (Bird, Spanier, Schwartz, 1978). An increased activity of these proteases has been associated with an increased rate of myofibrillar protein degradation (Bird, Spanier, Schwartz, 1978).

Exercise, either through stimulation by hormones or through some other mechanism, causes an increase in proteolytic enzyme activity. Day and Ashmore (1984) compared the activity of cathepsin C and D in normal chicks and those undergoing a passive stretch of the patagialis muscle. The stretched muscle demonstrated hypertrophy and an increased activity of cathepsin C and D. They concluded that the changes in proteolytic enzymes closely parallel changes in muscle weight. Increased cathepsin B activity has been seen in mature rats 24 and 48 hours following exhaustive aerobic exercise (Berry and Berry, 1984). It has been suggested that the increase in cathepsin B and other lysosomal enzyme activity are responsible for weakening the myofilaments thereby allowing for greater protein breakdown (Berry and Berry, 1984). Seene, Alev, and Pehme (1986) examined the effect of six hours of swimming on the autolytic activity in the gastrocnemius of rats. Autolytic activity during the exercise was increased and remained elevated during the six hour period following exercise. This increased proteolytic activity resulted in an increased breakdown of myosin heavy chain components of the muscle fibre. The severity of exercise may play a role in increasing the activity of

proteolytic enzymes. Schott and Terjung (1979) ran rats for 2h but not to exhaustion. They did not find increases in cathepsin D following the exercise session. Exhaustive long duration exercise causes increases in cathepsin D activity (Pilstrom, Vihko, Astrom, and Arstila, 1978; Vihko, Salminen, and Rantamaki, 1979; Salminen and Vihko, 1981) that may last for several days (Vihko, Salminen, and Rantamaki, 1979).

The studies on proteolytic enzyme activity following exercise have all been performed in animals because the removal of the muscle is necessary in order to measure the enzyme activity. Since there seems to be a consensus that exercise does increase proteolytic enzyme activity in animals a similar mechanism is probably responsible for protein turnover in man. Research still needs to be done on the effect of high intensity exercise on proteolytic enzyme activity

The breakdown of proteins that results from increased proteolytic activity may in turn stimulate hypertrophy. The breakdown buildup theory, according to MacDougall (1986), is very popular amongst the "bodybuilding" subculture and others involved in strength training. This theory has received some endorsement from researchers as well. Hickson and Hinkelman (1985) interpret their data on increased protein degradation following exercise as "reflecting a reorganizational aspect of exercise induced growth". This hypothesis is further supported by the work of Tomas, Ballard and Pope (1979) who found a direct relationship between protein degradation and growth.

Viru (1987) has suggested that during the recovery period both the rates of protein degradation and synthesis are increased. This would result in an accelerated rate of protein turnover designed to eliminate the physiologically exhausted structures and insure an improvement in contractile function.

If proteolytic activity in a muscle can be seen as causing a form of muscle damage or trauma then it is plausible that proteolytic activity may result in the activation of satellite cells. This hypothesis may explain both the breakdown build up theory and the removal of

exhausted elements theory proposed by Viru. If this does in fact occur a measure of protein degradation following exercise may indicate that a training stimulus has been reached.

3-Methylhistidine as an Indicator of Skeletal Muscle Turnover

Increased or decreased muscle mass due to exercise or a pathological condition are the direct result of changes in synthesis and degradation. An indicator of skeletal muscle breakdown could be a valuable tool to the clinician and sport scientist who need to know how various treatments are affecting changes in skeletal muscle. Due to its presence in skeletal muscle it was proposed that 3-mh might be a good indicator of skeletal muscle degradation (Young and Munro 1978).

Ballard and Tomas (1983) in reviewing the use of 3-mh as an indicator of muscle protein breakdown indicated that three criteria would have to be met in order for 3-mh to be considered a reliable tool. (A) 3-mh would have to be present exclusively in muscle and at a constant amount. (B) The 3-mh that was released after protein degradation was neither reused for protein synthesis or metabolized. (C) free 3-mh was quickly and quantitatively excreted.

Methylation of histidine in the 1 and 3 positions of the imidazole ring has been well established (Meister, 1965). The methyl group is removed from methionine and by the action of methylating enzymes and is attached to histidine (Young and Munro 1978). 3-mh is found in the globular head of the myosin heavy chain of white (fast) fibres and actin of all fibre types but it is absent from the myosin of slow fibres (Johnson, Harris and Perry 1967). The absence from slow fibres is probably due to a lack of methylating enzymes in these fibres.

3-mh has been found in the portion of one mole 3-mh per mole of actin and one mole 3-mh per mole of white fibre myosin heavy chain (Krzysik, Vergnes and Mcmanus 1971). The predominance of 3-mh in fast twitch fibres may make this amino acid particularly useful in the evaluation of exercise stimuli which require the recruitment of fast twitch motor units. Since

strength training exercises result in greater hypertrophy of fast twitch fibres than slow twitch fibres (MacDougall, 1986) 3-mh may be useful in the assessment of what is happening to this fibre pool following strength training exercise.

The possible contribution of non skeletal muscle protein pools to the excretion of 3-mh was discussed by Rennie and Millward (1983). They suggest that the rate of protein turnover in muscle is as important to the excretion of 3-mh as the size of the protein pool involved. Millward et al. (1980) found that in the rat the turnover of 3-mh in skeletal muscle is too slow to account for more than about half of the total excretion rate. They suggest that in the rat the smooth muscle of the GI tract and skin contribute significantly to the excretion of 3-mh. Long, Dillard, Bodzin, Geiger, and Blakemore (1988) investigated the contribution of the GI tract in the excretion of 3-mh in humans. The subjects involved in the study all required the removal of a section of their GI tract. The group on average had 72% of their GI tract removed. Pre-post measures of 3-mh excretion revealed no significant difference in excretion rate with more than 70% of the GI tract removed. The subjects did not experience any significant change in skeletal muscle mass following the surgery. From these results it was concluded that the GI tract is not a significant contributor to the excretion of 3-mh in humans.

There appears to be no reutilization or metabolism of 3-mh. Young, Alexis, Baliga, and Munro (1972) administered radioactive 3-mh to rats and examined the uptake of this amino acid by tRNA. There was no detectable uptake of the 3-mh. They also found that 93% of the administered 3-mh was excreted in a period of 72 hours. From these results they concluded that 3-mh was not used in protein synthesis, it was not metabolized, and 3-mh is quantitatively excreted in the urine. In humans similar results were achieved by Long, Haverberg, Young, Kinney, Munro, and Geiger (1975) who administered radioactive 3-mh to human subjects. 95%-104% of the administered 3-mh was recovered within 48 hours. From this they also concluded that 3-mh was not metabolized or reutilized.

3-mh is quantitatively excreted in the urine (Long, Haverberg, Young, Kinney, Munro,

and Geiger, 1975; Young, Alexis, Baliga, and Munro, 1972). As long as kidney clearance remains constant during the period of measurement the breakdown of muscle protein will result in a quantitative excretion of 3-mh in the urine (Young and Munro, 1978). During heavy exercise increased hydration inhibits the decreases in glomerular filtration rate seen in lighter exercise (Poortmans, 1984). Even if kidney function was impaired 3-mh could still be used if analysis was performed using blood sampling techniques (Rennie and Millward 1983). Since 3-mh can be measured in either blood or urine the choice of sampling technique depends upon the desired sampling times. Blood samples allow the researcher to obtain readings immediately following the exercise session whereas urine samples may not be obtainable until several hours following exercise.

The evidence seems to indicate that, in healthy subjects, all the conditions necessary for the use of 3-mh as an indicator of skeletal muscle degradation have been met (Ballard and Tomas, 1983; Young and Munro, 1978; Long, Dillard, Bodzin, Geiger, and Blakemore, 1988). Therefore, this can be a useful tool in studying the effects of exercise on skeletal muscle protein breakdown.

3-methylhistidine following exercise

The literature reveals that there is no consensus on the effects of exercise on the excretion of 3-methylhistidine(3-mh). Hickson et al. (1986) performed a study that used a bodybuilding program that consisted of two 36 minute parts. The first consisted of exercises for the chest, shoulders and arms. The second session worked the back and legs. Each part of the program consisted of six exercises performed for three sets of 30 s work followed by 150 s rest, 6-9 repetitions were performed in each set. The intensity was set at 75-80% of the subjects 1RM. They found no significant difference in the excretion of urinary ammonia, creatinine, 3-mh, urea or total nitrogen following exercise, as measured through a 24 hour urine collection. Hickson and Hinkleman (1985) using the same exercise methodology as

above manipulated the dietary intake of protein. They found that exercise increased the 3-mh excretion of the group that received the RDA for protein and of the group that received 3x RDA for protein. Paul et al. (1989) examined the effects of a strength training session on the excretion of 3-mh, creatinine, CK, and myoglobin. They used a group of experienced weightlifters and a group of untrained subjects and had them perform three sets of six exercises to fatigue with 70-80% of 1RM. They found no differences between the groups in 3-mh, creatinine, and the 3-mh/creatinine ratio. They also found that 24 and 48 hour measures of 3-mh were lower than the baseline readings. There were significant increases in myoglobin and CK that were interpreted as meaning that muscle damage had occurred. The increased presence of indicators of muscle damage in the absence of indicators of protein degradation indicate that muscle damage and muscle degradation are different events.

These three studies outlined above used similar intensity ranges and work volumes but observed different results. One explanation for this phenomena comes from the work of Dohm, Israel, Breedlove, Williams, and Askew (1985). They performed a series of studies to determine the time course for changes in 3-mh and the 3-mh/creatinine ratio. They found that there are no diurnal effects on these variables. There appears to be a biphasic response of the urinary 3-mh/creatinine ratio with exercise. There is a decrease in the ratio during the exercise period that is followed by an increased ratio during the recovery period. They have suggested that this effect may explain the mixed results in studies that only use 24 hour urine collections for the analysis of 3-mh. The amount of 3-mh seen in a mixed 24 hour sample is directly related to the magnitude of the decrease in excretion during the exercise period. Serum measurements of 3-mh in this study indicated an immediate increase in 3-mh excretion that persisted for 48 hours following the exercise.

Dohm, Williams, Kasperek, and van Rij (1982) had four subjects perform a standard powerlifting workout consisting of standing press, squats, and curls. Urine samples were collected for the 24 hour period before and after the exercise session. They found a significant

increase in the 3-mh/creatinine ratio following the exercise. This work has been criticized (Paul, DeLany, Snook, Seifert, Kirby, 1989) because the meat intake for these subjects was controlled rather than eliminated. The study by Dohm, Israel, Breedlove, Williams, and Askew (1985) attempted the same procedure with one of the four groups they used and found no effect of meat intake if the intake was controlled. This study may provide a second possible explanation for the lack of results in other studies. Dohm et al. give an incomplete description of the exact nature of their exercise stimulus for this investigation. They do state that it was a "typical" powerlifting workout. A typical powerlifting workout would be performed with intensities of 85% 1RM or above. The total volume of work would be higher than in other studies because 3-5 warm up sets would be performed before the 4-5 work sets would be performed. The warm up sets would normally range in intensity from 60-85% of 1RM. It is not possible to say if this is the sort of workout performed but if it was, the intensity and volume differences could explain the reason increased 3-mh was seen here and not in other studies. Pivarnik, Hickson, and Wolinsky (1989) used an exercise protocol that resembles the hypertrophy stage of the Stone and O'Bryant periodized strength training model. Their subjects trained 12 consecutive days alternating upper and lower body workouts. They performed four sets of ten repetitions with 60-70% of 1RM. They found that increases in 3-mh did not occur until the third consecutive day of strength training. They believe that the reason that Dohm et al. (1982) found a difference in excretion of 3-mh immediately following a strength training session and they did not was because of the longer duration and greater intensities used in the Dohm study.

The idea that 3-mh is a product of skeletal muscle protein degradation has lead some researchers investigate the relationship between 3-mh and post exercise muscle soreness. Horswill, Layman, Boileau, Williams, and Massey (1988) hypothesized that an exercise session that would induce muscle soreness would also increase the release of 3-mh. They found no significant difference in the excretion when the exercise group was compared to the control. Their experimental design, although it did result in post exercise muscle soreness,

probably was not of sufficient intensity to cause muscle degradation. They had their subjects perform three circuit of nine exercises, one circuit at 80% 1RM and one circuit at 60% and 40% of 1RM. They suggest that muscle mass involved in the exercise session may be an important factor in the 3-mh response to exercise.

Not only is muscle mass an important factor in the 3-mh response to exercise but fibre type employed in the exercise also probably plays a major role. If it is accepted that strength exercises performed at moderate to slow speed require the orderly recruitment of muscle fibres according to the size principle (Sale, 1992), then exercise intensities that stimulate maximal recruitment of large fast twitch motor units should result in a greater production of 3-mh than exercise sessions of lower intensity that may not recruit as many fast twitch motor units.

Tyrosine as an Indicator of Protein Degradation

The branched chain amino acid tyrosine has been suggested as another indicator of general muscle protein catabolism (Varrik, Viru, Oopik, and Viru, 1992). This amino acid is neither synthesized nor catabolized by muscle thus its release may reflect net muscle protein catabolism (Davis, Karl, Tegtmeier, Osborne, Klahr, and Harter, 1985).

Several methods of using tyrosine as an indicator of muscle degradation have been employed. The release of ¹⁴C labeled tyrosine from skeletal muscle was used by Davis, Karl, Tegtmeier, Osborne, Klahr, and Harter, (1985) on rats who swam for 2h/day 5days/week for 4 weeks. They found a decreased release of labeled tyrosine from the exercising rats compared to the controls. Dohm, Kasperek, Tapscott, and Beecher (1980) used the dilution of ¹⁴C labeled tyrosine by free tyrosine as an indicator of skeletal muscle breakdown in rats. They found an increased rate of breakdown following treadmill running to exhaustion as well as an increased activity of proteolytic enzymes. The dilution of labeled tyrosine by free tyrosine led these investigators to suggest that free tyrosine could be used as an indicator of protein degradation in the absence of labeled tyrosine. Both of the methods just discussed require

surgical procedures to remove the muscle that is being investigated. Whole muscle analysis is not viable for use on human subjects but changes in serum tyrosine may suggest that protein degradation is occurring. Using rats, Viru (1987) found an increase in serum tyrosine following 10 hours of swimming. The increased tyrosine was accompanied by increased 3-mh and serum total protein. The time course of changes in 3-mh and tyrosine were similar for the first 6 hours after exercise. Varrik, Viru, Oopik, and Viru, (1992) used rats to examine tyrosine release from different muscle fibre types following 10 hours of swimming. They found greater tyrosine release from white fibres than red fibres following the exercise. They also found increases in serum tyrosine following the exercise session. Haralambie and Berg (1976) examined the effects of exercise duration on serum amino acid concentration. They used eight groups of male subjects who exercised for durations of 15-765 minutes. Blood samples were obtained 8-10 min post exercise. Serum tyrosine increased for all groups except the 15 min exercise group. There was a significant correlation between the duration of exercise and serum tyrosine concentration ($r = 0.841$). Since muscle has no tyrosine deaminase they felt that the increased tyrosine represented a degradation of muscle protein caused by the exercise. All the work on tyrosine responses to exercise have focused on long duration exercise. If tyrosine responds to resistance exercise in a similar manner to 3-mh then tyrosine may be a good indicator of catabolic activity in the muscle. Tyrosine changes are not dependant upon food intake (Haralambie and Berg, 1976). This may make tyrosine a more viable indicator of protein degradation than 3-mh for athletes who may not want to follow a meat free diet.

Summary by Section

Adaptations to Resistance Training Training

The adaptations to resistance training that contribute to increased strength are brought about through improved neural functioning and increased muscle cross sectional area. During

the first 4-6 weeks of a strength training program the adaptations are primarily neural. These adaptations include synchronization of motor unit recruitment, more frequent firing of motor units and recruitment of more motor units. Following this six week period hypertrophy of muscle fibres becomes the primary strength adaptation. Hypertrophy occurs in both fast and slow muscle fibres but it occurs to a greater extent in fast fibres than in slow fibres.

Strength Training Methods

Strength training methods have been developed to bring about neural or hypertrophic adaptations. There seems to be a consensus that a high volume of work is necessary to bring about muscular hypertrophy. There are different opinions about the intensity that is necessary for muscular hypertrophy. Some practitioners and researchers feel that sets of 8-12 repetitions with 60-75% 1RM are effective for hypertrophy. Others feel that a resistance of 80-90% 1RM is necessary to stimulate hypertrophy.

The Stimulus for Muscular Hypertrophy

Two mechanisms have been proposed as being responsible for hypertrophy. Passive stretch has been found to induce hypertrophy in a variety of animals. Protein degradation has been suggested as a stimulant to protein synthesis. This latter view receives support from some work that has shown increased protein degradation and synthesis occurring simultaneously following exercise.

3-Methylhistidine as an Indicator of Skeletal Muscle Turnover

3-mh is an amino acid found predominately in white muscle fibres. 3-mh has met the criteria that would make it a valid indicator of skeletal muscle breakdown. It is released from skeletal muscle, it is not metabolized or reutilized in protein synthesis, and it is quickly and

completely excreted in the urine.

3-methylhistidine following exercise

3-mh has been shown to increase in serum and urine following strength training exercises. 3-mh exhibits a biphasic response to exercise. It decreases during the exercise period and then climbs for up to 48 hours post exercise. The magnitude of the decrease during the exercise phase has been proposed as an explanation for the lack of results seen by investigators who only used a 24 hour post exercise urine collection in their work. Intensity of exercise may play an important role in determining the duration and magnitude of 3-mh increases following exercise. The studies that have been conducted using high intensity strength training have shown greater increases than those that have used lower intensity exercises.

Tyrosine as an Indicator of Protein Degradation

Serum tyrosine like 3-mh is not reutilized or metabolized by skeletal muscle. It has been found to increase during exercise and has been suggested as an indicator of protein degradation. The time course of response for tyrosine is similar to that of 3-mh.

III

METHODOLOGY

Introduction

The purpose of this section is to describe the subjects, protocol, dependant variables, independant variables and statistical methods to be employed in this study. The purpose of this study was to observe the effects of two resistance training sessions, one at 70% and one at 85% of maximum, on the degradation of skeletal muscle proteins, in trained male subjects aged 20-35 years, as determined by changes in serum concentrations of 3-mh and tyrosine.

Subjects

Informed consent (Appendix VI) was obtained from 8 trained male subjects between the ages of 18 years and 30 years. The subjects had at least one year of strength training experience and had been incorporating the bench press movement in their training program for at least 6-8 weeks before their involvement in the study. The subjects were volunteers from local powerlifting and weightlifting clubs who, after a presentation on the purpose of the study, decided that they were interested in participating. No compensation or reward was offered to the subjects. It was assumed that subjects with at least one year of strength training would be familiar with correct bench press techniques. This criteria was adopted in order to avoid strength increases from one training testing day to the other because of a steep learning curve in becoming familiar with the exercise protocol. Moritani and DeVries (1979) found rapid strength increases as the result of neural adaptations during the first four to six weeks of strength training (see "neural adaptations" in chapter 2). By requiring a minimum of 6-8 weeks

training before the study, neural adaptations between the testing sessions were minimized (Moritani and DeVries ,1979).

Table 3. Description of Subjects

| Subject | Age | Height | Weight | Experience | 1 RM | 70% 1RM | 85% 1RM |
|---------|-----|--------|--------|------------|------|------------|------------|
| 1 | | | | | | | |
| 2 | | | | | | | |
| 3 | | | | | | | |
| 4 | | | | | | | |
| 5 | | | | | | | |
| 6 | | | | | | | |

Protocol

A nine day protocol was employed in this study. The subjects were required to follow a meat free diet throughout the nine days of the study. Prior to starting an exercise session the subjects were interviewed to determine if they had followed a meat free diet prior to the exercise session. During the period of the study, exercise was limited to the two training sessions and the 1RM test required by the experimental design. The subjects were requested to avoid any sort of activity that would result in a heart rate of 120 beats per minute or greater for more than two minutes at a time. If any of these conditions were violated the subject was required to drop out of the study. They were allowed to remain in the study provided they started over from day 1. Due to the close relationship between the investigator and the subjects daily contact was easily maintained and insured that all the criteria for participation in

the study were met.

Table 4. Chronological Outline

| Day 1 | Day 2 | Day 3 | Day 4 | Day 5 | Day 6 | Day 7 | Day 8 | Day 9 |
|-------|-------|-------|------------|-------------------------------------|-----------|-------|-------------------------------------|-----------|
| 1 RM | | | Baseline A | Baseline B Exercise1 Post 0-1 | Post 24-1 | | Baseline C Exercise2 Post 0-2 | Post 24-2 |

Day 1

A 1 RM on the bench press was determined using the protocol described by Sale and Norman (1982)(see appendix I). The maximum weight lifted was determined to the nearest 5 pounds. The bench press exercise was chosen because it involves a large amount of upper body mass and, according to Horswill, Layman, Boileau, Williams, and Massey, (1988) the mass of muscle involved may have an effect on 3-mh readings . The bench press is a common exercise and is very safe because a spotter is positioned at the end of the bench and can easily grab the bar should the subject have problems during the exercise. Familiarity with the exercise and safe spotting decreases the risk of injury to the subjects.

In order to insure that there were no differences in how the exercise was performed, the subjects had to employ the following technique. Failure to comply with this technique resulted in a warning the first time. If a second technical failure occurred the subjects were considered fatigued and the exercise session was stopped. This procedure was followed for all the exercise sessions.

Bench Press Technique:

Starting Position:

- Lie flat on the bench
- Feet flat on the floor
- Buttocks, shoulders and head are in contact with the bench
- The bar is grasped with a shoulder width or wider grip
- The same grip must be used for all sets

The Ready Position:

- The bar is removed from the rack to arms length
- Inhale deeply to stabilize the upper body
- The legs push against the floor to prevent slipping

The Descent:

- Slowly lower the bar to the chest
- The bar should touch a point level with the nipples

The Ascent:

- The bar is rapidly accelerated off the chest
- The line the bar follows is arced
- The bar should end over the neck
- Exhale as the bar passes through the sticking point
- Press until the bar comes to arms length and arms lock

Important Tips:

- Head shoulders and buttocks should remain in contact with the bench through out the movement
- Don't bounce the weight off the chest this can damage the sternum
- The movement of the bar should be followed with the eyes throughout the movement
- Excessive arching of the back can result in injury and take the buttocks off the bench.
- Don't place the feet on the bench as this decreases the stability of the body

Day 1- Day 3 and Day 7

Baseline measures on 3-mh and tyrosine were established on days 1, 3 and 7. Three days preceding the first sample were required to eliminate the effects of meat consumption on 3-mh excretion (Lukaski, Mendez, Buskirk, and Cohn, 1981). Following the three day

acclimatization period daily variations in 3-mh are negligible (Lukaski, Mendez, Buskirk, and Cohn, 1981; Pivarnik, Hickson, and Wolinsky, 1989). Day 7 was a recovery day between training sessions and provided time for the baseline to be reestablished. Dohm, Isreal, Breedlove, Williams, and Askew (1985) found serum 3-mh to be returning towards normal 48 hours after exercise. 72 hours between exercise sessions was used in this study to ensure adequate time for the re-establishment of baseline values.

The subjects were provided with counselling on meat free protein sources and an information sheet (see appendix II). The subjects were provided with 20 g/day of a protein supplement to insure that adequate amounts of all essential amino acids were consumed.

Day 4

The first baseline reading was measured. 50 ml of venous blood was drawn by a certified medical laboratory technologist. 50 ml was drawn in order to provide 10 ml of plasma for the analysis and 10 ml of plasma as a back up. The subjects were required to lie on their backs for 20 minutes before drawing the blood sample. This period should have allowed enough time for fluid compartments in the body to adjust to the change of body position (Senay and Pivarnik, 1985). Hematocrit measures were made for the baseline and all subsequent samples to evaluate if corrections were necessary to compensate for plasma volume changes that can occur during prolonged exercise.

Day 5 and Day 8

Baseline readings were established following the procedure outlined in Day 4. On Day 5, following the baseline measure, the subjects were randomly assigned to one of two treatments for the first training session. The other training session was performed on Day 8. Treatment (A) consisted of 3 sets of 10 repetitions of the bench press at 70% of 1RM following

one warm up sets of 6 repetitions at each of 50% and 60% of 1RM. A three minute rest was given between each of the warm up and exercise sets. A set was stopped if the subject was unable to finish a repetition without assistance or broke with the required technique. Treatment (B) consisted of 6 sets of 5 repetitions of the bench press at 85% of 1RM following warm up sets of 6 repetitions at 50% and 60% of 1RM. Three minutes rest was given between each set, as timed by a stopwatch. A set was stopped if the subject was unable to finish a repetition without assistance or broke with the technique described above.

The subjects were required to lie on the bench throughout the training session. Immediately following the final set a venous blood sample was obtained. The time was noted so that the time for the 24 hour post sample could be determined.

Day 6 and Day 9

The subjects were required to report to the lab at least 30 minutes before the 24 hour post exercise sample was collected. The subjects lay on their backs for 20 minutes before the collection of the sample. The sample was collected as described in Day 4.

Intensity

The intensities of exercise were set at 70% and 85% 1RM. 70% 1RM is what is commonly recommended during a strength training phase designed to elicit muscular hypertrophy. 85% 1RM is usually used during strength training phases where increases in strength are desired but may also result in muscular hypertrophy (see strength methods in chapter 2). Both strength training sessions in this study were designed to simulate what is currently practiced.

Time of Sampling

Sampling times were set at immediately post and 24 hour post exercise. If the process

of protein degradation is involved in the elimination of physiologically exhausted elements (Virus, 1987), then an immediate post sample can be used to evaluate whether or not this process is active during exercise. This time also represents the peak in serum 3-mh found by Dohm, Isreal, Breedlove, Williams, and Askew (1985). In their work subsequent samples showed decreased 3-mh until the 24 hour sample where it peaked again. The 24 hour sample enabled comparisons to other studies that have generally used only a 24 hour post exercise measure (see chapter 2). The 24 hour sample also provided evidence about whether or not the remodeling process for skeletal muscle includes an increased period of protein degradation after the exercise stimulus had been removed.

3-methylhistidine and Tyrosine

10 ml of serum were frozen at -20 C for storage and transport until it was sent to Hospitals In Common Lab where it was analyzed for 3-mh and tyrosine. A second aliquot of 10 ml of serum was kept frozen at -20C and used as a back up in case of problems during the analysis procedure. 3- Methylhistidine and tyrosine were analyzed with a blind protocol using a Beckman 6300 high performance amino acid analyzer. The average coefficient of variability for this analyzer is 1.03% . The assay recommended by Beckman industries was used for all analyses (see appendix VII for partial details).

Changes in Plasma Volume

Concentrations of 3-mh and tyrosine were corrected for changes in plasma volume. The plasma volume values of the experimental days were adjusted to equal the plasma volume of the corresponding baseline using the following formula: (Harrison, 1985)

$$\% \Delta PV = 100 (hct1-hct2/hct2) \times (1/1-hct1)$$

Where: %PV is the percentage change in plasma volume
 hct1 is the baseline hematocrit
 hct2 is the post exercise hematocrit

The percent plasma volume was then used to determine the change in 3-mh and tyrosine concentration using;

$$V_b/PV_b = V_e/PV_e$$

where: V_b is the baseline reading (for 3-mh or tyrosine)
 PV_b is baseline plasma volume (assumed as unity)
 V_e is post exercise reading (for 3-mh or tyrosine)
 PV_e is post exercise plasma volume (percent difference from baseline)

Statistical Methods

Descriptive statistics, means and standard deviations, were calculated for the subject profiles. A 2 x 3 way ANOVA with fixed effects was used with subjects, intensity, and sampling time as the independent variables, and 3-methylhistidine and tyrosine as the dependent variables. A one way ANOVA was used to determine if there was a difference between the baseline values. A Neuman-Keuls post hoc procedure was performed to determine where the differences occurred.

IV RESULTS

Description of Subjects:

Descriptive statistics and data for each subject are found in table 5. Subjects 2, 3, 4 and 6 have all been involved in competitive weight lifting or powerlifting (cl). The other subjects used weight training as conditioning for other competitive or recreational sports.

Table 5: Descriptive Data and Means for Each Subject

| Subject | Age (years) | Height (cm) | Weight (kg) | Experience (years) | Maximum Bench Press (lbs) |
|-------------|-------------|--------------|--------------|--------------------|---------------------------|
| 1 | 22 | 180 | 88 | 4 | 185 |
| (cl) 2 | 25 | 182 | 102.5 | 6 | 270 |
| (cl) 3 | 25 | 172 | 98.6 | 6 | 270 |
| (cl) 4 | 25 | 177 | 90 | 4 | 300 |
| 5 | 27 | 178 | 85 | 8 | 255 |
| (cl) 6 | 24 | 182 | 110 | 5 | 325 |
| 7 | 25 | 173 | 82.3 | 5 | 230 |
| 8 | 30 | 168 | 75.1 | 2 | 195 |
| Mean | 25.4 | 176.5 | 91.4 | 5 | 253.8 |
| STD | 2.18 | 4.74 | 10.77 | 1.66 | 45.38 |

Baseline Values:

The mean baseline values for 3-methylhistidine (Figure 3, Table 7) were not statistically different ($p=0.09$). Tyrosine baseline readings (Figure 2, Table 6) were not statistically different ($p=0.39$). The baseline mean hematocrit values were not statistically different ($p=0.67$) (B1 44.7 (STD= 1.46), B2 44.8 (STD= 2.44), and B3 44.4 (STD= 1.60)). Data analysis was performed using values that were corrected for plasma volume and uncorrected values.

Correcting the concentrations of 3-mh and tyrosine for changes in plasma volume did not change the results. Therefore, only the corrected data are presented.

Figure 2. Tyrosine Baseline values

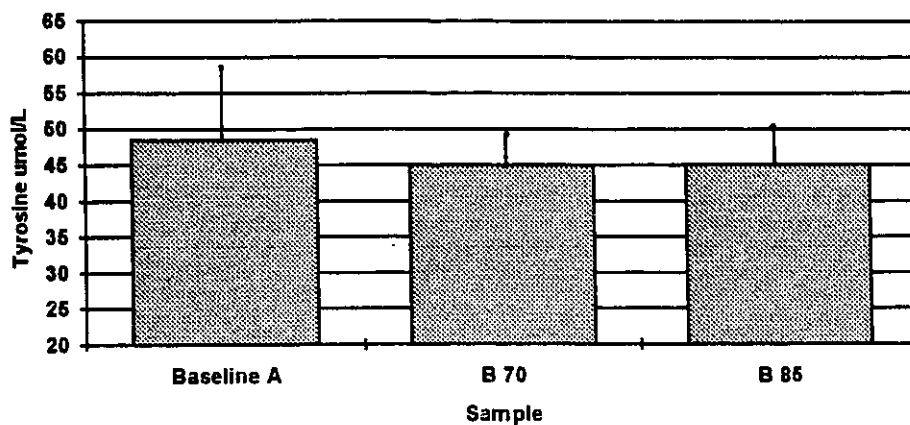


Table 6. Means and Standard Deviation for Tyrosine Baseline Readings

| <u>Sample</u> | <u>Mean</u> (µmol/L) | <u>STD</u> |
|---------------|-------------------------|------------|
| B1 | 48.5 | 9.80 |
| B 70 | 45 | 4.69 |
| B 85 | 45.1 | 5.64 |

Figure 3. 3-Methylhistidine Baseline Values

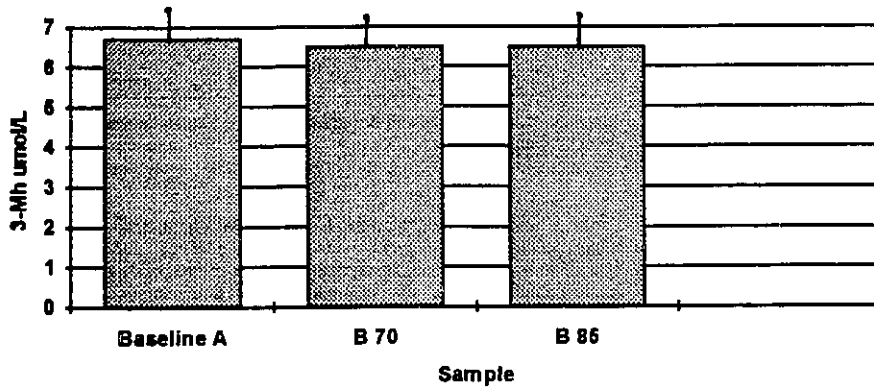


Table 7. Means and Standard Deviation for 3-Methylhistidine Baseline Readings

| <u>Sample</u> | <u>Mean</u> (μmol/L) | <u>STD</u> |
|---------------|-------------------------|------------|
| Baseline A | 6.7 | 0.59 |
| B 70 | 6.5 | 0.55 |
| B 85 | 6.5 | 0.56 |

Post Exercise Values:

1. 3-Methylhistidine

Table 8. presents the post exercise 3-methylhistidine values for the 70% and 85% 1RM test protocols. Figure 3 shows the pattern of 3-mh results throughout the study.

Table 8. Means and Standard Deviations for Post Exercise 3-mh for Each Intensity.

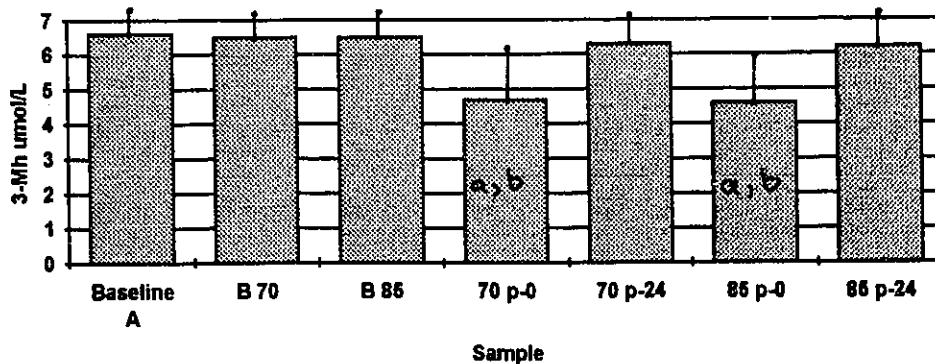
| Protocol | Baseline (μmol/L) | Immediate Post Exercise (μmol/L) | 24 Hour Post Exercise (μmol/L) |
|----------|-------------------|----------------------------------|--------------------------------|
| 70% 1 RM | 6.5(0.55) | 4.74 (1.37) ^{a,b} | 6.27 (0.76) |
| 85% 1RM | 6.5(0.56) | 4.55 (1.47) ^{a,b} | 6.20 (0.87) |

a = significantly different than the baseline $p < 0.05$

b = significantly different than the 24 hour Post Exercise $p < 0.05$

The immediate post exercise sample is significantly lower than both the baseline and 24 hour post exercise sample values. There is a statistically insignificant tendency for the 24 hour post exercise sample to be lower than the baseline values.

Figure 4. 3-Methylhistidine Concentration for each Sample Throughout the Study



a = significantly different than the baseline $p < 0.05$

b = significantly different than the 24 hour Post Exercise $p < 0.05$

When the data is analyzed according to trial order the baseline readings are not different. The immediate post exercise samples are still significantly lower than the baseline values ($p < 0.05$). The trial 1 immediate post exercise value is also lower than the trial 1, 24 hour post exercise value. The trial 2 immediate post exercise value is not different than the trial 2, 24 hour post exercise value. The trial 2 immediate post exercise value is higher than the trial 1 immediate post exercise value. Table 9. and Figure 5 show the results of the data when analyzed according to trial order.

Table 9. Means and Standard Deviations for Post Exercise 3-mh for Each Trial

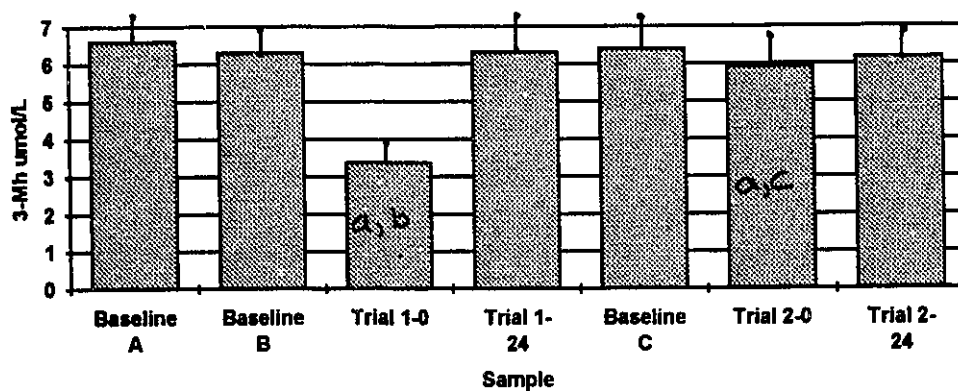
| Protocol | Baseline ($\mu\text{mol/L}$) | Immediate Post Exercise ($\mu\text{mol/L}$) | 24 Hour Post Exercise ($\mu\text{mol/L}$) |
|----------|--------------------------------|---|---|
| Trial 1 | 6.3 (0.51) | 3.38 (0.36) ^{a,b} | 6.31 (0.95) |
| Trial 2 | 6.4 (0.52) | 5.91 (0.50) ^{a,c} | 6.12 (0.65) |

a = significantly different than the baseline $p < 0.05$

b = significantly different than the 24 hour Post Exercise $p < 0.05$

c = different than trial 1 $p < 0.05$

Figure 5. Mean 3-Methylhistidine concentration for each Sample in Chronological Order



a = significantly different than the baseline $p < 0.05$

b = significantly different than the 24 hour Post Exercise $p < 0.05$

c = different than trial 1 $p < 0.05$

2. Tyrosine

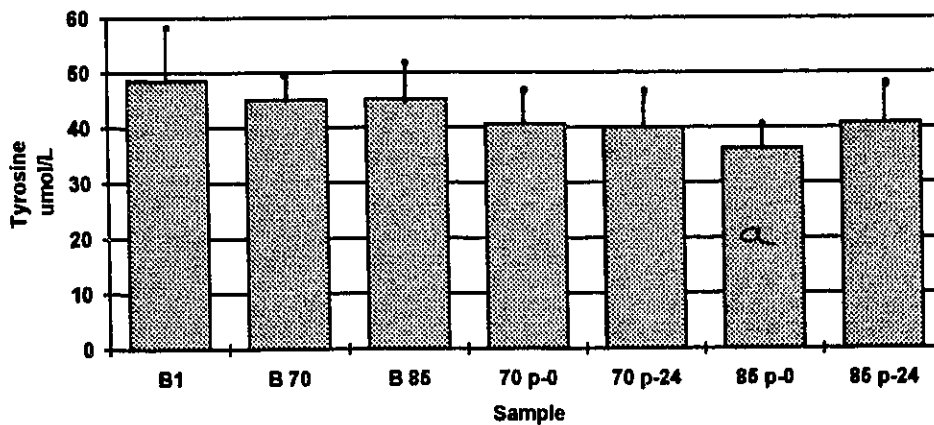
Table 10. Means and Standard Deviations for Post Exercise Tyrosine.

| Protocol | Baseline ($\mu\text{mol/L}$) | Immediate Post Exercise ($\mu\text{mol/L}$) | 24 Hour Post Exercise ($\mu\text{mol/L}$) |
|----------|-----------------------------------|---|---|
| 70% 1 RM | 45.0 (4.69) | 40.6(5.88) | 40.0(5.71) |
| 85% 1RM | 45.1(5.64) | 36.1(3.98) ^a | 40.8 (7.13) |

a = significantly different than the baseline $p < 0.05$

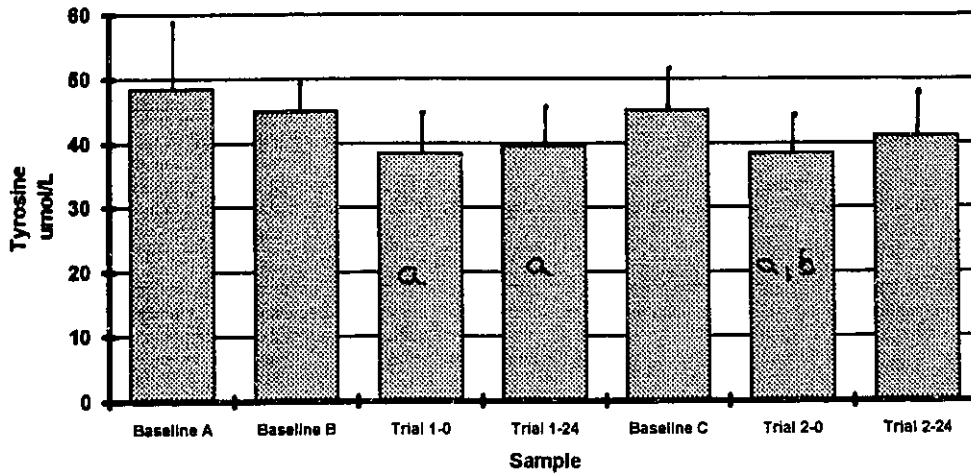
Figure 4. shows the pattern of tyrosine production throughout the study. The only significant change in tyrosine occurred immediately following the 85% 1RM protocol. There was a non significant trend for tyrosine production to be decreased 24 hours following both exercise intensities. There was also a non significant trend for the 85% 1RM protocol to elicit a greater suppression of tyrosine than the 70% 1 RM protocol.

Figure 6. Tyrosine Concentration for each Sample Throughout the Study



a = significantly different than the baseline $p < 0.05$

Figure 7. Mean Tyrosine Concentration for each Sample in Chronological Order



a = significantly different than the baseline $p < 0.05$
 b = significantly different than the 24 hour Post Exercise $p < 0.05$

Table 11. Means and Standard Deviations for Post Exercise Tyrosine for each Trial.

| Protocol | Baseline (µmol/L) | Immediate Post Exercise (µmol/L) | 24 Hour Post Exercise (µmol/L) |
|----------|-------------------|----------------------------------|--------------------------------|
| Trial 1 | 45(4.69) | 38.4(6.5)a | 39.6 (6.02)a |
| Trial 2 | 45.1(5.64) | 38.4(4.44)a,b | 41.1(6.79) |

a = significantly different than the baseline $p < 0.05$
 b = significantly different than the 24 hour Post Exercise $p < 0.05$

Table 4. shows that the immediate post exercise tyrosine values were different than the baseline values for both trials. The 24 hour post exercise sample for the first trial was lower than the baseline reading. The 24 hour post exercise sample for the second trial was not lower than the baseline reading.

V

DISCUSSION

The primary finding was that the present study was not able to support the hypothesis that exercise results in a degradation of muscle protein. The contention that protein degradation increases following exercise has received support from several studies. Dohm, Israel, Breedlove, Williams and Askew (1985) found decreases in 3-mh during the exercise period for humans running at 70-75% of VO₂ max for 90 minutes. The decreases ranged from about 20-35 % of pre exercise values. They also found that urine collections 24 hours following the exercise period, showed an overall increase in 3-mh release. Dohm, Williams, Kasperek and Van Rij (1982), using 24 hour urine collections, found increased 3-mh in humans following 10-12 miles of running. In the same set of experiments they found increased 3-mh in rats run to exhaustion. They did not find a significant increase in humans following a 1 hour weight lifting session. Decombaz, et al. (1979) found increased tyrosine levels following a 100 km run. Haralambie and Berg (1976) found that after 60-70 minutes of aerobic exercise there was a rise in serum tyrosine levels. This rise did not occur in exercise sessions of shorter duration and varying the exercise intensity, between that of a 12 km cross country ski race and a 4 hour military march, did not seem to change the response. They also found a significant correlation between the duration of an exercise session and the changes in tyrosine. The studies that show increased 3-mh in both humans and rats all seemed to involve exercise to exhaustion or for extended periods of time. Kasperek, Conway, Kraveski and Lohne (1992) suggested that the increased energy demand of exercise acts like a catalyst for the degradation of non-active muscle fibres so that their amino acids can be oxidized. This theory has received support from Varrik, Oopik and Viru (1992) who swam rats for 10 hours and then examined glycogen depletion and 3-mh release. The glycogen depleted muscles did not show

an increased rate of 3-mh release during a 2 hour period immediately following the exercise session. The non glycogen depleted muscles showed an increase in 3-mh release for 6 hours following the exercise session. Haralambie and Berg (1976) have suggested that amino acids may not serve as a fuel for energy production but that they may exert regulatory functions in exercise metabolism by acting as transporters of hydrogen into the mitochondria or of carbon compounds into the glucose alanine cycle.

One explanation for the differences in results between the present study and others cited above may be the differences in protocol. The above cited studies all involve aerobic exercise to exhaustion or for extended periods of time. The work period during the present study was probably too short to require the degradation of protein for energy production. The intensity of exercise in this study was supramaximal while in the studies that did find protein degradation in the myofibrils the exercise intensity was maximal or submaximal. In the present study the intensity was high enough that the majority of the energy demand was probably not met through oxidative metabolism, so the catabolism of protein to aid in energy production was probably unnecessary.

Decreases in 3-methylhistidine excretion following exercise, as seen in this study, have also been found by several other researchers. Kasperek, Conway, Kraveski and Lohne (1992) performed several experiments using a muscle perfusion technique to measure 3-mh and tyrosine release in which they ran rats for 200-215 minutes and found that immediately following exercise there was no change in 3-mh. They also found that there was a downward drift in the rate of 3-mh during the 90 minute post exercise period. Unlike the present results, Kasperek and co-workers (1992) found that tyrosine levels were elevated immediately after exercise but then decreased throughout the 90 minute post exercise period. They offer no explanation for this downward drift of both 3-mh and tyrosine. In their other experiment Kasperek and co-workers (1992) measured 3-mh and tyrosine while a muscle was electrically stimulated. They used two trains of 100-ms/s, which they felt was near the maximal stimulation

that could be sustained for 20 minutes. They found decreases of 41% in the rate of 3-mh release and a 20% decrease in the rate of tyrosine release. These decreases are of similar magnitude to that seen in the immediate post exercise readings in the present study. Bylund-Fellenius et al. (1984) also used an electrical stimulation protocol to measure 3-mh release from working muscle. They found a 77% decrease in the rate of 3-mh release in contracting muscle when compared to controls. There was no change seen in tyrosine in their study.

Studies involving strength training and 3-mh have shown variable results. The present study, and the work of Hickson et al. (1986) and Horswill, Layman, Boileau, Williams and Massey (1988) have all shown no change in 3-mh release 24 hours following a weight training session. Paul, DeLany, Snook, Seifert, and Kirby found no change in 3-mh release 24 hours after weight training but did find a decrease in the 3-mh/creatinine ratio.

Hickson and Hinkleman (1985) and Pivarnik, Hickson, and Wolinsky (1989) both found increases in 3-mh release following several consecutive days of weight training exercise. The present study did not use this protocol but there was an insignificant tendency for the decrease in immediate post exercise 3-mh values to be lower during the second training session in the week. The greater release of 3-mh when multiple training sessions were performed in a week may be the result of an alteration in the ratios between catabolic and anabolic hormones. Decreases of testosterone/cortisol ratios are known to occur with repeated bouts of exercise (see chapter 2).

The exercise sessions that consist of work periods of less than 60 minutes (i.e. the weight training and electrical stimulation studies) did not seem to produce increases in 3-mh or tyrosine release. Like in the present study, intensity did not appear to be a factor (see chapter 2 for a review of the intensities in the weight training studies). In the present study 70 and 85% of 1RM were chosen as they represent intensities that are commonly used in strength training. There was no difference between the 70% and 85% protocols for either 3-mh or tyrosine in this study. The lack of difference between the two protocols may have been because the same

muscle fibres were involved in both exercise sessions. When an increase in force generation is required, either more motor units can be recruited or the motor units can be recruited more often (Sale 1992). The difference between the 70% and 85% protocols may not have been great enough to have required a significantly different number of motor units and the required force may have been met simply through increasing motor unit activation frequency.

The recruitment of the same motor units may also partially explain why increases in muscle mass appear to have occurred using either intensity. Moritani and DeVries (1979) used two sets of 10 repetitions of elbow flexion, at 66% of maximum, three times a week for 8 weeks. They found significant increases in the muscle girth of the exercised arm following the training period. Dudley, Tesch, Miller, and Buchanan, (1991) found that increased 3 RM strength in the leg press was highly correlated with the increase in resistance used during training and not the volume. They also found that those subjects who displayed the greatest increase in resistance and 3 RM also displayed the greatest hypertrophic effect. In his review on strengthening muscle Atha (1981) points out that 5-6 contractions at 90% of maximal strength will prove effective in increasing muscle size and strength. If the same muscle fibres were recruited for both levels of intensity, and frequency of contraction does not affect protein degradation, as seems to be the case in this study, then the same fibres should have received stimulation using either intensity.

The purpose of the present research was to observe the changes in protein degradation following selected different exercise intensities. It is not possible on the basis of these results to make conclusive statements about the mechanisms behind an exercise mediated decrease in protein degradation. In fact, the mechanisms said to be responsible for a decreased rate of protein degradation during or immediately following an exercise period are not completely understood. Insulin like growth factors have been found to inhibit proteolysis in mouse skeletal muscle (Gulve, Mabuchi, and Dice, 1989, 1991). Vandeburgh (1992) has outlined several possible mechanisms whereby mechanical deformation of the cell may alter protein

degradation. One such mechanism hypothesizes that mechanical forces may alter the growth factor receptor sites on the muscle cell so as to increase their effectiveness at producing second messengers, such as Na, K, and ATP-ase, that help regulate either protein synthesis or degradation. In support of this hypothesis, he cites unpublished observations that mechanical stimulation of cultured skeletal muscle increases the sensitivity to insulin like growth factors. Hammond, Wieben, and Markert (1979) have found an increased efflux and/or production of endogenous growth factors from myocardial cells undergoing mechanical stimulation. The same mechanism may occur in skeletal muscle since fibroblast growth factor is stored in the fibres extracellular matrix and is released as a result of muscle trauma (Yamada, Buffinger, Dimario, and Strohman, 1989). Vandeburgh (1992) suggests that growth factor mediated alterations in protein synthesis and degradation could be stimulated in the absence of growth factors. Vandeburgh hypothesizes that mechanical forces applied to the external surface of a cell may generate conformational changes in the plasma membrane molecules such as G proteins that are identical to the conformational changes brought about by growth factor binding. Whether this in fact occurs or not is unclear at this time.

There are several scenarios that may explain the apparent decrease in the rate of protein degradation observed immediately following the exercise sessions of the present study. The sampling times and interval between samples in this study make it possible to comment only on the rate of protein degradation at the specific points analysed because the rates of protein degradation between sampling times are not known. The release of 3-mh and tyrosine from the cell may have been altered because of the exercise stimulus. If the rate of release of these amino acids from the cell was decreased during exercise, it might appear that the rate of degradation was decreased. Ballard and Tomas (1983) in reviewing the use of 3-mh as an indicator of muscle protein breakdown indicated that (A) 3-mh would have to be present exclusively in muscle and at a constant amount. (B) The 3-mh that was released after protein degradation was neither reused for protein synthesis or metabolized. (C) 3-mh would have to

be quickly and quantitatively excreted. If 3-mh were to be used as an indicator of muscle protein degradation. Ballard and Tomas, 1983; Young and Munro, 1978; Long, Dillard, Bodzin, Geiger, and Blakemore, 1988 in reviewing the criteria necessary for the use of 3-mh as an indicator of protein degradation felt that all the criteria for the use of 3-mh as an indicator of protein degradation had been met (see chapter 2 for details). The present study involved analysis of individual amino acids. If 3-mh and tyrosine were released and eliminated as parts of larger peptide chains, they may not have shown up during the analysis. If this were the case other authors would probably have encountered similar difficulties and changes in other indicators of protein degradation would have occurred. Measurements of blood urea nitrogen during the pilot work for the present study showed that they did not increase following exercise. Hickson et al. (1986) found no significant difference in the excretion of urinary ammonia, creatinine, 3-mh, urea or total nitrogen following exercise, as measured through a 24 hour urine collection. The lack of change in ammonia, urea and total nitrogen suggests that there was not an increased presence of peptide chains following the exercise session. These factors have not been measured but have been taken into consideration and controlled as much as possible in the design of this project. Therefore, as no reason could be found for the decreased protein degradation, it would seem reasonable to hypothesize that the results of the present study do not support the concept of an increase in the amount of protein degradation following exercise of the type and intensity used in this study.

VI

CONCLUSIONS AND RECOMMENDATIONS

The purpose of this study was to compare the effects of two resistance training sessions, one at 70% and one at 85% of maximum, on the degradation of skeletal muscle proteins, in trained male subjects aged 20-35 years, as measured by changes in serum concentrations of 3-methylhistidine and tyrosine. It was hypothesized that:

1. Strength training exercise would result in an increase in the serum concentration of 3-mh and tyrosine.
2. The 85% 1RM protocol would produce a greater change in 3-mh and tyrosine than the 70% 1RM protocol.

From the data gathered it is concluded that:

1. In trained subjects there was no difference found in the concentration of 3-methylhistidine between 70% and 85% of 1 RM immediately following exercise or 24 hours after exercise.
2. In trained subjects there was no difference found in the concentration of tyrosine between 70% and 85% of 1 RM immediately following exercise or 24 hours after exercise
3. Strength training exercise at 70% and 85% of 1 RM resulted in an immediate decrease in the concentration of 3-mh in the blood.
4. The 3-mh level had returned to baseline seventy two hours following the first exercise stimulus .

Recommendations

Caution should be used in applying the results of this study to the development of a strength training program. There appears to be no difference in the rate of skeletal muscle protein degradation between 70% and 85% of maximum but many questions still remain unanswered. (1) Work is required to determine the effect of work volume on protein degradation. (2) The protein response of other exercise intensities is still unclear, the effects of both volume and intensity on protein degradation in untrained subjects should be investigated. (3) There is a need to determine if the amount of decrease in protein degradation seen in this or the other strength training studies is sufficient to produce whole muscle hypertrophy.

APPENDIX I

Individual Data -3-Methylhistidine (Omol/L)

1. Corrected Data

| Subject | B1 | B70 | B85 | P0-70 | P24-70 | P0-85 | P24-85 |
|----------------|------------|------------|------------|--------------|---------------|--------------|---------------|
| 1 (70) | 6.1 | 6.0 | 6.3 | 3.0 | 5.7 | 5.5 | 5.5 |
| 2 (85) | 5.9 | 5.8 | 5.7 | 5.3 | 5.5 | 3.6 | 4.6 |
| 3 (70) | 7.2 | 7.3 | 7.3 | 3.5 | 7.1 | 6.5 | 7.2 |
| 4 (85) | 6.4 | 6.4 | 6.2 | 6.3 | 6.2 | 3.1 | 6.8 |
| 5 (70) | 6.1 | 6.1 | 6.0 | 3.9 | 5.9 | 5.4 | 5.8 |
| 6 (85) | 6.7 | 6.3 | 6.6 | 6.5 | 6.3 | 3.3 | 6.3 |
| 7 (70) | 7.6 | 7.2 | 7.2 | 3.7 | 7.7 | 6.1 | 7.0 |
| 8 (85) | 6.8 | 6.6 | 6.4 | 5.7 | 5.8 | 2.9 | 6.4 |

2. Uncorrected Data

| Subject | B1 | B70 | B85 | P0-70 | P24-70 | P0-85 | P24-85 |
|---------|-----|-----|-----|-------|--------|-------|--------|
| 1 (70) | 6.1 | 6.0 | 6.3 | 3.2 | 5.9 | 6.1 | 5.9 |
| 2 (85) | 5.9 | 5.8 | 5.7 | 5.3 | 5.5 | 4.9 | 5.6 |
| 3 (70) | 7.2 | 7.3 | 7.3 | 3.6 | 7.1 | 7.0 | 7.2 |
| 4 (85) | 6.4 | 6.4 | 6.2 | 6.5 | 6.6 | 3.7 | 5.8 |
| 5 (70) | 6.1 | 6.1 | 6.0 | 4.1 | 6.2 | 5.8 | 5.7 |
| 6 (85) | 6.7 | 6.3 | 6.6 | 6.5 | 6.4 | 3.5 | 6.3 |
| 7 (70) | 7.6 | 7.2 | 7.2 | 3.8 | 7.7 | 7.1 | 6.8 |
| 8 (85) | 6.8 | 6.6 | 6.4 | 6.5 | 6.5 | 3.2 | 5.9 |

APPENDIX II

Individual Data - Tyrosine (Omol/L)

1. Corrected Data

| Subject | B1 | B70 | B85 | P0-70 | P24-70 | P0-85 | P24-85 |
|---------|----|-----|-----|-------|--------|-------|--------|
| 1 (70) | 39 | 39 | 37 | 31 | 39 | 35 | 35 |
| 2 (85) | 70 | 44 | 43 | 47 | 43 | 29 | 34 |
| 3 (70) | 52 | 52 | 51 | 37 | 30 | 32 | 30 |
| 4 (85) | 48 | 44 | 49 | 44 | 40 | 39 | 51 |
| 5 (70) | 40 | 41 | 42 | 40 | 37 | 37 | 42 |
| 6 (85) | 44 | 44 | 42 | 40 | 46 | 39 | 44 |
| 7 (70) | 50 | 52 | 54 | 49 | 48 | 41 | 47 |
| 8 (85) | 45 | 44 | 43 | 37 | 37 | 37 | 43 |

2. Uncorrected Data

| Subject | B1 | B70 | B85 | P0-70 | P24-70 | P0-85 | P24-85 |
|----------------|-----------|------------|------------|--------------|---------------|--------------|---------------|
| 1 (70) | 39 | 39 | 37 | 33 | 40 | 38 | 38 |
| 2 (85) | 70 | 44 | 43 | 47 | 43 | 40 | 42 |
| 3 (70) | 52 | 52 | 51 | 38 | 30 | 34 | 30 |
| 4 (85) | 48 | 44 | 49 | 45 | 42 | 46 | 43 |
| 5 (70) | 40 | 41 | 42 | 42 | 39 | 40 | 41 |
| 6 (85) | 44 | 44 | 42 | 40 | 47 | 42 | 44 |
| 7 (70) | 50 | 52 | 54 | 50 | 48 | 47 | 46 |
| 8 (85) | 45 | 44 | 43 | 42 | 42 | 41 | 40 |

APPENDIX III

Individual Data- Hematocrit (%)

| Subject | B1 | B2 | B3 | P0-70 | P24-70 | P0-85 | P24-85 |
|----------------|-----------|-----------|-----------|--------------|---------------|--------------|---------------|
| 1 (70) | 43.5 | 44 | 44 | 45 | 44.5 | 46 | 45.5 |
| 2 (85) | 44 | 40 | 42 | 42 | 41 | 47 | 44 |
| 3 (70) | 46.5 | 48.5 | 46.5 | 49 | 48.5 | 48 | 46.5 |
| 4 (85) | 42 | 42.5 | 42 | 42.5 | 43 | 46 | 40 |
| 5 (70) | 46 | 46 | 46 | 47 | 47 | 47.5 | 45.5 |
| 6 (85) | 46 | 46 | 45.5 | 45.5 | 42 | 47.5 | 46 |
| 7 (70) | 44 | 46 | 45 | 46.5 | 46 | 48.5 | 44.5 |
| 8 (85) | 45.5 | 45.5 | 44 | 47 | 46.5 | 48 | 44 |

APPENDIX IV

1RM Test Protocol

Positioning and Instructions to Subject:

Starting Position:

- Lie flat on the bench
- Feet flat on the floor
- Buttocks, shoulders and head are in contact with the bench
- The bar is grasped with a shoulder width or wider grip
- The same grip must be used for all sets

The Ready Position:

- The bar is removed from the rack to arms length
- Inhale deeply to stabilize the upper body
- The legs push against the floor to prevent slipping

The Descent:

- Slowly lower the bar to the chest
- The bar should touch a point level with the nipples

The Ascent:

- The bar is rapidly accelerated off the chest
- The line the bar follows is arced
- The bar should end over the neck
- Exhale as the bar passes through the sticking point
- Press until the bar comes to arms length and arms lock

Important Tips:

- Head shoulders and buttocks should remain in contact with the bench through out the movement
- Don't bounce the weight off the chest this can damage the sternum
- The movement of the bar should be followed with the eyes throughout the movement
- Excessive arching of the back can result in injury and take the buttocks off the bench.
- Don't place the feet on the bench as this decreases the stability of the body

Number of Trials:

5-7 sets of one repetition were performed. With each set the weight was increased. When the subject could not lift the weight without help the preceding successful lift was considered the maximum. 3 min rest, as timed by a stopwatch, were left between sets. The initial weight was set at a weight that the subjects felt they could do 10 times. The weight was increased by 10-15% for the second lift. Smaller increases were used for the subsequent lifts. The size of the increase was dependant upon feedback provided by the subject.

APPENDIX V

Meat Alternatives (Williams, 1988)

It is recommended that you consume 0.8 grams of protein per kilogram of body weight (Williams, 1988). The following Table contains protein sources that do not contain meat.

| Food | Serving Size | Grams of Protein |
|----------------------|--------------|------------------|
| *Milk | 1 cup | 9 |
| *Cheddar Cheese | 1 ounce | 7 |
| *Yogurt | 1 cup | 8 |
| *Eggs | 3 whites | 7 |
| | 1 whole | 7 |
| *Cottage Cheese | 1/4 cup | 7 |
| Peanut Butter | 1 Tbsp | 4 |
| Green Peas | 1/2 cup | 4 |
| Baked Potato | 1 | 3 |
| Navy Beans | 1/2 cup | 7 |
| Wheat Bread | 1 slice | 3 |
| *Macaroni and Cheese | 1/2 cup | 9 |

Those foods that are marked with an asterisk * are complete proteins and can be used by the body in their present form. Those foods without an asterisk* will need to be combined before the body can use them.

Food Combinations:

Pasta with milk or cheese

Cereal with milk

Cheese sandwich

Cheese on Nachos

Cheese on refried beans

Wheat bread and baked beans

Pea soup and toast

Peanut butter sandwich

If there are any concerns about the foods that you want to eat during this study do not hesitate to ask me about them.

APPENDIX VI

Informed Consent Form

SCHOOL OF HUMAN KINETICS THESIS RESEARCH CONSENT FORM

Studies involving human subjects require informed written consent of the participants. I, _____, authorize Ed McNeely (737-4742) of the school of Human Kinetics, University of Ottawa, to administer and conduct the maximal bench press strength test, the 85% maximal bench press strength testing and the 70% maximal bench press strength testing. This study will be conducted under the supervision of Dr. J. Thoden and Dr. A. Reed, School of Human Kinetics (564-9123). Information on the University screening of research is available from M. Loyer, Chairperson, Faculty of Health Sciences Human Research Ethics Committee, 787-6550.

I understand the purpose of this study is to compare the effects of two exercise intensities on serum levels of 3-methylhistidine. I understand that no compensation is offered for participation in this study but that the information obtained from this study will be useful for the design of strength training programs for general and athletic populations.

I understand that prior to the maximal, 85% maximal and 70% maximal testing I will undergo a complete physical warm-up through general exercises. I understand that there will be a pre-screening of blood pressure and that if my resting blood pressure exceeds 150 systolic or 100 diastolic I will not be allowed to take part in the project. I understand that I will be required to fill out a physical activity readiness questionnaire (PAR-Q). I acknowledge that I have at least 1 year of strength training experience. I understand that the maximal strength testing involves lifting progressively heavier weights until a weight that cannot be lifted is obtained. I understand that the 85% maximal testing session involves lifting a weight that is equivalent to 85% of the maximum weight determined in the maximal strength testing session. This weight will be lifted five times followed by a three minute rest. This routine will be performed eight times or until the weight can no longer be lifted. I understand that the 70% maximal testing session involves lifting a weight that is equivalent to 70% of the maximum weight determined in the maximal strength testing session. This weight will be lifted ten times followed by a three minute rest. This routine will be performed four times or until the weight can no longer be lifted. The maximal strength testing session will take approximately 45 minutes, the 85% maximal and the 70% maximal sessions will last approximately 1.5 hours each. All the testing will be completed within a 9 day period. I understand that I will be required to follow a meat free diet for the 9 days required to complete the testing. I understand that I will be required to refrain from strength training during the 9 days of the project except where it is required for testing. I understand that information regarding meat alternatives will be provided to me should I request it.

3-methylhistidine excretion will be measured by amino acid analysis of 10 ml of serum. I understand that a venous blood sample will be drawn on 7 occasions, by a trained medical laboratory technologist. I understand that there may be some discomfort associated with this procedure. The samples will be drawn the day before the first exercise session as well as immediately before and after the 70 and 85 % maximal exercise sessions and 24 hours after the exercise sessions. I understand that I may experience some local muscular fatigue similar to what is experienced during strength training. However, I understand that there are potential risks to some individuals while performing an exercise test, these include episodes of transient light headedness, fainting, nausea, and cramping of the arm, shoulder or chest muscles. I am a healthy, active individual under the age of 30. I do not suffer any chronic medical problems and am physically active. I further understand that it is my responsibility to inform the testing personnel of any injury, illness, infection or other condition which would prevent me from fully participating in this session.

I understand that all information collected will be kept confidential and used in an anonymous form in any written presentation. I understand that I am entitled to see my results and have them explained to me upon the completion of the study.

I understand that I have the right to withdraw from the study at any time and that my refusal to participate or withdraw from this study will have no effect upon any present or future status with The University of Ottawa.

I acknowledge that I have read this form and have had the opportunity to get answers to my complete satisfaction of any questions I had about this form or any aspects regarding my participation in this research project prior to affixing my signature below

SUBJECT: _____

DATE: _____

WITNESS: _____

APPENDIX VII

Procedure for Amino Acid Analysis Using the Beckman System 6300 High Performance Amino Acid Analyzer

Sample Preparation:

1. Add 80 ul of sample to 80 ul of 0.5% SDS in a 1.5 ml microcentrifuge tube and mix with the end of the pipette tip.
2. Incubate at room temperature for 15 minutes.
3. Add 100 ul of sulfosalicylic acid solution (10% solution), mix well on vortex.
4. Add 140 ul of 0.5 M LiOH, mix well on vortex.
5. Centrifuge at 1500 rpm for 14 minutes.
6. Decant supernatant into 1.5 ml microcentrifuge tube and freeze at -70 degrees C.

On the Day of the Assay:

1. Thaw sample and mix well.
2. Centrifuge at 1500 rpm for 15 minutes.
3. Add 100 ul of the sample to 100 ul of dilution solution.
4. Mix well.
5. Samples are ready to load into the analyzer.

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