THE MECHANICAL, HORMONAL AND METABOLIC RESPONSES TO TWO RESISTANCE LOADING SCHEMES

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A Thesis submitted to Auckland University of Technology in partial fulfilment of the degree of Master of Health Science

2004
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DECLARATION

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

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ACKNOWLEDGEMENTS

I would like to express my thanks to the many individuals who have contributed towards this thesis. Firstly to my primary supervisor, John Cronin, thank you for your continual guidance and mentorship. You have provided invaluable assistance with this thesis and have been a pivotal role in my learning throughout my undergraduate and postgraduate studies at AUT. Thanks also to my secondary supervisor, Justin Keogh for your input with this project and my other research activities. Your knowledge and experience have also contributed to this thesis in many ways.

To Christian Cook and Martyn Beaven a sincere thanks for your assistance and continual feedback with the research project and literature review. Thanks also to the many other individuals who contributed to this project in some way; Will Hopkins, Ian Wilcock, my many readers and all the subjects who volunteered to participate.

To the other postgraduate students and staff in the Division of Sport and Recreation, thanks for providing an exceptional atmosphere from which I have been able to learn. I especially wish to thank Anne Kleyn for your love and support the last four years. You have always believed in my potential and encouraged me with all my achievements, and for that I am truly thankful.

This project was supported by a grant from the Health Research Council of New Zealand and by the Foundation for Research, Science and Technology. Ethical approval was provided by the Human Subject Ethics Committee of the Auckland University of Technology, 30th May 2003 – ethics number 03/40.
ABSTRACT

The effective prescription of resistance exercise for strength and power development has been a source of debate amongst practitioners and sport scientists alike. One of the key issues in this area relates to the training load that would best facilitate strength and power adaptation. Heavy loads (>60-70% 1RM) have been traditionally used for maximal strength development by facilitating changes in neural function (strength) and muscle size (hypertrophy). However, many studies have now found lighter load (>45% 1RM) training equally effective in improving both strength and hypertrophy. Similarly, many studies have found heavy load training effective in enhancing various measures of power though lighter loads (e.g. 45% 1RM) are thought to maximise the mechanical power output of muscle. Realising that adaptation depends upon some interaction between the mechanical, hormonal and metabolic stimuli, examining these responses would enhance our understanding of the underlying determinants of strength and power, and thereby improve strength and conditioning practice. Therefore, the purpose of this study was to examine the mechanical, hormonal and metabolic responses to equal-volume light and heavy loading schemes.

Eleven males (mean 26.6 ± 6.7 years; mean 79.0 ± 8.1 kg) with a minimum of 12 months weight training experience each performed two exercises (Smith squat and supine squat) at a light (45% 1RM) and heavy (88% 1RM) intensity. The light scheme consisted of eight sets of six repetitions, with six sets of four repetitions performed in the heavy scheme. Rest periods of three and four minutes respectively were used. Saliva sampling was used to determine the hormonal (cortisol and testosterone) and metabolic (lactate) responses. Samples were collected at rest (pre-), immediately after the first exercise (mid-), at the conclusion of the second exercise (P0) and every 15
minutes thereafter for one hour (P15, P30, P45, P60). Mean values for all variables were analysed with a paired sample T-test. Chances that the true effects were substantial (% and qualitative) were also calculated.

No significant (P>0.05) difference in total forces was found between schemes; however, the light scheme produced significantly greater total time under tension (36%), total work (37%) and total power output (115%). Total impulse (38%) was the only variable found to be greater in the heavy scheme. A decrease in testosterone (TST) was observed in the heavy scheme (-4 to -29%) with no significant changes found across the light scheme (1 to 12%). Cortisol decreased in the light (-6 to -30%) and heavy (-14 to -44%) schemes until P45. An increase in the TST/cortisol ratio was observed in both the light (17 to 49%) and heavy (2 to 44%) schemes. Both loading schemes resulted in similar increases in lactate (0.3 to 1.0nmol/l). Equating two schemes by volume resulted in differential responses, many of which favoured the lighter scheme in terms of mechanical, hormonal and metabolic outputs. These findings suggest that load or intensity employed may be not as important as initially proposed and that other factors (e.g. volume, technique) may explain the similar strength and hypertrophy adaptation reported in studies comparing light and heavy schemes.
CHAPTER ONE - INTRODUCTION

Muscular strength and power are important components of many athletic pursuits and everyday activities (Komi & Hakkinen, 1988). However, debate exists as to the resistance training methods that would best develop strength and power. One key issue is which load, expressed as a percentage of one repetition maximum (% 1RM), best facilitates strength and power development. Improvements in maximal strength are largely attributed to changes in muscle cross-sectional area (CSA) or size (hypertrophy) and improved neural function (Bloomer & Ives, 2000; Sale, 1992). Loading the muscle with intensities greater than 60-70% 1RM is thought fundamental to the development of maximal strength and an important stimulus for hypertrophy (MacDougall, 1992; McDonagh & Davies, 1984). Although the exact mechanism for stimulating protein synthesis has yet to be determined, MacDougall (1992) stated, that loading intensity is the main factor that determines whether or not an increase in strength and/or size will occur. In strength trained athletes even greater loading intensities (85-100% 1RM) are thought critical for the improvement of maximal strength (Komi & Hakkinen, 1988).

The importance of heavy training loads in inducing maximal strength and hypertrophic changes however, may be questioned in relation to recent research findings. Maximum power training using a load of 30% 1RM was found to be equally effective in enhancing a variety of performance measures (including bench press and squat 1RM strength), as compared to combined weight training (75-85% 1RM) and plyometric training (Lyttle, Wilson & Ostrowski, 1996). Using a similar methodology high force training (80-85% 1RM) and high power training (30% 1RM) were found to be equally effective in improving ¼ squat 1RM and mid-thigh pull 1RM strength.
(Harris, Stone, O'Bryant, Proulx & Johnson, 2000). Other studies have also found light and heavy load training equally effective in improving a number of performance measures, including 1RM strength and hypertrophy (Dahl, Aaserud & Jensen, 1992; Moss, Refsnes, Abildgaard, Nicolaysen & Jensen, 1997; Schmidtbleicher & Buehrle, 1987). It would seem from this literature that load is not as important as initially proposed for the improvement of maximal strength.

In terms of power development lighter intensities (e.g. ~45% 1RM) are thought to provide the optimal training stimulus as such loads maximise the mechanical power output of muscle (Newton, Kraemer, Hakkinen, Humphries & Murphy, 1996; Stone et al., 1998). Despite this, many studies have found heavy load training (> 70% 1RM) as effective as lighter load training in the development of muscular power. Adams, O’Shea, O’Shea and Climstein (1992) found heavy load (70-100% 1RM) squat training equally effective in improving jump performance compared to light load (body weight) plyometric training. Similarly, Lyttle and colleagues (1996) found heavy load (6-10RM) training to be equally effective as light load (30% 1RM) training in enhancing a variety of performance measures such as jumping, throwing, cycling and lifting. Other studies have also found heavy load training effective in improving various measures of power performance (Cronin, McNair & Marshall, 2001b; Fatouros et al., 2000; McBride, Triplett-McBride, Davie & Newton, 2002; Schmidtbleicher & Buehrle, 1987; Wenzel & Perfetto, 1992). Such findings further confound understanding in this area regarding the stimulus afforded by different resistance exercise programmes and the adaptive response of the body to such stimuli.
Many of the discrepancies in the literature may be explained by research failing to equate training volume (total repetitions x load) between treatment groups. As such any reported changes in maximal strength and/or power may simply reflect differences in volume between interventions, rather than the specific kinematic and kinetic characteristics associated with a particular training scheme. Unfortunately, the greater majority of research in this area does not equate volume between schemes (Bauer, Thayer & Baras, 1990; Fatouros et al., 2000; Harris et al., 2000; Jones & Rutherford, 1987; Jones, Bishop, Hunter & Fleisig, 2001; Lyttle et al., 1996; Schmidtleicher & Buehrle, 1987; Weiss, Coney & Clark, 1999; Weiss & Relyea, 2001; Wenzel & Perfetto, 1992; Wilson, Newton, Murphy & Humphries, 1993) and consequently many of the suggested conclusions and applications are fundamentally flawed.

In terms of the underlying determinants of strength and power, three stimuli (mechanical, hormonal and metabolic) are thought important (Enoka, 2002; Jones, Rutherford & Parker, 1989). The mechanical stimulus (e.g. high tension, stretch, time under tension) afforded by resistance exercise is thought to be the most important stimuli for training-induced adaptations to occur (Enoka, 2002; McDonagh & Davies, 1984). Many studies have investigated the repetition kinematics and kinetics associated with different resistance exercises and loads (Baker, 2001; Baker, Nance & Moore, 2001; Cronin, McNair & Marshall, 2001a; Murphy, Wilson & Pryor, 1994; Newton et al., 1996; Newton et al., 1997) and as such much is known about the mechanical response of a single repetition. However, to our knowledge no studies have examined these effects over multiple sets and/or exercises. As a typical training session is characterised by multiple repetitions, sets and exercises, such an analysis
appears fundamental to improving our understanding of how mechanical stimuli contribute to strength and power development.

Endocrine responses to resistance exercise are also thought important in the development of strength and power (Hakkinen, 1989). The interaction between the primary anabolic (e.g. testosterone and growth hormone) and catabolic (e.g. cortisol) hormones regulate the balance between protein synthesis and degradation (Deschenes, Kraemer, Maresh & Crivello, 1991; Kraemer, 1992a). Resistance exercise is known to elicit acute and chronic changes in blood hormone levels, thereby mediating the long term process of muscle tissue growth (Kraemer, 1992a; Kraemer, 1992b). As different training protocols produce differential hormonal responses, program design plays an important role in modulating muscle growth. However, few studies have examined the acute hormonal response to loading schemes and/or techniques commonly used to improve muscular strength and power (e.g. maximal strength and light load power training). Such an analysis would also add to our understanding of the mechanisms underlying strength and power development.

The metabolic response to resistance exercise may also be important for strength and to a lesser extent power development (Abernethy, Jurimae, Logan, Taylor & Thayer, 1994; Enoka, 2002). The stimulus for strength and power adaptation may result from changes in the level of circulating metabolites (e.g. lactate, creatine kinase, glycogen, etc.) as a consequence of resistance exercise and the effect of these metabolites upon the muscular environment. Such changes may contribute to the training stimulus by stimulating greater anabolic hormone release and/or greater motor recruitment (Carey Smith & Rutherford, 1995; Takarada et al., 2000), and as a result of greater muscle damage (Ebbeling & Clarkson, 1989). Again, little is known regarding the metabolic
responses imposed by different strength and power schemes and how these differences relate to subsequent adaptation.

**Purpose Statement**

The purpose of this thesis is to provide an overview of knowledge regarding the mechanical, hormonal and metabolic responses to resistance training and the response of the human body to such stimuli, in terms of maximal strength and power adaptation. First, literature examining the mechanical, hormonal and metabolic responses to resistance exercise will be reviewed. Second, the mechanical, hormonal and metabolic response to a light and heavy loading scheme of equal volume will be investigated.

**Aims**

The purpose of this study is to compare the mechanical, hormonal and metabolic responses to a light (45% 1RM) and heavy (88% 1RM) loading scheme, equated by total load (total repetitions x load) lifted.

**Hypothesis**

It is hypothesised that the mechanical, hormonal and metabolic responses to each loading scheme would differ even though training volume was equated.

**Significance**

Muscular strength and power are important components of everyday fitness and athletic performance. It is well recognised that strength and power are vital components for sporting success for many athletes, elite or otherwise. Developing such qualities is also important for those individuals wanting general health benefits and others seeking to improve functional performance (i.e. elderly, injured, etc.).
Thus, improving the prescription of resistance exercise for strength and power adaptation would appear of considerable importance for many populations. In developing a better understanding of the responses to different loading schemes we may be able to better understand the stimuli for training-induced adaptations. As a result we may prescribe resistance exercise more effectively and in doing so enhance strength and conditioning practice. This study may also provide a framework for further research in this area.

**Notes to reader**

This thesis is presented as two major chapters, a review that summarises the literature in this area and then the subsequent experimental chapter. Please note that some of the information provided in this thesis appears repetitive in parts, which is due to the chosen format for this thesis application. Nonetheless, this thesis fulfils the AUT Master of Health Science guidelines for thesis submissions.

**Authorship Contribution**

The contributions of the authors to the literature review and research paper submitted within this thesis are as follows:

The mechanical, hormonal and metabolic profile of two resistance loading schemes.

Crewther, B., (80%) Cronin, J., (10%) Keogh, J (5%) and Cook, C. (5%).

Potential stimuli for training-induced adaptation: Parts 1 and 2 mechanical, hormonal and metabolic stimuli.

Crewther, B., (80%) Cronin, J., (10%) Keogh, J (5%) and Cook, C. (5%).
INTRODUCTION

Muscular strength and power are important components of many athletic pursuits and everyday activities (Komi & Hakkinen, 1988). One key issue is which load, expressed as a percentage of one repetition maximum (% 1RM), best facilitates maximal strength and power development. Improvements in maximal strength are largely attributed to changes in muscle cross-sectional area (CSA) and neural function (Bloomer & Ives, 2000; Sale, 1992). Training programmes designed to increase muscle CSA or hypertrophy schemes are characterised by loads of 60-70% 1RM (MacDougall, 1992; McDonagh & Davies, 1984), whilst programmes designed to improve strength through enhanced neural function or neuronal schemes are typified by intensities of 85-100% 1RM (Komi & Hakkinen, 1988). However, recent research would suggest that such a perspective is somewhat simplistic given that lighter load (<45% 1RM) training has been found equally effective in improving strength and/or hypertrophy compared to heavy load training (Dahl et al., 1992; Harris et al., 2000; Lyttle et al., 1996; Moss et al., 1997; Schmidtbleicher & Buehrle, 1987).

In terms of power development lighter loads (e.g. 45% 1RM) are thought important based upon the mechanical power output of muscle (Newton et al., 1996; Stone et al., 1998). Despite this, many studies have found heavy load training (> 70% 1RM) effective in enhancing various measures of muscular power (Adams, O'Shea, O'Shea & Climstein, 1992; Fatouros et al., 2000; Hoff & Almasbakk, 1995; McBride et al., 2002; Schmidtbleicher & Buehrle, 1987). Therefore, it would seem from literature
that debate exists as to the training loads that would best facilitate improvement in strength and power. With many studies in this area further characterised by different movement and contraction types, which technique maximises strength and power adaptation above any other is also a contentious issue. Such issues illustrate an apparent lack of understanding regarding the stimulus afforded by different training methods and the adaptive response of the body to such stimuli.

In terms of the adaptations associated with resistance exercise, three stimuli (i.e. mechanical, hormonal and metabolic) are thought fundamental to strength and power development (Enoka, 2002). The mechanical stimulus (e.g. high tension, time under tension, work, etc.) afforded by resistance exercise is thought to be the most important stimuli for adaptations to occur (McDonagh & Davies, 1984). Many studies have investigated repetition kinematics and kinetics associated with different resistance exercises and loads (Baker, 2001; Baker et al., 2001; Cronin et al., 2001a; Murphy et al., 1994; Newton et al., 1996; Newton et al., 1997), and as such much is known about the mechanical response of single repetitions. However, to our knowledge no studies have systematically examined these effects over multiple sets and/or exercises. Considering the inherent nature of a typical training session (i.e. multiple repetitions, sets and exercises) such an analysis appears fundamental to improving our understanding of the kinematic and kinetic response to a single training session and thus, how mechanical stimuli contribute to strength and power development.

Endocrine responses to resistance exercise are also thought important in the development of strength and power (Hakkinen, 1989). The interaction between the primary anabolic (e.g. testosterone and growth hormone) and catabolic (e.g. cortisol)
hormones regulate the dynamic balance between protein synthesis and protein
degradation (Deschenes et al., 1991; Kraemer, 1992a). Resistance exercise has been
shown to elicit acute and chronic changes in circulating hormone levels, thereby
mediating the long-term physiological process of muscle growth (Kraemer, 1992a;
Kraemer, 1992b). However, few studies have examined the acute hormonal response
to different training schemes commonly used for strength and power development
(e.g. maximal strength and maximal power). Such an analysis would seem
fundamental in understanding how various training schemes affect the hormonal
milieu of the muscle and adaptations thereafter.

The metabolic response to resistance exercise may also be important for strength and
to a lesser extent power development (Abernethy et al., 1994; Enoka, 2002). The
stimulus for adaptation may result directly from changes in the metabolic (e.g. lactate,
glycogen, creatine kinase, ammonia, etc.) environment as a consequence of resistance
exercise and the effect of these metabolites upon the muscular environment. The
potential mechanisms for adaptation include an increase in anabolic hormone release
and/or enhanced motor unit recruitment (Carey Smith & Rutherford, 1995; Takarada
et al., 2000). Changes in the metabolic environment (e.g. lactate, hydrogen ions, etc.)
may also result in greater muscle damage (Ebbeling & Clarkson, 1989) further
contributing to the training stimulus. Again, little is known regarding the metabolic
stress imposed on the system by different strength and power schemes, and how these
differences relate to subsequent adaptation.

Given the importance of these mechanical, hormonal and metabolic factors, it is
disconcerting to note that not much is known about how these factors and their
interaction, might affect the development of maximal strength and power. As such a true understanding of the adaptations elicited by various resistance training protocols are for the most part not well understood. The purpose of this review therefore is to discern how various resistance-training movements and loading schemes differ in terms of their mechanical, hormonal and metabolic responses. Where possible the influence of these factors will be differentiated in order to determine those mechanisms underpinning strength and power development. It is hoped such a treatise will enable better understanding of how best to integrate these stimuli, to optimise the development of strength and/or power, and as a consequence improve strength and conditioning practice.

**Mechanical response to resistance exercise**

There is no doubt that the mechanical stimuli associated with resistance training are necessary prerequisites for strength and power adaptation to occur. It is thought that training loads need to be maximal or near maximal and of sufficiently long duration if strength and muscle cross-sectional area (CSA) are to increase (Komi & Hakkinen, 1988; MacDougall, 1992). The distance over which that force acts (work) may also be an important stimulus for changes in strength and muscle CSA to occur (Moss et al., 1997; Stone et al., 1998). In order to adequately train power, movements producing high power output are the training method of choice (Newton et al., 1996; Newton et al., 1997; Stone et al., 1998). As the training load used often determines these responses, the prescription of load becomes possibly the most important variable to consider for strength and power adaptation. However, due to discrepancies within research the effect of load might be less important than the kinematics and kinetics associated with that load. That is, how a load is moved will have a varied effect on the
kinematics and kinetics of that movement and thereafter adaptation. This section will
discuss the kinematics and kinetics associated with different loads, contractions and
techniques. In the first instance an analysis of the literature profiling a single
repetition will be discussed and thereafter, multiple repetition and set kinematics-
kinetics will be addressed.

**Kinematics and kinetics of a single repetition – load effects (% 1RM)**
The relationship between force and velocity is important for understanding movement,
as all movement is a combination of these two qualities. According to the concentric
force-velocity relationship of muscle, the ability to generate force increases at slower
contraction velocities during shortening, as there is a greater number of cross-bridges
available for attachment and more time for these cross-bridges to generate tension
(Gregor, 1993; Herzog, 2000). A typical concentric force-velocity curve, obtained
from isolated muscle, is shown in Figure 1.

![Concentric force-velocity relationship of muscle. Modified from Edman (1992).](image)

**Figure 1:** Concentric force-velocity relationship of muscle. Modified from Edman (1992).
The ability to produce greater forces at slower contraction velocities is further related to changes in the internal viscosity of muscle during movement. With an increase in shortening velocity there is greater fluid resistance in the sarcomere, requiring greater internal force to overcome and thereby resulting in lower total force production (Enoka, 2002). Accordingly, the force generation capabilities of muscle is greater at zero velocity (isometric action) compared to that achieved during shortening velocities (concentric actions).

A number of studies have examined the effect that load (% 1RM) has on the force characteristics of muscle (Cronin & Crewther, 2003; Cronin et al., 2001a; Cronin, McNair & Marshall, 2003; McBride, Triplett-McBride, Davie & Newton, 1999; Murphy et al., 1994; Newton et al., 1997), which clearly show that force output increases with an increase in load as per the concentric force-velocity relationship (see Table 1). This is true for both mean and peak force output irrespective of the exercise performed. As expected greater force outputs are associated with lower body exercises (~ 2700 N) compared to upper body exercises (~ 950 N) across the various studies (see Table 1). This is likely to be a function of the larger muscle groups in the lower limbs, multiarticular nature of many lower body exercises and the longer distances travelled (e.g. squats). Nonetheless, there appears to be a relationship (force-load relationship) between the load lifted (% 1RM) and the amount of force generated, with an increase in load producing a concomitant increase in force output. It can be observed from Table 1 that on average a 10% increase in load resulted in a 10% increase in force output for the different exercises assessed.
Table 1: Effect of load (single repetitions) upon force output.

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<th>Maximum - load</th>
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<td>30% 1RM CO</td>
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<td>~420-560 N</td>
<td>90% 1RM (PF) - all techniques</td>
<td>~900-1150 N</td>
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<td></td>
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<td>15% 1RM (PF) - all techniques</td>
<td>~710-860 N</td>
<td>90% 1RM (PF) - all techniques</td>
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<td>90% 1RM (PF) - all techniques</td>
<td>2687 N</td>
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<td>BW (PF) - sprinters</td>
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<td>90% 1RM (PF) - sprinters</td>
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<td>27 Males - UT</td>
<td>CO and RB bench press and bench press throws (30, 40, 50, 60, 70, 80% 1RM)</td>
<td>BW (PF) - OL 30% 1RM (PF) - all techniques</td>
<td>2022 N</td>
<td>90% 1RM (PF) - OL 80% 1RM (PF) - all techniques</td>
<td>3717 N</td>
</tr>
<tr>
<td>Cronin and Crewther (2003)</td>
<td>10 Males - T</td>
<td>Supine squats (30, 60, 90% 1RM)</td>
<td>30% 1RM (PF)</td>
<td>970 N - ECC</td>
<td>90% 1RM (PF)</td>
<td>2778 N - ECC</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>943 N - CON</td>
<td></td>
<td>2214 N - CON</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>30% 1RM (MF)</td>
<td>804 N - ECC</td>
<td>90% 1RM (MF)</td>
<td>1962 N - ECC</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>847 N - CON</td>
<td></td>
<td>1937 N - CON</td>
</tr>
<tr>
<td>Cronin, McNair and Marshall (2003)</td>
<td>27 Males - UT</td>
<td>CO and RB bench press and bench press throws (30, 40, 50, 60, 70, 80% 1RM)</td>
<td>30% 1RM(MF) - all techniques</td>
<td>~260 N</td>
<td>80% 1RM (MF) - all techniques</td>
<td>~680 N</td>
</tr>
</tbody>
</table>

NOTE: CO, concentric only; EO, eccentric only; CON, concentric phase; ECC, eccentric phase; RB, rebound; CMJ, counter movement jump; MF, mean force; PF, peak force; BW, bodyweight; PL, power lifters; OL, olympic lifters; T, trained; UT, untrained.
Although greater forces are developed with heavier training loads (force-load relationship), research in this area must be interpreted with caution. Comparing findings between studies is made difficult due to differences in the pieces of equipment (e.g. force platform, linear transducers, etc.) and mode of dynamometry (e.g. isoinertial, isotonic) used to determine force output. Some studies (Cronin & Crewther, 2003; McBride et al., 1999) have also employed techniques where the load or individual is projected (i.e. ballistic) whilst others have used more traditional (i.e. non-projection) methods (Murphy et al., 1994). With ballistic techniques producing enhanced kinematics and kinetics compared to traditional movements (Newton et al., 1996) the technique employed also warrants consideration, as would the type of contraction assessed (e.g. concentric v eccentric). The influence of technique and contraction type will be addressed in later sections.

As heavy loads are characterised by slower velocities (concentric force-velocity relationship) the use of such loads would also result in longer contraction durations. This relationship is directly observable in Table 2 where heavy loads have been shown to maximise time under tension (TUT) during a single repetition (Cronin & Crewther, 2003; Cronin et al., 2001a; Newton et al., 1997). It can be observed that on average, a 10% increase in load resulted in a 14% increase in TUT for the various exercises assessed (see Table 2). These findings do not however, reflect differences in training tempo as the movements in these studies were performed with both maximal effort and intent. A given load may be moved with maximal or submaximal effort and accordingly, will have a significant impact upon resultant load velocities and the duration of contraction thereafter. Again, different techniques have been assessed in these studies (e.g. traditional v ballistic) which may further influence TUT.
Table 2: Effect of load (single repetitions) upon time under tension, impulse and work.

<table>
<thead>
<tr>
<th>Author/s</th>
<th>Subjects</th>
<th>Protocols (load)</th>
<th>Minimum - load</th>
<th>Value</th>
<th>Maximum - load</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time under tension</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Newton et al. (1997)</td>
<td>17 Males - T</td>
<td>CO and RB bench press throws (15, 30, 45, 60, 75, 90% 1RM)</td>
<td>15% 1RM CO</td>
<td>0.45 sec</td>
<td>90% 1RM CO</td>
<td>2.23 sec</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>15% 1RM RB</td>
<td>0.33 sec</td>
<td>90% 1RM RB</td>
<td>1.54 sec</td>
</tr>
<tr>
<td>Cronin, McNair and Marshall (2001)</td>
<td>27 Males - UT</td>
<td>CO and RB bench press and bench press throws (30, 40, 50, 60, 70, 80% 1RM)</td>
<td>30% 1RM - all techniques</td>
<td>0.61-0.79 sec</td>
<td>80% 1RM - all techniques</td>
<td>1.12-1.23 sec</td>
</tr>
<tr>
<td>Cronin and Crewther (2003)</td>
<td>10 Males - T</td>
<td>Supine squats (30, 60, 90% 1 RM)</td>
<td>30% 1RM</td>
<td>0.44 sec - CON</td>
<td>90% 1RM</td>
<td>0.93 sec - CON</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>30% 1RM</td>
<td>0.42 sec - ECC</td>
<td>90% 1RM</td>
<td>0.64 sec - ECC</td>
</tr>
<tr>
<td><strong>Impulse</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cronin and Crewther (2003)</td>
<td>10 Males - T</td>
<td>Supine squats (30, 60, 90% 1 RM)</td>
<td>30% 1RM</td>
<td>351 N/sec(^{-1}) - CON</td>
<td>90% 1RM</td>
<td>1816 N/sec(^{-1}) - CON</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>30% 1RM</td>
<td>352 N/sec(^{-1}) - ECC</td>
<td>90% 1RM</td>
<td>1239 N/sec(^{-1}) - ECC</td>
</tr>
<tr>
<td><strong>Work</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brown et al. (1990)</td>
<td>15 Males - T, ET, UT</td>
<td>Leg press to failure (60, 70, 80% 1RM)</td>
<td>60% 1RM - T</td>
<td>718 J</td>
<td>80% 1RM - T</td>
<td>911 J</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>60% 1RM - ET</td>
<td>506 J</td>
<td>80% 1RM - ET</td>
<td>691 J</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>60% 1RM - UT</td>
<td>522 J</td>
<td>80% 1RM - UT</td>
<td>728 J</td>
</tr>
<tr>
<td>Craig and Kang (1994)</td>
<td>4 Males - T</td>
<td>Half squat in 15 sec (75, 90% 1 RM)</td>
<td>75% 1RM</td>
<td>570 J</td>
<td>90% 1RM</td>
<td>684 J</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>25RM - leg press</td>
<td>473 J</td>
<td>3RM - leg press</td>
<td>841 J</td>
</tr>
<tr>
<td>Cronin and Crewther (2003)</td>
<td>10 Males - T</td>
<td>Supine squats (30, 60, 90% 1 RM)</td>
<td>30% 1RM</td>
<td>262 J - CON</td>
<td>90% 1RM</td>
<td>576 J - CON</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>30% 1RM</td>
<td>239 J - ECC</td>
<td>90% 1RM</td>
<td>583 J - ECC</td>
</tr>
</tbody>
</table>

NOTE: CO, concentric only; CON, concentric phase; ECC, eccentric phase; RB, rebound; T, trained; UT, untrained, ET, endurance trained.
As heavier loads produce greater forces and result in longer periods of muscular tension than lighter loads, it is not surprising that the product of these variables (impulse) is also maximised with heavier loads (see Table 2). For example, impulse production increased with an increase in mass for both the concentric and eccentric phases when examining three different loads (30, 60, 90% 1RM), each performed explosively on a supine squat machine (Cronin & Crewther, 2003). Given that impulse is the product of force and time this variable may therefore be a more appropriate measure of mechanical performance than either variable alone. The impulse-momentum relationship is certainly important for understanding much of human movement. Unfortunately, the assessment and/or practical significance of impulse in terms of strength and power adaptation are not well documented.

A number of studies have assessed the amount of work performed at different loads (Brown et al., 1990; Craig & Kang, 1994; Cronin & Crewther, 2003; Kang, Martino, Russo, Ryder & Craig, 1996) and have reported a concomitant increase in work performed with an increase in load (see Table 2). This is not surprising where for a given exercise the force component of the work formula (work = force x distance) is enhanced when heavier training loads are utilised. On average a 15% increase in work done was noted with a 10% increase in training load for the various exercises performed (see Table 2). As a function of force, the amount of work performed is likely to be constrained by those factors influencing force production such as maximal strength. For example, Brown et al. (1990) reported greater work done amongst resistance-trained individuals (~911 J), compared to endurance trained (~691 J) and untrained individuals (~728 J), when performing a leg press exercise at the same relative load (80% 1RM). The difference in work performed may be attributed to
differences in 1RM strength between groups and hence, the absolute load utilised. With the distance of force application also important the range of movement afforded by different exercises and/or techniques in the assessment of work are other considerations. This is evident in the various types of squatting movements (i.e. half squats, ballistic supine squats, full squats) used within research to assess work (see Table 2), the effects of which will be addressed in a later section.

A number of researchers have studied the effect that load (% 1RM) has on the power output of muscle (see Table 3). In contrast to force output, mechanical power is the product of force and velocity therefore, a specific combination of these variables will maximise power output. A force-power-velocity curve from isolated muscle is shown in Figure 2. Earlier research in vivo indicated that maximal power output occurred at approximately 30% of maximum isometric strength and 30% of maximum shortening velocity (Faulkner, Claflin & Cully, 1986; Kaneko, Fuchimoto, Toji & Suei, 1983; Moritani, 1992; Perrine, 1986). However, researchers using multiarticular movements in vivo have reported that heavier loading intensities (50-70% 1RM) may be superior in maximizing the power output of muscle (see Table 3). This appears true for the development of both mean and peak power output. In light of these findings a “band width” approach for load intensity (e.g. 30-60% 1RM) is often prescribed for maximising power performance (Kraemer et al., 2002). Similar to force output, greater power values have been reported in the performance of lower body (~2000 W) exercises compared to those found during upper body (~400 W) exercises (see Table 3). This may again be explained by the larger muscle groups of the lower limbs and multiarticular nature of many lower body exercises (e.g. squats).
Interestingly, the greatest power values (peak) were achieved with resistance (i.e. 20kg) much lighter than that found in other research (McBride et al., 1999). Furthermore, the values reported in this study (~ 3700-4900 W) were much greater than that reported by other research (~ 900-1800 W) using a similar population (i.e. trained males) and assessing the power output of the lower limbs (see Table 3). These findings may be attributed to several factors including the exercise and technique assessed (e.g. jump squats v parallel squats), and differences in 1RM strength between populations. The specific training experience of the subjects in this study (i.e. Olympic lifters, power lifters, sprinters) may also be important. For example, individuals who perform exercises within their own practice similar to those used during assessment, are likely to perform better than trained individuals who do not. Thus, the greater values reported in this study might be partially attributed to the fact that the assessment used (i.e. jump squats) may be of similar nature to those exercises performed in the training regime of the subjects used in this study.
Table 3: Effect of load (single repetitions) upon power output.

<table>
<thead>
<tr>
<th>Author/s</th>
<th>Subjects</th>
<th>Protocols (load)</th>
<th>Maximum - load</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thomas et al. (1996)</td>
<td>19 Females - UT</td>
<td>Double leg - leg press</td>
<td>56-78% 1RM (PP)</td>
<td>404 W</td>
</tr>
<tr>
<td>Newton et al. (1997)</td>
<td>17 Males - T</td>
<td>CO and RB bench press throws (15, 30, 45, 60, 75, 90% 1RM)</td>
<td>30-45% 1RM (MP)</td>
<td>560-563 W</td>
</tr>
<tr>
<td>McBride et al. (1999)</td>
<td>28 Males - PL, OL, sprints and controls</td>
<td>Jump squats (BW, 20KG, 40KG, 30, 60, 90% 1RM)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baker, Nance and Moore (2001)</td>
<td>32 Males - T</td>
<td>Jump squats (40, 60, 80, 100 kg - system mass)</td>
<td>55-59% 1RM (MP)</td>
<td>1851 W</td>
</tr>
<tr>
<td>Baker (2001)</td>
<td>49 Males - T</td>
<td>CO bench press throw (40, 50, 60, 70, 80 kg)</td>
<td>51% 1RM (MP) - NRL</td>
<td>600 W</td>
</tr>
<tr>
<td>Baker, Nance and Moore (2001)</td>
<td>31 Males - T</td>
<td>CO bench press throw (40, 50, 60, 70, 80 kg)</td>
<td>55% 1RM (MP) - SRL</td>
<td>502 W</td>
</tr>
<tr>
<td>Cronin, McNair and Marshall (2001)</td>
<td>27 Males - UT</td>
<td>CO and RB bench press and bench press throws (30, 40, 50, 60, 70, 80% 1RM)</td>
<td>50-70% 1RM (MP) - all techniques</td>
<td>~270-340 W</td>
</tr>
<tr>
<td>Izquierdo et al. (2001)</td>
<td>47 Males - UT</td>
<td>CO half squats (15, 30, 45, 60, 70% 1RM)</td>
<td>50-60% 1RM (PP) - all techniques</td>
<td>~550-625 W</td>
</tr>
<tr>
<td>Izquierdo et al. (2002)</td>
<td>70 Males - WL, MDR, HBP, cyclists and controls</td>
<td>CO bench press (30, 45, 60, 70, 80, 90% 1RM)</td>
<td>60% 1RM (PP) - 42 years</td>
<td>486 W</td>
</tr>
<tr>
<td>Siegel et al. (2002)</td>
<td>25 Males - T</td>
<td>CO half squats (30, 45, 60, 70, 80, 90% 1RM)</td>
<td>70% 1RM (PP) - 65 years</td>
<td>391 W</td>
</tr>
<tr>
<td>Weiss et al. (2002)</td>
<td>31 Males - T</td>
<td>Squat (30, 40, 50, 60, 70, 80, 90% 1RM)</td>
<td>45-60% 1RM (MP)</td>
<td>385-755 W</td>
</tr>
<tr>
<td>Cronin and Crewther (2003)</td>
<td>10 Males - T</td>
<td>CO squats (Parallel)</td>
<td>50-70% 1RM (PP)</td>
<td>~950 W</td>
</tr>
</tbody>
</table>

NOTE: CO, concentric only; CON, concentric phase; ECC, eccentric phase; RB, rebound; MP, mean power; PP, peak power; BW, bodyweight; PL, power lifters; OL, Olympic lifters; T, trained; UT, untrained; WL, weight lifters; MDR, middle distance runners; HBP, handball players; NRL, national rugby league; SRL, state rugby league. * Similarly effective to loads that maximised power output
Comparing findings between studies are again made difficult due to differences in the equipment used (e.g. force platform, accelerometers, linear transducers, etc.) and the exercise and/or technique assessed (e.g. jump squats, half squats, concentric only squats, etc.). The training experience of subjects also makes comparisons problematic. It was reported that the stronger subjects achieved peak power at a higher percentage of individual 1RM compared to the weaker subjects (Stone et al., 2003). This suggests that the ability to exert power output is transient and affected by changes in maximal strength of the population assessed. Baker (2001) however, found that the percentage 1RM that maximised power output was significantly lower in stronger professional (51%) as compared to state rugby league players (55%) (see Table 2). It would seem as athletes become stronger they can produce greater power outputs with any absolute load, but the ability to produce power at a given percentage of their 1RM remains similar as relative resistances increase proportionally to strength levels. Other issues require consideration when interpreting research. For example, some studies (Kaneko et al., 1983) assessed only a limited range of loads (e.g. 30, 60% 1RM) and as such the load that maximises power may well lie between these loads, whilst other research (Baker et al., 2001) have found no differences in power output between different loads (e.g. 46-62% 1RM). The different formulae used to assess power output (e.g. mean, peak, relative and instantaneous) further confound understanding in this area.

Implications for strength and power development

Strength adaptation is largely attributed to changes in neural function (motor unit recruitment, firing frequency, synchronisation and reflex activity) and muscle morphology or muscle CSA (Sale, 1992; Schmidtbleicher, 1992). The mechanism for these adaptations may be explained by the “stimulus-tension” theory, that is, the
intensity (% 1RM) and the duration of muscular tension (i.e. forces) are responsible for neural and morphological adaptation (Ritzdorf, 1998). Heavy loads would seem fundamental to strength development, as high forces are associated with maximal motor unit recruitment according to the “size principle” (Behm, 1995; McDonagh & Davies, 1984). The size principle states that motor units are recruited in order from the low threshold units to the high threshold units when greater forces are required, with these units also firing at higher frequencies (McDonagh & Davies, 1984). The development of high load forces may further inhibit the force-feedback (Golgi tendon organs) reflex mechanisms (Schmidtbleicher, 1992) and further improve the ability of muscle to generate greater forces (maximal strength).

The development of high forces, particularly when the muscle is actively stretched, is also thought important in modulating protein balance following resistance exercise (Fowles et al., 2000; Lieber & Friden, 1993). According to the “break-down build-up” theory, muscle protein is broken down during training resulting in tissue regeneration or protein accretion in the recovery period. On this basis heavy load training (>60-70% 1RM) is thought to provide the superior stimulus for neural and morphological adaptation and hence, maximal strength development (MacDougall, 1992; McDonagh & Davies, 1984). However, it remains to be seen if muscle damage is a prerequisite for hypertrophy, as other factors (e.g. age, gender, genetic potential, diet, etc.) will further influence whether or not muscle growth will occur. Other hormonal and metabolic factors may also determine if changes in muscle morphological will occur in response to a given exercise training program.
Time under tension is also important for strength and hypertrophic adaptation (Bloomer & Ives, 2000; Komi & Hakkinen, 1988). In theory the longer a muscle is subjected to a given training stimulus the greater the potential for adaptation. Given the longer contraction durations associated with heavier loads the prescription of such loading intensities would again seem better suited to produce changes in maximal strength. However, it is unlikely that TUT alone is the critical stimulus for adaptation to occur. Strength endurance training for example, is typically characterised by high volume training (high total repetitions = high time under tension) and is not thought to result in substantial hypertrophy and/or strength gains (Fleck & Kraemer, 1997; Kraemer et al., 2002). This may be attributed to the lighter loads utilised with strength endurance training, which suggests that a tension (load/force) threshold also exists for strength and/or hypertrophy adaptation to occur. Such a notion further underscores the importance of heavier training loads for the improvement of maximal strength.

It may be that force and time alone does not adequately account for changes in strength and hypertrophy but rather the distance over which force acts or work (Moss et al., 1997; Stone et al., 1998). Such benefits are partially supported by the larger training-induced strength gains found with high volume programmes (greater volume = greater work) compared to low volume programmes (Borst et al., 2001; Kramer et al., 1997; Marx et al., 1998; Marx et al., 2001; Schlumberger, Schmidtbleicher & Stec, 2001). An increase in mechanical work may further result in greater metabolic activity, which may also be important for increasing strength and muscle CSA (Enoka, 2002; Jones, 1992). If the amount of work done were an important stimulus to maximal strength adaptation, then the prescription of heavy loads would again appear the superior training option for a given displacement. It should be noted that this
interpretation of work is somewhat oversimplified, as technique will also have an important influence on total work done. For example, lighter load ballistic training may elicit greater work for a given load by increasing the distance travelled and thereby contribute to greater adaptation.

Another kinetic variable that may be important for maximal strength and power adaptation is impulse. Given the importance of high tension and TUT to strength development, it may be speculated that impulse production is the critical stimulus for strength and hypertrophic adaptation. Unfortunately, such a contention has not been investigated. In terms of explosive activities impulse would also appear an important training factor. For example, producing high forces in the shortest period of time or developing greater forces in the same period of time, would no doubt aid performance in those activities that require the generation of forces in such a manner (e.g. sprint running). Therefore, weight training techniques that maximise force and/or minimise the time over which the force is applied and their subsequent effect upon performance, need to be investigated. Whether changes in impulse affect the power output of muscle is another area requiring examination.

Power is the ability to produce force quickly (power = force x velocity) therefore, the slower velocities associated with heavy load-high force training would seem less important. The longer contraction durations associated with heavy loads would also appear undesirable given that the ability to produce force in the shortest period of time (i.e. displacement / time) would imply greater power. It has been suggested that loads that maximise mechanical power output would optimise power performance (Newton et al., 1996; Newton et al., 1997; Wilson et al., 1993). Though the prescription of load
based upon mechanical power is an attractive proposition, what may be critical is the ability to exert force at speeds (i.e. power) specific to a sporting or athletic movement. For example, whilst power is typically associated with high movement velocities (e.g. throwing, sprinting, jumping, etc.) other activities such as Olympic weight lifting and power lifting may also be classified as powerful activities. As these activities are characterised by slower movement velocities it would seem appropriate to train power at similar velocities for optimal adaptation. Similarly, as kicking a ball, throwing a shot put, and tackling an opponent involve different masses and hence, force-velocity characteristics, moving one load (i.e. load that maximises mechanical power output) for all activities would seem fundamentally flawed.

*Kinematics and kinetics of a single repetition – contraction type*

It is known that eccentric muscle actions generate greater forces in the active muscles than concentric only muscle actions (Edman, 1992; Gregor, 1993; Jones, 1992). Where greater forces are generated at slower contraction velocities during shortening, during lengthening contractions greater forces are developed at faster contraction velocities, until the load is no longer under muscular control (see Figure 3). During eccentric actions it is thought that there is greater contribution from the elastic components of the musculo-tendinous unit increasing the potential for force generation (Edman, 1992; Gregor, 1993). As such eccentric actions generate greater maximal tension and further, produce similar tension with less motor units recruited compared to concentric only actions. On this basis eccentric actions are thought to produce greater “mechanical” efficiency than concentric actions (Enoka, 2002). Whilst a general trend in the force-velocity curve holds true with regards to the magnitude of force output between these actions, differences in force production would also depend
upon other variables such as the assessment conditions, mode of testing and training status of subjects (Bilcheck & Maresh, 1992).

Due to the greater mechanical efficiency afforded by eccentric contractions supramaximal loads (>100% concentric 1RM) may be employed and high load forces developed. A study by Murphy, Wilson and Pryor (1994) examined the effect of concentric only (30, 60, 100% 1RM) and eccentric only (100, 130, 150% 1RM) contractions utilising bench press movements. As expected force output increased with an increase in mass in the concentric conditions and further again with the heavier masses in the eccentric conditions. However, not much is known about the kinematics and kinetics associated with supramaximal eccentric loading. Whilst some studies have investigated the kinematics and kinetics associated with eccentric actions (movements), submaximal training loads were employed (Cronin & Crewther, 2003; Newton et al., 1997). Given the wider use of heavy eccentric loading, it is recommended that research investigate the mechanical response to such training.

**Figure 3:** Concentric/eccentric force-velocity relationship of muscle. Modified from Gregor (1993).
Implications for strength and power development

As resistance training is generally performed with eccentric and concentric muscle actions the combined effect of both contractions are likely to contribute to the cumulative training response. As mentioned previously, heavy eccentric loading is often used within practice as an advanced overloading technique, allowing “maximal” loads to be utilised and high forces to be developed for greater strength and/or hypertrophic gains (stimulus-tension theory). In the development of high muscular forces heavy eccentric loading affords other training benefits. For example, the larger high threshold motor units may be preferentially recruited under supramaximal loading conditions due to the greater forces developed (Behm, 1995). If these units contributed to a greater proportion of hypertrophy than the low threshold motor units (Deschenes & Kraemer, 2002; MacDougall, 1986), their selective recruitment through such actions may further augment morphological adaptation. Another benefit of supramaximal eccentric loading is the higher incidence of microscopic muscle injury (Jamurtas et al., 2000; Nosaka & Newton, 2002), which would also seem ideal stimuli for adaptation in terms of the “break-down build-up” theory.

The potential benefits afforded by eccentric loading are not without some controversy. Concentric only training has been found equally effective in stimulating strength and hypertrophy changes compared to eccentric only training (Carey Smith & Rutherford, 1995; Jones & Rutherford, 1987; Marler, Motl, Johnson, Walker & Subudhi, 1999; Mayhew, Rothstein, Finucane & Lamb, 1995). This may be explained by the fact that many of these studies employed submaximal loads (< 100% concentric 1RM) and thus, the benefits of eccentric training may not be fully realized, or the greater metabolic costs associated with concentric only actions (Carey Smith & Rutherford,
Whether or not eccentric actions provide the superior training stimulus to concentric actions with the same relative load remains a topic of debate. Eccentric muscle actions are also characterised by reduced motor unit activation compared to concentric actions (Behm, 1995; Enoka, 2002), which would suggest that less muscle fibres are recruited and trained. Still, there may be greater relative tension produced with less motor units activated. Such issues limit our understanding of eccentrics and the prescription of such exercise.

One of the main limitations when employing eccentric or concentric only loading for power development is the lack of specificity (e.g. contraction type, velocity, etc.). For example, it is known that training-induced performance gains, related to concentric or eccentric only actions, are likely to result in contraction specific adaptation (Hortobagyi et al., 1996; Seger, Arvidsson & Thorstensson, 1998). In contrast most athletic activities utilise both eccentric and concentric actions during movement and further, are performed at greater velocities than that achieved during typical weight training. Still, training in this manner may aid power development in those activities requiring high levels of contraction-specific strength. For example, activities that involve landing under high loading conditions (e.g. gymnastics) are likely to require a certain amount of absorbing or eccentric strength for successful performance. It must also be remembered that many sporting activities such as swimming and cycling, are in fact performed with predominantly concentric only muscle actions and would no doubt benefit from some type of training in this manner.
Kinematics and kinetics of a single repetition – rebound vs non rebound

The stretch shortening cycle (SSC) is a common pattern of muscle activation that occurs when an eccentric muscle action precedes a concentric muscle action (Enoka, 2002). As most sporting and everyday activities elicit some combination of eccentric and concentric movement, SSC muscle actions would appear fundamental to human movement. Cronin, McNair and Marshall (2001) examined the effect of concentric only and rebound bench press movements (throw and non-throw) across a range of loads (30, 40, 50, 60, 70 and 80% 1RM). In utilising rebound they reported greater force (peak - 14.1%) and power output (mean - 11.7%) on average across all loads, compared to the non-rebound conditions (see Table 4). Another study examined the effect of rebound squat jumps and static (concentric only) squat jumps, performed over a wide spectrum of loads from 10% to 100% 1RM (Stone et al., 2003). On average an increase in power output (mean - 4%) was also found across all loads with the use of rebound. Other research have reported similar findings (Cronin et al., 2003; Newton et al., 1997), thereby demonstrating that the use of rebound potentiates the kinematics and kinetics associated with that movement.

The mechanical enhancement reported within research may be attributed to the enhanced kinematic profile associated with SSC movements. For example, load greater accelerations (peak-38.5%) and velocities (mean-12.4%) were reported across all loads assessed when utilising rebound, whether or not the load was held or thrown (see Table 4). With an increase in the velocity and acceleration profile across the various loads, resultant power and force values are enhanced. The augmentation from SSC muscle action is typically attributed to a number of factors including; the storage and re-utilisation of elastic energy stored in the series elastic component of the
musculo-tendinous unit (Asmussen & Bonde-Petersen, 1974; Komi & Bosco, 1978); spinal reflexes (Dietz, Noth & Schmidtbleicher, 1981), as well as long latency responses (Melville-Jones & Watt, 1971) that increase muscle stimulation, allowing the muscle to reach maximum activation prior to the concentric muscle action (Van Ingen Schenau, 1984).

Due to enhanced kinematic responses it is not surprising that the concentric phase of SSC movements generally result in shorter contraction durations than non-rebound movements, regardless of the technique employed (Cronin et al., 2001a, 2003; Newton et al., 1997). No research has specifically examined work done or impulse production during rebound and non-rebound conditions. In spite of this it may be speculated that with an increase in force production across the various loads (see Table 4), greater work would also result. An increase in force output with rebound would further suggest enhanced impulse production for any given load. However, as studies in this area have indicated a reduction in contraction duration with rebound movements, the combined effect of force (increase) and time (decrease) upon impulse values, remains as yet unknown. Assessment of these variables (work, impulse) warrant some consideration within research in order to determine their response to different techniques and thus, their contribution to strength and conditioning practice.
Table 4: Effect of technique (single repetitions) upon peak and mean velocities, peak accelerations, peak and mean forces, peak and mean power, and duration of concentric contraction during bench press movements at different loading intensities (30-80 % 1RM). Modified from Cronin, McNair and Marshall (2001, 2003).

<table>
<thead>
<tr>
<th>% 1RM</th>
<th>PV (m/s)</th>
<th>MV (m/s)</th>
<th>PA (m/s²)</th>
<th>PF (N)</th>
<th>MF (N)</th>
<th>PP (W)</th>
<th>MP (W)</th>
<th>DOCC (s)</th>
<th>PV (m/s)</th>
<th>MV (m/s)</th>
<th>PA (m/s²)</th>
<th>PF (N)</th>
<th>MF (N)</th>
<th>PP (W)</th>
<th>MP (W)</th>
<th>DOCC (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>30</td>
<td>1.49</td>
<td>0.82</td>
<td>5.87</td>
<td>414.1</td>
<td>257.9</td>
<td>467.4</td>
<td>211.5</td>
<td>0.629</td>
<td>1.63</td>
<td>0.87</td>
<td>5.74</td>
<td>411.1</td>
<td>257.9</td>
<td>467.4</td>
<td>211.5</td>
<td>0.629</td>
</tr>
<tr>
<td>40</td>
<td>1.35</td>
<td>0.73</td>
<td>4.63</td>
<td>505.9</td>
<td>339.5</td>
<td>532.0</td>
<td>250.0</td>
<td>0.683</td>
<td>1.45</td>
<td>0.77</td>
<td>4.73</td>
<td>505.2</td>
<td>340.3</td>
<td>575.0</td>
<td>263.3</td>
<td>0.816</td>
</tr>
<tr>
<td>50</td>
<td>1.16</td>
<td>0.64</td>
<td>3.78</td>
<td>583.8</td>
<td>429.2</td>
<td>552.1</td>
<td>271.7</td>
<td>0.761</td>
<td>1.27</td>
<td>0.68</td>
<td>3.76</td>
<td>589.2</td>
<td>430.6</td>
<td>522.0</td>
<td>223.5</td>
<td>0.792</td>
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<tr>
<td>60</td>
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<td>0.55</td>
<td>2.96</td>
<td>660.9</td>
<td>509.4</td>
<td>549.3</td>
<td>280.8</td>
<td>0.818</td>
<td>1.05</td>
<td>0.57</td>
<td>2.85</td>
<td>657.3</td>
<td>508.8</td>
<td>609.3</td>
<td>290.9</td>
<td>0.883</td>
</tr>
<tr>
<td>70</td>
<td>0.86</td>
<td>0.36</td>
<td>2.43</td>
<td>766.4</td>
<td>588.8</td>
<td>542.4</td>
<td>266.8</td>
<td>0.964</td>
<td>0.89</td>
<td>0.47</td>
<td>2.45</td>
<td>772.8</td>
<td>594.8</td>
<td>580.0</td>
<td>280.9</td>
<td>0.953</td>
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<tr>
<td>80</td>
<td>0.68</td>
<td>0.33</td>
<td>1.93</td>
<td>826.3</td>
<td>677.8</td>
<td>478.2</td>
<td>222.0</td>
<td>0.72</td>
<td>0.36</td>
<td>2.06</td>
<td>820.5</td>
<td>677.8</td>
<td>527.5</td>
<td>232.2</td>
<td>1.213</td>
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</tr>
</tbody>
</table>

Rebound bench press

<table>
<thead>
<tr>
<th>% 1RM</th>
<th>PV (m/s)</th>
<th>MV (m/s)</th>
<th>PA (m/s²)</th>
<th>PF (N)</th>
<th>MF (N)</th>
<th>PP (W)</th>
<th>MP (W)</th>
<th>DOCC (s)</th>
<th>PV (m/s)</th>
<th>MV (m/s)</th>
<th>PA (m/s²)</th>
<th>PF (N)</th>
<th>MF (N)</th>
<th>PP (W)</th>
<th>MP (W)</th>
<th>DOCC (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>30</td>
<td>1.52</td>
<td>0.91</td>
<td>8.54</td>
<td>491.8</td>
<td>262.1</td>
<td>463.2</td>
<td>237.0</td>
<td>0.614</td>
<td>1.68</td>
<td>0.98</td>
<td>8.51</td>
<td>488.8</td>
<td>261.6</td>
<td>531.8</td>
<td>252.3</td>
<td>0.782</td>
</tr>
<tr>
<td>40</td>
<td>1.37</td>
<td>0.83</td>
<td>7.11</td>
<td>596.1</td>
<td>340.5</td>
<td>536.3</td>
<td>283.1</td>
<td>0.642</td>
<td>1.48</td>
<td>0.87</td>
<td>7.34</td>
<td>593.5</td>
<td>341.2</td>
<td>590.6</td>
<td>296.5</td>
<td>0.779</td>
</tr>
<tr>
<td>50</td>
<td>1.21</td>
<td>0.75</td>
<td>6.33</td>
<td>692.7</td>
<td>427.2</td>
<td>557.9</td>
<td>312.8</td>
<td>0.739</td>
<td>1.30</td>
<td>0.78</td>
<td>6.67</td>
<td>714.3</td>
<td>424.7</td>
<td>626.4</td>
<td>325.5</td>
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</tr>
<tr>
<td>60</td>
<td>0.99</td>
<td>0.62</td>
<td>4.97</td>
<td>786.4</td>
<td>513.4</td>
<td>550.6</td>
<td>315.4</td>
<td>0.803</td>
<td>1.08</td>
<td>0.66</td>
<td>5.01</td>
<td>777.3</td>
<td>511.4</td>
<td>609.4</td>
<td>336.6</td>
<td>0.844</td>
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<td>70</td>
<td>0.83</td>
<td>0.53</td>
<td>4.12</td>
<td>876.6</td>
<td>599.1</td>
<td>542.3</td>
<td>316.3</td>
<td>0.945</td>
<td>0.88</td>
<td>0.53</td>
<td>4.33</td>
<td>866.1</td>
<td>594.8</td>
<td>681.7</td>
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<td>0.944</td>
</tr>
<tr>
<td>80</td>
<td>0.65</td>
<td>0.39</td>
<td>3.19</td>
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<td>679.0</td>
<td>468.9</td>
<td>261.3</td>
<td>0.69</td>
<td>0.39</td>
<td>3.25</td>
<td>914.1</td>
<td>678.7</td>
<td>499.3</td>
<td>271.7</td>
<td>1.180</td>
<td></td>
</tr>
</tbody>
</table>

NOTE: PV, peak velocity; MV, mean velocity; PA, peak acceleration; PF, peak force; MF, mean force; PP, peak power; MP, mean power; DOCC, duration of concentric contraction.
Whilst the potentiating effects of rebound to the concentric muscle action are well recognised within literature, such effects may be limited to the initial period of the concentric movement. In comparing the effects of rebound and concentric only actions during bench press movements, Cronin, McNair and Marshall (2001, 2003) found that the use of rebound did not enhance peak velocities between these conditions (see Table 4). A study by Newton, Murphy, Humphries, Wilson, Kraemer and Hakkinen (1997) reported similar findings and attributed this to the recovery of stored elastic energy. That is, elastic and reflex potentiation only enhance the initial phase of the concentric movement and that peak velocities occur later where the effects of SSC has diminished (Newton et al., 1997).

An interaction between the potentiating effects of the SSC and the load utilised has also been revealed within research (see Table 4). It can be observed that greater enhancement (peak force) occurred with a load of 30% 1RM (18-19%), compared to that found with a load of 80% 1RM (10-11%), when concentric only and rebound movements were compared. This data indicates that the potentiating effect of rebound decreases with an increase in load. The mechanisms for this are likely to involve a slower rate of eccentric muscle action, longer duration eccentric muscle action and slower coupling times when utilising heavier loads (Cronin et al., 2001a; Newton et al., 1997). The loss of this enhancement may be further attributed to the inability of the muscle to generate force at high shortening velocities (Cronin et al., 2003).

*Implications for strength and power development*

The SSC has been shown to augment a variety of kinematic and kinetic variables across a spectrum of loads. As most training programmes are performed with rebound
it is assumed that changes in maximal strength are, in some capacity, related to the potentiating effects of both the elastic (i.e. tendon, epimysium, etc.) and active components (i.e. cross-bridges) of muscle. Quantifying the contribution of the SSC to changes in performance however remains difficult. Realising that pre-stretching the muscle enhances the concentric muscle action, eliminating the influence of the SSC may intuitively elicit greater contribution from the contractile machinery. If changes in strength and hypertrophy were largely attributed to adaptations within the contractile elements, concentric only training would appear an attractive training option. More advanced weight training techniques are based upon this premise. For example, techniques such as rest-pause and super slow training may be implemented to minimize the potentiating effects of the SSC and stress the contractile elements of the muscle for greater strength and hypertrophic gains. Since pre-stretch shortens the duration of the concentric phase eliminating the eccentric contribution will also result in greater TUT and thereby provide additional stimulus for adaptation.

As mentioned previously, concentric only actions may aid power performance for those sporting activities predominantly characterised by such muscle actions. Unlike strength however, the utilisation of the SSC is an important consideration for power adaptation. Pre-stretch is an inherent aspect of most athletic activities (e.g. running, jumping, throwing and catching) therefore the use of rebound during training would appear fundamental to improving power performance. Equally important in power development is the efficient use of the SSC phenomenon. The prescription of plyometric training is based upon such a premise. That is, the performance of powerful movements utilising coupled eccentric and concentric muscle actions improve the storage and utilisation of elastic energy, and reflex activity (Komi &
Hakkinen, 1988). Furthermore, it is likely that the velocity and acceleration profile of rebound movements, particularly when performed explosively with lighter loads, more closely simulate those occurring during sporting performance and everyday activities compared to non-rebound movement (Newton et al., 1997).

*Kinematics and kinetics of a single repetition – traditional v ballistic*

As described previously, ballistic refers to techniques in which the load (e.g. the bar, oneself, etc.) is projected or released at the end of the concentric phase. A number of studies have reported enhanced kinematic and kinetic responses to exercise utilising ballistic movements (Cronin et al., 2001a, 2003; Newton & Wilson, 1993; Newton et al., 1996). For example, an increase in power output (mean - 5.8%, peak - 9.1%) on average was found across all loads when comparing traditional and ballistic bench press movements, performed with and without rebound (see Table 4). Newton, Kraemer, Hakkinen, Humphries and Murphy (1996) also reported greater power enhancement (mean - 70%, peak - 67%) when comparing traditional and bench press throw movements utilising a load of 45% 1RM. A significant increase in force output (mean - 35%) was also found in response to the ballistic movements in this study. Although no changes in force output (mean and peak) were reported in the previous study, comparing findings between these studies is made difficult due to differences in research design (e.g. multiple loads v single load, trained v untrained subjects, linear transducer v force plate). Nonetheless, these findings still suggest greater mechanical responses with ballistic techniques compared to more traditional techniques.

Similar to rebound, the potentiation afforded by ballistic techniques may be attributed to altered load kinematics. The bench press throw with a load of 45% 1RM allowed
the bar to be accelerated for 96% of the throw movement as opposed to 60% for a traditional movement (Newton et al., 1996). With longer periods of acceleration the ballistic movement had a significant effect upon load velocities (mean - 27%, peak - 36%) and related kinetics thereafter. Due to the enhanced velocities, ballistic movements usually result in early completion of the concentric phase (Newton et al., 1996). However, other studies have reported a longer concentric phase with ballistic movements (Cronin et al., 2001a, 2003). These studies also reported an interaction between load and concentric contraction duration (see Table 4). For example, the mean increase in concentric duration was greater at 30% 1RM than that found at 80% 1RM (26% v 2% respectively). This may be explained by the measurement of work. Muscle activation will only be achieved up to the point of release in spite of the load being thrown a greater distance. Some studies (Cronin & Crewther, 2003; Newton et al., 1996) have accounted for this and reported “muscular work” whilst others have examined “system work” (Cronin et al., 2001a, 2003), explaining differences in literature. This is an important consideration when examining mechanical data given the relationship between displacement and load kinematics and kinetics.

Although not reported within research, it may be speculated that the use of ballistic techniques result in greater work done for a given exercise and load, if force output were enhanced. With an increase in force output but a reduction in TUT though, what influence ballistic movements would have upon impulse production remains speculative. With this in mind further research is warranted to elucidate the responsiveness of work done and impulse, with the use of ballistic weight training techniques. Given that research in this area has only examined upper body exercises (i.e. bench press), what influence the larger muscle groups of the lower limbs and
The multiarticular nature of many leg exercises would have upon resultant kinematics and kinetics, utilising projection techniques, is another area for investigation.

**Implications for strength and power development**

If ballistic techniques produced greater forces compared to traditional techniques, then training in this manner would appear to provide a more effective stimulus for maximal strength development (stimulus-tension theory). Ballistic techniques may further enhance the training stimulus in developing high eccentric forces when “catching” and lowering the load. Whether or not ballistic techniques provide the superior stimulus compared to traditional techniques, using the same relative load remains unknown. Previously, it was thought that heavier loads (>60-70% 1RM) were necessary for strength and hypertrophic adaptation given the greater forces associated with heavier loads. However, such beliefs were based on techniques that did not involve the projection of the load. Ballistic techniques allow greater forces to be produced for a given load and hence, lighter load (<60% 1RM) ballistic training may offer an alternative strategy for maximal strength adaptation (Dahl et al., 1992; Lyttle et al., 1996; Moss et al., 1997). It should be realised that the benefits afforded by such techniques may decrease as load increases, due to the inability to project the bar or oneself at heavier loads. The benefits of ballistics may be further limited by the fact that such techniques result in a shorter duration of the concentric phase and hence, a reduction in the amount of time muscle is under tension.

Ballistic techniques would appear to offer an ideal stimulus for power development considering the high power outputs produced. Also, a major limitation of traditional resistance training for transference of strength and power to functional performance is
the fact that a large portion of the movement is spent decelerating the load. Ballistic training overcomes such a limitation and in this process develops a velocity profile more closely resembling that occurring during most athletic activities (i.e. longer periods of acceleration, higher velocities). With longer periods of acceleration, ballistic techniques may increase the “loading” period of muscle, further enhancing the stimulus for power adaptation. It should however be realised that the likelihood for injury may increase due to excessive loading upon the muscular and skeletal systems. This may explain the use of lighter loads (<50% 1RM) among research when examining the effects of ballistic weight training (Hammett & Hey, 2003; McEvoy & Newton, 1998; Olsen & Hopkins, 2003). A further issue may be the availability of appropriate machines within practice to allow such movements to be performed correctly and in a safe manner (e.g. Smith machine).

*Kinematics and kinetics of multiple repetitions/sets*

Given that resistance exercise usually involves multiple repetitions, sets and exercises the kinematic/kinetic response to a single repetition has little practical significance, in terms of understanding the stimulus imposed by resistance exercise during a typical training session. For example, Cronin and Crewther (2003) found that force output with a single repetition at 90% 1RM produced superior forces (mean and peak) during both the eccentric and concentric phases. However, greater total eccentric forces and concentric forces were observed in the 30% 1RM condition. This was also true for time under tension and power output where the 30% 1RM condition produced superior responses, compared to the two heavier loading conditions (see Table 5). An analysis of multiple repetitions would also provide a better understanding of any kinematic and kinetic changes across a given set and/or exercise. For example, different weight
training schemes (e.g. hypertrophy) are characterised by fatigue and thus, force and power outputs recorded over the last few repetitions are likely to be quite different from the initial few repetitions.

The impulse produced under the different conditions appears less clear. No differences were found between three loading conditions (30, 60, 90% 1RM) when comparing total eccentric impulse; however, the 90% 1RM condition resulted in greater total concentric impulse (Cronin & Crewther, 2003). Where the amount of work performed increased with heavier masses during single repetitions (Brown et al., 1990; Craig & Kang, 1994; Cronin & Crewther, 2003; Kang et al., 1996), these same studies reported greater total work (set responses) with the lightest loads examined (see Table 5). Such findings may be explained by differences in training volume, where the greater number of repetitions associated with the lighter loads, performed to failure, accounted for the greater forces produced with the heavier loads. With most studies having assessed work alone, the kinematic and kinetic response to multiple repetitions and/or sets of resistance exercise remains relatively unknown.
### Table 5: Effect of load (multiple repetitions) upon total work, total forces, total power, total time under tension and total impulse.

<table>
<thead>
<tr>
<th>Author/s</th>
<th>Subjects</th>
<th>Protocols (load)</th>
<th>Maximum - load</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brown et al. (1990)</td>
<td>15 Males - T, ET, UT</td>
<td>Leg press to failure (60, 70, 80% 1RM)</td>
<td>60% 1RM - T</td>
<td>Work - 33208 J</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>60% 1RM - ET</td>
<td>Work - 22074 J</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>60% 1RM - UT</td>
<td>Work - 19330 J</td>
</tr>
<tr>
<td>Craig and Kang (1994)</td>
<td>4 Males - T</td>
<td>Half squat in 15 sec (75, 90% 1RM)</td>
<td>75% 1RM</td>
<td>Work - ~6200 J</td>
</tr>
<tr>
<td>Kang et al. (1996)</td>
<td>3 Males - T</td>
<td>Squat and leg press to failure (3, 10, 25RM)</td>
<td>25RM - squat</td>
<td>Work - 9875 J</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>25RM - leg press</td>
<td>Work - 11831 J</td>
</tr>
<tr>
<td>Cronin and Crewther</td>
<td>10 Males - T</td>
<td>Supine squats equated by volume (6 x 30%, 3 x 60%, 2 x 90% 1RM)</td>
<td>30% 1RM</td>
<td>Impulse - 2116-2535 N/sec⁻¹</td>
</tr>
<tr>
<td>(2003)</td>
<td></td>
<td></td>
<td>30, 60, 90% 1RM</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
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<td>CON values</td>
<td></td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Time - 2.53 sec</td>
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<td>Force - 5084 N</td>
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<td>Power - 3626 W</td>
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<td></td>
<td></td>
<td>Work - 1510 J</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Impulse - 3632 N/sec⁻¹</td>
</tr>
</tbody>
</table>

**NOTE:** T, trained; UT, untrained; ET, endurance trained; CON, concentric phase; ECC, eccentric phase.
A number of issues warrant consideration when examining research in this area. For example, the total amount of work performed differed considerably (e.g. ~6000-33000 J) between studies (see Table 5). This may be due to several factors including differences in exercise technique (e.g. half squat, full squat, ballistic supine squat) and the assessment procedures used (e.g. repetitions to failure, repetitions in 15 seconds, equated by volume, etc.). The strength of the population assessed is also important, as stronger individuals not only use greater absolute loads compared to weaker individuals, but may also perform a greater number of repetitions at any given load (% 1RM). In their study, Brown et al. (1990) found that trained males performed significantly more repetitions to failure at 70% 1RM compared to a group of untrained males (e.g. 22 v 14 repetitions respectively). This may be explained by the “trained” status of these individuals in combination with other factors such as familiarity with testing procedures and greater tolerance to fatigue.

Manipulating weight-training technique is common within practice to overload the muscular system (Bloomer & Ives, 2000). For example, a reduction of lifting tempo may reduce the contribution of SSC during rebound movements and elicit greater contractile contribution. A slow eccentric phase in comparison to the concentric phase may also serve to exhaust eccentric strength and promote specific training-induced gains. However, the mechanical response to different techniques, over multiple repetitions, has received little attention. Keogh, Wilson and Weatherby (1999) examined the mechanical response of seven techniques (isokinetics, eccentrics, functional isometrics, super slow motion, rest pause, break-downs, maximal power training), using a bench press movement each performed over six repetitions. Understandably many of these techniques produced specific responses. For example,
maximal power training (30% 1RM) maximised power output per repetition, whilst super slow motion training (55% 1RM) maximised contraction duration over the six repetitions. However, extrapolating this information is difficult given that few variables were assessed and with data reported for the first, middle and last repetitions alone. Also, contraction duration was only reported as a combined total over the six repetitions. No other studies to our knowledge have examined the effects of different weight training techniques over multiple repetitions.

Given the importance of force and TUT to maximal strength adaptation, it is interesting to note that no studies have reported the cumulative effect of force and time over the course of different loading schemes (i.e. multiple repetitions, sets and exercises). This is not withstanding the importance of other mechanical variables to the training environment. Thus, the kinematic and kinetic response to a typical weight training session remains unknown. Research has, in the examination of endocrine responses to resistance exercise, reported total work between hypertrophy and neuronal loading schemes (Kraemer et al., 1993a; Kraemer et al., 1993b; Kraemer et al., 1991; Kraemer et al., 1990). Greater total work was reported for the hypertrophy schemes, compared to the neuronal schemes, amongst males (60,000J v 50,000J) and females (32,000 v 25,000J) respectively. The differences in total work between schemes may be explained by the greater training volume associated with hypertrophy training. However, the assessment of work alone does not adequately reflect the nature of the mechanical stimulus imposed by different loading schemes. With limited data available further research is warranted to determine the mechanical response to different strength and power schemes, as performed in practice.
Implications for strength and power development

The limitations to our discussion about the practical applications of the kinematics and kinetics of a single repetition are obvious. Strength and power adaptation necessitate the use of loading schemes characterised by multiple repetitions, sets and exercises, varied intensities (% 1RM), rest periods and training volume (Hedrick, 1995; Kraemer et al., 2002; Tan, 1999). It is the specific configuration of these variables that ultimately determines the mechanical response to the weight training session and adaptation thereafter. For example, neuronal schemes are characterised by the utilisation of very heavy loads (e.g. 85-100% 1RM), few repetitions performed per set (e.g. 1-6) and longer rest periods between sets (e.g. 3-5 minutes). Conversely, hypertrophy schemes are characterised by lower intensities (e.g. 60-70% 1RM), moderate repetitions (e.g. 8-12) and shorter rest periods (e.g. 1 minute). Hypertrophy training is further typified by high training volume (i.e. multiples sets and exercises per body-part) and more so than other training schemes.

Whilst heavy loads are thought necessary for maximal strength adaptation it may be speculated that maximal loads are needed to induce such changes through neural rather than morphological mediated adaptation (Bloomer & Ives, 2000; Schmidtbleicher, 1992). This is supported by the use of greater intensities in neuronal schemes, combined with performance of lower training volume and longer rest periods. It may also be speculated that TUT is of greater importance for morphological adaptation to occur. As the total number of repetitions performed largely regulates the duration of muscle activity, the greater volume of training associated with hypertrophy schemes would result in greater TUT compared to neuronal training. Such a notion is further supported by the fact that movement during hypertrophy training is deliberately...
controlled and often performed to muscular fatigue (Hedrick, 1995; Kraemer et al., 2002). The loading parameters associated with hypertrophy training (i.e. high volume, short rest, greater TUT) may also elicit greater hormonal and metabolic activity, which are also thought important for muscle growth to occur (Carey Smith & Rutherford, 1995; Hedrick, 1995; Kraemer, 1992a).

Interestingly however, many studies have found light load (<45% 1RM) training equally effective in increasing muscle CSA and strength compared to heavier load training methods (Dahl et al., 1992; Harris et al., 2000; Lyttle et al., 1996; Moss et al., 1997; Schmidtbleicher & Buehrle, 1987). Therefore, it would seem the use of heavier loads for maximal strength development is not as important as originally thought. Heavy training loads no doubt maximise the mechanical response to resistance exercise (i.e. force output, TUT, work and impulse) when single repetitions are compared and thus, the basis for the prescription of such intensities for strength and hypertrophy. However, superior kinematics and kinetics (e.g. total forces, total TUT, total work) have been reported using lighter loads when examining the mechanical response to multiple repetitions. Whilst load appears important in determining the mechanical response to a single repetition, other factors (e.g. volume and/or technique) play an important role in modulating these responses over multiple repetitions. These findings may help explain the efficacy of lighter load training for strength and hypertrophy development.

Confounding understanding in this area is the fact that few studies have equated different interventions by the volume of load lifted (i.e. equal volume). As such any reported findings may simply reflect differences in volume rather than the specific
kinematic and kinetic characteristics associated with a particular training scheme.
Unfortunately, the great majority of research does not adopt such an approach (Bauer
et al., 1990; Fatouros et al., 2000; Harris et al., 2000; Lyttle et al., 1996;
Schmidtbleicher & Buehrle, 1987; Weiss et al., 1999; Weiss & Relyea, 2001; Wenzel
& Perfetto, 1992) and as a result many of the suggested conclusions and practical
applications, based upon this data, are fundamentally flawed. Research that has used
this approach (equal volume training) have reported similar changes in strength and
muscle CSA across a range of loading (e.g. 6-8RM v 15-20RM v 30-40RM) schemes
(Chestnut & Docherty, 1999; Stone & Coulter, 1994; Taaffe, Pruitt, Pyka, Guido &
Marcus, 1996). Such findings again challenge the prescription of heavy training loads
as the only means for maximal strength adaptation.

A common training method (dynamic power loading) used for power development
involves the use of lighter loads (e.g. 45% 1RM) where movements are performed in
an explosive and/or ballistic manner (Newton et al., 1996; Stone et al., 1998).
Training in this manner is further typified by low volume and longer rest periods (e.g.
2-3 minutes) to maintain the “quality” of performance (i.e. high power output).
However, as previously mentioned the rationale for the prescription of load, based
upon maximising mechanical power output, could be questioned in terms of the power
demands of a particular activity. A further benefit of lighter loads exists in the fact
that such loads allow greater velocities to be achieved and thus, the better transfer of
effects to functional performance. However, research indicates that such effects
(velocity specific) may only be realised after a base level of strength and power is
developed (Jones et al., 2001; McBride et al., 2002; Wilson et al., 1993). Given that
the internal velocity of muscle may be independent of external load velocity, the
“intention” to move a load at high velocity may be more important than actual velocities achieved (Behm & Sale, 1993; Zehr & Sale, 1994). With this in mind the load utilised may be less important for high velocity adaptation to occur if training were performed with both maximal effort and intent.

Studies have found heavy load training (> 70% 1RM) effective in enhancing various measures of muscular power (Adams et al., 1992; Fatouros et al., 2000; Hoff & Almasbakk, 1995; McBride et al., 2002; Schmidtbleicher & Buehrle, 1987). These findings may be explained by the ambiguous nature of power performance and issues surrounding velocity-specific training effects. Other training methods (e.g. maximal strength training, plyometric training, isometric weight training and explosive power training) have also been found effective in enhancing power and functional performance (Lyttle, 1994; Young, 1989). Given the efficacy of such techniques, it may be of greater importance to determine the method that maximises power development above any other. For example, some studies have found combined strength (heavy load) and power (light load) training superior to either method alone in improving various measures of power (Adams et al., 1992; Fatouros et al., 2000; Harris et al., 2000). Again, none of these studies equated volume between training interventions. Determining the most effective training method for power development is again limited by the lack of available data (kinematics and kinetics) and thus, our understanding of the stimulus afforded by different loading schemes is also limited.

It would seem that our understanding of the kinematic and kinetic responses to various weight training methods is in its infancy. Making conclusions about the efficacy and/or adaptations of different training methods without such an understanding
appears highly questionable. It is suggested that research adopt a multiple set/exercise approach in their analysis to develop a better understanding as to the mechanical stimuli afforded by different strength and power programmes. Equating between parameters (e.g. time under tension, force, power, etc.) would also enable a better understanding of the importance of such variables in the development of strength and power.

**Hormonal response to resistance exercise**

The interaction between the primary anabolic (e.g. testosterone, growth hormone) and catabolic (e.g. cortisol) hormones are thought to regulate the remodelling of muscle tissue (Deschenes et al., 1991; Kraemer, 1992a). Resistance exercise has been shown to stimulate acute changes in blood borne hormone levels and through these mediate those cellular processors involved in muscle tissue growth (Kraemer, 1992a; Kraemer, 1992b). As the acute hormonal response to resistance exercise is largely determined by the specific configuration of the various training variables (e.g. volume, rest, etc.), program design plays an important role in determining the contribution of the endocrine system to adaptation. Examining the responsiveness of the primary anabolic and catabolic hormones to different strength and power schemes, will provide better understanding of the contribution of the hormonal stimulus in developing these qualities. This section will examine the testosterone, growth hormone and cortisol response to hypertrophic, neuronal and power loading schemes.

**Acute anabolic hormone response**

The majority of evidence supports the contention that testosterone (TST) has a considerable anabolic effect, directly and indirectly, upon muscle tissue growth
(Deschenes et al., 1991; Kraemer, 1992a). Secreted from the testes, TST is thought to contribute to muscle growth by increasing protein synthesis and decreasing protein degradation (Kraemer, 1992a; Kraemer, 1992b). The release of TST is also thought to enhance the training environment by augmenting the release of other anabolic hormones (i.e. growth hormone). The biologically active form of TST is in the free or unbound form which accounts for approximately 2% of all TST, with 38% bound to albumin whilst the remaining 60% is bound to sex hormone-binding globulin (SHBG) (Loebel & Kraemer, 1998). The 40% of TST not bound to SHBG is believed available for metabolism, the importance of which relates to the “free hormone” hypothesis that states that only the free component of TST is transported to the target tissues. This is partially supported by the larger increase (%) in free TST compared to total TST within research (see Tables 6 and 7). However, the validity of the free hormone hypothesis has not yet been established and the importance of the bound component may lie in the fact that the bound fraction dictates the amount of hormone available for receptor interactions (Kraemer, 1992a; Loebel & Kraemer, 1998).

Programmes designed to induce muscle growth (hypertrophy schemes) have generally been shown to elicit large increases in circulating TST (up to 72%) following a single training session (see Table 6). In comparison those programmes designed to enhance maximal strength through neural adaptations (neuronal schemes) have indicated smaller (up to 30%) or non-significant TST responses (see Table 7). For example, Kraemer and colleagues (1991) compared the hormonal response to eight exercises performed with a five repetition maximum load (5RM) for 3-5 sets per exercise and three minutes rest between sets (neuronal scheme), or a 10RM load (3 sets per exercise) with one minutes rest (hypertrophy scheme). The total TST response to the
hypertrophy scheme (~72%) was greater than that found following the neuronal scheme (~27%). Similarly, Hakkinen and Pakarinen (1993) reported an increase in total TST (24%) and free TST (22%) to a hypertrophy squat session (10 sets x 10 repetitions, 10RM and 3 minutes rest). However, a squat session employing neuronal protocols (20 sets x 1 repetition, 1RM and 3 minutes rest) revealed no changes in circulating TST (free and total). These findings confirm the importance of program design in modulating the acute TST response to resistance exercise.

Dynamic power schemes have also been found to induce significant TST responses (see Table 7). Mero, Komi, Kyllonen, Pullinen and Pakarinen (1991) examined the effect of half squat lifts performed with either moderate repetitions (10 sets x 6 repetitions, 50% 1RM, 1 and 4 minutes rest) or high repetitions (2 sets x 30 repetitions, 50% 1RM and 2 minutes rest). Both schemes resulted in a significant increase in total TST (~18-30%) immediately post exercise (single assay). Another study reported a 15% increase in total TST (single assay) immediately following a series of jump squats (5 sets x 10 repetitions, 30% 1RM and 2 minutes rest), performed by a group of trained males (Volek, Kraemer, Bush, Incledon & Boetes, 1997). It can be observed from Tables 6 and 7 that on average the TST response to dynamic power schemes (15%) are of similar magnitude to those found in response to hypertrophy schemes (15%). Whilst neuronal schemes have been found to elicit the smallest increase in TST (10%), it is important to recognise that a greater number of studies have examined different hypertrophy schemes. Further research is therefore warranted to examine the hormonal response to different neuronal and dynamic power schemes.
Table 6: Acute hormonal response to hypertrophy schemes.

<table>
<thead>
<tr>
<th>Author/s</th>
<th>Subjects (age)</th>
<th>Protocols Exercise/s - sets x reps (load)</th>
<th>Hormone (% or fold change)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>TST</td>
</tr>
<tr>
<td>Kraemer et al. (1990)</td>
<td>9 Males - T</td>
<td>8 ex - 3 x 10 (10RM)</td>
<td>↑ ~30</td>
</tr>
<tr>
<td>Boone et al. (1990)</td>
<td>11 Males - T</td>
<td>1 ex - 10 x 1-10 (50-110% 1RM) - SU</td>
<td>Nil</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 ex - 10 x 1-10 (50-110% 1RM) - NU</td>
<td>↑ ~20</td>
</tr>
<tr>
<td>Kraemer et al. (1991)</td>
<td>8 Males - T</td>
<td>8 ex - 3 x 10 (10RM)</td>
<td>↑ ~72</td>
</tr>
<tr>
<td>Kraemer et al. (1991)</td>
<td>8 Females - T</td>
<td>8 ex - 3 x 10 (10RM)</td>
<td>Nil</td>
</tr>
<tr>
<td>Kraemer et al. (1992)</td>
<td>8 Males - UT</td>
<td>4 ex - 3 x 10 (10RM)</td>
<td>Nil</td>
</tr>
<tr>
<td>Kraemer et al. (1993)</td>
<td>8 Males - T</td>
<td>8 ex - 3 x 10 (10RM)</td>
<td>Nil</td>
</tr>
<tr>
<td>Hakkinen and Pakarinen (1993)</td>
<td>10 Males - T</td>
<td>1 ex - 10 x 10 (10RM)</td>
<td>↑ ~24</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ ~1,70 fold</td>
</tr>
<tr>
<td>Hakkinen and Pakarinen (1995)</td>
<td>8 Males - UT (27yr)</td>
<td>1 ex - 5 x 10 (10RM)</td>
<td>↑ 9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ ~200 fold</td>
</tr>
<tr>
<td>Hakkinen and Pakarinen (1995)</td>
<td>8 Males - UT (47yr)</td>
<td>1 ex - 5 x 10 (10RM)</td>
<td>↑ 15</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ ~19 fold</td>
</tr>
<tr>
<td>Mulligan et al. (1996)</td>
<td>10 Females - T</td>
<td>8 ex - 1 x 10 (10RM)</td>
<td>↑ ~14</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ ~350</td>
</tr>
<tr>
<td>Gotshalk et al. (1997)</td>
<td>8 Males - T</td>
<td>8 ex - 1 x 10 (10RM)</td>
<td>↑ ~32</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ ~700</td>
</tr>
<tr>
<td>Volek et al. (1997)</td>
<td>12 Males - T</td>
<td>1 ex - 5 x 10 (10RM)</td>
<td>↑ 7</td>
</tr>
<tr>
<td>Kraemer et al. (1998)</td>
<td>9 Males - T</td>
<td>4 ex - 4 x 10 (10RM)</td>
<td>↑ ~20</td>
</tr>
<tr>
<td>Hakkinen et al. (1998)</td>
<td>10 Males - UT</td>
<td>2 ex - 4 x10 (100% MVC)</td>
<td>↑ ~27</td>
</tr>
<tr>
<td>Kraemer et al. (1998)</td>
<td>8 Males - UT (30yr)</td>
<td>1 ex - 4 x 10 (10RM)</td>
<td>↑ ~38</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ ~16 fold</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ ~78</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ ~40</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑ ~45</td>
</tr>
<tr>
<td>Study (Year)</td>
<td>Group</td>
<td>Protocol</td>
<td>Reps</td>
</tr>
<tr>
<td>-------------------</td>
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<td>----------</td>
<td>------</td>
</tr>
<tr>
<td>Kraemer et al. (1998)</td>
<td>8 Females - UT</td>
<td>3 ex - 3 x 6-8 (6-8RM)</td>
<td>Nil</td>
</tr>
<tr>
<td>Kraemer et al. (1998)</td>
<td>13 Males - UT</td>
<td>3 ex - 3 x 6-8 (6-8RM)</td>
<td>Nil</td>
</tr>
<tr>
<td>Bosco et al. (2000)</td>
<td>6 Males - T</td>
<td>3 ex - 12 x 8-12 (70-75% 1RM)</td>
<td>↓ ~70</td>
</tr>
<tr>
<td>Taylor et al. (2000)</td>
<td>6 Females - T</td>
<td>7 ex - 3-4 x 10 (10RM)</td>
<td>-</td>
</tr>
<tr>
<td>Taylor et al. (2000)</td>
<td>6 Females - UT</td>
<td>7 ex - 3-4 x 10 (10RM)</td>
<td>-</td>
</tr>
<tr>
<td>Smilios et al. (2003)</td>
<td>11 Males - T</td>
<td>4 ex - 2 x 10 (75% 1RM)</td>
<td>Nil</td>
</tr>
<tr>
<td>Smilios et al. (2003)</td>
<td></td>
<td>4 ex - 4 x 10 (75% 1RM)</td>
<td>↑ ~10</td>
</tr>
<tr>
<td>Smilios et al. (2003)</td>
<td></td>
<td>4 ex - 6 x 10 (75% 1RM)</td>
<td>Nil</td>
</tr>
</tbody>
</table>

**NOTE:** T, trained; UT, untrained; MVC, maximal voluntary contraction; TST, testosterone; GH, growth hormone; NU, non steroid users; SU, steroid users. ¹free testosterone
## Table 7: Acute hormonal response to neuronal and dynamic power schemes.

<table>
<thead>
<tr>
<th>Author/s</th>
<th>Subjects (age)</th>
<th>Protocols Exercises/s - sets x reps (load)</th>
<th>Hormone (% change)</th>
<th>TST</th>
<th>GH</th>
<th>Cortisol</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Neuronal schemes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kraemer et al. (1990)</td>
<td>9 Males - T</td>
<td>8 ex - 3/5 x 5 (5RM)</td>
<td>↑ ~30</td>
<td>↑ ~275</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Kraemer et al. (1991)</td>
<td>8 Males - T</td>
<td>8 ex - 3/5 x 5 (5RM)</td>
<td>↑ ~30</td>
<td>↑ ~275</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Kraemer et al. (1991)</td>
<td>8 Females - T</td>
<td>8 ex - 3/5 x 5 (5RM)</td>
<td>Nil</td>
<td>Nil</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Kraemer et al. (1993)</td>
<td>9 Females - T</td>
<td>8 ex - 3/5 x 5 (5RM)</td>
<td>Nil</td>
<td>↓ 70</td>
<td>Nil</td>
<td></td>
</tr>
<tr>
<td>Hakkinen and Pakarinen (1993)</td>
<td>10 Males - T</td>
<td>1 ex - 20 x 1 (100% 1RM)</td>
<td>Nil</td>
<td>↑ 361</td>
<td>Nil</td>
<td></td>
</tr>
<tr>
<td>Kraemer et al. (1993)</td>
<td>8 Males - T</td>
<td>8 ex - 3/5 x 5 (5RM)</td>
<td>-</td>
<td>-</td>
<td>Nil</td>
<td></td>
</tr>
<tr>
<td>Smilios et al. (2003)</td>
<td>11 Males - T</td>
<td>4 ex - 2 x 5 (88% 1RM)</td>
<td>Nil</td>
<td>Nil</td>
<td>↓ ~25</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 ex - 4 x 5 (88% 1RM)</td>
<td>Nil</td>
<td>↑ ~300</td>
<td>↓ ~22</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 ex - 6 x 5 (88% 1RM)</td>
<td>Nil</td>
<td>↑ ~300</td>
<td>↓ ~70</td>
<td></td>
</tr>
<tr>
<td><strong>Dynamic power schemes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mero et al. (1991)</td>
<td>9 Males - T</td>
<td>1 ex - 10 x 6 (50% 1RM)</td>
<td>↑ ~18-30</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 ex - 2 x 30 (50% 1RM)</td>
<td>↑ ~30</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Mero et al. (1993)</td>
<td>6 Males - (24 yr)</td>
<td>1 ex - 10 x 6 (50% 1RM)</td>
<td>↑ 16</td>
<td>-</td>
<td>Nil</td>
<td></td>
</tr>
</tbody>
</table>
|                     |                | 1 ex - 10 x 6 (50% 1RM)  
                   | ↑ 18               | -     | -     |          |
|                     |                | 1 ex - 10 x 6 (50% 1RM)          | ↑ 19^1             | -     | ↑ 33-67 |          |
|                     |                | 6 Males - (15 yr)  
                   | Nil               | -     | -     |          |
|                     |                | 1 ex - 10 x 6 (50% 1RM)          | ↑ 13               | -     | -     |          |
|                     |                | 1 ex - 10 x 6 (50% 1RM)          | ↑ 11^1             | -     | -     |          |
| Bosco et al. (1996) | 16 Males - T   | 60 seconds jumping (BW)                 | ↑ 12               | Nil   | ↑ 14  |          |
| Volek et al. (1997) | 12 Males - T   | 1 ex - 5 x 10 (30% 1RM)               | ↑ 15               | -     | Nil   |          |

NOTE: T, trained; BW, body weight; TST, testosterone; GH, growth hormone.  
^1 free testosterone, ^24-min rest, ^31-min rest
Whilst males generally exhibit increases in TST following resistance exercise such a response is not evident among females (see Tables 6 and 7). An exercise session performed with either 5RM and three minutes rest or 10RM with one minutes rest, resulted in an increase in total TST in males but not in females (Kraemer et al., 1993b; Kraemer et al., 1991). This may be attributed to the different production/release mechanisms of TST between genders. In males the luteinising hormone and follicle-stimulating hormone stimulates the Leydig cells of the testes to synthesis and release large amounts of TST into the blood stream. Within females smaller quantities of TST are produced in the ovaries and the adrenal glands, with much smaller amounts released into the blood (Kraemer, 1992a). Males also demonstrate higher resting concentrations of total TST than females (Hakkinen & Pakarinen, 1995; Kraemer et al., 1991; Kraemer et al., 1998b), which may again be related to the aforementioned mechanisms. These responses are not surprising as differences in strength and hypertrophy between genders have traditionally been attributed to the anabolic actions of TST (Burger, 2002; Kraemer, 1992b).

Interestingly, individuals who specifically train to increase muscle mass (i.e. bodybuilders) have revealed an inhibited TST response to resistance exercise (see Table 6). Bosco, Colli, Bonomi, Von Duvillard and Viru (2000) examined the hormonal response of six male bodybuilders performing an exercise bout consisting of half squat, leg press and leg extension exercises (4 sets x 8-12 repetitions, 70-75% 1RM, 1-2 minutes rest). A 70% reduction in total TST (single assay) was found after the exercise bout. Another study examined the hormonal response among a group of bodybuilders and power lifters assigned into two groups, anabolic steroid users (SU) and non anabolic steroid users (NU) (Boone et al., 1990). Following the performance
of an exhaustive squat session (10 sets x 1-10 repetitions, 50-110% 1RM, 4 minutes rest) no changes in total TST (single assay) were found in either group. This is somewhat surprising given that anabolic steroid usage is often associated with increased muscle mass. The importance of such a response (lowered TST) is difficult to discuss as the subsequent actions of other hormones (e.g. cortisol) may also be important to the hormonal environment and muscle growth.

One of the difficulties in interpreting research in this area exists in the fact that many studies have examined only a single assay when investigating the endocrine response to resistance exercise. Such an analysis does not adequately reflect the time course release of the various hormones within the body. Due to the spasmodic and continual secretion of most hormones frequent sampling is needed to characterise the actual dynamics of the hormonal response to the resistance exercise stimulus. Another difficulty surrounding the interpretation of research in this area lies in the time of sample extraction. Such assays are typically taken upon the immediate completion of the exercise program. Given that hormone secretion may occur from the onset of exercise additional samples may be required throughout a training session. One must therefore remain cognizant of limitations associated with the analysis of a single hormone and/or assay when extrapolating findings.

Chronic periods of weight training may also influence the acute TST response to the exercise stimulus (Kraemer et al., 1998b). Kraemer et al. (1998) examined the effects of a 9-week training program among a group of untrained males and untrained females. An exercise session comprising of three exercises (3 sets x 6-8 repetitions, 6-8RM and 2 minutes rest) was performed pre- and post- training to ascertain the
adaptive response of the endocrine system. After the training period an enhanced total TST response (~12%) compared to pre-training (no change) was found in males, which may be indicative of enhanced sensitivity of TST release to the resistance exercise stimulus. This may be attributed to various biological adaptations including; changes in hormone storage, transport mechanisms, hepatic clearance rates, fluid shifts, receptor affinity and receptor binding (Kraemer, 1992b; Kraemer & Mazzetti, 2003). Both groups also reported a change in resting hormone levels (increase total TST, decrease cortisol) compared to pre-training values. Given the relative importance of TST and cortisol such a response may be coupled to the remodelling of muscle tissue that is known to occur with weight training. Similar findings have been reported (Hakkinen et al., 2001; Kraemer et al., 1999; Marx et al., 2001; Staron et al., 1994), which may partly explain time course changes in strength where the contribution of hypertrophy is thought to occur later (>8 weeks) in the training period (Deschenes & Kraemer, 2002; Komi & Hakkinen, 1988).

It can be observed that TST secretion decreases as a consequence of aging (see Table 6). Kraemer et al. (1998) examined the hormonal response among a group of adult males (30yrs) and elderly males (62yrs), each performing a single exercise session (4 sets x 10 repetitions, 10RM and 90 seconds rest). The TST response was found to be greater in the younger group for both free TST (40% v 26%) and total TST (38% v 20%) respectively. Such a finding is supported by other research (Hakkinen & Pakarinen, 1995; Kraemer et al., 1999) thereby indicating a reduced TST response among elderly males to the same exercise stimulus. These differential responses may well explain the reduced ability of individuals to maintain strength and muscle mass (atrophy) with increasing age (Hopp, 1993; Lexell, Taylor & Sjostrom, 1988).
Proposed mechanisms for this include; failure of the hypothalamic-pituitary axis, changes in testicular function, an increase in SHBG levels and/or increased sensitivity of gonadotropin secretion to androgen negative-feedback inhibition (Izquierdo et al., 2001; Lexell et al., 1988). Considering the benefits of weight training (i.e. increase exercise-induced responses, altered basal levels), restoring endocrine function through resistance exercise remains an attractive hypothesis, which may help ameliorate the age-related decline in strength and muscle mass.

Although the acute endocrine response to resistance exercise has been studied extensively the nocturnal hormonal response has received much less attention. Given that the recovery period is critical for muscle regeneration to occur, nocturnal hormonal responses (e.g. 24 hours) may be of greater significance than the acute measurements (e.g. <90 minutes) commonly used within research. McMurray, Eubank and Hackney (1995) examined the nocturnal hormonal response among a group of untrained males to a resistance training session comprising of six exercises (3 sets x 6-8 repetitions, 88% 1RM). The exercise session was performed from 1900 to 2000 hours with hormone data (total TST, GH and cortisol) collected hourly from 2100 hours until 700 hours. The hormonal response to a non-active control group was also measured. The nocturnal response of cortisol and growth hormone was found to be no different from control values; however, the exercise group revealed significantly greater total TST compared to controls from 0500 to 0700 (P<0.05). This is suggestive of an early morning anabolic effect, particularly as cortisol levels during the same period remained unchanged. Due to the paucity of literature in this area however, the importance of nocturnal hormonal responses remains speculative.
Similar to TST, GH is thought to facilitate muscle growth by increasing protein synthesis and reducing protein degradation (Kraemer, 1992b; Widerman, Weltman, Hartman, Veldhuis & Weltman, 2002). Released from the anterior pituitary GH represents a family of proteins rather than a single hormone, though the function of its various derivatives has not yet been fully established. Some of the effects of GH may be mediated through polypeptides known as insulin-like growth factors or IGF’s. It has been further suggested that these polypeptides prolong the growth-promoting effects of GH secretion (Viru & Viru, 2001). However, the influence of IGF’s upon muscle tissue growth may depend upon the release mechanism of these hormones as IGF’s are known to be secreted by way of autocrine and paracrine pathways (Baechle & Earle, 2000). Regardless, less is known about the responsiveness of IGF’s to different resistance exercise schemes. This is not withstanding the fact that IGF’s may not follow a classic endocrine response (i.e. stimulus of gland resulting in hormone release into blood) as indicated by studies in this area (Baechle & Earle, 2000).

Exercise programmes designed to induce muscle growth have been shown to produce large GH responses (up to 200 fold) whilst neuronal schemes generally elicit much smaller (up to 4 fold) responses (see Tables 6 and 7). A study by Kraemer et al. (1990) examined the effects of load (5RM v 10RM), rest interval (1 v 3 minutes) and total work (high v low) on the hormonal responses among a group of trained males. The hypertrophy scheme (10RM, 1 minute, high total work) produced a much greater response than that found to the neuronal scheme (5RM, 3 minutes, low total work), with respective increases of 11-fold and 3-fold in GH reported. Other research supports these findings (Hakkinen & Pakarinen, 1993; Kraemer et al., 1993b; Kraemer et al., 1991; Smilios, Pilianidis, Karamouzis & Tokmakidis, 2003). On average the
GH response to hypertrophy schemes (30-fold) are larger than that found in response to neuronal schemes (3-fold) (see Tables 6 and 7). To our knowledge no research has examined the GH response to dynamic power loading schemes. Interpreting this information is again limited due to the lack of data available.

It can be observed that males generally elicit greater GH responses to resistance exercise compared to females (see Tables 6 and 7). Interestingly, some studies have reported greater exercise-induced responses of GH (absolute values) among females performing the same exercise protocol as males (Kraemer et al., 1991; Kraemer et al., 1998b). This may be related to the estrogen sensitization of the somatotrophs, which are known to give an increased responsiveness to a variety of stimuli among females (Kraemer et al., 1991). Differences in baseline levels between genders may also explain this finding. Kraemer et al. (1991) reported a significant difference in baseline levels between genders with females GH levels five times greater than that found in males (P<0.05). As males still demonstrate greater muscle mass and strength than females the importance of these elevated responses remains unknown. With most studies examining the hormonal response among women in the follicular phase of menstruation (Kraemer et al., 1993b; Kraemer et al., 1991; Kraemer et al., 1998b; Mulligan, Fleck, Gordon, Koziris & Triplett-McBride, 1996; Taylor, Thompson, Clarkson, Miles & De Souza, 2000) the effect of different protocols on hormone release, in different phases of the menstrual cycle, requires further investigation.

As with TST the training status of subjects may also influence the GH response to the stimulus of resistance exercise. A recent study compared the GH response between a group of trained females and untrained females, each performing the same resistance
exercise program (7 exercises, 3-4 sets x 10RM and 1 minute rest) (Taylor et al., 2000). A 90% increase in circulating GH was found in the trained group with a 30% increase reported in the untrained group. This may be partially due to the lower resting GH levels in the trained group (P<0.01). Differences in exercise-induced responses and basal levels of GH again reflect positive adaptations to chronic periods of weight training, as demonstrated by the trained group. The menstrual cycle in females is another important consideration when interpreting such data. It is known that the menstrual cycle may influence both the amplitude and frequency of GH secretion to exercise (Baechle & Earle, 2000). As with the majority of research in this area the women assessed in this study were in the follicular phase of menstruation.

Similar to TST the GH response to resistance exercise is also lowered with increasing age (see Table 6). A study by Hakkinen and Pakarinen (1995) examined the GH response to a single resistance exercise session (5 sets x 10 repetitions, 10RM and 3 minutes rest), among different age groups of untrained males (27yrs, 47yrs, 68yrs) and untrained females (25yrs, 48yrs, 68yrs). Within the male groups a reduction in GH was found with increasing age from 27yrs (200 fold) up to 47yrs (19 fold) and 68yrs (nil). Similar responses were reported within the various female groups. Although the 48yr group revealed a much larger increase in GH (20 fold) than the 25yr group (225%) following the exercise protocol, a reduced response was found in the 68yr group (nil). Other research have reported similar findings (Kraemer et al., 1998a; Kraemer et al., 1999), again supporting suggestions that the decline in muscle mass and strength with age may be partially attributed to changes in endocrine function.
**Acute catabolic hormone response**

Cortisol is considered the primary catabolic hormone as it inhibits protein synthesis and increases the level of enzymes that break down muscle protein (Burger, 2002; Viru & Viru, 2001). The anti-anabolic properties of cortisol are also related to its attenuation of the principal anabolic hormones such as TST and GH (Deschenes & Kraemer, 2002). Cortisol is often linked to various stressors such as exercise, trauma and overtraining, and in this capacity is widely considered a “stress” hormone (Urhausen & Kindermann, 2002). As such it may be speculated that the cortisol response is related to the nature of the training stress, afforded by the design of the exercise scheme. Similar to other hormones, hypertrophy schemes produce larger increases in circulating cortisol (up to 175%) compared to neuronal schemes, following a single exercise session (see Tables 6 and 7). For example, Kraemer et al. (1993) compared the hormonal response among a group of trained females to a hypertrophy (3 sets x 10 repetitions, 10RM, 1 minutes rest) and a neuronal scheme (3-5 sets x 5 repetitions, 5RM, 3 minutes rest), each performed over eight exercises. The cortisol response to the hypertrophy scheme (~125%) was much greater than that found after the neuronal scheme (Nil). Other research has reported similar findings (Hakkinen & Pakarinen, 1993; Kraemer et al., 1993a) thereby demonstrating greater cortisol responses to hypertrophy loading schemes.

Dynamic power schemes have also been shown to elicit a “stress” response as indicated by an increase in circulating cortisol (see Table 7). Mero, Pullinen, Komi, Pakarinen, Kyllonen and MacDonald (1993) investigated the hormonal response to boys and men performing a half squat exercise session (10 sets x 6 repetitions, 50% 1RM) with both one and four minute rest periods. The boys reported significant
increases in serum cortisol (37-67%) with both rest periods with no changes in the adult males (P<0.05). This is indicative of a greater stress response in the younger males, which may be explained by differences in endocrine function between pubertal and adult males (Fry & Schilling, 2002). For example, adult males demonstrate higher resting TST levels compared to pubertal males that may enable, in some capacity, adult males to tolerate more stressful training events. However, only a single assay was performed in this study. It can be observed in Tables 6 and 7 that on average the cortisol response to hypertrophy schemes (35%) are of similar magnitude to that found to dynamic power schemes (38%). In contrast the cortisol response to neuronal schemes reveals a reduction in cortisol (-30%). Again, with scant data available an accurate analysis of such information is limited. Further research is therefore necessary to determine the responsiveness of cortisol to different neuronal and power loading schemes.

As a stress hormone cortisol release may be further modulated by other factors independent of the scheme employed and subject age. An analysis of the different hypertrophy schemes revealed on average a greater increase in cortisol amongst untrained males (40%) compared to males (31%) with training experience (see Table 6). With this in mind it may be speculated that trained individuals possess greater tolerance to the stress of resistance exercise. Gender differences may also exist with regards to the ability of individuals to tolerate stressful exercise. It can be seen in Table 6 that females performing hypertrophy schemes have on average revealed greater cortisol responses compare to males (45% v 35% respectively). Unfortunately, little scientific data directly supports these suggestions and as such the influence of training experience and/or gender upon cortisol release remains speculative. Adding
to the difficulty of this analysis is the fact that cortisol release is modulated by other factors such as emotional strain, fatigue, diet and trauma (Viru, Smirnova, Kareelson, Snegovskaya & Viru, 1996; Viru & Viru, 2001). Whilst the cortisol response to stress, as mediated by the stimulus of exercise is well recognised, labelling cortisol as the major stress hormone may itself be controversial. Given that TST and GH levels show differential responses according to the nature of the training stress (i.e. loading scheme), they too may be classified as stress hormones.

An interesting dichotomy is raised in that schemes designed to induce muscle growth elicit large catabolic or muscle inhibiting responses. This may be explained by the complex nature of cortisol and its function within the body. In breaking down muscle protein the catabolic actions of the glucocorticoids may create an increased pool of free amino acids, or the “building blocks” for protein synthesis to occur (Viru & Viru, 2001). Viru and Viru (2001) further suggested that the catabolic effects of glucocorticoids might be essential to increase protein turnover rate in previously active muscles during the recovery period. The secretion of cortisol may therefore aid in the remodelling of muscle tissue. Importantly, cortisol has a wide spectrum of metabolic effects including, stimulation of the glucose-alanine cycle, decreased glucose use by the cells and reduction of cellular protein stores (O'Shea, 1984; Viru & Viru, 2001), and as such changes in cortisol levels may simply reflect an increase in hormone activity to maintain homeostasis. Despite this other hormones (i.e. anabolic) are released concurrently in response to the exercise stimulus and may work to counter any negative effects of cortisol. This is evident in Table 6 with hypertrophy schemes also producing large increases in circulating concentrations of TST and GH.
Whilst most research indicates that cortisol levels are acutely amplified following a single bout of resistance exercise, some data has indicated a depression of pituitary-adrenocortical activity as indicated by lowered cortisol levels (see Tables 6 and 7). Smilios, Pilianidis, Karamouzis and Tömjakidis (2003) reported a reduction in cortisol levels among resistance-trained males performing a neuronal loading scheme (4 exercises, 5 repetitions, 88% 1RM, 3 minutes rest) using either a 2-set, 4-set or 6-set per exercise protocol. This response appeared related to the volume of training performed with a greater reduction found in the 6-set protocol (~70%) compared to the 2- and 4-set protocols (~22-25%). A study by Bosco et al. (2000) also reported a reduction in cortisol (single assay) among six male sprinters performing an exercise bout comprising of half and full squats (6 series x 6 + 6 + 4 repetitions, 80% 1RM, 8 minutes rest). Due to differences in program design and other methodological issues (e.g. single assay, low subject numbers) determining the mechanisms for a lowered cortisol response is difficult. Nonetheless, the importance of such a response, in terms of resultant adaptations, is difficult to discuss given the added importance of anabolic hormone activity.

Implications for strength and power development

The endocrine response to resistance exercise plays an important role in facilitating changes in muscular performance through morphological adaptation. In conjunction with other stimuli (e.g. high forces, TUT, stretch) the hormonal stimulus is thought to modulate the dynamic balance between protein synthesis and protein degradation. The stimulus of resistance exercise is also known to modulate the “quality” of protein synthesized (Goldspink, 1992) however, less is know about such adaptations and their importance to the expression of strength, hypertrophy and power. In terms of muscle
quantity”, an increase in the physiological CSA of muscle increases the potential for force generation and through this strength and power performance. The events involved in this process are outlined in Figure 4.

**Figure 4**: Flow chart of events involving the endocrine system and the adaptive processors associated with resistance training. Modified from Kraemer (1992).

The hormonal environment (e.g. increase TST, GH and cortisol) afforded by hypertrophy schemes appears conducive for morphological adaptation to occur. That is, an increase in circulating hormone levels increases the likelihood of receptor interactions and cell metabolism thereafter. In particular a much greater increase in anabolic activity has been observed in those programmes designed to induce muscle growth, which would appear necessary for greater protein turnover (synthesis and degradation) and the subsequent accretion of muscle protein. The smaller hormonal responses found after the different neuronal schemes suggests that training in this manner is less likely to result in muscle growth. The fact that such schemes are thought to induce changes in maximal strength through neural rather than morphological adaptation (Bloomer & Ives, 2000) is partially supported by this data.
The importance of the endocrine system to power adaptation appears less clear. Although the TST and cortisol response to hypertrophy and power schemes appear similar, there exists a lack of data to substantiate the hormonal response to different schemes (i.e. dynamic power). Furthermore, the mechanical and metabolic responses to hypertrophy and power schemes are likely to be quite different and may also determine if morphological adaptation will occur. A key issue is whether or not muscle growth is a desirable adaptation for enhancing power performance. An increase in muscle CSA is known to enhance the force generation capabilities of muscle and in doing so may improve power performance. If muscle growth were important for power adaptation then individuals may be better advised to perform hypertrophy type schemes. However, training in this manner (i.e. controlled velocities, fatigue) may limit the extent to which muscular power may be fully realised. An increase in muscle CSA via hypertrophy training may also be undesirable for many activities if accompanied by an increase in body weight.

Those mechanisms that mediate muscle growth remain controversial and may involve many factors such as hormone receptors, binding hormones, releasing/inhibiting hormones, target organs, etc. For example, it is the resultant binding with hormone receptors that ultimately determines whether or not alterations in cell metabolism will occur (Baechle & Earle, 2000; Kraemer & Mazzetti, 2003). The biological actions of the hormone-receptor complex will itself be determined by factors such as the receptor domain, number of receptors, receptor binding sensitivity, etc. It may be that body builders exhibit more effective hormone-receptor interactions than other individuals, explaining the limited hormone response found in these individuals following resistance exercise. This also means that different loading schemes may elicit similar
hormone activity yet elicit different responses. Such a notion may partially explain the similar hormonal response found between hypertrophy and power schemes in the literature reviewed. The importance of target organs must also be recognised as it is known that training one muscle does not result in global adaptive changes. This is likely to be related to the additional mechanical stimuli afforded by resistance exercise when training a given muscle and/or muscle group.

It is apparent that the contribution of the endocrine system to strength and power has yet to be fully elucidated. The endocrine system is complex and involves the actions/interactions of multiple hormones, including the primary anabolic and catabolic hormones. This is not withstanding the controversy surrounding the mechanisms by which muscle growth occurs. In terms of the data presented it must be acknowledged that the percentage change in hormonal responses only reflects the endocrine response to resistance exercise. Such data does not represent any volume changes in hormone levels or the time integrated response (i.e. area under the curve) of hormone secretion, the importance of which remains unknown. It is suggested that research examine the endocrine response to different strength and power training schemes and further, adopt a more systematic approach in the analysis of hormone activity (i.e. more samples and assays, nocturnal responses, etc.). Such an analysis would enable a better understanding of the importance of the endocrine system in the development of maximal strength and power.

**Metabolic response to resistance exercise**

The metabolic response to resistance exercise is thought important for strength and to a lesser extent power development (Abernethy et al., 1994; Enoka, 2002). Resistance
exercise has been shown to elicit acute changes in the levels of various circulating metabolites (e.g. lactate, glycogen, creatine kinase, ammonia, etc.) and through these changes contribute to resultant adaptation (Carey Smith & Rutherford, 1995; Tesch, 1987). As the metabolic response is determined by the configuration of the various training variables (e.g. volume, rest, etc.), program design plays an important role in determining the contribution of the metabolic stimulus to subsequent adaptation. Examining the metabolic response to loading schemes used to improve strength and power will provide some understanding of the contribution of the metabolic stimulus in developing these qualities. This section will investigate the responsiveness of various metabolites to hypertrophic, neuronal and power loading schemes.

**Acute metabolic response**

Resistance exercise is known to produce marked changes in various metabolic enzymes, substrates and by-products as indicated in Tables 8 and 9. For example, Tesch, Colliander and Kaiser (1986) examined the metabolic response to a typical training session comprising of four lower body exercises (4 sets x 6-12 repetitions, 6-12RM, 1 minute rest). A significant reduction in adenosine triphosphate (ATP), creatine phosphate and muscle glycogen was reported (P<0.05). Increases in lactate, creatine, glycerol 3-phosphate, glucose, glucose-6-phosphate levels and plasma glycerol were also found (P<0.05). As many of these metabolites are used to indicate energetic requirements during exercise (i.e. anaerobic and aerobic pathways) the metabolic response to resistance exercise appears linked to the contribution of the different energy systems, as determined by program design. Importantly, the majority of research has examined lactate alone which does not adequately reflect the metabolic response to exercise. It is therefore recommended that a greater range of metabolites...
be examined within research in order to develop a more complete metabolic profile to resistance exercise.

Dynamic power schemes have also been shown to elicit some metabolic responses (see Table 9). A similar increase (~50%) in serum lactate (single assay) was found between two groups of males performing half squat exercises (10 sets x 6 repetitions, 50% 1RM), performed with either 1 or 4 minute rest periods (Mero, Komi, Kyllonen, Pullinen & Pakarinen, 1991). When performing an equal volume of training with 30 repetitions (per set) a much larger response was found (482%). This finding confirms the importance of program design in modulating the metabolic (lactate) response to exercise. Another study investigated the effect of six sets of leg extensions with loads of 70% (I-70) and 35% (I-35) 1RM (Robergs et al., 1991). In spite of the difference in load, both schemes produced a similar increase in muscle lactate (~13-14 fold), which may be attributed to the number of repetitions performed per set in the I-35 and I-70 conditions (13 v 6 respectively). On average hypertrophy schemes have revealed greater metabolic (lactate) responses compared to neuronal and dynamic power schemes (see Tables 8 and 9). Again, such findings must be interpreted cautiously with little metabolic data available and few metabolites assessed.

It can be observed that males generally elicit greater metabolic (lactate) responses compared to females (see Tables 8 and 9). For example, when examining the lactate response to hypertrophy schemes, males on average have demonstrated a larger increase (~7 fold) compared to females (~5 fold). Kraemer and colleagues (1998) compared the lactate response among a group of untrained males (n = 13) and untrained females (n = 8), each performing the same exercise program consisting of
three exercises (3 sets x 6-8 reps, 6-8RM, 2 minutes rest). Following this program the male subjects reported a 13-fold increase in serum lactate whilst the females reported a much lower response (7-fold). This may be attributed to factors such as the greater relative loads used by males and differences in relative muscle mass between genders. With only limited research directly examining such responses, the influence of gender upon lactate responses to different exercise schemes remains highly speculative.

The lactate response to resistance exercise may be further modulated by subject training experience. It can be observed in Tables 8 and 9 that on average trained males produced a 600% increase in lactate concentrations, compared to 740% among untrained males. Such a notion is supported by other data. Kraemer and colleagues (1998) examined the effects of a 10 week training program among two groups of untrained males (30yr and 60yr). Both groups revealed a reduction in serum lactate responses, when comparing a single exercise bout performed pre- (700% v 380% respectively) and post training (460% v 350% respectively). It may be speculated that trained individuals possess a greater aerobic capacity than non-trained individuals, thereby delaying the onset of anaerobic glycolysis and hence, a reduction in blood lactate concentrations. This may be attributed to factors such as increased capillarization, improved oxygen extraction, alterations in muscle fibre size and distribution, and changes in enzyme activity and substrate levels with resistance training (Baechle & Earle, 2001; Abernethy et al., 1994).
Table 8: Acute metabolic response to hypertrophy schemes.

<table>
<thead>
<tr>
<th>Author/s</th>
<th>Subjects (age)</th>
<th>Protocols</th>
<th>Metabolite (% or fold change)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Exercise/s - sets x reps (load)</td>
<td>Lactate</td>
</tr>
<tr>
<td>Tesch, Colliander and Kaiser (1986)</td>
<td>9 Males - T</td>
<td>4 ex - 5 x 6 - 12 (6-12RM)</td>
<td>↑ 391</td>
</tr>
<tr>
<td>Kraemer et al. (1990)</td>
<td>9 Males - T</td>
<td>8 ex - 3 x 10 (10RM)</td>
<td>↑ 592</td>
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<tr>
<td>Robergs et al. (1991)</td>
<td>8 Males - T</td>
<td>1 ex - 6 x 6 (70 % 1RM)</td>
<td>↑ ~13 fold</td>
</tr>
<tr>
<td>Kraemer et al. (1991)</td>
<td>8 Males - T</td>
<td>8 ex - 3 x 10 (10RM)</td>
<td>↑ 648</td>
</tr>
<tr>
<td>Kraemer et al. (1991)</td>
<td>8 Females - T</td>
<td>8 ex - 3 x 10 (10RM)</td>
<td>↑ 478</td>
</tr>
<tr>
<td>Kraemer et al. (1991)</td>
<td>8 Females - T</td>
<td>8 ex - 3 x 10 (10RM)</td>
<td></td>
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<tr>
<td>Kraemer et al. (1992)</td>
<td>8 Males - UT</td>
<td>4 ex - 3 x 10 (10RM)</td>
<td>↑ ~750</td>
</tr>
<tr>
<td>Kraemer et al. (1993)</td>
<td>8 Males - T</td>
<td>8 ex - 3 x 10 (10RM)</td>
<td>↑ ~600</td>
</tr>
<tr>
<td>Kraemer et al. (1993)</td>
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<td>8 ex - 3 x 10 (10RM)</td>
<td>↑ 433</td>
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<tr>
<td>Hakkinen and Pakarinen (1993)</td>
<td>10 Males - T</td>
<td>1 ex - 10 x 10 (10RM)</td>
<td>↑ 971</td>
</tr>
<tr>
<td>Mulligan et al. (1996)</td>
<td>10 Females - T</td>
<td>8 ex - 1 x 10 (10RM)</td>
<td>↑ ~550</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8 ex - 3 x 10 (10RM)</td>
<td>↑ ~430</td>
</tr>
<tr>
<td>Gotshalk et al. (1997)</td>
<td>8 Males - T</td>
<td>8 ex - 1 x 10 (10RM)</td>
<td>↑ ~440</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8 ex - 3 x 10 (10RM)</td>
<td>↑ ~690</td>
</tr>
<tr>
<td>Kraemer et al. (1998)</td>
<td>9 Males - T</td>
<td>4 ex - 4 x 10 (10RM)</td>
<td>↑ 718</td>
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<tr>
<td>Hakkinen et al. (1998)</td>
<td>10 Males - UT</td>
<td>2 ex - 4 x10 (10RM)</td>
<td>↑ ~190</td>
</tr>
<tr>
<td>Kraemer et al. (1998)</td>
<td>8 Males - UT</td>
<td>1 ex - 4 x 10 (10RM)</td>
<td>↑ ~700</td>
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<tr>
<td>Kraemer et al. (1998)</td>
<td>13 Males - UT</td>
<td>3 ex - 3 x 6-8 (6-8RM)</td>
<td>↑ ~13 fold</td>
</tr>
<tr>
<td>Kraemer et al. (1998)</td>
<td>8 Females - UT</td>
<td>3 ex - 3 x 6-8 (6-8RM)</td>
<td>↑ ~700</td>
</tr>
<tr>
<td>MacDougal et al. (1999)</td>
<td>8 Males - T</td>
<td>1 ex - 1 x failure (80% 1RM)</td>
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<td></td>
<td>1 ex - 3 x failure (80% 1RM)</td>
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<tr>
<td>Smilios et al. (2003)</td>
<td>11 Males - T</td>
<td>4 ex - 2 x 10 (75% 1RM)</td>
<td>↑ ~700</td>
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<td></td>
<td></td>
<td>4 ex - 4 x 10 (75% 1RM)</td>
<td>↑ ~850</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 ex - 6 x 10 (75% 1RM)</td>
<td>↑ ~760</td>
</tr>
</tbody>
</table>

NOTE: T, trained; UT, untrained; ATP, adenosine triphosphate; CK, creatine kinase; G-3-P, glycerol 3-phosphate. ¹glucose-6-phosphate, ²creatine phosphate
Table 9: Acute metabolic response to neuronal and dynamic power schemes.

<table>
<thead>
<tr>
<th>Author/s</th>
<th>Subjects (age)</th>
<th>Protocols</th>
<th>Metabolite (% or fold change)</th>
<th>Lactate</th>
<th>Glucose</th>
<th>Ammonia</th>
<th>Other</th>
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<td></td>
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<tr>
<td>Kraemer et al. (1990)</td>
<td>9 Males - T</td>
<td>8 ex - 3/5 x 5 (5RM)</td>
<td>↑460</td>
<td>Nil</td>
<td>-</td>
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<tr>
<td>Kraemer et al. (1991)</td>
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<td>8 ex - 3/5 x 5 (5RM)</td>
<td>Nil</td>
<td>Nil</td>
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<tr>
<td>Kraemer et al. (1991)</td>
<td>8 Females - T</td>
<td>8 ex - 3/5 x 5 (5RM)</td>
<td>↑140</td>
<td>Nil</td>
<td>-</td>
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<td>Kraemer et al. (1993)</td>
<td>9 Females - T</td>
<td>8 ex - 3/5 x 5 (5RM)</td>
<td>↑133</td>
<td>↓11</td>
<td>Nil</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Hakkinen and Pakarinen (1993)</td>
<td>10 Males - T</td>
<td>1 ex - 20 x 1 (100% 1RM)</td>
<td>↑84</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Kraemer et al. (1993)</td>
<td>8 Males - T</td>
<td>8 ex - 3/5 x 5 (5RM)</td>
<td>↑~400</td>
<td>-</td>
<td>Nil</td>
<td>CK ↑~110</td>
<td></td>
</tr>
<tr>
<td>Smilos et al. (2003)</td>
<td>11 Males - T</td>
<td>4 ex - 2 x 5 (88% 1RM)</td>
<td>↑~350</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 ex - 4 x 5 (88% 1RM)</td>
<td>↑~400</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 ex - 6 x 5 (88% 1RM)</td>
<td>↑~350</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Dynamic power schemes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Robergs et al. (1991)</td>
<td>8 Males - T</td>
<td>1 ex - 6 x 13 (35% 1RM)</td>
<td>↑~14 fold</td>
<td>-</td>
<td>-</td>
<td>Glycogen ↓38</td>
<td></td>
</tr>
<tr>
<td>Mero et al. (1991)</td>
<td>9 Males - T</td>
<td>1 ex - 10 x 6 (50% 1RM)</td>
<td>↑~50</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 ex - 10 x 6 (50% 1RM)</td>
<td>↑~50</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 ex - 2 x 30 (50% 1RM)</td>
<td>↑~480</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Mero et al. (1993)</td>
<td>6 Males - (24 yr)</td>
<td>1 ex - 10 x 6 (50% 1RM)</td>
<td>Nil</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 ex - 10 x 6 (50% 1RM)</td>
<td>↑27</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>6 Males - (15 yr)</td>
<td>↑35</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 ex - 10 x 6 (50% 1RM)</td>
<td>↑94</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

NOTE: T, trained; CK, creatine kinase. 14-min rest, 21-min rest
It can be observed in Tables 8 and 9 that an acute bout of exercise elicits an increase in creatine kinase levels from baseline. Creatine kinase is an enzyme found within the muscle and often used within research as a metabolic marker of muscle damage. As such it may be speculated that an increase in creatine kinase levels reflects an increase in muscle damage following resistance exercise, thereby supporting the "break-down build-up" theory of muscle growth. However, with limited data reporting the magnitude of tissue damage (as indicated by creatine kinase responses), the adaptive response to different loading schemes remains unclear. It must also be recognised that creatine kinase levels in the blood only provides an indirect measure of tissue damage and is often used as part of a battery of assessments (e.g. range of motion, isometric force generation, swelling, delayed onset of muscle soreness, etc.). Whilst such assessments are common within practice they too provide only indirect measures of muscle tissue damage.

Similar to the examination of hormonal responses an accurate interpretation of research in this area is limited by various methodological issues, such as the examination of a single assay and/or metabolite. Another important consideration is the different metabolic sampling methods used within research in this area (e.g. blood samples, muscle biopsies, saliva samples). For example, saliva samples only provide an indirect measure of circulating metabolite levels in the blood and itself, an indirect measure of muscle metabolite concentrations. Given the time course difference in partitioning between the various systems and biological membranes in the body, this data should be interpreted accordingly. A further consideration in the collection of muscle samples exists in the fact that the invasiveness of some methods (e.g. muscle biopsies) may itself induce a metabolic and/or hormonal response, thereby
confounding any results reported. These issues limit an accurate interpretation of the metabolic/hormonal response to resistance exercise and require careful consideration when extrapolating findings.

Implications for strength and power development

Whilst the direct contribution of the mechanical and hormonal stimuli is well recognised, the metabolic response to resistance exercise may play some indirect role in strength and power adaptation. It is known that training programmes designed to promote muscle mass tend to be more muscle fatiguing (Lambert & Flynn, 2002; Rooney, Herbert & Balnave, 1994) and with this it may be speculated the metabolic stimulus is important for muscle growth. That is, programmes characterised by high muscular fatigue are likely to elicit greater metabolic responses than non-fatiguing programmes. Such a notion is partially confirmed by the greater lactate response to hypertrophy schemes, which in combination with an increase in hydrogen ions and changes in muscle pH levels, is often associated with muscular fatigue (Bilcheck & Maresh, 1992; Deschenes & Kraemer, 1989). As a consequence of fatigue or greater metabolic activity other potential mechanisms for adaptation may be stimulated (e.g. motor unit recruitment, hormone release). The smaller metabolic response to neuronal and dynamic power schemes suggests fatigue is less likely to occur and thus, little metabolic influence to subsequent adaptations.

Whilst fatigue is often thought to mediate adaptation such a notion may be overly simplistic. The mechanisms of fatigue are complex and may involve changes in the central nervous system drive to the motor neurons, changes in neuromuscular propagation, availability of metabolic substrates and dehydration (Bilcheck & Maresh,
1992; Deschenes & Kraemer, 1989). The mechanisms by which the metabolic stimulus contributes to adaptation also remain speculative. For example, Takarada, Sato and Ishii (2002) found that integrated electromyography (iEMG) of a low intensity (40% 1RM) exercise performed with occlusion was almost equal to that of a high intensity (80% 1RM) exercise without occlusion. This partially supports suggestions that an increase in metabolic activity may enhance motor unit recruitment (Carey Smith & Rutherford, 1995). Other literature suggests that an increase in blood lactate and hydrogen ion concentrations may augment the hypophyseal secretion of GH (Kraemer et al., 1998b; Taylor et al., 2000). A 290-fold increase in plasma GH concentrations was found with a low intensity (20% 1RM) exercise with occlusion, whereas no such effect was found after the same exercise without occlusion (Takarada et al., 2000). However, given this type of methodology (i.e. occlusion), the metabolic responses found may not reflect that occurring under normal training conditions.

It has been proposed that an increase in metabolic waste products (e.g. lactate) combined with altered hydrogen ion concentrations and ATP deficiency may further explain exercise-induced muscle damage (Ebbeling & Clarkson, 1989). If muscle damage were a precursor to muscle growth then the metabolic stimulus may further contribute to the training environment. However, most data indicates that the mechanisms for muscle damage are more likely to involve mechanical factors (e.g. high forces, stretch) rather than metabolic factors (Lieber & Friden, 1993; Nosaka & Newton, 2002; Tarnopolsky & Roy, 2000). In spite of this it remains to be seen if muscle damage is a prerequisite for muscle growth to occur as other factors (e.g. age, genetic potential, diet, etc.) may also be important. Whilst these data elucidate
potential mechanisms for adaptation, the contribution of the metabolic stimulus to strength and power adaptation remains controversial.

Our understanding of the metabolic stimulus and its contribution to strength and power is also in its infancy. The metabolic response to resistance exercise is complex and involves various energetic systems and their combined influence upon substrate use, enzyme activity, resultant by-products, etc. Given the limited research in this area the importance of the metabolic stimulus and further, mechanisms for adaptation remain largely unknown. It is suggested that more research be conducted in this area to develop a better understanding as to the metabolic response afforded by different strength and power programmes. To develop a more complete metabolic profile research should further examine a greater number of metabolites and/or assays. Such an analysis would enable a better understanding of the importance of the metabolic stimulus in the development of strength and power. As with the hormonal responses, the metabolic data presented does not represent any volume changes in metabolic activity or time-integrated responses.

**Conclusion**

Given the importance of these mechanical, hormonal and metabolic factors, it is disconcerting to note the paucity of literature that has investigated how these factors and their interaction, might affect the development of strength and power. Therefore, a true understanding of the adaptations elicited by various resistance training protocols are for the most part not well understood. Making conclusions about the efficacy and/or adaptations of various training without such an understanding of the interaction appear highly questionable. Until strength and power research examine the
mechanical, hormonal and metabolic responses to multiple repetitions, sets and exercises, much of the research will not contribute greatly to our understanding of how various training schemes optimise strength and power development. Consequently, one should remain cognizant of the limitations that exist in the interpretation of research data in this field.
CHAPTER THREE – THE MECHANICAL, HORMONAL AND METABOLIC PROFILE OF TWO LOADING SCHEMES

Prelude

It would seem from the literature that the training load utilised is not as important as a training stimulus as initially proposed. Both heavy and light load training have found to be equally effective in the improvement of strength, hypertrophy and power. Realising that adaptation of muscle will depend on some interaction between the mechanical, hormonal and metabolic responses, examining such responses during light and heavy load training would develop our understanding as to the stimuli afforded by different loading schemes. Therefore, the purpose of this study was to determine the mechanical, hormonal and metabolic responses to two different loading schemes (light and heavy), of equal volume.

Introduction

Muscular strength and power are important components of many athletic pursuits and everyday activities (Komi & Hakkinen, 1988). If the muscle is not overloaded above a certain intensity (load-force), which is thought to be in the vicinity of 60-70% 1RM (MacDougall, 1992; McDonagh & Davies, 1984), it is generally believed that no increase in strength and/or hypertrophy will occur. However, some studies have found lighter loads (<45% 1RM) to be as effective as heavier loading schemes in facilitating changes in muscular strength, hypertrophy and power (Dahl et al., 1992; Harris et al., 2000; Lyttle et al., 1996; Moss et al., 1997; Schmidtbleicher & Buehrle, 1987). In order to adequately train speed and power, training loads allowing high power output (e.g. 45% 1RM) are thought necessary (Kraemer et al., 2002; Stone et al., 1998).
Again, improvements in muscular power have been reported using loads (>70% 1RM) traditionally prescribed for the improvement of maximal strength and muscular size (Adams et al., 1992; Fatouros et al., 2000; Hoff & Almasbakk, 1995; Schmidtbleicher & Buehrle, 1987). Consequently, there exists some debate as to the load (% 1RM) that would best facilitate changes in maximal strength and power.

In terms of the underlying determinants of strength and power development, three stimuli (mechanical, hormonal and metabolic) appear important (Enoka, 2002). The mechanical or kinematic/kinetic stimuli (e.g. time under tension, force, power, etc.) associated with resistance exercise are thought most important for training-induced adaptation to occur (Enoka, 2002; McDonagh & Davies, 1984). Many studies have investigated repetition kinematics and kinetics associated with different resistance exercises and loads (Baker, 2001; Baker et al., 2001; Cronin et al., 2001a; Newton et al., 1996; Newton et al., 1997), and as such much is known about the mechanical response to a single repetition. To our knowledge though, no studies have systematically examined these effects over multiple sets and/or exercises. Given the inherent nature of a typical resistance training session (i.e. multiple repetitions, sets and exercises) such an analysis would appear fundamental to improving our understanding of how mechanical stimuli contribute to strength and power development.

Endocrine responses to resistance exercise are also important for strength and power development (Hakkinen, 1989). The interaction between the primary anabolic (i.e. testosterone, growth hormone) and catabolic (i.e. cortisol) hormones are thought to regulate the dynamic balance between protein synthesis and degradation (Deschenes &
Kraemer, 2002; Kraemer, 1992b), the results of which producing either a net gain or loss in muscle tissue. Resistance exercise is known to elicit acute and chronic changes in blood hormone levels, thereby contributing to the long-term process of muscle growth (Kraemer, 1992b). However, few studies have examined the acute response to loading schemes and/or techniques commonly used to improve muscular strength and power (e.g. maximal strength and light load power training). Understanding the differential response of the endocrine system to such loading parameters would also add to our understanding on how best to improve strength and power.

The metabolic response to resistance exercise may also be important for strength and to a lesser extent power development (Abernethy et al., 1994; Enoka, 2002). Training conditions associated with greater “metabolic stress” have been found equally effective in inducing strength and hypertrophic changes compared to training conditions associated with greater mechanical stress (Carey Smith & Rutherford, 1995; Higbie, Cureton, Warren & Prior, 1996; Marler et al., 1999). Changes in the metabolic environment subsequent to resistance exercise may stimulate anabolic hormone release and/or contribute to greater motor unit recruitment (Carey Smith & Rutherford, 1995; Takarada et al., 2000). An accumulation of metabolic by-products (i.e. lactate) combined with altered ion concentrations may also contribute to greater muscle damage (Ebbeling & Clarkson, 1989), further contributing to adaptation. Unfortunately, there is little scientific evidence as to how various metabolites change when different strength and power programmes are used.

It would seem from the literature that a minimum load or threshold tension is not as important as a training stimulus as initially proposed. Both light and heavy load
training have been found to be equally effective in the improvement of strength, hypertrophy and power. Realising that adaptation of muscle will depend on some interaction between mechanical, hormonal and metabolic responses, examination of such responses would provide greater understanding as to the stimuli afforded by different training schemes. Therefore, the purpose of this study was to determine the mechanical, hormonal and metabolic responses to a light (45% 1RM) and heavy (88% 1RM) loading scheme of equal volume.

**Methods**

*Experimental Design*

An acute randomised study using a cross-over design was performed to examine the mechanical, hormonal and metabolic responses to two different loading schemes.

*Subjects*

Eleven male subjects volunteered to participate in this study. The mean (SD) age and mass of the participants were 26.6 (6.7) years and 79.0 (8.1) kg respectively. All subjects had a minimum of 12 months weight training experience (3-4 times per week) and were considered healthy. Each subject had the risks of the investigation explained to them and signed an informed consent prior to their participation in this research. The Human Subject Ethics Committee of the Auckland University of Technology approved all procedures undertaken.
**Equipment**

Subjects performed their assessments on an isoinertial supine squat machine (Fitness Works, Auckland, NZ) and a modified Smith machine (Fitness Works, Auckland, NZ). The supine squat machine incorporated a 300kg pin loaded weight stack attached to a sled, allowing horizontal movement on low friction sliders (see Figure 5). It was designed to allow subjects to perform maximal squats or explosive squat jumps, with the back supported, thus minimizing the risk associated with such exercises in an upright position. The undercarriage of the supine squat machine enabled the sled’s range of motion to be adjusted in 2cm increments, allowing lower limb joint angles to be standardized between subjects.

![Supine squat machine](image)

**Figure 5:** Supine squat machine.

The Smith machine (see Figure 6) allowed the squats to be performed in the vertical direction. The squatting position was adjusted in 2cm increments using a mechanical brake, again allowing lower limb joint angles to be standardized. With additional free
plates the load used for each exercise was adjustable in 2.5kg increments. A linear transducer (P-80A, Unimeasure, Oregon – mean sensitivity 0.499mV/V/mm, linearity 0.05% full scale) was attached to each apparatus which measured displacement for each exercise with an accuracy of 0.01cm. Data was sampled at 200Hz and collected via a custom-built computer based data acquisition program (Labview™ 6.0).

![Modified Smith machine](image)

**Figure 6:** Modified Smith machine.

**Protocols**

Testing for this study was conducted over three sessions. Subjects were familiarized with test procedures in the first session during which their one repetition maximum (1RM) strength was determined on the supine squat and Smith machines. Strength assessment on the supine squat machine involved subjects maintaining a flat back with shoulders placed firmly against the carriage pads. In this position subjects placed their feet upon the pushing plate (toes aligned to the top of the plate) whilst adopting a shoulder width stance. Assistance was provided to move the carriage and subject into
a fully extended position prior to each trial. Following 30 minutes rest the Smith machine squat assessment was performed. Bar placement on the shoulders was between C7 and the superior aspect of the scapula. A shoulder width stance was again used with heels placed directly under the bar. Squat depth for both exercises was performed to a knee angle of 90° (measured with a manual goniometer). Hip angle at this depth was not measured. A repetition to failure protocol was used to establish subject 1RM for each exercise as previously described (Heyward, 1991). Trials were considered valid if performed with good technique (i.e. knees in line with toes, back flat, no bouncing) and without assistance.

The following two sessions involved subjects completing the light (45% 1RM) and heavy (88% 1RM) schemes. A standardized warm-up was performed prior to each session consisting of a five minute cycle (moderate load) at 60rpm, followed by stretches for the calf, hamstring and quadriceps muscle groups. Two light warm-up sets (10 repetitions per set) were then performed on the supine squat machine with loads of 100% and 150% of bodyweight respectively. After the warm-up subjects were randomly assigned to perform the light or heavy scheme. The light scheme consisted of eight sets of six repetitions, with six sets of four repetitions performed in the heavy scheme. Respective rest periods (between sets) of three and four minutes were employed. The loading parameters employed were based upon previous recommendations for power development using either light or heavy loads (Kraemer et al., 2001; Newton et al., 1996; Wilson et al., 1993), with each scheme equated to ensure training volume was approximately equal (total repetitions x load). For each session half the sets were performed on the supine squat and Smith machines, with the supine squat exercise preceding the Smith squat in both sessions. Movements began
in an extended position to better simulate a typical weight training session with assistance provided (if necessary) to ensure the prescribed number of repetitions were performed per set. At least 72 hours recovery was given between schemes with no other training performed during this period.

Subjects were instructed to control the load on the eccentric phase of each repetition and perform the concentric phase in an explosive manner, irrespective of the load utilized. The heavy scheme was performed using a traditional weight training technique (i.e. non-projection) for both exercises. With the light scheme, concentric movements on the supine squat were performed in a ballistic manner (i.e. jump squats), the subjects instructed to move the load as explosively as possible, which resulted in the feet leaving the plate. The concentric movements on the Smith squat were also explosive; however, subjects were instructed to extend up to their toes only to minimize the risk of injury. The mechanical brake on each machine was adjusted to ensure the knee angle achieved at the bottom position of each exercise (90°) was standardized between subjects and replicated across exercises, sets and repetitions.

Data Analysis

The displacement time data were filtered using a low pass second order Butterworth filter with a cut-off frequency of 5 Hz. The filtered displacement values were then differentiated to determine velocity and acceleration. From this data the various temporal (time under tension) and kinetic (force, impulse, work, power) variables of interest were determined (Cronin, McNair & Marshall, 2000; Cronin et al., 2001a). Data was calculated for the eccentric and concentric phase of each repetition, as well as each set and each exercise. This data was then combined to provide total values for
each mechanical variable. The eccentric phase of each repetition was defined as the period of time from maximum to minimum vertical displacement (i.e. lowering the load). The concentric phase was the period of time from the minimum vertical displacement to maximum vertical displacement (i.e. lifting the load).

Saliva Sampling

Saliva samples were used to determine the hormonal (testosterone and cortisol) and metabolic (lactate) responses in this study. Prior to assessment all subjects were advised to avoid eating any coarse textured food, brushing their teeth, drinking coffee or other hot drinks two hours prior to supplying saliva samples (Cook, 2002). The sampling procedure required subjects to deposit saliva samples (about 5ml) into 25ml sterile containers (Labserve, Auckland, NZ), which were then stored at -20°C until assay. These samples were collected at rest (pre-), immediately after the first exercise (mid-), at the conclusion of the second exercise (P0) and every 15 minutes thereafter (P15, P30, P45, P60) for a period of one hour (see Figure 7).

![45% 1RM loading scheme](image1)

![88% 1RM loading scheme](image2)

Figure 7: Time course sampling for each loading scheme.
After each training session subjects sat passively in the laboratory until all post exercise samples had been collected. No food was taken during this period. Each subject performed their respective sessions at approximately the same time of the day (±30 minutes) to account for the effects of circadian rhythm upon hormonal responses (Kraemer et al., 2001). Subjects were instructed to replicate diet and hydration 24 hours prior to testing to reduce the influence of dietary variations upon the various samples of interest. Verbal confirmation of these instructions was gained before any testing was carried out.

**Saliva Analysis**

The hormonal samples were analysed in duplicate by Enzyme-Linked Immunosorbent Assay (ELISA) methods with samples centrifuged for 15 minutes prior to analysis. The analysis of testosterone (TST) was performed as per manufacturer’s instructions (Salivary Testosterone Enzyme Immunoassay Kit, Salimetrics). Briefly, 50 µl of TST standards and 50 µl of saliva were pipetted into wells coated with antibodies to TST after which a diluted conjugate solution (150 µl) was added to each well. The plate was then mixed on a plate rotator (500 rpm) at room temperature for 60 minutes, washed using an electronic plate washer, after which 200 µl of tetramethylbenzidine (TMB) was added to each well and mixed for five minutes. The plate was then incubated at room temperature in the dark for 25 minutes. After incubation 50 µl of stop solution was pipetted into each well followed by three minutes mixing.

Cortisol analysis was also performed using the manufacturer’s instructions (Salivary Cortisol Enzyme Immunoassay Kit, Salimetrics). Briefly, 25 µl of TST standards and 25 µl of saliva were pipetted into wells, coated with antibodies to cortisol. A diluted
conjugate solution (200 µl) was then added to each well. The plate was mixed (500 rpm) at room temperature for five minutes and left to incubate for 55 minutes. The plate was again washed, after which 200 µl of TMB was added to each well, mixed for five minutes and left to incubate in the dark at room temperature for 25 minutes. After incubation 50 µl of stop solution was pipetted into each well and mixed for three minutes. Intra assay variance for TST and cortisol samples were 3.4% and 3.0%, respectively (Bioengineering group, HortResearch, Hamilton, New Zealand).

Lactate was measured in saliva samples following an initial step in which nitrogen was pumped over the liquid sample at approximately 150 kPa (1125 mm Hg) for five minutes to concentrate the sample. Direct measurement of a 10 µl sample was then performed using an electrochemical sensor for lactate oxidase incorporated as a test strip into a Lactate Pro, LT-1710 (KDK Corporation, Kyoto Daiichi Kagaku Co., Ltd Nishi Aketa-Cho, Higashi-Kujo, Minami-Ku, Kyoto 601-8045, Japan). The procedures for this analysis has been previously described (Shimojo et al., 1993; Westgren et al., 1995) with an accuracy range of 0.05nmol/l provided for the lactate samples obtained (Bioengineering group, HortResearch, Hamilton, New Zealand). Salivary amylase was also measured to assess changes in concentration of saliva secretion across experiment, as previously described (Searcy, Wilding & Berk, 1967).

Statistical Analysis

Means and standard deviations were used throughout as measures of centrality and spread of data. Change scores were computed for the various mechanical, hormonal and metabolic variables with a spreadsheet (Hopkins, 2003). The spreadsheet also computed chances (% and qualitative) that the true effects were substantial. Raw
values of change scores were used in the analysis of the mechanical variables (force output, time under tension, impulse, work and power output) and for the lactate values; otherwise, each subject’s change score was expressed as a percent baseline score, to reduce bias arising from non-uniformity of error (Hopkins, 2003). The mechanical data was further divided into eccentric and concentric phases for analysis. Testosterone and cortisol were log-transformed before analysis to reduce non-uniformity bias with standard deviations back-transformed to coefficients of variation. The fixed effects were data of interest (mechanical, hormonal and metabolic), group (light and heavy scheme) and their interaction. The random effects were within group and between group variance. Mean values for all variables were further analysed with a paired sample T-test. A statistical significance of P<0.05 was used in this analysis.

Results

The mechanical responses to the light and heavy loading schemes can be observed in Table 10. In terms of the eccentric phase significantly greater total time under tension, total work and total power outputs were associated with the 45% 1RM protocol. These findings were similar for the concentric phase. Total impulse was the only variable that was found to be significantly greater during the 88% 1RM scheme. Differences in time under tension, work, power and impulse between schemes were almost certain to be substantial (100% probability). No significant difference in total forces was observed between schemes though it is very likely that the difference between loading schemes in concentric force output (797 N) was substantial (96% probability).
Table 10: Total force output, total time under tension, total impulse, total work and total power output during eccentric and concentric phases of the 45% and 88% 1RM loading schemes.

<table>
<thead>
<tr>
<th>Variables</th>
<th>45% 1RM Mean (SD)</th>
<th>88% 1RM Mean (SD)</th>
<th>Difference (raw)</th>
<th>Chances that the true differences are substantial Qualitative</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eccentric phase</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Force (N)</td>
<td>31767 (2452)</td>
<td>31524 (3358)</td>
<td>243</td>
<td>63</td>
<td>Possibly not</td>
</tr>
<tr>
<td>Time (sec)</td>
<td>51.87 (2.89)</td>
<td>34.81 (2.30)</td>
<td>17.06</td>
<td>100</td>
<td>Almost certain</td>
</tr>
<tr>
<td>Impulse (N sec(^{-1}))</td>
<td>36027 (2233)</td>
<td>45368 (4248)</td>
<td>9341</td>
<td>100</td>
<td>Almost certain</td>
</tr>
<tr>
<td>Work (J)</td>
<td>18214 (2560)</td>
<td>14291 (1703)</td>
<td>3924</td>
<td>100</td>
<td>Almost certain</td>
</tr>
<tr>
<td>Power (W)</td>
<td>19557 (3187)</td>
<td>8844 (1248)</td>
<td>10713</td>
<td>100</td>
<td>Almost certain</td>
</tr>
<tr>
<td><strong>Concentric phase</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Force (N)</td>
<td>37853 (2909)</td>
<td>37056 (3192)</td>
<td>797</td>
<td>96</td>
<td>Very likely</td>
</tr>
<tr>
<td>Time (sec)</td>
<td>42.57 (1.48)</td>
<td>34.81 (2.07)</td>
<td>7.77</td>
<td>100</td>
<td>Almost certain</td>
</tr>
<tr>
<td>Impulse (N sec(^{-1}))</td>
<td>35189 (2406)</td>
<td>52855 (4572)</td>
<td>17666</td>
<td>100</td>
<td>Almost certain</td>
</tr>
<tr>
<td>Work (J)</td>
<td>17225 (2784)</td>
<td>11659 (1920)</td>
<td>5566</td>
<td>100</td>
<td>Almost certain</td>
</tr>
<tr>
<td>Power (W)</td>
<td>23466 (3437)</td>
<td>11190 (1904)</td>
<td>12276</td>
<td>100</td>
<td>Almost certain</td>
</tr>
</tbody>
</table>
The salivary testosterone (TST) response to the light and heavy schemes can be observed in Figure 8. A decrease in TST as compared to baseline was observed over time for the heavy scheme (-4 to -29%) whereas a non-significant increase was observed for the light scheme (1 to 12%). Only the TST response at P60 was significantly less (-29%) than baseline for the heavy scheme (P=0.01). However, it was likely (77-86% probability) that there were substantial decreases in TST at P15 (-23%), P30 (-17%) and P45 (-20%). Significant differences between schemes were found at P30 (32%) and P60 (58%). It is likely that the differences between the light and heavy schemes at mid-, P15 and P45 were also substantial (86-93% probability).

![Graph of saliva testosterone concentrations](https://example.com/graph.png)

**Figure 8:** Changes in saliva testosterone concentrations (log transformed) of the 45% and 88% 1RM loading schemes. Means (± SD) are presented for testosterone (pg/ml).

The salivary cortisol responses for both schemes are plotted in Figure 9. In relation to baseline measurements cortisol decreased for both the light (-6 to -30%) and heavy (-14 to -44%) schemes until 45 minutes post-workout, thereafter a slight increase was
observed for both schemes. For the light scheme a significant reduction in cortisol was found at P45 (-30%) and it is likely (85-93% probability) that the differences from baseline at P15 (-21%) and P30 (-28%) were substantial. Compared to baseline significant reductions in cortisol were found at mid- (-14%), P0 (-19%), P15 (-31%) and P45 (-44%) following the heavy scheme, with the differences at P30 (-31%) and P60 (-24%) also likely to be substantial (79-92% probability). No differences in the cortisol response were found between the two loading schemes.

![Graph showing changes in saliva cortisol concentrations](image)

* Significantly different from baseline P<0.05

**Figure 9:** Changes in saliva cortisol concentrations (log transformed) of the 45% and 88% 1RM loading schemes. Means (± SD) are presented for cortisol (ng/ml).

The TST/cortisol ratio to each loading scheme is shown in Figure 10. Compared to the baseline an increase in the TST/cortisol ratio was observed over time for the light scheme (17 to 49%), whereas an increase up to P45 (2 to 44%) was observed in the heavy scheme and thereafter a decrease occurred at P60 (-6%). Significant differences to baseline were found in the light scheme at P15 (41%) and P45 (44%), whilst the
differences at P30 (49%) and P60 (27%) were likely to be substantial (77-93% probability). For the heavy scheme a significant increase was found at P45 (44%) with the difference at P0 (19%) likely to be substantial (79% probability). Between group analyses showed that the light scheme produced a greater TST/cortisol ratio response across all time periods. Significant differences were found at P15 (53%) and P30 (51%), with the differences found at mid- (40%), P45 (21%) and P60 (65%) were all likely to be substantial (78-89% probability).

![Figure 10: Changes in testosterone/cortisol concentrations (log transformed) of the 45% and 88% 1RM loading schemes. Means (± SD) are presented for testosterone/cortisol (arbitrary units).](image)

* * Significantly different from baseline P<0.05
** Significantly different from light scheme P<0.05

Pre and post exercise results for salivary lactate were analysed only up to P30 due to test kit availability. Significant increases in lactate were found in both loading schemes as compared to baseline values for all the time intervals (see Figure 11). Increases in lactate concentrations of 0.8-1.0nmol/l and 0.3-0.5nmol/l were found across the light and heavy schemes respectively. No significant differences between
loading scheme lactate responses were observed. Amylase results (data not presented) showed no significant differences between groups or across each workout schedule.

**Figure 11:** Changes in saliva lactate concentrations (log transformed) of the 45% and 88% 1RM loading schemes. Means (± SD) are presented for lactate (nmol/l).

**Discussion**

The mechanical stimulus of resistance exercise (e.g. forces, power, time under tension, etc.) is believed to be the most important stimulus for training-induced adaptations to occur (Enoka, 2002). The development of high mechanical forces is thought to be one of the most important mechanical stimuli for strength and hypertrophy adaptation (McDonagh & Davies, 1984). High forces are required to stimulate a greater number of motor units, according to the “size principle”, for neural (maximal strength) and morphological (hypertrophy) adaptation to occur. Both loading schemes produced similar total forces over the eccentric and concentric phases in this study. Research *in vivo* shows that force output during a single repetition increases with an increase in
load (Cronin et al., 2001a; Newton et al., 1997). It is on this basis (high load tension) that heavy training loads are thought to provide the superior stimulus for inducing strength and hypertrophic changes. However, examination of forces in this manner does not adequately reflect the nature of the weight-training stimulus, where it is the cumulative effects of multiple repetitions (i.e. total forces) that elicit the training response. Cronin and Crewther (2003) reported greater total forces with a light load (30% 1RM) when examining the response to three equal-volume loading conditions (30, 60, 90% 1RM) performed on a supine squat machine. Although mean forces increased with load, differences in the number of repetitions performed in each condition (6, 3 and 2 respectively) resulted in greater total forces for the 30% 1RM load. The similarity in total force output in this study would be largely attributed to the number of repetitions performed in light (48) and heavy (24) schemes.

The high force output found in the light scheme may be further attributed to the ballistic technique employed (supine squat only). Newton, Kraemer, Hakkinen, Humphries and Murphy (1996) found that the bench press throw allowed the bar to be accelerated for 96% of the throw movement as opposed to 60% for a traditional bench press movement. With longer acceleration periods the throw technique resulted in enhanced velocities (27-36%) and subsequently, greater forces (3-35%) and power (67-70%) compared to the non-throw technique. Similar findings have been reported (Cronin et al., 2001a, 2003; Newton et al., 1997; Stone et al., 2003) thereby demonstrating superior mechanical responses when a given training load is projected in some manner. Therefore, the manner in which the load is moved and/or the volume lifted may be of equal importance to the magnitude (%1RM) of the load lifted. That is, techniques that utilise lighter loads but are explosive in intent and ballistic in nature...
produce high force outputs and if equated by volume, may be as effective as heavier loading (>70% 1RM) schemes for producing high force outputs. This may in part explain studies that have found both light and heavy load training to be equally effective in increasing muscle CSA, strength and power (Dahl et al., 1992; Harris et al., 2000; Lyttle et al., 1996; Moss et al., 1997; Schmidtbleicher & Buehrle, 1987).

The amount of time under tension (TUT) is also thought important for strength and hypertrophy (Komi & Hakkinen, 1988). In theory the longer a muscle is subjected to a stimulus the greater the potential for adaptation to occur. The light scheme resulted in greater TUT for both the eccentric and concentric phases. As greater forces are developed at slower velocities (concentric force-velocity relationship) an increase in load would produce longer contraction durations (Cronin & Crewther, 2003; Cronin et al., 2001a; Newton et al., 1997). The prescription of heavy loads on this basis is again flawed given the repetitive nature of weight training. The number of repetitions performed in the light and heavy schemes would explain the greater amount of time with the 45% 1RM load. It should be however be recognized that such findings do not take into account different weight-training techniques used within practice (e.g. super slow, rest-pause, etc.) and their influence upon contraction duration. Nonetheless, if time under tension were important for strength and hypertrophic adaptation, the similar results reported previously using lighter training intensities, as compared to the heavier paradigms, might also be explained by this response.

This analysis of TUT however, may over simplify our understanding of TUT as a training stimulus and it may be that TUT is less important than the duration of each epoch. That is, the heavy scheme with long rest periods but fewer epochs offers a
different stimulus to the lighter scheme that has shorter rest periods but more epochs. It may be speculated that the heavier scheme results in greater vascular occlusion thereby decreasing blood flow (Bond et al., 1996; Edwards, Hill & McDonnell, 1972; Tamaki, Uchiyama, Tamura & Nakano, 1994). It has been suggested that the occlusion and subsequent muscle anoxia associated with this type of training may cause muscles to become of the slow type as evidenced in the high proportions of slow muscle fibres and myosin heavy chain (MHC) (Sale, MacDougall, Jacobs & Garner, 1990; Tesch & Larsson, 1982). If this were the case, greater lactate accumulation and phosphocreatine degradation would be expected with decreases in muscle pH and increases in ADP and inorganic phosphates. However, the lactate response did not differ significantly between schemes. As such it may be that the lighter load scheme with a greater number of epochs offers a better alternative to traditional continuous high intensity strength training in terms of rest and recovery, maintenance of fibre type/MHC expression and hence, increased explosive force and power production.

If high forces and TUT were the most important mechanical stimuli for adaptation, then it may be speculated that the development of large impulses (force x time) may be the critical stimulus for improvement of strength and muscle CSA. Accordingly, heavier loads would provide the superior training stimulus to achieve these changes, as the heavy scheme was associated with greater total impulse over the eccentric and concentric phases. Unfortunately, the assessment and/or practical significance of impulse are not well documented. A comparison of single repetitions (eccentric and concentric) showed that impulse increased with heavier masses (30, 60, 90% 1RM) and whilst the 90% 1RM load produced greater total concentric impulse, no significant differences were found between conditions in terms of total eccentric impulse (Cronin
& Crewther, 2003). Our understanding of strength and power adaptation may benefit from research differentiating between impulse as opposed to force and time under tension and mapping adaptation thereafter.

In theory the greatest gains in power performance would be achieved with training loads that maximise mechanical power output. Early research indicated that maximal power output occurred at approximately 30% of maximum isometric strength and 30% of maximum shortening velocity (Faulkner et al., 1986; Moritani, 1992; Perrine, 1986). However, recent findings using isoinertial (constant gravitational load) multiarticular movements in vivo suggest that heavier loading intensities (50-70% 1RM) maximize power output (Baker, 2001; Baker et al., 2001; Cronin & Crewther, 2003; Cronin et al., 2001a). Disparate findings may be explained by factors such as the mode of dynamometry, subject training status, assessment technique and the exercise performed. Unfortunately, this data is again based upon the findings of single repetitions, which may have little practical relevance. The light scheme in this study produced greater total power over the eccentric and concentric phases, with these values more than double that found in the heavy scheme. Cronin and Crewther (2003) reported a similar finding with greater total power in the 30% 1RM condition for both the eccentric (25-48%) and concentric (40-69%) phases, compared to the 60% and 90% 1RM conditions. These findings suggest that lighter schemes produce greater total power if compared to heavier schemes of equal volume.

It may be that force alone does not adequately account for changes in strength and muscle CSA but rather the distance over which that force acts or work (Stone et al., 1998). In attempting to explain the increases in 1RM strength and CSA of their 35%
1RM-training group (G35), Moss and colleagues (1997) found that G35 performed 70% more work than the 90% 1RM-training group. Other studies also emphasize the importance of work performed (Carey Smith & Rutherford, 1995; Shinohara, Kouzaki & Fukunaga, 1997). From our understanding of single repetitions an increase in load produces an increase in work, as the force component (work = force x distance) is enhanced (Brown et al., 1990; Craig & Kang, 1994; Cronin & Crewther, 2003; Kang et al., 1996). However, these studies also reported greater total work (set response) with the lightest loads examined, due to the performance of high total repetitions accounting for the heavier loads. The findings of the present study also found greater total concentric and eccentric work performed in the lighter scheme, which may be again be attributed to differences in repetitions performed and technique. If total work performed were important for adaptation, then light load training appears an attractive training option compared to heavier paradigms of equal volume.

One of the difficulties of interpreting training studies exists in the fact that few have equated different interventions by volume. As such any reported findings may simply reflect differences in volume rather than the specific kinetic and kinematic characteristics associated with a particular training program. Research adopting this approach (equal volume) has reported similar changes in maximal strength and muscle CSA between different (e.g. 6-8RM, 15-20RM or 30-40RM) loading programmes (Chestnut & Docherty, 1999; Stone & Coulter, 1994; Taaffe et al., 1996). These results provide further evidence that a minimum load threshold exists for strength and hypertrophic adaptation to occur. The similar adaptations cited within research may be partially explained by the mechanical responses reported in this study. That is, similar total forces were produced and although the lighter scheme resulted in greater...
TUT, power and work, this may be accounted for by the greater impulse in the heavier scheme. Although resistance exercise is not typically prescribed in this manner, such an approach provides a context from which we may better interpret research findings.

Whilst an examination of multiple repetitions enables a better understanding of the mechanical stimulus to a single resistance exercise session (i.e. total responses), such an analysis also provides an understanding as to the kinematic and kinetic changes occurring across a given set and/or exercise. For example, different weight training schemes employed within practice (e.g. hypertrophy) are characterised by large amounts of muscular fatigue and thus, force and power outputs recorded over the last few repetitions are likely to be quite different from the initial few repetitions. This approach certainly provides more valid data rather than simply multiplying repetition kinetics and kinematics by the number of repetitions performed to ascertain total responses. Accordingly, a multiple repetition approach may allow us to differentiate mechanical responses between schemes as well as responses within a given scheme.

As observed, data in this study was presented as combined values for all the mechanical variables assessed.

The endocrine system and it’s response to resistance exercise is also important for mediating training-induced changes in strength, hypertrophy and power. The dynamic interaction between the anabolic (e.g. testosterone) and catabolic (e.g. cortisol) are thought to regulate the balance between protein synthesis and degradation (Kraemer, 1992b). Secreted from the testes in men and the ovaries and adrenal glands in women, testosterone (TST) is an important anabolic hormone affecting muscle tissue growth through various direct and indirect mechanisms (Kraemer, 1992b). Directly, TST is
transported to the muscle, after which it associates with a cytosolic receptor and migrates to the cell nucleus, where interactions with nuclear receptors on the DNA take place, resulting in protein synthesis (Kraemer, 1992b, Baechle & Earle, 2001). When protein synthesis is greater than protein degradation the gross accretion of muscle protein occurs (muscle growth). Indirectly, TST may further mediate the complex process of muscle growth by stimulating other anabolic hormones such as growth hormone (GH).

Temporal analysis of salivary TST resulted in no significant changes across the light scheme; however, a substantial and significant decrease (-17 to -29%) in TST was observed from P15 to P60 in the heavy scheme. Substantial and significant differences in TST concentrations were also found between the two loading schemes from P15 to P60 (29 to 58%), with the lighter scheme resulting in greater TST levels. Not unexpectedly, hypertrophy schemes (high total work, moderate loads, short rest periods) have been shown to elicit relatively large TST responses (Hakkinen & Pakarinen, 1993; Kraemer et al., 1991; Kraemer et al., 1998a; Kraemer et al., 1990; Smilios et al., 2003). These same studies found that schemes characterised by lower total work, heavier loads and longer rest periods (neuronal schemes), elicit reduced or non-significant TST responses. Training programmes characterised by lighter loads and explosive movements, as per this study, have reported small or non-significant TST responses (Mero et al., 1991; Mero et al., 1993). Such data underscores the importance of program design in modulating the TST response to resistance exercise.

Whilst the light scheme appears consistent with other research, with either an increase or no changes in TST, the response in the heavy scheme (reduction from baseline)
appears a more novel response. A recent study reported a reduction in TST among two groups of males (sprinters, bodybuilders), each performing two different exercise programmes (Bosco, Colli, Bonomi, Von Duvillard & Viru, 2000). The male sprinters \( (n = 6) \) performed half and full squat exercises (6 sets x 6 + 6 + 4 repetitions, 80% 1RM) with the bodybuilders \( (n = 6) \) performing half squat, leg press and leg extension exercises (4 sets x 8-12 repetitions, 70-75% 1RM). The lowered TST response (single assay) in each group was partially attributed to the effect of fatigue. It may be that the way in which load volume was lifted in the heavy scheme (reduced quantity of work:rest epochs and greater impulse) resulted in greater fatigue, hence the lowered TST response. An accurate interpretation of the above study is however limited by low subject numbers and the measurement of a single assay. Regardless, such a response is difficult to discuss, as the analysis of TST alone does not adequately reflect the “hormonal environment” afforded by resistance exercise.

Secreted from the adrenal cortex, cortisol is generally considered a catabolic hormone as it inhibits protein synthesis, increases protein degradation and further, by attenuating other hormones such as TST and growth hormone (Deschenes & Kraemer, 2002). Cortisol is often linked to various stressors (e.g. exercise, trauma, overtraining, etc.) and in this capacity is widely considered a “stress” hormone (Urhausen, 2002). Substantial and significant decreases (-14 to -44%) in salivary cortisol were observed for both schemes until P45, with no differences in salivary cortisol found between schemes. Similar to TST, hypertrophy schemes produce large cortisol responses with neuronal schemes resulting in smaller or non-significant responses (Hakkinen & Pakarinen, 1993; Kraemer et al., 1993a; Kraemer et al., 1993b; Kraemer et al., 1998a). Dynamic power schemes, similar to the lighter scheme performed in this study, have
also produced non-significant cortisol responses (Mero et al., 1993; Volek et al., 1997). The findings of these studies again underscore the importance of program design in modulating the hormonal response to exercise.

Given the similar cortisol response between schemes it may be speculated that cortisol was less sensitive to how the load was lifted but influenced more by the total volume lifted. The findings of Smilios, Piliandis, Karamouzis and Tokmakidis (2003) give limited support to such a contention. These researchers reported a reduced cortisol response after the completion of a heavy loading scheme (5 repetitions per set, 88% 1RM, 3-minute rest) performed for either 2-, 4- or 6-sets per exercise. The magnitude of this response appeared related to training volume with the cortisol response in the 6-set protocol (~70%), greater than that found in the 2- and 4-set protocols (~22-25%). Irrespective of this finding a reduction in cortisol coupled with either an increase or no changes in TST would appear to produce a more favourable hormonal environment for muscular adaptation to occur. With this in mind the hormonal response to the light scheme appears conducive to muscle protein accretion in the recovery period. These findings give further insight as to how a minimal threshold load (e.g. 60-70% 1RM) may be less important than initially thought for strength and hypertrophy.

The TST/cortisol ratio has been used within research in an attempt to mark the “anabolic” response to resistance exercise (Fry, Kraemer & Ramsey, 1998; Gorostiaga, Izquierdo, Iturralde, Ruesta & Ibanez, 1999; Potteiger, Judge, Cerny & Potteiger, 1995). The use of this ratio reduces systematic error in interpretation (e.g. single hormone analysis) and further, guards against the problems associated with changes in salivary secretion or hydration. Both schemes produced an increase in the
TST/cortisol ratio, though greater variability was found across the heavy scheme (-6 to 44%) compared to the light scheme (17 to 49%). Such findings suggest a potential anabolic response to both schemes. However, this ratio has been used with only limited success, which may be partly attributed to the complex role of cortisol in the body. For example, cortisol has a wide spectrum of metabolic effects including stimulation of the glucose-alanine cycle, decreased glucose use, reduction of cellular protein stores and anti-inflammatory effects (Viru & Viru, 2001). Cortisol release is also modulated by other factors including emotional strain, temperature, fatigue, diet and trauma (Viru et al., 1996; Viru & Viru, 2001). As stress is relative by nature the “perception” of individuals, when performing different loading schemes, may further influence cortisol responses. Such a response was not addressed in this study.

Although TST and cortisol are widely recognised as important hormones for adaptation to occur, the release and subsequent actions/interactions of other hormones (e.g. growth hormone, somatomedins, catecholamines, etc.) to the exercise stimulus may also determine whether or not muscle growth will occur. It must be further recognised that resultant adaptations occur after the repeated application of multiple training bouts. That is, the accumulated hormonal effects over multiple training sessions will ultimately determine the training response in terms of strength, hypertrophy and power development. As such the importance of the hormonal response following a single exercise bout, as performed in this study, may not adequately account for any long-term adaptive changes associated with a given exercise stimulus. With this in mind the acute hormonal responses reported within literature should be extrapolated with some caution.
It has been suggested that the metabolic response to resistance exercise may contribute to the training stimulus (Carey Smith & Rutherford, 1995). As a by-product of anaerobic metabolism the lactate response to resistance exercise is determined by the relative contribution of energy supply via the glycolytic pathway. Both loading schemes produced an increase in salivary lactate across all time intervals with no significant differences between schemes. Peak lactate concentrations reached 2.50nmol/l and 2.51nmol/l in the heavy and light schemes respectively. The lactate response to hypertrophy programmes (7.5-10.5mmol/l) are typically greater than that found after neuronal (3.3-5.0mmol/l) (Kraemer et al., 1993a; Kraemer et al., 1993b; Kraemer et al., 1990; Kraemer et al., 1991; Smilios et al., 2003) and light load programmes (3.1-3.7mmol/l) (Mero et al., 1991; Mero et al., 1993). As with hormonal responses, these data suggest that lactate responses may be modulated by program design. Whilst these data suggest smaller lactate responses in this study, comparing research is made difficult due to the fact that most studies have employed blood or plasma sampling methods (i.e. different units of measure).

Saliva sampling offers a compliant collection method that is non-invasive and can be applied frequently (Cook, 2002). Saliva will however, only relay information concerning free concentrations of hormones or substances that are not strongly protein bound (Stupnicki & Obminski, 1992). A further limitation is that salivary secretion is autonomically controlled and subject to changes in concentration due to secretory rate (Ohkuwa, Itoh, Yamazaki & Sato, 1995). Still, if interpretation takes into account salivary secretion and partitioning, it does provide an opportunity to extend otherwise mechanical studies to include endocrine and metabolic responses, particularly if subjects are unwilling to give frequent blood samples. The transfer of analytes across
the various biological membranes also requires consideration. Given the lag time in partitioning between blood and saliva this method would require additional time course samples. This is not withstanding the fact that the lag time in partitioning between blood and saliva may not be linear (Cook, 2002). It should also be remembered that salivary analysis only provides an indirect measure of circulating blood levels and further, from the muscle itself. Thus, one must remain cognizant of limitations associated with the utilisation and interpretation of saliva sampling.

The importance of fatigue as a precursor for adaptation partially supports the metabolic stimulus. It may be speculated that schemes characterised by fatigue elicit greater metabolic responses than non-fatiguing conditions, thereby providing additional stimuli for adaptation. With only small increases in lactate (up to 1nmol/l) from baseline in each scheme, the contribution of the metabolic stimulus appears minimal. Still, the mechanisms for subsequent adaptations remain speculative. These may include an increase in anabolic hormone release, increased motor unit recruitment (Carey Smith & Rutherford, 1995; Takarada et al., 2000) and/or by way of exercise-induced muscle damage (Ebbeling & Clarkson, 1989). Whilst lactate is an important metabolite, it does not account for any “metabolic” responses alone. It is therefore recommended that research examine a range of metabolic markers to gain a better appreciation of the metabolic profile to different weight training methods and/or techniques. Such an analysis may also provide understanding as to the mechanisms that underlie training-induced adaptations, related to the metabolic stimulus.
Conclusion

Much of our understanding about resistance exercise is based on the mechanical response (e.g. forces, time, power, etc.) associated with single repetitions. Such an understanding does not adequately reflect the nature of the resistance-training stimulus (multiple repetitions) and as such many of the suggested conclusions/practical applications may be fundamentally flawed. For example, many strength and conditioning coaches and/or sport scientists have proposed that a minimum threshold of tension (> 60-70% 1RM) is required for strength and hypertrophic adaptation. Recent research would suggest that such assertions are questionable. It was found that equating a light and heavy scheme by volume resulted in a great deal of difference in terms of the kinematic and kinetic responses from that found in a single repetition. These differences may explain some of the disparate findings in research where light and heavy loading schemes have been found equally effective in developing strength, hypertrophy and power. Thus, the magnitude of load may be less important than how a load is moved (technique) and/or the volume of load lifted. In terms of mechanical stimuli it would appear that the importance of variables such as total impulse and total work done as determinants of adaptation need to be differentiated and investigated.

As seen with other research the configuration of the various program variables imposed a specific activation pattern in the hormonal and metabolic responses. The endocrine response to the light scheme (no change TST, decreased cortisol, increased TST/cortisol ratio) is suggestive of a hormonal response more conducive to the accretion of muscle protein post exercise. This may again explain some of the findings within research regarding the effectiveness of light load training as a stimulus for strength and hypertrophy. With only small changes in lactate it would seem that
the contribution of the metabolic stimulus in each scheme is somewhat limited. However, one must exercise caution given that only a single metabolite was assessed in this study. This is not withstanding those issues relating to mechanisms for adaptation relating to the metabolic stimulus. It is suggested that future research examine a range of metabolites in order to develop a more complete metabolic profile of the different resistance exercise programmes employed within practice.

Acknowledgements

This project was supported by a grant from the Health Research Council of New Zealand and by the Foundation for Research, Science and Technology.
CHAPTER FOUR – SUMMARY

Summary

In terms of the adaptations associated with resistance exercise, three stimuli (i.e. mechanical, hormonal and metabolic) are thought fundamental to strength and power development. However, our understanding of the responses of these three stimuli to various loading schemes is rudimentary at best. It has been acknowledged in the literature that body building type loading schemes (8-12RM, slow tempo, short rest periods ~60 s) elicit very different responses to power lifting type schemes (1-6RM, explosive tempo, long rest periods ~180 s). This is not surprising given the very different kinematic and kinetic characteristics of each loading scheme. That is, the greater total time under tension, total forces and total work done in body building schemes, result in elevated hormonal and metabolic responses as compared to power lifting type-loading schemes. It is acknowledged that such loading results in greater hypertrophy, though the exact mechanisms of growth and the interaction of the mechanical, hormonal and metabolic stimuli are not well understood.

As the training load employed often determines such responses, the prescribed load is often considered the most important variable. That is, loads greater than 60-70% 1RM are thought fundamental to strength and hypertrophy increases. Of interest to this research however, were a number of studies describing strength and hypertrophic adaptation with substantially lower loads (e.g. 10-50% 1RM). It would seem in these cases that the effect of load might be less important than the kinematics and kinetics associated with that load. That is, the manner in which a load is moved will have a varied effect on the kinematics and kinetics of that movement and thereafter,
adaptation. However, the problem with research in this area is that very little consideration is given to understanding the kinematics and kinetics associated with various loading schemes before prescription of that scheme. There is no doubt that much is known about the single repetition kinematics and kinetics associated with different resistance exercises and loads. However, to our knowledge no studies have analysed the kinematic and kinetic profiles of various loading schemes across multiple sets and/or exercises, and the associated hormonal and metabolic responses. Considering the inherent nature of a typical training session (i.e. multiple repetitions, sets and exercises) such an analysis appears essential to improving our understanding of strength and power adaptation.

Given the conjecture in the literature, this study sought to determine if the similar adaptations resulting from light and heavy loads are a function of these schemes imposing similar mechanical, hormonal and metabolic stresses on the body. Therefore the mechanical, hormonal and metabolic responses of two equal-volume schemes (45% 1RM v 88% 1RM) were investigated. The light scheme produced greater time under tension (36%), work (37%) and power output (115%). Impulse (38%) was the only variable found to be greater in the heavy scheme. A decrease in testosterone (TST) was observed in the heavy scheme (-4 to -29%) with no changes found across the light scheme (1 to 12%). Cortisol decreased in the light (-6 to -30%) and heavy (-14 to -44%) schemes until P45. An increase in the TST/cortisol ratio was observed in both the light (17 to 49%) and heavy (2 to 44%) schemes. Both schemes also revealed similar increases in lactate from baseline (0.3 to 1.0nmol/l). Such findings may help explain the similar strength, hypertrophic and power adaptation reported in studies comparing light and heavy schemes. It would seem that while the training load
utilised is an important stimulus for strength and power adaptation, the way in which that load is moved (technique) and/or volume of training performed may be of greater importance.

**Practical Applications**

Based upon the findings of this study there exist many opportunities to enhance the prescription of resistance exercise within practice:

1) In terms of general prescription a greater number of variables (e.g. volume, technique, etc.) may be manipulated, beyond load, to induce strength and hypertrophic adaptation.

2) For athletes light load-ballistic type schemes may be employed to maintain sports specificity (e.g. velocity, acceleration, etc.) within a periodised training program, whilst enhancing and/or maintaining other muscular qualities (e.g. strength).

3) Elite athletes may further benefit from the variety afforded by lighter load training if used in conjunction with typical strength/hypertrophy loading schemes.

4) If lighter loading schemes enable the maintenance/development of strength and/or hypertrophy then such training methods may be employed within those populations more susceptible to injury through heavy loading (e.g. elderly and children).

5) The above benefits would no doubt enhance the prescription of resistance exercise as a tool for muscular and skeletal rehabilitation in a variety of populations.
Limitations

The authors note and acknowledge the following limitations and delimitations of the research performed:

1) As a cross-sectional study we may only infer as to the responses found with the repeated application of such loading schemes and adaptations thereafter.

2) Due to subject inclusion criteria (males with weight-training experience) the findings of this study may only be applied to this population.

3) Only 11 subjects participated in this study.

4) The two exercise machines in this study were chosen for instrumentation purposes and availability. However, the supine squat is a research based machine and not typically used within practice. The performance of ballistic movements on the supine squat exercise also required some degree of coordination.

5) To assist with the analysis of results, mechanical data was combined to provide total values for each scheme. The analysis of data in this manner in no way reflects individual responses to each exercise or any interactive effects (i.e. fatigue) within each scheme and/or exercise.

6) The data analysis program was unable to differentiate when the sled (load) was under muscular control or projected beyond this, during the ballistic movements on the supine squat exercise.

7) The protocols employed for each loading scheme were designed to replicate a typical training scheme as performed in practice. However, the protocols employed may have differed from that performed in practice or by the subjects who participated in this study.
Recommendations

A number of areas require further investigation and in doing so will contribute to greater understanding within strength and conditioning practice. Firstly, it is recommended that research adopt a multiple set and/or exercise approach in the analysis of the mechanical responses to resistance training. Such an analysis would help elucidate the mechanical profile associated with different weight training methods and thereby develop greater understanding with regards to the stimulus for strength, hypertrophy and power adaptation. Research should also examine a wider range of hormonal and metabolic responses to different loading schemes, as performed within practice. This analysis would not only provide a more complete hormonal/metabolic profile to resistance exercise but further, assist in determining those mechanisms for adaptation relating to these responses. It is also recommended that acute and longitudinal research in this area equate different interventions in some manner (e.g. training volume, TUT, etc.). Such an approach would assist in the analysis and subsequent interpretation of research in this area.
REFERENCES


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**Appendix 1.** Hormonal and metabolic responses of the 45% 1RM loading scheme.

<table>
<thead>
<tr>
<th>Testosterone</th>
<th>Change (%)</th>
<th>Substantial Difference (%)</th>
<th>P value</th>
<th>Cortisol</th>
<th>Change (%)</th>
<th>Substantial Difference (%)</th>
<th>P value</th>
<th>TST/cortisol ratio</th>
<th>Change (%)</th>
<th>Substantial Difference (%)</th>
<th>P value</th>
<th>Lactate</th>
<th>Change (raw)</th>
<th>Substantial Difference (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mid</td>
<td>10</td>
<td>Possibly (50)</td>
<td>0.1244</td>
<td>-6</td>
<td>Possibly (60)</td>
<td>0.4371</td>
<td>17</td>
<td>Possibly (73)</td>
<td>0.1471</td>
<td>1.0</td>
<td>Possibly (55)</td>
<td>0.0005</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P0</td>
<td>6</td>
<td>Likely (79)</td>
<td>0.2483</td>
<td>-8</td>
<td>Possibly (46)</td>
<td>0.5872</td>
<td>15</td>
<td>Possibly (61)</td>
<td>0.3889</td>
<td>1.0</td>
<td>Possibly (60)</td>
<td>0.0001</td>
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<tr>
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<td>12</td>
<td>Possibly (59)</td>
<td>0.1180</td>
<td>-21</td>
<td>Likely (85)</td>
<td>0.0980</td>
<td>41</td>
<td>Very likely (96)</td>
<td>0.0235</td>
<td>0.7</td>
<td>Very likely (99)</td>
<td>0.0012</td>
<td></td>
<td></td>
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</tr>
<tr>
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<td>Possibly (63)</td>
<td>0.3820</td>
<td>-28</td>
<td>Likely (93)</td>
<td>0.0528</td>
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<td>Likely (93)</td>
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<td>0.8</td>
<td>Likely (86)</td>
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<td>-30</td>
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<td>Possibly (63)</td>
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NS – no sample
Appendix 2. Hormonal and metabolic responses of the 88% 1RM loading scheme.

<table>
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<th>Testosterone</th>
<th>Cortisol</th>
<th>TST/cortisol ratio</th>
<th>Lactate</th>
</tr>
</thead>
<tbody>
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<td>Substantial Difference (%)</td>
<td>P value</td>
<td>Change (%)</td>
</tr>
<tr>
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<td>-14</td>
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NS – no sample
Appendix 3. Hormonal and metabolic responses between the 45% 1RM and 88% 1RM loading schemes.

<table>
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<th>TST/cortisol ratio</th>
<th>Lactate</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Change (%)</td>
<td>Substantial Difference (%)</td>
<td>P value</td>
<td>Change (%)</td>
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<td>-12</td>
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<tr>
<td>P45</td>
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<td>Likely (86)</td>
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<tr>
<td>P60</td>
<td>58</td>
<td>Very likely (98)</td>
<td>0.0100</td>
<td>-4</td>
</tr>
</tbody>
</table>

NS – no sample
Appendix 4. Labview data collection program (front panel).
Appendix 5. Labview data collection program (back panel).
Appendix 6. Labview data analysis program (front panel).
Appendix 7. Labview data analysis program (back panel).
Appendix 8: Subject information sheet.

Participant Information Sheet

Project Title: Mechanical, hormonal, and metabolic responses of two resistance loading schemes.
Project Supervisors: John Cronin, Justin Keogh
Researcher: Blair Crewther

You are invited to participate in a study investigating the effects of two resistance-loading schemes. Participation is completely voluntary and you may withdraw at any stage without giving a reason or being disadvantaged.

What is the purpose of the study?
Resistance exercise is prescribed on the basis that the training scheme employed will improve various aspects of muscular function (e.g. strength, hypertrophy). However, those stimuli contributing to these adaptations remain relatively unknown. Though mechanical stresses (i.e. high forces, time under tension, etc.) are believed the primary training stimulus, hormonal and metabolic responses to training may also be important. Thus, the aim of this study is to examine the mechanical, hormonal and metabolic responses of two loading schemes (light and heavy) of equal volume.

Can I join the study?
If you have at least 12 months weight training experience, no injuries and do not suffer from high blood pressure then you are eligible to participate in this study.

What happens in the study?
This project will be performed over three testing occasions. During the first session subject 1RM (repetition maximum) will be determined on the supine squat and Smith squat machines. Subjects will then randomly perform the light (8 sets x 6 reps, 45% 1RM) and heavy (6 sets x 4 reps, 88% 1RM) schemes over subsequent sessions. Adequate rest (72 hours) will be provided between each test occasion.

Cost of participation?
Subjects will not incur any monetary costs participating in this study. Subjects will be required to attend three assessment sessions (1 x 45 minutes; 2 x 90 minutes), with all travel costs reimbursed.

What are the benefits?
These results will improve our understanding of resistance exercise and provide prescriptive information for the development of strength, hypertrophy and power). Such data will assist all areas where resistance training is prescribed including performance enhancement, rehabilitation and general health and fitness. An additional benefit will be the validation of equipment employed in this study. Subjects will gain strength assessment information for the lower body (1RM).

What are the discomforts and risks?
The risks involved in this study are minimal. Subjects may experience mild muscular discomfort from the research procedures. If an injury occurs due to unforeseen
circumstances, you will receive immediate attention from the AUT physiotherapists, located thirty metres from the testing premises. Hormonal and metabolic data will be determined by saliva sampling with all care will be taken when extracting samples.

**How is my privacy protected?**
All records will be kept in a locked limited access cabinet. Data will be treated as confidential and will be used only for the purpose of this study.

**Results**
The results of this project will be published in a scientific journal and presented at a national or international conference.

**Participant Concerns**
Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor. Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEC, Madeline Banda, madeline.banda@aut.ac.nz, 917 9999 ext 8044.

Project Supervisors
Dr John Cronin
Deputy Director of Sport Science Research
Division of Sport and Recreation
Auckland University of Technology
Phone 917 9999 ext 7353

Mr Justin Keogh
Lecturer
Division of Sport and Recreation
Auckland University of Technology
Phone 917 9999 ext 7617

Principal Investigator
Mr Blair Crewther
Division of Sport and Recreation
Auckland University of Technology
Phone 917 9999 ext 7119

Approved by the Auckland University of Technology Ethics Committee (AUTEC). AUTEC reference number 03/40.
Appendix 9: Subject consent form.

Consent to Participation in Research

Title of Project: Mechanical, hormonal, and metabolic responses of two resistance loading schemes.
Project Supervisors: John Cronin, Justin Keogh
Researcher: Blair Crewther

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

Participant signature: .......................................................
Participant name: ....................................................... 
Date:

Supervisor contact details:
Dr John Cronin
Deputy Director of Sport Science Research
Division of Sport and Recreation
Auckland University of Technology
John.cronin@aut.ac.nz
Phone (09) 917-9999 Ext. 7353 (Work)

Approved by the Auckland University of Technology Ethics Committee (AUTEC). AUTEC reference number 03/40.
Appendix 10: Ethics approval sheet.

Memorandum

Student Services Group - Academic Services

To: John Cronin  
From: Madeline Banda  
Date: 30 May 2003  
Subject: 03/40 Mechanical, hormonal, and metabolic profiles of two equal-volume strength training loads

Dear John  

Thank you for providing amendment and clarification of your ethics application as requested by AUTEC. Your application is approved for a period of two years until 30 May 2005.

You are required to submit the following to AUTEC:
- A brief annual progress report indicating compliance with the ethical approval given.
- A brief statement on the status of the project at the end of the period of approval or on completion of the project, whichever comes sooner.
- A request for renewal of approval if the project has not been completed by the end of the period of approval.

Please note that the Committee grants ethical approval only. If management approval from an institution/organisation is required, it is your responsibility to obtain this. The Committee wishes you well with your research. Please include the application number and study title in all correspondence and telephone queries.

Yours sincerely

Madeline Banda  
Executive Secretary  
AUTEC

CC: 9900799 Blair Crewther