Possible Stimuli for Strength and Power Adaptation
Acute Metabolic Responses

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Abstract

The metabolic response to resistance exercise, in particular lactic acid or lactate, has a marked influence upon the muscular environment, which may enhance the training stimulus (e.g. motor unit activation, hormones or muscle damage) and thereby contribute to strength and power adaptation. Hypertrophy schemes have resulted in greater lactate responses (%) than neuronal and dynamic power schemes, suggesting possible metabolic-mediated changes in muscle growth. Factors such as age, sex, training experience and nutrition may also influence the lactate responses to resistance exercise and thereafter, muscular adaptation. Although the importance of the mechanical and hormonal stimulus to strength and power adaptation is well recognised, the contribution of the metabolic stimulus is largely unknown. Relatively few studies for example, have examined metabolic change across neuronal and dynamic power schemes, and not withstanding the fact that those mechanisms underpinning muscular adaptation, in relation to the metabolic stimulus, remain highly speculative. Inconsistent findings and methodological limitations within research (e.g. programme design, sampling period, number of samples) make interpretation further difficult. We contend that strength and power research needs to investigate those metabolic mechanisms likely to contribute to weight-training adaptation. Further research is
also needed to examine the metabolic responses to different loading schemes, as well as interactions across age, sex and training status, so our understanding of how to optimise strength and power development is improved.

The importance of the acute mechanical and hormonal stimulus for strength and power adaptation is well recognised.\(^{1-5}\) Literature now suggests that the acute metabolic stimulus may also be important for resistance training adaptation to occur.\(^{6,7}\) Comparison of eccentric and concentric training provides some insight into the importance of the metabolic stimulus. That is, concentric-only training has been found equally effective in stimulating strength and hypertrophy compared with eccentric-only training.\(^{8-11}\) despite the mechanical benefits afforded by eccentric actions (e.g. greater forces, greater muscle damage).\(^{12-14}\) This may be attributed to the greater metabolic costs associated with concentric actions.\(^{15,16}\) The responses to weight training combined with vascular occlusion, provide further evidence of the metabolic contribution to adaptation. Occluding the muscle during resistance exercise leads to greater accumulation of metabolic by-products, enhancing the metabolic stimulus and the adaptive responses to such exercise. As a result, similar adaptations have been found when comparing light-load training with occlusion and heavy-load training,\(^{17}\) and greater adaptation with light-load training with occlusion, than that found without occlusion.\(^{18,19}\)

The acute metabolic stimulus is itself related to the build-up of metabolic by-products, in particular lactic acid, within the muscular environment. A build-up of these products may increase the secretion of various anabolic hormones (e.g. growth hormone [GH])\(^{20-22}\) or lead to greater motor unit activation at a given load,\(^{20,23}\) thereby contributing to the weight-training stimulus. An increase in metabolic waste products, combined with altered hydrogen ion concentrations and adenosine triphosphate (ATP) deficiency, may also produce some muscle damage,\(^{24}\) further enhancing the training stimulus. Over time, it may be expected that these acute responses (i.e. greater hormone secretion, increased motor unit recruitment, muscle damage) would facilitate adaptations leading to changes in the force-generating capacity of muscle and thus, greater strength and/or power. Traditionally, it is believed that large ‘metabolic responses’, such as those seen during endurance-type exercises, does not produce any significant changes in strength and power. However, during weight training, blood supply is occluded during movement and in the presence of other mechanical and hormonal stimuli, produce adaptations that may vary quite considerably from that seen with endurance exercise.

The measurement of lactic acid, or lactate, is frequently used to determine the anaerobic contribution from glycolysis during exercise.\(^{25,26}\) Thus, programme design (e.g. load intensity, volume, rest periods) plays an important role in determining the metabolic response to such exercise and the likely contribution of the metabolic stimulus. For example, those programmes designed to induce the greatest change in morphological adaptation, or muscle growth, are characterised by relatively large increases (%) in blood lactate levels,\(^{27-33}\) hence, greater contribution from the metabolic stimulus. However, little is known regarding the metabolic stress imposed on the system by other strength and power schemes used within practice (e.g. neuronal and dynamic power). Debate also surrounds those mechanisms that may potentially underlie weight-training induced adaptation. The aim of this review is to therefore critique what literature there is available in this area, so that we may develop a better understanding as to the possible contribution of the metabolic stimulus to strength and power development. Such an understanding may enable resistance exercise to be prescribed more effectively for inducing these changes, thereby improving strength and conditioning practice.
1. Metabolic Stimulus and Resistance Exercise

Weight training is a powerful exercise stimulus for strength and power adaptation, which may be partly attributed to the lactic acid response, or its blood equivalent (lactate), during a single exercise bout. Examining those schemes specifically designed to improve strength and power will provide understanding as to the importance of the metabolic stimulus in the development of these qualities. In the first instance, those energetic processors influencing lactic acid production will be briefly reviewed, followed by an examination of those mechanisms that underlie weight training-induced adaptation, in relation to the metabolic stimulus. This article will then examine the acute lactate responses to three broad types of programmes often used within practice: (i) hypertrophy (controlled movements, moderate loads, short rest periods, high total work); (ii) neuronal (explosive intent, heavy loads, long rest periods, lower total work); and (iii) dynamic power (explosive and/or ballistic movements, light loads, moderate rest periods, lower total work) schemes. The influence of age, sex, training status and nutrition, upon the acute lactate response to resistance exercise, will also be investigated.

1.1 Lactate Production

The energy source for all muscular contractions is ATP and due to the fact that only small amounts of this compound are stored inside the muscle cell, it must be continually resynthesized at its rate of use. The resynthesis of ATP is predominantly achieved through the three classic energy systems or pathways: (i) phosphagen system; (ii) anaerobic glycolysis; and (iii) oxidative phosphorylation. Briefly, when the muscle is contracting at low intensities the energy for muscular activity is provided primarily through the aerobic energy system, oxidative phosphorylation. During high-intensity activities, however, this system is unable to supply ATP at the rate at which it is required. Energy supply for intense activities of short duration is met through the phosphagen system, which relies on stored creatine phosphate to synthesize ATP. Given that this system has only a limited capacity for energy restoration, intense activities of longer duration require a greater proportion of ATP to be resynthesized via glycolysis. This process involves the breakdown of carbohydrates (CHO), in the form of glucose or glycogen, to synthesize ATP and in doing so, results in lactic acid formation within the muscle. Once lactic acid is formed in the muscle, it diffuses rapidly into the blood, where it is buffered to form lactate. Thus, an accumulation of lactic acid or blood lactate may be observed when there is greater reliance upon glycolysis, to meet the energetic demands of exercise. Although blood lactate concentration is not a definitive measure of lactic acid production, it does provide an indirect measure of lactic acid and one that is less invasive to extract compared with muscle biopsies, and more widely used within research. For these reasons, this article will focus primarily upon blood lactate responses to resistance exercise.

1.2 Mechanisms for Adaptation

The contribution of the mechanical and hormonal stimuli to resistance training adaptation is well recognized. For instance, the production of high forces and time under tension are important mechanical factors thought necessary for improving maximal strength. Changes in muscle morphology are further mediated by the interaction of the anabolic (e.g. testosterone, GH, insulin) and catabolic (e.g. cortisol) hormones. In order to adequately train power, movements producing high power outputs with high contractile velocities are thought important. The role of the metabolic stimulus in facilitating improvement in strength and power would appear to be more indirect. First, an accumulation of lactic acid in response to resistance exercise produces a shift in the acid balance of muscle, reducing muscle pH, increasing muscle acidity and the build-up of hydrogen ions. As a result of these changes in the muscular environment, various other training mechanisms may be stimulated (e.g. motor unit activation, hormones, muscle damage), thereby enhancing the weight-training stimulus and signaling pathways for long-term adaptation to occur.
during the recovery period. These events are outlined in figure 1.

The mechanisms by which the metabolic stimulus contributes to adaptation are often based on research where the metabolic environment is artificially altered (i.e. occlusion). By occluding the muscle during voluntary contractions, greater lactate accumulation occurs, enhancing the metabolic stimulus. Takarada and Ishii[23] for example, found that the integrated electromyography (EMG – an index of muscle activity) of a low intensity (40% one repetition maximum [1RM]) exercise performed with occlusion, was almost equal to that of a high-intensity (80% 1RM) exercise performed without occlusion. Enhanced EMG has also been found with an exercise performed with a light load (20% 1RM) in an occluded state, compared with that performed with the same load in a non-occluded state. It would seem from these findings that an increase in lactic acid accumulation, at a given load, would produce a concomitant increase in motor unit activation. Takarada et al.[20] also reported a 290-fold increase in plasma GH concentrations with the low-intensity exercise (with occlusion), whereas no such effect was found after the same exercise without occlusion. This finding supports suggestions that an increase in blood lactate and hydrogen ion concentrations may augment the hypophyseal secretion of GH. Research supports such a notion with the largest GH responses observed in those protocols producing the greatest lactate responses.[27,29,33,40]

Although occlusion enhances metabolic activity within the muscle, this type of methodology may not reflect the metabolic environment occurring under normal weight-training conditions.

There is some speculation that an accumulation of metabolic by-products (e.g. lactate), combined with altered hydrogen ion concentrations and ATP deficiency, plays a role in mediating muscle tissue damage.[24] Greater muscle damage, as indicated by elevated creatine kinase levels, has also been observed in those schemes producing the greatest lactate responses.[20] Thereby possibly demonstrating a causal relationship between lactate accumulation and muscle tissue breakdown. The importance of muscle damage as a mediator for adaptation, lies in the overcompensation processors associated with muscle recovery and repair (e.g. greater protein [PRO] synthesis). If muscle damage mediated the remodelling of muscle tissue and subsequently, muscle growth, then the metabolic stimulus may be an important component for inducing changes in muscle morphology. However, most data indicate that the mechanisms for muscle damage are more likely to involve mechanical (e.g. high forces, greater active strain), rather than metabolic factors.[14] It also remains to be seen if muscle damage is a prerequisite for muscle growth to occur, as changes in muscle morphology are also linked to factors such as age, nutrition, sex, genetic predisposition and training status.

Fatigue is often thought an important component for strength adaptation to occur[43] and as such, places some emphasis on the metabolic stimulus (i.e. increase lactate accumulation = greater fatigue). The rationale for training in this manner lies in the belief that additional motor units, in particular the high-threshold motor units, are recruited when trying to maintain performance when fatigued.[44] If motor units are trained upon recruitment, then training to fatigue would stimulate a greater number of

![Fig. 1. Schematic representation of those events signalling long-term adaptation.](image-url)
fibres (and greater adaptation) than non-fatigue training. The findings of Takarada et al.[20] and Takarada and Ishii[23] lend support to this contention. Although bodybuilders often train in this manner, there is still little scientific data to support such a practice. In fact, studies have found that training to failure or exhaustion is not necessary for optimal adaptation to occur.[45-48] which may be partially explained by the complex nature of fatigue. As well as lactate accumulation, fatigue may also result from changes in CNS drive to the motor neurons, changes in neuromuscular propagation, dehydration and the availability of various metabolic substrates.[43,49,50] Such mechanisms may also vary depending on factors such as muscle fibre type,[43] age or sex.[51] Regardless of the mechanism, it should be recognised that exercising in a fatigued state is likely to compromise the ‘quality’ of performance (e.g. high power output). Further research is therefore needed to elucidate those mechanisms underpinning the metabolic stimulus, as well as the relative importance of these effects to weight training adaptation.

1.3 Acute Lactate Response

1.3.1 Programme Design

Those resistance exercise schemes designed to increase strength through primarily morphological adaptation (i.e. hypertrophy) generally result in large increases (%) in circulating blood lactate levels from pre-exercise (see table I). In comparison, those programmes designed to increase strength through mainly neural adaptation (i.e. neuronal) have generally resulted in much lower blood lactate responses (see table II). For example, Kraemer et al.[27] compared the lactate response to eight exercises performed with a 5RM load for 3–5 sets per exercise and 3 minutes of rest between sets or a 10RM load (three sets per exercise) with 1-minute rest periods. The peak lactate response to the hypertrophy scheme (433%) was found to be much greater than that reported following the neuronal scheme (133%). This may be attributed to the greater amount of work performed in the hypertrophy scheme (greater work = greater time under tension) in conjunction with the much shorter rest periods between sets and exercises. Such a result is in agreement with other research.[28-33]

Dynamic power schemes have also been shown to elicit significant lactate responses (see table II). A 50% increase in serum lactate was found in response to an exercise session consisting of half-squat exercises (ten sets × six repetitions), performed with 1- and 4-minute rest periods.[67] Another study investigated the effect of six sets of leg extensions with loads of 70% (I-70) and 35% (I-35) 1RM.[54] The response found in the I-35 condition was similar to the I-70 condition (13- to 14-fold increase in lactate), which may be due to the average number of repetitions performed in each set, with I-35 more than twice that performed in I-70 (13 vs 6, respectively). This value is much greater than other dynamic power schemes and may be explained by extraction from a muscle biopsy (lactic acid) and with each set performed to failure. Whilst subtle differences in programme design make it difficult to determine the overall response to each scheme, on average, hypertrophy schemes have produced greater lactate responses (500%) than the neuronal (200%) and dynamic power (70%) schemes. It is evident, however, that much less research has profiled the lactate response to neuronal and dynamic power, than hypertrophy schemes. Additional research into the metabolic nature of neuronal and dynamic power schemes would improve our understanding as to the contribution of the metabolic stimulus to adaptive changes associated with these training methods.

1.3.2 Sex

It has been reported that males exhibit greater lactate responses to a bout of resistance exercise compared with females.[31,69-71] For instance, Kraemer et al.[31] observed greater lactate responses among recreationally trained males to a hypertrophy scheme consisting of eight exercises (three sets × ten repetitions, 10RM), when compared with a similar female population (648% vs 468%, respectively). A similar result was found, in the same study, when examining the lactate responses to a neuronal type scheme in both males (230%) and females (140%). The observed differences may be attributed to males
Table I. Acute lactate response to hypertrophy schemes

<table>
<thead>
<tr>
<th>Study</th>
<th>Subjects</th>
<th>Protocols</th>
<th>Lactate response (%) change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vanhelder et al. [52]</td>
<td>5 M, UT</td>
<td>1 ex, 7 × 10 (10RM)</td>
<td>↑ 650</td>
</tr>
<tr>
<td>Tesch et al. [53]</td>
<td>9 M, T</td>
<td>4 ex, 5 × 6–12 (6–12RM)</td>
<td>↑ 391</td>
</tr>
<tr>
<td>Kraemer et al. [29]</td>
<td>9 M, T</td>
<td>8 ex, 3 × 10 (10RM)</td>
<td>↑ 592</td>
</tr>
<tr>
<td>Robbergs et al. [54]</td>
<td>8 M, T</td>
<td>1 ex, 6 × 6 (70% 1RM)</td>
<td>↑ 1300</td>
</tr>
<tr>
<td>Kraemer et al. [31]</td>
<td>8 M, T</td>
<td>8 ex, 3 × 10 (10RM)</td>
<td>↑ 648</td>
</tr>
<tr>
<td>Kraemer et al. [55]</td>
<td>8 M, UT</td>
<td>4 ex, 3 × 10 (10RM)</td>
<td>↑ 750</td>
</tr>
<tr>
<td>Kraemer et al. [28]</td>
<td>8 M, T</td>
<td>8 ex, 3 × 10 (10RM)</td>
<td>↑ 600</td>
</tr>
<tr>
<td>Kraemer et al. [27]</td>
<td>9 F, T</td>
<td>8 ex, 3 × 10 (10RM)</td>
<td>↑ 433</td>
</tr>
<tr>
<td>Hakkinen and Pakarinen [56]</td>
<td>10 M, T</td>
<td>1 ex, 10 × 10 (10RM)</td>
<td>↑ 971</td>
</tr>
<tr>
<td>Fahey et al. [54]</td>
<td>10 M, T</td>
<td>9 ex, 5 × 8–12 (70% 1RM) SUP</td>
<td>↑ 313</td>
</tr>
<tr>
<td>McBride et al. [57]</td>
<td>8 F, UT (RJ)</td>
<td>9 ex, 3 × 10 (10RM)</td>
<td>↑ 623</td>
</tr>
<tr>
<td>Mulligan et al. [54]</td>
<td>10 F, T</td>
<td>8 ex, 1 × 10 (10RM)</td>
<td>↑ 550</td>
</tr>
<tr>
<td>Gotshalk et al. [59]</td>
<td>8 M, T</td>
<td>8 ex, 1 × 10 (10RM)</td>
<td>↑ 430</td>
</tr>
<tr>
<td>Kraemer et al. [56]</td>
<td>9 M, T</td>
<td>4 ex, 4 × 10 (10RM)</td>
<td>↑ 718</td>
</tr>
<tr>
<td>Hakkinen et al. [51]</td>
<td>10 M (26y), UT</td>
<td>2 ex, 4 × 10 (100% MVC)</td>
<td>↑ 200</td>
</tr>
<tr>
<td>Kraemer et al. [32]</td>
<td>10 M (70y), UT</td>
<td>2 ex, 4 × 10 (100% MVC)</td>
<td>↑ 75</td>
</tr>
<tr>
<td>Kraemer et al. [53]</td>
<td>8 M (30y), UT</td>
<td>1 ex, 4 × 10 (10RM)</td>
<td>↑ 700</td>
</tr>
<tr>
<td>Kraemer et al. [63]</td>
<td>9 M (62y), UT</td>
<td>1 ex, 4 × 10 (10RM)</td>
<td>↑ 420</td>
</tr>
<tr>
<td>Kraemer et al. [54]</td>
<td>7 M, T</td>
<td>1 ex, 1 × failure (80% 1RM)</td>
<td>↑ 340</td>
</tr>
<tr>
<td>MacDougall et al. [56]</td>
<td>8 M, T</td>
<td>1 ex, 1 × failure (80% 1RM)</td>
<td>↑ 310</td>
</tr>
<tr>
<td>Linamo et al. [58]</td>
<td>8 M, UT</td>
<td>1 ex, 5 × 10 (67% MVC)</td>
<td>↑ 105</td>
</tr>
<tr>
<td>Smilios et al. [31]</td>
<td>11 M, T</td>
<td>4 ex, 4 × 10 (75% 1RM)</td>
<td>↑ 742</td>
</tr>
<tr>
<td>Zafeiridis et al. [33]</td>
<td>10 M, T</td>
<td>4 ex, 2 × 10 (75% 1RM)</td>
<td>↑ 850</td>
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<tr>
<td></td>
<td></td>
<td>4 ex, 6 × 10 (75% 1RM)</td>
<td>↑ 760</td>
</tr>
</tbody>
</table>

a Lactic acid.

CR = competitive runners; ex = exercises; F = females; M = males; MVC = maximal voluntary contraction; PLA = placebo; reps = repetitions; RJ = recreational joggers; RM = repetition maximum; SUP = supplement; T = trained; UT = untrained; ↑ indicates increase.

generally possessing greater lean muscle mass and using heavier relative loads than females. Muscle composition may also be important, one study reporting differences in the relative cross-sectional area of the three major fibres, in the vastus lateralis muscle, in males (IIA > I > IIB) and females (I > IIA > IIB), respectively.[72] Given that the type II or fast twitch fibres have a greater glycolytic capacity than the type I or slow twitch fibres,[26,73] male muscle is likely to exhibit greater potential for lactate production than female muscle. Still, some researchers have reported similar lactate responses between males and females, performing the same resistance exercise programmes.[21,74] Disparate findings may be explained by differences in programme design, sampling procedures (e.g. number) post-exercise and the relative training experience of subjects.

1.3.3 Age

Subject age would appear to be an important factor modulating the lactate response to a single
resistance exercise session. Mero and colleagues\textsuperscript{[68]} examined the lactate response of adult and pubertal males, to an exercise programme consisting of half-squat lifts (ten sets × six repetitions, 1- and 4-minute rest periods), performed with a relative load of 50% 1RM. The lactate responses were found to be lower among the adult males compared with the younger males, for both the 1- (94% vs 27%) and 4-minute (35% vs 0%) rest periods. Another study\textsuperscript{[71]} also reported a greater lactate response among pubescent boys, than men, when examining the lactate response to a single exercise bout of knee extensions (five sets × ten repetitions, 40% 1RM), followed by two additional sets to exhaustion. The larger responses in the younger males may be partially attributed to differences in maturation, trainability and the ability to tolerate stressful exercise. To our knowledge no research has examined the same age-related effects among female populations. Given the limited data in this area, it is suggested that further research be conducted in order to differentiate the lactate responses across these groups (pubescent and adult), in both males and females.

A reduction in the lactate response to resistance exercise, with increasing age after adulthood, has been reported previously. A group of adult males (30 years) for example, reported a 700% increase in blood lactate, whereas a group of older males (60 years) reported a 420% increase, performing an identical exercise session.\textsuperscript{[62]} Other researchers have also reported greater lactate responses among young adult males, when compared with elderly males.\textsuperscript{[61,63,70]} The greater lactate responses for younger adults may be explained by the use of heavier absolute loads (i.e. greater 1RM strength) and their ability to exercise at a greater relative intensity. The differential lactate responses may be further attributed to a reduction in the size of the

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Subjects</th>
<th>Protocols exercise(s), sets × reps (load)</th>
<th>Lactate response (% change)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuronal schemes</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Kraemer et al.\textsuperscript{[29]}</td>
<td>9 M, T</td>
<td>8 ex, 3/5 × 5 (5RM)</td>
<td>↑ 180</td>
</tr>
<tr>
<td>Kraemer et al.\textsuperscript{[31]}</td>
<td>8 M, T</td>
<td>8 ex, 3/5 × 5 (5RM)</td>
<td>↑ 230</td>
</tr>
<tr>
<td>Kraemer et al.\textsuperscript{[31]}</td>
<td>8 F, T</td>
<td>8 ex, 3/5 × 5 (5RM)</td>
<td>↑ 140</td>
</tr>
<tr>
<td>Kraemer et al.\textsuperscript{[27]}</td>
<td>9 F, T</td>
<td>8 ex, 3/5 × 5 (5RM)</td>
<td>↑ 133</td>
</tr>
<tr>
<td>Hakkinen and Pakkarinen\textsuperscript{[30]}</td>
<td>10 M, T</td>
<td>1 ex, 20 × 1 (100% 1RM)</td>
<td>↑ 84</td>
</tr>
<tr>
<td>Kraemer et al.\textsuperscript{[28]}</td>
<td>8 M, T</td>
<td>8 ex, 3/5 × 5 (5RM)</td>
<td>↑ ~223</td>
</tr>
<tr>
<td>Zafeiridis et al.\textsuperscript{[33]}</td>
<td>10 M, T</td>
<td>4 ex, 4 × 5 (88% 1RM)</td>
<td>↑ ~319</td>
</tr>
<tr>
<td>Smilios et al.\textsuperscript{[32]}</td>
<td>11 M, T</td>
<td>4 ex, 2 × 5 (88% 1RM)</td>
<td>↑ ~350</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 ex, 4 × 5 (88% 1RM)</td>
<td>↑ ~350</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 ex, 6 × 5 (88% 1RM)</td>
<td>↑ ~350</td>
</tr>
<tr>
<td>Dynamic power schemes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Robergs et al.\textsuperscript{[54]}</td>
<td>8 M, T</td>
<td>1 ex, 6 × 13 (35% 1RM)</td>
<td>↑ ~1400\textsuperscript{a}</td>
</tr>
<tr>
<td>Mero et al.\textsuperscript{[68]}</td>
<td>6 M (24y)</td>
<td>1 ex, 10 × 6 (50% 1RM)</td>
<td>↑ ~50</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 ex, 10 × 6 (50% 1RM)</td>
<td>↑ ~50</td>
</tr>
<tr>
<td>Mero et al.\textsuperscript{[68]}</td>
<td>6 M (15y)</td>
<td>1 ex, 10 × 6 (50% 1RM)</td>
<td>Nil</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 ex, 10 × 6 (50% 1RM)</td>
<td>↑ 27</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 ex, 10 × 6 (50% 1RM)</td>
<td>↑ 35</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 ex, 10 × 6 (50% 1RM)</td>
<td>↑ 94</td>
</tr>
<tr>
<td>Linnamo et al.\textsuperscript{[66]}</td>
<td>8 M, UT</td>
<td>1 ex, 5 × 10 (40% MVC)</td>
<td>↑ 209</td>
</tr>
</tbody>
</table>

\textsuperscript{a} Lactic acid.
\textsuperscript{b} 4-minute rest.
\textsuperscript{c} 1-minute rest.
\textsuperscript{ex} = exercises; \textsuperscript{F} = females; \textsuperscript{M} = males; \textsuperscript{MVC} = maximal voluntary contraction; \textsuperscript{reps} = repetitions; \textsuperscript{RM} = repetition maximum; \textsuperscript{T} = trained; \textsuperscript{UT} = untrained; ↑ indicates increase.
type II muscle fibres, with the aging process\cite{75,76} and hence, a reduction in the glycolytic capabilities of muscle. Whilst increasing age would appear to lower lactate responses among males, age-related differences among females are less clear. Copeland et al.\cite{77} found no differences in the lactate response to a single resistance exercise bout, performed across five different age groups (mean age 25, 34.5, 43.8, 52.4 and 62.3 years). The findings of Hakkinen et al.\cite{70} however, support those found among males, with greater lactate responses among middle-aged (39 years) women, than elderly (67 years) women, to a single exercise bout. Differences in programme design and in the age groups assessed again make comparisons difficult. More research is needed to determine if elderly females show an inhibited lactate response to resistance exercise.

### 1.3.5 Weight Training Status (Highly Trained vs Untrained)

Given that weight training is often performed in such a manner as to activate the glycolytic system, one may also expect highly trained individuals to exhibit training adaptations, leading to increased production of lactate (e.g., increase glycolytic enzymes, increase fast twitch fibre size). Brown et al.\cite{86} for instance, reported a greater lactate response among weight-trained males (570%), than untrained males (330%), performing three sets of leg press movements to failure. Such a finding supports the earlier work by Stone et al.\cite{87} who also reported a greater lactate response in well-trained individuals. The differential responses were attributed to the greater 1RM strength of those with weight-training experience and the greater muscle mass of these subjects. Weight-trained individuals also exhibit greater levels of lactate dehydrogenase, an important mediator in pyruvate conversion to lactate, in both fast and slow twitch muscle.\cite{7,79,80} Studies examining the effect of different strength programmes, among those previously untrained, have reported an increase in the percentage of type IIA and decrease in the percentage of type IIB fibres.\cite{81,84} Combined with proportional increases in capillary density,\cite{84,85} such changes may enhance the oxidative potential of muscle and reduce lactate accumulation. However, the above findings are not necessarily typical, with another study reporting no changes in acute lactate responses with training.\cite{21}

A major factor to consider is the type of training programme employed, with endurance-based programmes likely to lead to adaptations that would improve oxidative capacity, more so than maximal strength or power-based programmes. The health status of subjects may be another point to consider, with untrained sedentary adults less likely to experience training specific adaptations than those who are untrained but active.

### 1.3.4 Weight Training Status (Trained vs Untrained)

A group of previously untrained males reported a reduced blood lactate response (700% vs 500%), to an acute exercise bout (4 sets of 10RM squats), after 10 weeks of periodised weight training.\cite{63} A similar finding was reported by Pierce et al.\cite{78} after 8 weeks of weight training. It would seem from these findings that short periods of resistance training elicits those adaptations more commonly observed with aerobic training (e.g., lowered lactate response to exercise) among previously untrained individuals. Such training effects may, however, diminish with increasing age.\cite{63} It is accepted that these effects are the result of adaptations in muscle fibre capillarisation, oxygen extraction, fibre conversion, enzyme activity and substrate levels.\cite{17,79,80} For instance, studies examining the effect of different strength programmes, among those previously untrained, have reported an increase in the percentage of type IIA and decrease in the percentage of type IIB fibres.\cite{81,84} Combined with proportional increases in capillary density,\cite{84,85} such changes may enhance the oxidative potential of muscle and reduce lactate accumulation. However, the above findings are not necessarily typical, with another study reporting no changes in acute lactate responses with training.\cite{21}

A major factor to consider is the type of training programme employed, with endurance-based programmes likely to lead to adaptations that would improve oxidative capacity, more so than maximal strength or power-based programmes. The health status of subjects may be another point to consider, with untrained sedentary adults less likely to experience training specific adaptations than those who are untrained but active.
trained individuals may only exhibit greater lactate responses when lifting maximal or near maximal loads. \textsuperscript{[86]} McMillan et al.\textsuperscript{[90]} did, however, report lower lactate concentrations at 90 minutes post-exercise in the trained group, which suggests greater lactate clearance in those who are well trained. When comparing data, one must also account for the nature of the weight-training experience. Olympic/power lifting is typically characterised by heavier loads, lower volume and longer rest periods than bodybuilding-type programmes. The different training strategies may explain the greater percentage of slow twitch fibres and greater mitochondrial density seen among bodybuilders, than Olympic/power lifters.\textsuperscript{[88,92]} and consequently, different lactate responses to resistance exercise. Still, Kraemer et al.\textsuperscript{[93]} found no differences in the lactate response to a heavy resistance exercise session performed by competitive bodybuilders and power lifters, although the bodybuilders appeared to better tolerate exercise based upon enhanced performance and reduced sympatomatology.

One of the difficulties with research interpretation lies in the different sampling procedures employed (e.g. sampling duration and intervals), when determining the metabolic response to resistance exercise. For example, a single sample collected after exercise may not adequately characterise the metabolic environment afforded by such exercise, particularly given the time-course delay in lactate accumulation, due to lactic acid diffusion into the blood. Employing longer sampling periods, and collecting a greater number of samples post-exercise, would no doubt prove beneficial in determining the lactate responses afforded by different exercise programmes, as well as differentiating the effects of age, sex, training status, etc. The sampling of blood in such a manner would also help determine what, if any, interactive effects exist between these factors and the lactate responses to resistance exercise.

### 1.3.6 Training Status (Weight Trained vs Endurance Trained)

The type of training experience is important in modulating the acute lactate response to resistance exercise. Brown and colleagues\textsuperscript{[86]} compared the responses among two trained groups (weight trained and endurance trained), each performing three sets of leg press movements to exhaustion. The lactate response in the weight-trained group was significantly elevated above the other group after the last set and remained so throughout the 15-minute recovery period. Regan and Potteiger\textsuperscript{[94]} reported a similar response (weight trained > endurance trained), examining blood lactate concentrations in strength/power and endurance athletes at three different velocities, on an isokinetic dynamometer. Such results are not surprising given that endurance training is thought to reduce lactate accumulation during exercise.\textsuperscript{[26,95]} reflecting the adaptive response of the body to this type of training. That is, endurance athletes exhibit greater capillary supply and density, and a greater percentage of type I fibres than weight-trained athletes.\textsuperscript{[96,97]} Given that training of this nature is also known to increase the number and volume of mitochondria, increase oxidative and decrease glycolytic enzyme activity,\textsuperscript{[98,99]} such differences provide endurance-trained muscle with greater oxidative capacity (i.e. reduce lactate production, increase lactate removal) than weight-trained muscle. Central adaptations to endurance training (e.g. increase haemoglobin and haematocrit levels, increase stroke volume, greater fatty acid metabolism)\textsuperscript{[95,100,101]} would further inhibit the accumulation of lactate. Again, respective differences in 1RM strength and lean muscle mass, being typically greater in resistance-trained individuals, would also explain the greater responses found among weight-trained athletes.

### 1.3.7 Nutrition

It would appear that nutritional strategies (CHO and/or PRO) have little influence upon acute lactate responses to resistance exercise.\textsuperscript{[56,60,102,103]} Williams et al.\textsuperscript{[102]} examined the effect of a PRO and CHO supplement ingested immediately after the performance of three different workouts (low, moderate and high training volume). Supplementation produced no differences in blood lactate in any workout, from that observed in a placebo group. Kraemer et al.\textsuperscript{[60]} investigated the influence of PRO and CHO supplementation, taken before and after
exercise, and repeated over 3 consecutive days with exercise. Although no differences in lactate responses were found on day one, a lowered lactate response was reported over days two and three with supplementation. It was suggested that the lower lactate responses might be due to a relative increase in the proportion of lipid as a fuel source or greater conversion of lactate into glycogen. However, in practice resistance exercise is not typically performed in such a manner (i.e. 3 consecutive days of the same programme). Therefore, any changes in the metabolic environment occurring under these conditions, may not reflect those likely to occur in the ‘real world’. Other possibilities include a reduction in muscle glycogen levels, resynthesis of lactate back to glycogen or increased clearance of lactate via gluconeogenesis. Pre-training levels of glycogen are another important consideration, as lactate production may increase if muscle glycogen is increased to supramaximal levels prior to exercise. Alternatively, low muscle glycogen levels may lead to greater utilisation of lactate as a fuel source, resulting in greater lactate removal. Addressing such issues is beyond the scope of this review.

2. Implications for Strength and Power Development

From the data reviewed, it would appear that hypertrophy schemes elicit relatively large increases in blood lactate concentrations to a single training session. Furthermore, such responses are greater than that found across a single neuronal or dynamic power session. This may be attributed to the specific configuration of the various training variables and subsequently, the relative contribution of energy supply via glycolysis. Thus, where hypertrophy schemes (controlled movements, moderate loads, moderate repetitions, high total work, short rest periods) rely heavily on anaerobic glycolysis for energy production, neuronal (explosive intent, heavy loads, few repetitions, lower total work, long rest periods) and dynamic power schemes (explosive and/or ballistic movements, light loads, moderate repetitions, lower total work, moderate rest periods) involve much less glycolytic activity. The shorter rest periods associated with hypertrophy schemes also reduce the ability of muscle to clear blood lactate during exercise. Given these findings, it may be speculated that the metabolic stimulus plays a greater role in mediating strength changes through morphological adaptation. Anecdotal evidence supports such a notion with bodybuilders often utilising advanced training strategies (e.g. giant sets, drop sets, pause training) as part of their training regime, which are likely to produce large metabolic responses, contributing to resultant adaptation.

The different lactate responses observed appear to be largely mediated by differences in strength, muscle mass used and muscle composition. For example, the amount of work (force × distance) performed would appear an important factor determining lactate responses. Therefore, those with greater IRM strength would elicit greater work at a given relative intensity (i.e. heavier loads) and thereafter, produce more lactate than less strong individuals. The amount of muscle mass employed during movement is another consideration. Activities characterised by a large number of muscle groups (i.e. high total muscle mass), such as squats and deadlifts, would stimulate much higher blood lactate concentrations than activities using smaller quantities of muscle mass, such as bicep curls. By analogy those individuals utilising a greater amount of lean muscle mass, at a given relative intensity, may also produce greater lactate responses. As indicated throughout this review, the intracellular composition of muscle (e.g. enzyme activity, fibre distribution, capillary density, mitochondria size and volume) is also important in regulating the production and removal of lactate. Whilst the accumulation of lactate appears to be influenced by various factors, it is not yet known if the ability to increase the lactate response would also result in greater training benefits from the metabolic stimulus.

It would appear that nutritional strategies (CHO and/or PRO) do not influence the acute lactate response to a single bout of resistance exercise; however, over consecutive days of such exercise, a reduced lactate response may result from supplementation. Again, how this response may influence the
training benefits afforded by the metabolic stimulus remains highly speculative. Although CHO and PRO would appear to inhibit the metabolic stimulus, nutrition itself is a key component for weight training and resultant adaptation, particularly for energetic requirements and muscle recovery and repair.

It is evident from the literature that the importance of the acute metabolic stimulus, in facilitating strength and power adaptation, remains largely unknown. This is highlighted by the lack of scientific evidence surrounding those mechanisms that underlie the metabolic stimulus. Confounding our understanding is the fact that endurance-type schemes (light loads, high total repetitions, short rest periods) produce greater lactate responses than neuronal and hypertrophy-type schemes, which would suggest that the metabolic stimulus may be more important for eliciting endurance, rather than strength adaptation. However, light-load high-repetition training is another strategy often employed by bodybuilders to induce muscle growth. Still, with resistance exercise inducing numerous acute mechanical, hormonal and metabolic responses, it is likely that resultant adaptation is an integration of these factors, rather than a single mechanism. What role the metabolic stimulus plays in signalling those processors involved in long-term adaptive change is still speculative. As intimated throughout this review, there are a number of areas in need of further research and interactions to be addressed. Such an analysis would enhance understanding in this area and improve the prescription of resistance exercise for strength and power development.

3. Conclusions

It is obvious from the data presented that the current understanding of the metabolic stimulus and its contribution to strength and power adaptation remains largely unknown. The configuration of the various training variables did impose a specific activation pattern in the lactate responses (i.e. hypertrophy schemes > neuronal > dynamic power schemes); however, fewer studies have examined neuronal and dynamic power schemes. Factors such as age, sex, training experience and nutrition may also influence the lactate response to resistance exercise and thereafter, adaptation. Still, the importance of these differences remains, as yet, unclear. Those mechanisms underpinning muscular adaptation, in relation to the metabolic stimulus, remain highly speculative. Inconsistent findings and methodological limitations within research (e.g. programme design, sampling period, number of samples) also make interpretation difficult. More research is also needed to examine the lactate responses to different loading schemes, as well as interactions across age, sex and training status, so our understanding of the metabolic stimulus is improved.

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