

# Possible Stimuli for Strength and Power Adaptation

## Acute Hormonal Responses

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## Abstract

The endocrine system plays an important role in strength and power development by mediating the remodelling of muscle protein. Resistance training scheme design regulates muscle protein turnover by modifying the anabolic (testosterone, growth hormone) and catabolic (cortisol) responses to a workout. Although resistance exercise increases the concentrations of insulin-like growth factor 1 in blood following exercise, the effect of scheme design is less clear, most likely due to the different release mechanisms of this growth factor (liver vs muscle). Insulin is non-responsive to the exercise stimulus, but in the presence of appropriate nutritional intake, elevated blood insulin levels combined with resistance exercise promotes protein anabolism. Factors such as sex, age, training status and nutrition also impact upon the acute hormonal environment and, hence, the adaptive response to resistance training. However, gaps within research, as well as inconsistent findings, limit our understanding of the endocrine contribution to adaptation. Research interpretation is also difficult due to problems with experimental design (e.g. sampling errors) and various other issues (e.g. hormone rhythms, biological fluid examined). In addition to the hormonal responses to resistance exercise, the contribution of other acute training factors, particularly those relating to the mechanical stimulus (e.g. forces, work, time under tension) must also be appreciated. Enhancing our understanding in these areas would also improve the prescription of resistance training for stimulating strength and power adaptation.

Weight training or resistance training is widely recognised as an important training stimulus for the development of muscular strength and power. The endocrine system plays an important role in the training process by mediating the remodelling, or turnover (i.e. synthesis and degradation), of muscle protein.<sup>[1,2]</sup> Muscle protein turnover is an integral part of protein metabolism and necessary for the breakdown (during exercise) and subsequent repair (during recovery) of muscle tissue. The adaptive response reflects the overall summation of protein metabolism after exposure to multiple bouts of resistance exercise (i.e. training effect). A net accretion in muscle protein (synthesis > degradation) is typically manifested by an increase in muscle cross-sectional area (CSA), thereby enhancing the potential

for force generation (e.g. strength and power). Alternatively, a negative protein balance (degradation > synthesis) would result in a loss of muscle tissue and possibly diminish the force-generating capabilities of muscle.

A bout of resistance exercise produces acute changes in the hormonal environment, which have been linked to those cellular processors involved in protein turnover and muscle growth.<sup>[3-5]</sup> In brief, elevated hormone concentrations increase the likelihood of receptor interactions, thereby initiating a cascade of events leading to the acute (e.g. protein metabolism) and chronic (e.g. muscle growth) adaptive response.<sup>[6]</sup> How different resistance training schemes influence the hormonal environment is therefore fundamental to improving our understand-

ing of the effect of resistance exercise upon the hormonal milieu and long-term adaptation. Factors such as sex, age, training status and nutrition are also determinants of the adaptive response to resistance training. Therefore, further examination of any hormonal interactions with these factors, would also seem important in understanding the endocrine contribution to the adaptive process. This article examines the acute hormonal response to different resistance exercise schemes and, where possible, describes how these effects may be mediated by sex, age, training status and nutrition.

The design of the resistance exercise programme, or scheme design, underpins the adaptive response to resistance training by modifying the acute hormonal responses. Consequently, examining the hormonal response to different strength and power schemes would provide a better understanding of the hormonal contribution to adaptation associated with the repeated application of these lifting methods. The interactions between the anabolic (e.g. testosterone [TST], growth hormone [GH], insulin-like growth factors [IGFs], insulin) and catabolic (e.g. cortisol) hormones are also important in the training process, by modifying the balance between anabolism and catabolism. This article therefore examines the responsiveness of these hormones to three broad types of schemes often used within practice. That is, hypertrophy (controlled movements, moderate loads, short rest periods), neuronal (explosive intent, heavy loads, long rest periods) and dynamic power (explosive and/or ballistic movements, light loads, moderate rest periods) schemes. Additional interactions between the hormonal responses to resistance exercise and sex, age, training status and nutrition, will then be discussed.

## 1. Testosterone

The majority of evidence supports the notion that TST has a considerable anabolic effect upon muscle tissue.<sup>[3-5]</sup> Traditionally considered the primary an-

drogen, TST is synthesised and secreted from the Leydig cells of the testes, via the hypothalamic-pituitary-gonadal (HPG) axis, with a small amount also derived from the ovaries, adrenals and from the conversion of other androgens (e.g. androstenedione). This hormone contributes to muscle growth by increasing protein synthesis and decreasing protein degradation.<sup>[5]</sup> Indirectly, TST may also contribute to protein accretion by stimulating the release of other anabolic hormones (e.g. GH). Like all steroid hormones, TST is derived from cholesterol and not freely soluble in plasma. Most TST is bound to albumin (~38%) and sex hormone-binding globulin (SHBG) [~55–60%] with the remaining circulating freely or unbound (~2–5%).<sup>[7]</sup> The unbound steroid represents the biologically active fraction (i.e. available to the tissues), although TST that is weakly bound to albumin may be rendered active through its rapid dissociation from albumin. Thus, the pool of free and albumin-bound TST, or non-SHBG-bound TST, has often been termed the 'bioavailable' steroid. The 'free androgen index' is another measure of androgen status, calculated from the ratio of total TST to the concentration of SHBG. This article, however, focuses upon the more common measurements of total and free TST.

### 1.1 Programme Design

Resistance training schemes designed to improve maximal strength through primarily morphological adaptation (i.e. hypertrophy schemes) generally produce relatively large increases (%) in TST, with these increases greater than that observed for schemes designed to enhance strength through mainly neural (i.e. neuronal schemes) adaptation (see table I and table II). Kraemer et al.,<sup>[8]</sup> for instance, compared the hormonal response to eight exercises performed with either a five repetition maximum (5RM) load for 3–5 sets per exercise and 3 minutes rest between sets or a 10RM load (three sets per exercise) with 1-minute rest periods. The

**Table I.** Acute hormonal response to hypertrophy schemes

Study	Subjects (age)	Protocols [exercise(s), sets × reps (load)]	Hormone (% or fold change)				
			TST	GH	IGF-1	insulin	cortisol
Vanhelder et al. <sup>[10]</sup>	5 UT males	1 ex, 7 × 10 (10RM)				↑ ~10	↑ ~80
Kraemer et al. <sup>[11]</sup>	9 T males	8 ex, 3 × 10 (10RM)	↑ ~30	↑ ~11-fold	↑ ~26		
Craig et al. <sup>[12]</sup>	11 UT males	7 ex, 3 × 8–10 (75% 1RM)		↑ ~520			
Kraemer et al. <sup>[8]</sup>	8 T males	8 ex, 3 × 10 (10RM)	↑ ~72	↑ ~850	↑ ~34		
	8 T females	8 ex, 3 × 10 (10RM)	Nil	↑ ~106	↑ ~13		
Kraemer et al. <sup>[13]</sup>	8 UT males	4 ex, 3 × 10 (10RM)	Nil	↑ ~550	Nil		
Kraemer et al. <sup>[14]</sup>	8 T males	8 ex, 3 × 10 (10RM)					↑ ~65
Hakkinen and Pakarinen <sup>[9]a</sup>	10 T males	1 ex, 10 × 10 (10RM)	↑ 24 (↑ 22) <sup>b</sup>	↑ 170-fold			↑ 149
Kraemer et al. <sup>[15]</sup>	9 T females	8 ex, 3 × 10 (10RM)	Nil	↑ ~110	Nil		↑ ~125
Chandler et al. <sup>[16]</sup>	9 T males	8 ex, 2 × 8–10 (75% 1RM)	↑ ~20	↑ ~700	Nil	↑ ~20	
McMurray et al. <sup>[17]a</sup>	8 T males	6 ex, 3 × 6–8 (80% 1RM)	↑ 21	↑ 31-fold			↑ 21
Hakkinen and Pakarinen <sup>[18]a</sup>	8 UT males (27y)	1 ex, 5 × 10 (10RM)	↑ 9	↑ 200-fold			Nil
	8 UT females (25y)	1 ex, 5 × 10 (10RM)	Nil	↑ 225			Nil
Mulligan et al. <sup>[19]</sup>	10 T females	8 ex, 1 × 10 (10RM)		↑ ~70			↑ ~20
		8 ex, 3 × 10 (10RM)		↑ ~450			↑ ~175
Gotshalk et al. <sup>[20]</sup>	8 T males	8 ex, 1 × 10 (10RM)	↑ ~14	↑ ~400			↑ ~10
		8 ex, 3 × 10 (10RM)	↑ ~32	↑ ~700			↑ ~25
Volek et al. <sup>[21]</sup>	12 T males	1 ex, 5 × 10 (10RM)	↑ 7				Nil
McCall et al. <sup>[22]</sup>	10 RT males	8 ex, 3 × 10 (10RM)	Nil	↑ ~38-fold	Nil		↑ ~27
Kraemer et al. <sup>[23]</sup>	8 UT males (30y)	1 ex, 4 × 10 (10RM)	↑ ~38 (↑ 40) <sup>b</sup>	↑ ~16-fold		Nil	↑ ~78
Kraemer et al. <sup>[24]</sup>	8 UT males (30y)	1 ex, 4 × 10 (10RM)	↑ ~37 (↑ 29) <sup>b</sup>	↑ ~28-fold			↑ ~80
Hakkinen et al. <sup>[25]a</sup>	10 UT males (26y)	2 ex, 4 × 10 (100% MVC)	↑ ~27 (↑ 21) <sup>b</sup>	↑ ~31-fold			Nil
Bosco et al. <sup>[26]</sup>	6 T males	3 ex, 12 × 8–12 (70–75% 1RM)	↓ ~70	↑ ~50-fold			
Hakkinen et al. <sup>[27]a</sup>	10 UT males (40y)	1 ex, 5 × 10 (10RM)	↑ ~21	↑ ~340			
	11 UT females (40y)	1 ex, 5 × 10 (10RM)	(↑ 21) <sup>b</sup>	↑ ~170			
			Nil (nil)				
Taylor et al. <sup>[28]</sup>	6 T females	7 ex, 3-4 × 10 (10RM)		↑ ~90			
	6 UT females	7 ex, 3-4 × 10 (10RM)		↑ ~30			
Kraemer et al. <sup>[29]a</sup>	10 RT males	10 ex, 3 × 10 (10RM)	↓ ~29 <sup>c</sup>				

Continued next page

Table I. Contd

Study	Subjects (age)	Protocols [exercise(s), sets × reps (load)]	Hormone (% or fold change)				
			TST	GH	IGF-1	insulin	cortisol
Consitt et al. <sup>[30]a</sup>	16 T females	8 ex, 3 × 10 (10RM)	Nil	↑ ~350	Nil		Nil
Smilios et al. <sup>[31]a</sup>	11 T males	4 ex, 2 × 10 (75% 1RM)	Nil	↑ ~400			Nil
		4 ex, 4 × 10 (75% 1RM)	↑ ~10	↑ ~11-fold		↑ ~28	
		4 ex, 6 × 10 (75% 1RM)	Nil	↑ ~19-fold		↑ ~27	
Zafeiridis et al. <sup>[32]a</sup>	10 T males	4 ex, 4 × 10 (75% 1RM)		↑ ~13-fold			↑ ~38
McGuigan et al. <sup>[33]</sup>	8 RT males and 9 RT females	2 ex, 6 × 10 (75% 1RM)					↑ 97%
Rubin et al. <sup>[34]</sup>	9 T males	1 ex, 6 × 10 (10RM)		↑ ~13-fold	↑ 11		
	10 UT males	1 ex, 6 × 10 (10RM)		↑ ~9-fold	↑ 10		

a Control data provided.

b Free TST.

c Salivary hormones.

**ex** = exercises; **GH** = growth hormone; **IGF-1** = insulin-like growth factor 1; **MVC** = maximal voluntary contraction; **reps** = repetitions; **RM** = repetition maximum; **RT** = recreationally trained; **T** = trained; **TST** = testosterone; **UT** = untrained; ↓ indicates decrease; ↑ indicates increase.

total TST response to the hypertrophy scheme (72%) was much greater than that reported following the neuronal scheme (27%). Similarly, Hakkinen and Pakarinen<sup>[9]</sup> reported an increase in total TST (24%) and free TST (22%) to a hypertrophy squat session (ten sets × ten repetitions, 10RM). However, no significant changes in total or free TST occurred after the performance of a neuronal type squat session (20 sets × one repetition, 1RM). These findings confirm the importance of programme design in modulating the acute hormonal response to resistance exercise.

Dynamic power schemes, often employed to maximise explosive power and functional performance, have also produced significant androgen responses. For example, total (18%) and free TST (30%) increased in response to half-squat lifts performed with a load of 50% 1RM.<sup>[36]</sup> It would appear that, on average, the blood TST response (peak percentage change from pre-exercise) to hypertrophy and dynamic power schemes are of similar magnitude (14%), with neuronal schemes producing the smallest (7%) change (see tables I and II). When extrapolating research in such a manner the effect of sex upon TST activity (i.e. females non-responsive) is an important consideration. Subtle differences in the protocols implemented across research (e.g. exercises performed, total volume lifted), in each of the generic schemes, should also be acknowledged. In addition, fewer studies have examined the hormonal response to neuronal and dynamic power schemes, than hypertrophy schemes. Thus, it is suggested that further research examine the responsiveness of TST across these schemes, with an emphasis upon those training methods often used within practise, to improve the 'practical significance' of these findings.

## 1.2 Sex

Whilst resistance exercise generally produces an elevated TST profile among males, such a response

**Table II.** Acute hormonal response to neuronal and dynamic power schemes

Study	Subjects (age)	Protocols [exercise(s) sets × reps (load)]	Hormone (% or fold change)				
			TST	GH	IGF-1	insulin	cortisol
<b>Neuronal schemes</b>							
Kraemer et al. <sup>[11]</sup>	9 T males	8 ex, 3/5 × 5 (5RM)	↑ ~30	↑ ~175	↑ ~25		
Kraemer et al. <sup>[8]</sup>	8 T males	8 ex, 3/5 × 5 (5RM)	↑ ~27	↑ ~375	↑ ~27		
	8 T females	8 ex, 3/5 × 5 (5RM)	Nil	Nil	↑ ~13		
Kraemer et al. <sup>[15]</sup>	9 T females	8 ex, 3/5 × 5 (5RM)	Nil	↓ 70	Nil		Nil
Hakkinen and Pakarinen <sup>[9]a</sup>	10 T males	1 ex, 20 × 1 (100% 1RM)	Nil (nil) <sup>b</sup>	↑ 361			Nil
Raastad et al. <sup>[35]a</sup>	9 T males	3 ex, 3 × 3–6 (3–6RM)	↑ ~17	↑ ~14-fold	↓ ~7	↓ ~11	Nil
Kraemer et al. <sup>[14]</sup>	8 T males	8 ex, 3/5 × 5 (5RM)					Nil
Smilios et al. <sup>[31]a</sup>	11 T males	4 ex, 2 × 5 (88% 1RM)	Nil	Nil			Nil
		4 ex, 4 × 5 (88% 1RM)	Nil	Nil			Nil
		4 ex, 6 × 5 (88% 1RM)	Nil	↑ ~600			Nil
Zafeiridis et al. <sup>[32]a</sup>	10 T males	4 ex, 4 × 5 (88% 1RM)		↑ ~400			Nil
<b>Dynamic power schemes</b>							
Mero et al. <sup>[36]</sup>	9 T males	1 ex, 10 × 6 (50% 1RM)	↑ 18 (↑ 30) <sup>b</sup>				
Mero et al. <sup>[37]</sup>	6 males (24y)	1 ex, 10 × 6 (50% 1RM) <sup>c</sup>	↑ 16				Nil
		1 ex, 10 × 6 (50% 1RM) <sup>d</sup>	↑ 18 (↑ 19) <sup>b</sup>				Nil
	6 males (15y)	1 ex, 10 × 6 (50% 1RM) <sup>c</sup> 1 ex, 10 × 6 (50% 1RM) <sup>d</sup>	Nil ↑ 13 (↑ 11) <sup>b</sup>				↑ 67 ↑ 33
Volek et al. <sup>[21]</sup>	12 T males	1 ex, 5 × 10 (30% 1RM)	↑ 15				Nil
Pullinen et al. <sup>[38]</sup>	6 males (25y)	1 ex, 10 × 6 (50% 1RM) <sup>c</sup>	↑ ~16				
		1 ex, 10 × 6 (50% 1RM) <sup>d</sup>	↑ ~18				
	6 males (15y)	1 ex, 10 × 6 (50% 1RM) <sup>c</sup> 1 ex, 10 × 6 (50% 1RM) <sup>d</sup>	Nil ↑ ~13				

a Control data provided.

b Free TST.

c 4-minute rest periods.

d 1-minute rest periods.

**ex** = exercises; **GH** = growth hormone; **IGF-1** = insulin-like growth factor 1; **reps** = repetitions; **RM** = repetition maximum; **T** = trained; **TST** = testosterone; ↓ indicates decrease; ↑ indicates increase.

is usually not evident among females, regardless of the protocol performed (see tables I and II). For example, in response to a hypertrophy and neuronal scheme, males revealed an elevated total TST profile (72% and 27%, respectively).<sup>[8]</sup> In contrast, females produced no TST changes across these exercise schemes, when lifting the same relative loads. The average response to hypertrophy schemes performed by males (18%) and females (nil) also support these data (see table I). Sex differences at rest are also evident, with females demonstrating much lower pre-exercise (or baseline) total TST concentrations (5–10% of males), than that seen across male populations of similar age.<sup>[8,18,27,39-43]</sup> These differences may be attributed to the respective production and release mechanisms of this androgen. In males, luteinising hormone (LH) and follicle-stimulating hormone stimulates the Leydig cells of the testes to synthesise and secrete relatively large amounts of TST into the blood stream, at rest and in response to physical stimulation. Conversely, the female ovaries and adrenals produce much smaller quantities of TST. The different secretion patterns of this androgen are not unexpected, as sex differences in strength and hypertrophy have traditionally been attributed to the anabolic actions of TST.<sup>[5]</sup>

### 1.3 Age

Resistance exercise is known to increase blood TST levels among young males.<sup>[37,38,44,45]</sup> However, young males appear to elicit smaller hormonal responses than their adult counterparts. Pullinen et al.<sup>[39]</sup> compared the hormonal response to an exercise scheme with the same relative load, among men, women and pubescent boys. Adult males were the only group exhibiting an increase in TST (total and free), although this result may have been explained by plasma volume changes. Other studies<sup>[37,38]</sup> have also observed greater TST (total and free) responses among adult males, compared with young males, performing dynamic power schemes

with different rest periods. Baseline TST levels are also greater among adult males,<sup>[37,39]</sup> although younger males do exhibit a more pronounced diurnal rhythm. That is, TST samples drawn in the morning were not significantly different between groups, but by early afternoon (pre-exercise) these samples were significantly higher among men.<sup>[39]</sup> The differences between men and boys may be explained by the greater testicular volume (e.g. Leydig cells) in men and the regulatory control of the HPG axis, being more synchronised among adults.<sup>[46]</sup> This variability is likely to provide adult males with a more anabolic response for adaptation, thereby potentially explaining muscle mass and strength differences between groups.

It is generally accepted that the aging process is accompanied by alterations in endocrine function (e.g. decline in hormone secretion).<sup>[47]</sup> Such a notion is directly supported by research in this area.<sup>[23-25]</sup> Kraemer et al.,<sup>[23]</sup> for instance, compared the hormonal responses of adult and elderly males, each performing a hypertrophy scheme using the same relative load. In the adult group, total (38%) and free TST (40%) levels were elevated, with these changes greater than that observed in the elderly group (20% and 26%, respectively). In addition to these exercise-induced differences, adult males also exhibit greater resting TST levels, than their older counterparts.<sup>[23,25]</sup> The reduction in androgen activity with increasing age may, in part, explain the similar loss in muscle mass, as well as strength and power changes (i.e. reducing), that occurs as we age. Proposed mechanisms for these age-related changes 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.<sup>[48]</sup> Considering some of the potential benefits of resistance training (e.g. altered basal levels), restoring endocrine function in older adults through resistance exercise is an attractive hypothesis for

ameliorating the age-related decline in muscle mass and function.

#### 1.4 Training Status

Training status, or experience, appears an important factor regulating the hormonal response to resistance exercise. For example, previously untrained males produced an elevated total TST response (12%) to exercise, after 9 weeks of resistance training, whereas no change occurred when exercise was performed before training.<sup>[41]</sup> It has also been revealed that strength-trained athletes have greater TST (total and/or free) responses than non-athletes,<sup>[49,50]</sup> and that greater training experience is accompanied by enhanced responses.<sup>[44]</sup> These data are indicative of enhanced sensitivity of the HPG axis (and TST secretion) to resistance exercise with resistance training experience. Such adaptation would positively influence the training response (i.e. more anabolic), particularly if combined with a training-induced increase in resting TST.<sup>[24,41]</sup> Interactions between training status and age is less clear with some studies reporting enhanced secretion among elderly males after resistance training,<sup>[24,42]</sup> whilst others have reported no changes.<sup>[27,51]</sup> Still, any endocrine change would appear limited to male populations with females non-responsive to training.<sup>[41,42,52]</sup> Hormone secretion may be further sensitive to the type of training experience, with a less pronounced TST response (total and free) found in endurance-trained males, than resistance-trained and untrained males.<sup>[50]</sup> Lowered TST secretion among endurance athletes is not uncommon and may be explained by training-related dysfunction within the HPG axis (e.g. feedback control, LH secretion).<sup>[53]</sup>

Interestingly, those individuals who specifically train to increase muscle mass (e.g. bodybuilders and steroid users) have shown an inhibited hormone response to resistance exercise. For example, a 70% reduction in TST was observed among male

bodybuilders performing a lower body workout.<sup>[26]</sup> Another study<sup>[54]</sup> examined the endocrine responses among bodybuilders and power-lifters who were categorised into two groups: anabolic steroid users or non-steroid users. Following an exhaustive squat session, no significant changes in blood TST occurred in either group. Similarly, Rozenek et al.<sup>[55]</sup> reported a smaller TST response (%) to resistance exercise among steroid lifters, compared with non-steroid lifters. Given that bodybuilding and anabolic steroid usage is often associated with extreme muscle mass, these results are somewhat surprising. The responses observed may be explained by the greater TST levels in steroid lifters (and lower LH levels), than non-steroid lifters,<sup>[55]</sup> or increased binding affinity of steroids with muscle androgen receptors.<sup>[26]</sup> Although speculative, it is possible that TST uptake and utilisation is enhanced as an adaptive response to specific training practises (e.g. bodybuilding). The responsiveness of other hormones also need to be considered, as concomitant increases in anabolic activity (GH)<sup>[26]</sup> or lowered catabolic (cortisol) activity<sup>[54]</sup> may account for the attenuated TST response.

#### 1.5 Nutrition

Nutritional intake (e.g. carbohydrate [CHO] and protein [PRO]) is an important aspect of resistance training, in terms of energy maintenance and restoration, as well as assisting with muscle recovery and repair. Nutritional supplementation may further contribute to the training process by regulating the hormonal response to exercise.<sup>[16,56,57]</sup> Chandler et al.<sup>[16]</sup> examined acute hormone response to exercise, with CHO and/or PRO taken immediately after and 2 hours after exercise. Supplementation produced a reduction in total TST in the post-exercise period, compared with a placebo. A similar response was also observed over 3 consecutive days of exercise with CHO and PRO supplementation,<sup>[56]</sup> and in response to a mixed meal, an isocaloric beverage of

similar content and an isocaloric CHO beverage.<sup>[57]</sup> It therefore seems that supplementation of this nature may exaggerate the post-exercise decline in TST. The implications of this response are unclear, as this may be accredited to an increase in hormone uptake and/or greater clearance. For example, the decrease in TST observed was not associated with a decline in LH,<sup>[16]</sup> thereby suggesting greater hormone clearance. The ratio between TST and SHBG also remained unchanged after supplementation, which is indicative of stable free testosterone levels, despite lowered TST.<sup>[56]</sup> Further research is needed to differentiate those mechanisms (uptake or clearance) underpinning the exercise-induced changes in total TST, as well as the partitioning of the different TST fractions, with nutrition.

## 2. Growth Hormone

GH, also known as somatotropin, is another potent anabolic hormone influencing muscle tissue growth. This peptide is synthesised and released in a pulsatile manner from the somatotrope cells, located within the anterior segment of the pituitary gland.<sup>[58]</sup> Somatotropin is secreted via the somatotrophic axis, regulated by two hypothalamic peptides, GH-releasing hormone (GHRH), which stimulates GH synthesis and secretion, and GH-inhibiting hormone or somatostatin, which inhibits the release of GH.<sup>[59]</sup> Similar to TST, GH contributes to protein metabolism by increasing protein synthesis and reducing the degradation of muscle protein.<sup>[5,59]</sup> The secretion of GH may further enhance the training environment by stimulating the release of a family of polypeptide growth factors (i.e. somatomedins), also known for their anabolic effect upon muscle tissue.<sup>[4]</sup> As a peptide, human GH represents a family of proteins with over 100 different forms currently identified within plasma fluid;<sup>[58]</sup> however, the function of the different variants has yet to be fully established. The most dominant form of GH in circulation and the

most widely examined within research is the 22 kDa variant, which is the primary focus in this article.

### 2.1 Programme Design

The secretion of GH is extremely sensitive to the stimulus of resistance exercise, particularly those schemes designed to maximise muscle growth (see tables I and II). For example, the performance of a neuronal scheme, among trained males, produced a 3-fold increase in circulating GH, compared with an 11-fold increase after a hypertrophy scheme.<sup>[11]</sup> Similarly, GH increased more dramatically in response to a hypertrophy scheme (13-fold) than a neuronal scheme (4-fold), also among trained males.<sup>[32]</sup> Altogether, these data confirm the importance of programme design in modulating the GH response to resistive exercise. To our knowledge, no studies have examined the responsiveness of this hormone to dynamic power schemes. The observed increase in GH to hypertrophy schemes (23-fold) is, on average, ~8 times the response produced by neuronal schemes (3-fold) [see tables I and II]. Whilst these data confirm a greater anabolic response to muscle building schemes, fewer studies have again examined maximal strength schemes. Examination of the GH release patterns across neuronal and dynamic power schemes would also benefit our understanding regarding the hormonal contribution to strength and power adaptation.

Individual variability in the GH response to resistance exercise is an important consideration. Raastad et al.<sup>[35]</sup> examined the GH response of nine trained males to two exercise schemes, one performed at a high intensity (3RM) and the other at a moderate intensity (70% of 3RM). An examination of individual responses revealed that three subjects did not respond to either protocol, five subjects showed moderate responses to both protocols and one subject increased his GH level to twice that of the moderate responders, during the high and moderate schemes. These data suggest that even within a

homogenous population that there may be some individual differences (i.e. responders vs non-responders) in the hormonal response to a given workout. These differences may be attributed to individual factors relating to psychological and genetic make-up, ethnicity, subject chronotype or habitual training practises. The sensitivity of GH release to exercise may also magnify the effects of individual variability. Differentiating between 'responders' and 'non-responders' and tracking adaptation across different resistance training regimens, would improve our understanding regarding the contribution of the endocrine system to strength and power adaptation.

## 2.2 Sex

Males generally produce greater GH responses (%) to exercise than females, regardless of the scheme performed (see tables I and II). In response to a hypertrophy and neuronal scheme, male GH levels increased by 850% and 375%, respectively, whilst among females, much smaller hormonal change (106% and nil, respectively) occurred.<sup>[8]</sup> Despite this difference, females consistently exhibit greater blood GH levels after resistance exercise.<sup>[8,27,40,41]</sup> Sex differences in the basal concentrations of somatotropin offer a likely explanation, being much greater among female populations (up to four times) compared with males of similar age.<sup>[8,27,40-42]</sup> Sex differences in GH secretion at rest and in response to exercise may be attributed to several factors including: the orderliness of pulsatile GH release (males > females); the mass of secreted hormone per burst; and the sensitivity of GH to GHRH (females > males).<sup>[60]</sup> The maximal GH-releasing capacity, however, is thought to be equal between men and women, at least in healthy adult populations.<sup>[60]</sup> Given that males still demonstrate greater muscle mass and force-generating capabilities than females, the importance of these elevated

GH levels among females requires further investigation. As most studies have examined women in the follicular phase of menstruation,<sup>[8,15,19,28,39,41]</sup> the effect of exercise upon hormone release patterns in different phases of the menstrual cycle, is another area for enquiry.

## 2.3 Age

Scant data are available to characterise the release pattern of GH between young males and adults. As with TST (adults vs elderly), GH production shows attenuation with age, in terms of both total secretion and the amplitude of pulsatile release.<sup>[47]</sup> Hakkinen and Pakarinen<sup>[18]</sup> examined the endocrine response to a hypertrophy-type workout, among three groups of untrained males (27 years, 47 years, 68 years) and untrained females (25 years, 48 years, 68 years). Each group lifted the same relative load. For males, the youngest group recorded the greatest GH response (200-fold), followed by the 47-year-old group (19-fold) with no changes among the oldest group. Although the 48-year-old group, in females, produced a larger GH response (20-fold) than the youngest group (2-fold), the oldest female group were again non-responsive to the exercise protocol. Other studies have reported similar findings,<sup>[23-25,61]</sup> further indicating a diminished GH response with aging. In terms of basal hormone levels, aging is associated with a progressive fall in the 24-hour rate of GH secretion (~14% per decade), after adulthood.<sup>[62]</sup> Age-related changes in somatotrophic function may be due to several factors including reduced sensitivity of GH release to GHRH and age-related variations in the neurohormonal and neurotransmitter control of somatotropin secretion, as well as other peripheral factors (e.g. gonadal hormones, adiposity).<sup>[62]</sup> These data suggest that alterations in endocrine activity may contribute to the changes in muscle mass and functional performance with aging.

## 2.4 Training Status

The effect of training status upon GH release during resistance exercise is unclear. A greater GH response was found in untrained males, than trained males, each performing a leg workout.<sup>[63]</sup> Similarly, a training study reported a reduction in the GH response to exercise, among previously untrained males.<sup>[24]</sup> Data extrapolated from table I provide further evidence of training-related differences (i.e. decreasing with experience), with untrained males (on average) revealing greater blood GH responses (37-fold) than trained males (29-fold). Still, other studies have reported no differences between resistance-trained and untrained individuals,<sup>[49]</sup> or as a function of resistance training experience.<sup>[44]</sup> Many training studies have also reported little to no change in the GH response (exercise-induced), among previously untrained individuals,<sup>[12,41]</sup> in particular those of older age,<sup>[24,27,42,51]</sup> and among those with recreational experience.<sup>[22]</sup> Other data confound our understanding in this area, with trained individuals exhibiting greater GH responses than untrained,<sup>[28,34]</sup> and enhanced responses after resistance training.<sup>[42,52]</sup> The variability observed may be explained by training-related factors (e.g. length, type, periodisation), age and/or sex interactions, as well individual variability in GH response to exercise. Lowered resting GH levels as a result of training,<sup>[12,28]</sup> further complicates our understanding regarding the effect of training experience upon hormone release in response to a workout.

## 2.5 Nutrition

A number of studies have examined the effect of supplementation upon the GH response to resistance exercise.<sup>[16,45,56,64,65]</sup> A combined PRO and CHO supplement, taken 120 minutes before and immediately after exercise, produced an elevated GH profile compared with a placebo treatment.<sup>[56]</sup> This response was not, however, repeatable over 2 more

days of consecutive exercise under the same nutritional conditions. Another study also found supplementation (CHO and PRO, CHO) to positively influence the GH response to exercise, at some point in the recovery period.<sup>[16]</sup> In contrast, other experiments have reported no hormonal differences with PRO and CHO<sup>[64]</sup> or PRO (amino acids) supplementation.<sup>[45,65]</sup> This variability may be explained by differences in the procedures for supplementation (e.g. timing, content and volume) and the experimental design (e.g. single vs multiple workouts). It is also possible that the effects of nutrition upon the GH response to exercise may, in part, be masked by factors relating to training status, as these studies employed resistance-trained subjects, as well as the additional metabolic effects of GH (e.g. fatty acid mobilisation, blood glucose regulation). From the data presented in this section, it appears that somatotropin is characterised by variable responses to resistance exercise and an important consideration when interpreting and extrapolating research findings.

## 3. Insulin-Like Growth Factors

The identification of the IGFs (somatomedins) has added much to our understanding of the hormonal contribution to muscle growth. Whilst the anabolic role of GH is well established, literature now suggests that the effect of GH upon muscle tissue is largely mediated by these growth factors<sup>[47,59,66]</sup> by way of similar mechanisms (e.g. increase protein synthesis, decrease protein degradation).<sup>[2]</sup> In terms of muscle growth, the most important of these factors is believed to be IGF-1, also known as somatomedin-C. It was traditionally believed that IGF-1 was synthesised and released from the liver, under the regulatory control of GH (GH-IGF-1 axis), and then transported to the target tissue.<sup>[66]</sup> In addition to the liver, this factor is also produced from within the muscle in response to mechanical signals.<sup>[67]</sup> To distinguish the muscle-secreted growth factor from the liver variant, the term mecha-

no growth factor (MGF) has been used.<sup>[67]</sup> Overall, this growth factor may exert its biological actions through the blood (and liver secretion), as well as muscle release mechanisms, by way of autocrine (i.e. within muscle cells) and paracrine (i.e. between adjacent muscle cells) pathways.<sup>[68,69]</sup> It remains to be seen if circulating GH also regulates IGF-1 expression in skeletal muscle. This section will focus upon the IGF-1 variant of the somatomedin family.

### 3.1 Programme Design

Resistance exercise often modifies the blood levels of IGF-1, but in contrast to TST and GH, the effect of scheme design is less clear. On average, the IGF-1 response to hypertrophy (9%) and neuronal schemes (12%) are relatively small and somewhat similar (see tables I and II). Research findings in this area are still largely inconsistent (i.e. either increasing or no change), which may be partly explained by individual variability in the response to exercise or a possible increase in IGF-1 uptake. The different release mechanisms of this growth factor offer the most likely explanation for this inconsistency. That is, blood-borne measurements of IGF-1, as performed by most studies, may not reflect the overall response of this growth factor, given its release from both systemic (liver) and local (muscle) pathways. The influence of resistance exercise upon these growth factors may also lie in the manner in which IGF-1 is partitioned among its family of binding proteins.<sup>[70]</sup> Determining the responsiveness of the different growth factor variants to these lifting schemes, would therefore seem important to understanding their role in strength and power adaptation.

### 3.2 Sex

Research findings indicate similar somatomedin responses to resistance exercise, between males and females. For instance, the IGF-1 response to a workout exhibited no sex differences between elderly males and females.<sup>[40]</sup> When comparing adult

males and females, no differences were also found in the IGF-1 response to a hypertrophy scheme.<sup>[8]</sup> In response to a neuronal scheme, however, the time course of the growth factor response varied, with significant increases found immediately post-exercise in males and an hour after exercise among females. Unfortunately, characterising the temporal release of this growth factor is difficult due to the sampling periods ( $\leq 1$  hour) employed in these studies. That is, the subsequent stimulation of IGF-1 secretion from the liver, may occur some 16–28 hours after GH-stimulated release.<sup>[71]</sup> Although baseline GH levels are often much greater among females, compared with males, blood IGF-1 levels appear similar across sex.<sup>[8,40,43]</sup> Taken together, this highlights a sex difference in the feedback control of the GH-IGF-1 axis, which may also explain the greater adaptive capabilities of male muscle. It is also possible that sex differences may exist in the ability to express IGF-1 in non-hepatic tissue (muscle), in response to mechanical loading. Because only blood measures have been extracted from within research, such a notion requires further exploration.

### 3.3 Age

The effect of age on the growth factor response to resistance exercise has received little attention. Elderly individuals<sup>[40,71,72]</sup> have demonstrated elevated IGF-1 responses to resistance exercise, but in these studies age-related comparisons were not performed. A recent study<sup>[73]</sup> examined the expression of the IGF-1 muscle variant (from a muscle biopsy) within adult and elderly men, in response to a lower limb workout. Resistance exercise produced a significant increase in MGF expression in the adult, but not the elderly group. No differences existed in the resting levels of MGF. These data are suggestive of an attenuated MGF response to resistance exercise, in older males, and indicative of age-related desensitivity to mechanical loading. Resting levels of blood

IGF-1 have also revealed age-related interactions among males (adult > elderly).<sup>[24]</sup> It therefore appears that the aging process, at least in males, is accompanied by various changes in growth factor activity, and another endocrine mechanism likely to explain age-related differences in muscle mass and function. Although altered endocrine function is an attractive mechanism for explaining muscular differences between adults and the elderly, one must also take into account the general decline in physical activity among older populations. A decline in activity would not only reduce the mechanical loading upon the muscle, which is an important stimulus for muscle protein metabolism,<sup>[74]</sup> but may also exacerbate many of the hormonal changes seen among older populations.

### 3.4 Training Status

Training experience appears to have little influence upon the IGF-1 response to resistance exercise. For example, examination of recreationally trained men revealed no training-induced changes in the acute IGF-1 response to exercise.<sup>[22]</sup> No changes in the acute IGF-1 (total and free) response to a workout were also found among elderly subjects, after 8 weeks of strength training.<sup>[72]</sup> The examination of trained and untrained individuals provides further evidence to support these data.<sup>[34]</sup> In response to a hypertrophy type scheme, both resistance-trained and untrained males produced a similar blood IGF-1 response to exercise (11% and 10%, respectively), each lifting the same relative load. However, the absolute concentrations of IGF-1 were consistently greater among the resistance-trained group, at rest and throughout the exercise intervention, suggesting differences in basal concentrations. Other research have also reported greater resting blood levels of IGF-1 among active individuals than those who are sedentary.<sup>[40]</sup> Thus, those mechanisms controlling the systemic secretion (basal levels) of this growth factor would appear to be sensitive to the

effects of training or similar exercise. Examining the effect of different training practises (e.g. strength and power schemes) upon both growth factor variants would enhance our understanding of the endocrine contribution to the adaptive process.

### 3.5 Nutrition

Interactions between supplementation and the IGF-1 system are largely unclear, due to a paucity of research in this area. The few studies that have examined these interactions have found no differences between supplementation and placebo treatments upon the somatomedin response to resistance exercise.<sup>[16,56]</sup> Still, there may be some benefits for PRO and CHO supplementation, as a result of elevated IGF-1 concentrations (on days 2 and 3), when combined with 3 days of consecutive exercise.<sup>[56]</sup> Although speculative, it is possible that feeding may play an important role in the training process by regulating IGF binding protein levels and/or the expression of the muscle IGF-1 variant with exercise. The blood and muscle release patterns of this growth factor, in response to resistance exercise and supplementation, is an area for further investigation. Whilst the growth factors are known to have important anabolic effects upon muscle tissue, both circulating and locally produced IGF-1 are also thought to have important metabolic effects (e.g. stimulate glucose uptake).<sup>[75]</sup> Therefore, supplementation resulting in altered blood glucose levels, may well mask the effect of nutrition upon the hormonal response to exercise. One must remain cognizant of such issues within future studies in this area.

## 4. Insulin

Insulin is another peptide hormone that is known to have a strong anabolic effect upon muscle tissue.<sup>[4,5]</sup> This hormone is synthesised and released from the  $\beta$  cells of the islets of Langerhans, from within the pancreas.<sup>[4]</sup> The primary role of insulin, in conjunction with the hormone glucagon, is to regu-

late blood glucose concentrations and the metabolism of fatty acids. A secondary role of insulin is to increase the uptake of CHO and amino acids into the muscle cell.<sup>[4,76]</sup> In this capacity, insulin is thought to play an important role in the remodelling of muscle tissue, as protein intake (i.e. amino acids) is generally seen as an essential component to anabolism resulting from resistance training. In terms of muscle protein turnover, insulin release suppresses the degradation of muscle protein in the recovery period.<sup>[76]</sup> Under some conditions (e.g. amino acid infusion) insulin is also known to have a positive effect upon protein synthesis, although this response may also depend upon the normal levels of amino acids, in the intracellular compartment of the muscle.<sup>[2]</sup>

#### 4.1 Programme Design

Whilst insulin has been widely implicated in muscle growth, recovery and repair, little research has examined the responsiveness of this hormone to different lifting schemes. Hypertrophy schemes, the most widely examined in this area, have produced only small changes (10%) in blood insulin concentrations, despite the fact that anabolic hormone activity is critical to muscle tissue growth (see table I). As the primary regulator of glucose levels, however, insulin secretion is likely to be more sensitive to alterations in blood glucose levels. A number of studies support such a contention, having examined the endocrine response to exercise in conjunction with supplementation. That is, hypertrophy schemes when performed with a placebo treatment have produced no changes in the acute insulin response.<sup>[16,56,57,64,77,78]</sup> However, when this exercise was performed under the additional influence of nutrition (CHO and/or PRO), an ~5-fold increase (on average) was found following exercise. Thus, nutritional intake would appear to be the primary factor regulating insulin secretion, rather than the stimulus of resistance exercise itself or any interac-

tions thereof. For this reason, only the effects of nutrition will be examined in this section.

#### 4.2 Nutrition

Nutritional intake plays a key role in mediating the anabolic effects of insulin. The ingestion of CHO would increase insulin levels and in the presence of PRO (e.g. increased amino acid availability) promote greater protein anabolism.<sup>[1,79]</sup> A CHO and PRO supplement taken immediately after the performance of three different workouts, consisting of low, moderate or high volume, resulted in a 300% increase (on average) in insulin levels, whereas no changes were found with a placebo treatment.<sup>[64]</sup> Other studies are in agreement, having observed elevated insulin responses to resistance exercise, when combined with CHO and PRO (or amino acids) supplementation,<sup>[16,56,80-82]</sup> particularly when taken before exercise, as compared with post-exercise ingestion.<sup>[80]</sup> CHO alone not only improves the insulin response to resistance exercise,<sup>[77,78,83,84]</sup> but also produces a more favourable environment for muscle growth (i.e. reduces myofibrillar protein breakdown).<sup>[83,84]</sup> It does appear, however, that a combined CHO and PRO supplement produces a superior result than either alone.<sup>[82]</sup> Examination of long-term strategies is now required to determine the importance of these supplements and their associated endocrine responses to long-term adaptation, with emphasis upon target tissue effects.

### 5. Cortisol

Generally considered the primary catabolic hormone, cortisol is the main member of a family of steroid hormones called glucocorticoids. Corticosterone is the other glucocorticoid of interest; however, it is thought to be much less potent than cortisol, accounting for around 4–5% of total glucocorticoid activity.<sup>[85]</sup> Cortisol is synthesised and secreted from the adrenal cortex, via the hypothalamic-pituitary-adrenal (HPA) axis, with a small amount also de-

rived from the conversion of cortisone. The catabolic effects of cortisol are well recognised and attributed to a decrease in protein synthesis and increased protein degradation.<sup>[6]</sup> The anti-anabolic properties of this hormone are also related to the attenuation of other anabolic hormones (e.g. TST and GH).<sup>[3]</sup> Chronically elevated cortisol levels have been linked to various stressors (e.g. depression, trauma, over-training); hence, this glucocorticoid is also considered one of the primary stress hormones.<sup>[86]</sup> In blood, >90% of this hormone is bound with plasma proteins, mainly with cortisol binding globulin and the rest with albumin, with the remaining fraction (<10%) circulating freely.<sup>[85]</sup> No studies have sought to directly examine changes in blood-free cortisol under exercising conditions and, therefore, only data relating to total cortisol will be discussed.

### 5.1 Programme Design

Hypertrophy lifting schemes produce an elevated stress hormone (cortisol) response, with this greater than that found in neuronal lifting schemes (see tables I and II). For instance, a hypertrophy scheme performed by trained males produced a 65% increase in blood cortisol, whereas no cortisol change occurred across a neuronal scheme.<sup>[14]</sup> The performance of identical protocols, among trained females, also revealed a much greater stress response to a hypertrophy scheme (125%) than a neuronal scheme (nil).<sup>[87]</sup> Such data indicate that the design of the exercise scheme regulates the cortisol response to a workout. Dynamic power schemes have also resulted in elevated cortisol concentrations,<sup>[37]</sup> although not to the same extent as hypertrophy schemes. On average, a 45% increase in blood cortisol has been observed in response to hypertrophy schemes, whereas dynamic power schemes have resulted in a 20% increase (see tables I and II). Neuronal schemes, on the other hand, have not produced any change in cortisol concentrations across exercise. Fewer studies have again examined the hormonal

response to training methods often used to optimise maximal strength and explosive power, than bodybuilding-type training methods.

### 5.2 Sex

Research to date would indicate that no sex differences exist in the response of this stress hormone to resistance exercise when lifting the same relative load.<sup>[18,33,39,40]</sup> McGuigan et al.<sup>[33]</sup> examined the sex response of this glucocorticoid (in saliva) to two lifting schemes of different intensities (30% and 70% 1RM), each performed with a squat and a bench-press exercise. The authors found no significant differences between groups, in the cortisol response to these schemes. Furthermore, the rating of perceived exertion for each session also revealed no sex differences, suggesting that the 'perception of stress' was also the same. As an indirect measure of the free (biological active) steroid, salivary cortisol is thought to provide a better measure of adrenal function;<sup>[88]</sup> therefore, the values reported in this study may better reflect the stress response to exercise than blood (total hormone measures). The benefits and limitations of saliva and blood, as biological tools for hormone measurement, will be discussed in a later section. The above data are indicative of a similar stress hormone response with this type of exercise. In addition, baseline cortisol levels are also similar across male and female populations.<sup>[18,39-43]</sup> Given these similarities, it is possible that sex differences in muscle protein turnover (and functional differences) may depend, to a greater extent, upon the actions of the anabolic hormones and the variability discussed previously.

### 5.3 Age

Young males appear to produce a greater stress hormone response to resistance exercise than adults. For example, in response to a dynamic power scheme with two different rest periods, boys produced significant increases in serum cortisol, where-

as no changes were found among adults.<sup>[37]</sup> Also, when comparing men, women and pubescent boys, only boys experienced a significant cortisol response to a workout.<sup>[39]</sup> In addition, peak adrenaline concentrations, another stress hormone, were found to be twice as high in boys after the exercise bout compared with both men and women. No differences in morning and baseline hormone samples were found between men and boys. These findings are suggestive of a greater exercise-induced cortisol response, among younger males. Although the underlying mechanisms are difficult to define, differences in maturation, anxiety and adaptive capability to resistance exercise, may be contributing factors in this respect.<sup>[39]</sup> As young males exhibit a larger catabolic (cortisol) and lower anabolic (TST) response to resistance activity, it is perhaps not surprising that this population do not exhibit the same adaptive changes with training, often seen among adult males.

Adult and elderly populations have revealed similar cortisol responses to resistive exercise, irrespective of sex.<sup>[18,23-25]</sup> When interpreting these data it is, however, important to recognise that some of the interventions performed did not result in any significant changes in cortisol.<sup>[18,25]</sup> Comparing stress hormone responses to schemes that did not sufficiently stimulate the HPA axis (and cortisol secretion), would appear to be somewhat flawed. Baseline concentrations of cortisol are also similar between adult and elderly populations.<sup>[18,23-25]</sup> Hence, it would appear that cortisol production and secretion may be independent of the aging process. Despite these similarities, the tissue-specific availability of active glucocorticoids may be altered among elderly populations. It has been speculated that the activity of steroid metabolising enzymes (e.g. 11 $\beta$ -hydroxysteroid dehydrogenase) may increase with aging, leading to increased glucocorticoid availability and action within elderly muscle.<sup>[47]</sup> Such an adaptation, particularly when combined with a lowered anabolic

state, as indicated throughout this article, would result in a hormonal balance (e.g. less anabolic – more catabolic) that is less conducive to muscle recovery and repair.

#### 5.4 Training Status

The cortisol response to resistance exercise reveals ambivalent results, as a function of training status. For instance, a greater cortisol response was observed among untrained males, compared with trained males, after a lower body workout.<sup>[63]</sup> Similarly, a lowered cortisol response to exercise was found after 10 weeks training, among untrained males.<sup>[24]</sup> These differences (i.e. decreasing with experience) are supported by the data in table I with trained males exhibiting a 35% increase (on average) in cortisol, compared with a 48% increase among untrained males. In contrast, many studies have found no differences between strength-trained and untrained men,<sup>[49,50,89]</sup> or as a function of resistance training experience.<sup>[44]</sup> Training studies have also found little to no change in the exercise-induced cortisol response among previously untrained males<sup>[24,41]</sup> or among males with recreational experience.<sup>[22]</sup> Disparities may again be explained by training-related factors, age interactions, as well as altered resting hormone levels (i.e. decreasing) with training.<sup>[22,24,41]</sup> Variable stress responses to exercise (cortisol no change, catecholamines increasing), as well as training-related differences in the catecholamine response (trained > untrained) should be noted.<sup>[89]</sup> Thus, if any results are to be interpreted in the context of stress, then it may be more appropriate to identify and contrast a range of biological stress responses (e.g. glucocorticoids, catecholamines, cytokines).

Similar to TST, the cortisol response to resistance exercise would appear to be influenced by the type of training experience. Tremblay et al.,<sup>[50]</sup> for instance, observed an attenuated cortisol response among endurance-trained men to a resistance

workout with the same relative load compared with both resistance-trained and untrained men. Examination of different types of strength athletes (bodybuilders and power-lifters) has, however, revealed no differences in the catabolic response to a workout.<sup>[90]</sup> Thus, the cortisol response to resistance exercise may be more sensitive to athlete type and associated training practises (e.g. endurance vs strength), as opposed to different training methods among strength-trained athletes (e.g. bodybuilding vs power lifting). Training-related differences in strength, muscle size and composition, trainability to specific stimuli and energetic metabolism, are likely to explain this finding. When making comparisons in such a manner the nature of the assessment is an important consideration, as those individuals performing an exercise protocol similar to their own training methods, are more likely to tolerate such exercise than those who are unaccustomed but equally trained.<sup>[90]</sup>

### 5.5 Nutrition

Whilst nutritional supplementation appears to play some role in regulating many of the anabolic responses to resistance exercise, it would appear less important in regulating the catabolic response. Thyfault et al.,<sup>[77]</sup> for instance, examined two nutritional interventions (CHO or placebo), taken 10 minutes prior and immediately after a bout of resistance exercise. A similar increase in cortisol occurred in both groups examined, with no significant differences found between treatments. Several other studies have also found little or no differences in the cortisol response to resistance exercise, performed either with (CHO and/or PRO) or without (placebo) supplementation.<sup>[45,56,57,64]</sup> However, one study did observe a blunted response to an exercise bout with CHO ingestion, compared with a placebo treatment (7% vs 99%, respectively).<sup>[78]</sup> As most studies have found the total cortisol response to exercise non-responsive to supplementation, the effect of nutri-

tional strategies upon the different steroid fractions is another line of investigation.

## 6. Limitations of Research

Scrutiny of research evaluating the hormone responses to resistance exercise reveals several problems with interpretation. A major difficulty exists in the design of many experiments, which often involves a short sampling period ( $\leq 5$  minutes) and/or the collection of only a single biological sample after exercise.<sup>[9,18,21,22,25-27,36-39,41,51,54,63,72,89,90]</sup> Such an approach does not provide sufficient information to characterise the temporal dynamics of the endocrine system. Although many studies have monitored temporal hormonal change, longer intervals between samples (e.g.  $>30$  minutes) would introduce further sampling errors. In order to adequately monitor hormonal change in response to exercise, a more systematic approach is needed (e.g. more samples and shorter sampling intervals) in the design of these types of experiments. The importance of plasma volume shifts, as indicated throughout, has been discussed extensively. The need for correction depends upon the questions asked if, for example, they relate to hormone secretion then concerns over plasma volume may be warranted; however, if the questions relate to the biological response, dependent upon blood hormones levels, then plasma volume shifts may be less important. Regardless of the mechanism, an increase in blood hormone levels is thought to increase receptor interactions;<sup>[91]</sup> therefore, in the context of this review, plasma volume changes are deemed less important.

Few studies have accounted for the diurnal (or circadian) rhythm of hormonal secretion (see tables I and II), the importance of which, relates to fluctuations in daily hormone levels. For example, TST and cortisol both peak in the early morning and decline throughout the day.<sup>[92,93]</sup> Depending upon the rate of decline, hormone data that appear to be responsive to a particular exercise stimulus (i.e. reduced levels),

might also be an artefact of these normal biological fluctuations. Examining the hormone response to exercise, without reference to control data, may also lead to erroneously finding no significant changes across exercise (i.e. pre levels = post levels), when in fact a real increase occurred, or, alternatively, mask the magnitude of the hormonal increase. The circadian rhythm is also known to influence the hormonal response to the exercise stimulus,<sup>[94-96]</sup> thus, one must also take into consideration the time of assessment. The pulsatile secretion (or ultradian rhythm) exhibited by some hormones (e.g. GH)<sup>[97]</sup> confounds our understanding if, for example, a given sample was taken before, or after, one of these episodic patterns. This rhythm may also regulate the hormonal response to stress, at least in rats. That is, a noise stressor coinciding with a rising phase resulted in a significant hormone response, whereas the stress response coinciding with a falling phase resulted in no significant changes.<sup>[98,99]</sup> Exercise is an activator of a stress response, therefore, the application of resistance exercise (i.e. sets) in relation to these pulsatile fluctuations, may also determine the hormonal response. Such a notion warrants further investigation among humans.

Saliva is a relatively new biological tool within strength and conditioning practise. Compared with blood, saliva is an easy compliant method for steroid determination, which can be applied frequently and is a less stressful mechanism for fluid collection. The most salient feature of saliva is its ability to reflect the free hormone,<sup>[100-103]</sup> which is important as most studies have monitored the bound (total) steroid in blood. Although this fraction (bound) may be used as an indirect measure of the free hormone under resting conditions, this may not be the case during exercise. Under exercising conditions the free steroid may increase, relative to the bound fraction, due to the saturation of the binding globulins at higher blood hormone levels.<sup>[104]</sup> Changes in exercise-induced metabolic acidosis may also con-

tribute to a disproportionate increase in free hormone levels, by increasing the dissociation rate of the bound steroid.<sup>[105]</sup> Where possible the direct measurement of the free hormone in blood, or indirectly in saliva, should therefore be employed. Despite the advantages that saliva offers there are limitations.<sup>[91]</sup> The contamination of saliva with blood would falsely increase the saliva hormone level. Salivary protein from the mucosa may also interfere with the processing of these samples. Regardless of the fluid examined, one must remain cognizant of other issues relating to hormone evaluation in the exercising environment (e.g. statistical reporting, analysis procedures, techniques), which have been discussed in detail elsewhere.<sup>[91]</sup>

## 7. Implications for Strength and Power Development

The endocrine system plays an important role in mediating the remodelling of muscle protein and, as a result, strength and power development. Although the exact mechanisms underpinning muscle growth remain unknown, elevated hormone levels increase the likelihood of interactions with membrane-bound (i.e. peptides) or nuclear-bound (i.e. steroids) receptors. Hormone-receptor interactions then initiate a cascade of events leading to alterations in protein turnover rate. Over time, a net accretion in muscle protein would lead to measurable changes in muscle CSA, enabling muscle to exhibit greater potential for force generation (e.g. strength and power). Additional mechanisms of signal transduction have been recently identified for the steroid hormones, termed non-genomic, which are rapid onset (i.e. seconds to minutes) and non-compatible with gene expression, involving membrane-bound receptor interactions.<sup>[106]</sup> Thus, the steroid contribution to performance and adaptation may well involve both long-term (e.g. morphological) and short-term (e.g. neural) pathways. The non-genomic steroid effects upon skeletal muscle tissue remains largely unknown.

Hypertrophy schemes produce hormonal responses that, by analogy, would appear important for muscle growth to occur, including elevated concentrations of TST, GH and IGF-1 (see table III). Similarities in the TST responses to hypertrophy and dynamic power schemes is somewhat surprising given the anabolic (e.g. muscle building) properties of this hormone, which may be partly explained by an increase in TST uptake and utilisation as an adaptive response to specific training practises (e.g. bodybuilding) or sex differences (i.e. females non-responsive). Although the GH-IGF-1 axis is activated with such exercise, the overall IGF-1 response is unclear due to the different release mechanisms of this growth factor (liver vs muscle). Insulin is not responsive to the exercise stimulus, but still plays an important anabolic role, in the presence of appropriate nutritional intake. Hypertrophy schemes also produce the largest cortisol response, an integral part in protein metabolism. The glucocorticoids for example, are thought to create an increased pool of amino acids for protein synthesis to occur and may also increase protein turnover rate in previously active muscles.<sup>[85]</sup> A hormonal environment characterised by a large catabolic response, in conjunction with a 'larger' anabolic response, is therefore likely to benefit muscle protein accretion.

Neuronal schemes produce smaller anabolic (TST and GH) and catabolic (cortisol) responses than hypertrophy schemes (see table III). It may therefore be speculated that neuronal training meth-

ods are less likely to result in net protein accretion, to the same extent as hypertrophy training methods. Such a notion is supported by the different morphological profiles (e.g. fibre and muscle CSA) exhibited by bodybuilders versus power-lifters/Olympic lifters,<sup>[107-109]</sup> which may be attributed to the training methods employed by these athletes (bodybuilders = hypertrophy schemes, power/Olympic lifters = neuronal schemes) and associated hormonal responses. The IGF-1 response to this type of exercise is similar to hypertrophy lifting schemes; however, the practical significance of this finding is again complicated by the different mechanisms of release. Although the insulin response to neuronal schemes (and dynamic power schemes) remains virtually unknown, the effect of exercise alone, as demonstrated by several studies, is unlikely to modify the insulin response to any great extent.

The effect of dynamic power schemes upon GH and IGF-1 activity has not yet been determined. Exercise of this nature has, however, resulted in blood TST responses similar to hypertrophy schemes, yet this type of training does not generally produce significant muscle hypertrophy. The elevated TST response may be attributed to the non-genomic actions of this hormone. It has been proposed that TST contributes, not only to the morphological development of the fast twitch muscle fibres, but also the functioning of these fibres during powerful performance (e.g. jumping, sprinting).<sup>[110]</sup> If blood-borne TST levels regulated the functioning of these fibres, then an elevated TST response to explosive exercise (e.g. dynamic power schemes) may be expected among trained athletes, as an adaptive response to specific training practises.<sup>[111]</sup> Cortisol is also responsive to dynamic power schemes, but not to the same extent as hypertrophy schemes, possibly explaining the lack of muscle tissue change accredited to such training methods.

The premise that elevated hormone levels (and receptor reactions) regulates protein metabolism,

**Table III.** Summary of research examining the acute anabolic and catabolic hormone responses to hypertrophy, neuronal and dynamic power schemes

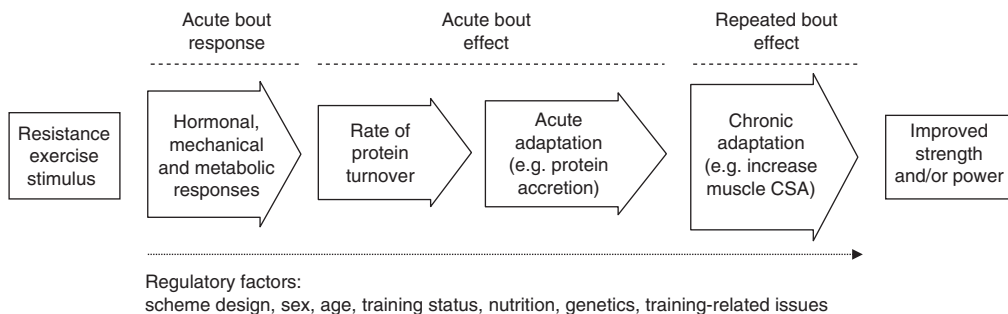
	Hypertrophy	Neuronal	Dynamic power
Testosterone	↑↑↑↑	↑↑	↑↑↑↑
GH	↑↑↑↑↑↑↑↑	↑	↔
IGF-1	↑↑↑	↑↑↑↑	↔
Insulin	↔	↔	↔
Cortisol	↑↑↑↑↑	↔	↑↑

**GH** = growth hormone; **IGF-1** = insulin growth-like factor 1; ↑ indicates increase, ↔ indicates no change or equivocal results.

and thereafter long-term adaptation, is too simplistic with many endocrine issues unresolved. These not only involve hormone release mechanisms, which are the focus for most investigations, but may also include hormone clearance rates, changes in binding proteins, fluid shifts and hormone degradation rates.<sup>[5,68]</sup> The biological actions of the hormone-receptor complex will itself be determined by the number and/or binding affinity of receptors, which are also dynamic structures adapting (e.g. up-regulating or down-regulating) in response to different hormonal and mechanical stimuli.<sup>[6]</sup> Thus, alterations in the hormonal environment, or the acute hormonal responses to resistance exercise, is but one of many endocrine factors mediating the training response and muscular adaptation. Although the primary anabolic and catabolic hormones examined have received considerable attention within research, the actions and interactions of other hormones (e.g. catecholamines,  $\beta$ -endorphins, thyroid hormones, estrogens) present other complex possibilities. An in-depth examination of those endocrine mechanisms possible underpinning strength and power expression is beyond the scope of this article, but may be sourced elsewhere.<sup>[68,69]</sup>

Factors such as sex, age, training status and nutrition may also regulate the acute hormonal environment. The influence of these factors upon the adaptive response to resistance training may therefore be due, in part, to the hormonal interactions observed.

However, gaps within research and inconsistent findings still limit our understanding of the endocrine contribution to resistance training adaptation. In addition to these hormonal factors, the importance of other mechanical (e.g. forces, work, time under tension) and, to a lesser extent, metabolic factors (e.g. lactic acid), should also be recognised. That is, strength endurance schemes (i.e. light loads, high total repetitions, short rest periods), and even aerobic exercise, often produce hormonal changes similar to, or greater than, hypertrophy schemes.<sup>[12,30-32]</sup> However, these training methods do not generally result in any substantial gains in muscle size, strength or power, due to the different mechanical stressors imposed.<sup>[74]</sup> Hence, the adaptive response is likely to be the integration of several acute training factors (e.g. hormonal, mechanical, metabolic), rather than any single factor alone (see figure 1). The overall influence of sex, age, training status and nutrition upon the adaptive response to resistance training, is therefore likely to depend upon the combined responses to, and effect of, these acute training factors and not withstanding the importance of other genetic factors (e.g. fibre distribution, psychological make-up) and training-related issues (e.g. overload, periodisation). Such is the nature of resistance training where a multitude of adaptive strategies may be used to facilitate strength and power development.



**Fig. 1.** Schematic representation of the morphological pathway to adaptation. **CSA** = cross-sectional area.

## 8. Conclusions

Scheme design produces a specific activation pattern in the acute hormonal (TST, GH and cortisol) response to resistance exercise, thereby mediating the remodelling of muscle tissue and long-term adaptation. The response of other hormones (IGF-1 and insulin) to, or in conjunction with, this type of exercise is also important to protein metabolism, but more difficult to quantify. Factors such as sex, age, training status and nutrition may also regulate the hormonal environment; however, gaps within research and inconsistent findings limit our understanding of the endocrine contribution to adaptation. This understanding is complicated by problems with research interpretation and the complex nature of the acute training stimulus. The endocrine system plays an important role in muscle protein turnover, leading to subsequent changes in strength and power, as part of an integrative response to the stimulus of resistance training.

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