Neuroendocrinology and resistance training in adult males

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Lead Summary

An understanding of the neuroendocrine system will assist the Strength and Conditioning coach in the design of progressive strength training programmes by allowing them to manipulate acute training variables according to hormone release profiles. For muscle hypertrophy, training programmes should utilise 3 sets of 10 repetitions at 10RM loads, with short rest periods of no longer than 1 minute. This will ensure the accumulation and maintenance of lactate and hydrogen ions, to which anabolic hormone release is correlated. For strength adaptations without concomitant muscle hypertrophy, the training load and the length of rest periods should be increased, (>85% 1RM and >2mins respectively), and body parts should be rotated (e.g. upper body to lower body or agonist to antagonist). Finally, catabolic hormones and neurohormones significantly affect training adaptations. Therefore the strength and conditioning coach should be cognisant of the specific exercise programming and psychological interventions that manipulate their release.

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Neuroendocrinology

Neuroendocrinology describes the partnership between the endocrine system and the nervous system and their collaboration to maintain homeostasis via hormonal regulation.[63,65] The nervous system functions quickly, but actions are short-lived and localised. In contrast, the endocrine system functions slowly, but actions are longer lasting and more general. The endocrine system includes all tissues and glands that secrete hormones into the circulatory system. Hormonal signals can also be secreted via paracrine and autocrine mechanisms.[23] The former describes a hormonal interaction between adjacent cells without transport from the circulatory system, whilst the latter describes releasing hormones within the cell itself for interaction with that cell. Similar to endocrine glands, neurons synthesize, store and secrete chemical messengers, namely neurotransmitters (e.g. acetylcholine) and neurohormones (i.e. catecholamines).[65]

The neuroendocrine system is directly affected by the following strength training variables: exercise modality (involved musculature), exercise sequence, intensity/load, sets and repetitions (volume) and rest period.[34,48,49,63,74] These variables must be skilfully manipulated within a periodised strength and conditioning (S&C) programme if increases in strength and/or hypertrophy are to be optimised. If however, significant miscalculations are made, the result may be regressive, catabolic and in

time could lead to overtraining.[19,54] An understanding of the neuroendocrine system will therefore assist the S&C coach in the design of progressive strength training programmes by allowing them to manipulate the aforementioned variables according to hormone release profiles, thereby optimising the hormonal environment required for specific goals. The purpose of this article therefore, is to discuss the hormones pertaining to resistance training programmes, and identify the optimal manipulation of acute training variables to optimise hormonal release. This article will review: (1) the main anabolic hormones (those that promote tissue building), testosterone, growth hormone, and insulin-like growth factor, (2) the catecholamine's (stress hormones released in response to the fight or flight phenomenon), principally adrenaline and noradrenalin, and (3) the catabolic hormones (those that promote tissue degradation), namely cortisol.

Before examining each hormone in turn however, this article will first describe the fundamental role of receptors and the significance of muscle remodelling.

Hormone-Receptor Complex

Hormones can be defined as chemical messengers that are transported to specific target cells, which possess specific hormone receptors. The specificity of a hormone and its receptor is often explained using the lock and key theory, whereby the receptor is the lock and the hormone is the key. It is important to note that the concentration of hormones may not be as important as the number of receptors available, as this ultimately determines the possibility of interactions.[23] For example, when a cell has reached its genetic ceiling for adaptation (e.g. through protein accretion), receptors may become non responsive and down-regulate, thus reducing the probability of hormonal binding.[65] Alternatively, receptors can up-regulate and increase the probability of interactions. For example, Kadi *et al*.,[47] reported that power lifters had a greater number of androgen receptors in response to continued resistance training and thus, an enhanced ability to use testosterone. In addition, Ratamess *et al*.,[80] have shown significant correlations between baseline androgen receptor content in the vastus lateralis and 1RM squat, further suggesting that androgen receptor content may assist in mediating strength changes during resistance training. Moreover, resistance training results in up-regulation of androgen receptors in fast twitch muscle fibres, which rely on protein accretion for hypertrophy, and down-regulation in slow twitch fibres, which instead resist protein degradation as hypertrophy may be disadvantageous.[19,70]

Muscle Remodelling

Muscle remodelling involves the disruption of muscle fibres, (stimulus/load dependent), in response to mechanical loading, resulting in the inflammatory process (immune cells and catabolic hormones), and subsequent release of anabolic hormones.[4,12] In addition, mechanical loading increases receptor and membrane permeability to hormones and nutrients, therefore tissue activation may be considered a precursor to anabolism.[64] Consequently, only the recruited muscle fibres can be remodelled,[65] emphasising the need to exercise muscle groups in a sport specific manner (including range of motion, muscle action, velocity of movement, force generation and relative intensity), and the need to utilise progressive overload. The latter will increase motor unit recruitment,[82] thereby exposing a greater number of muscle fibres to hormone-tissue interactions.[64]

Testosterone

Testosterone (TST) is responsible for the development of male secondary sex characteristics, spermatogenesis and the male skeletal system. Pertinent to this discussion, TST is involved in the muscle growth and protein retention observed during strength training through its direct (i.e. muscle growth), and indirect (stimulation of growth hormone and neuron receptors) affects on muscle tissue.[23,65] Moreover, due to its potent anabolic effects, the levels of circulating TST have been proposed as a physiological marker to evaluate the anabolic status of the body.[39] Further, Staron *et al*.,[85] linked muscle fibre transformations (IIx to IIa) and concomitant enhancement in strength, to increased serum TST concentrations. TST is also related to the inhibition of muscle glycogen breakdown and the displacement of glucocortcoids (e.g. cortisol).[70] The response of TST to offset protein metabolism may be vital for maintaining muscle size and function.[70]

TST is a steroid hormone derived from cholesterol (lipid soluble), and is therefore able to diffuse through the cell membrane (sarcolemma of muscle cells) and bind with its androgen receptors in the cytoplasm (sarcoplasm in muscle cells). This hormone-receptor complex then enters the nucleus and binds with the cell's DNA in a process referred to as direct gene activation. This stimulates the formation of mRNA (messenger ribonucleic acid), which enters the cytoplasm and promotes protein synthesis.[19,70,79] The synthesised protein may be an enzyme, which can have numerous effects, or a structural protein used for tissues growth and repair. Examples of structural proteins include titin, actin, myosin and myosin isoform changes (such as IIx to IIa). Following stimulation from Luteinizing hormone (LH) and Follicle-stimulating hormone (FSH), TST is released from the Leydig cells of the testes in men and the ovaries and adrenal glands in women.[16] Women consequently have TST concentrations 15-20 times lower than males,[65] which affects their capacity for muscle hypertrophy and strength.

Transport Proteins

After secretion, the vast majority of testosterone becomes either, tightly bound with a beta globulin called sex hormone binding globulin, or loosely bound with albumin. It is delivered to its target tissue via these methods (SHBG; ≈44-60% of total transportation), and albumin (≈38%), with the remainder remaining in its free biological state (≈0.2-2%).[16,92] The free-hormone hypothesis suggests that it is only the free hormones that interact with the target tissue receptors[16,64,70] with SHBG effectively inhibiting TST's action.[92] It may be no surprise therefore, that the ratio of SHBG to TST has been reported to correlate with isometric leg strength,[37,40,41,42] closely mirror strength changes,[38] and relate to actual competitive weightlifting performances.[41] Transport proteins however, are ultimately responsible for the rate of delivery of the hormone[70] and act much like a chaperone, protecting TST from degradation.[30] Moreover, albumin is considered to have only a loose connection with TST and therefore, although to a lesser extent than free TST, is considered biologically active.[16,92]

TST-Nervous Interactions

As well as cellular interactions, TST can also bind with receptors on neurons and therefore increase instantaneous muscle strength and recruited muscle mass.[63,64,65] This is achieved through an increase in neurotransmitter release and structural adaptations of the neuromuscular junction.[11,23,75] Moreover, TST-nervous interactions can regenerate nerves and increase the cell body size and dendrite length and diameter.[75] These neural adaptations may demonstrate an advanced strategy to increase force capability in subjects who have little potential for change in muscle hypertrophy.[23] Furthermore, initial pre-competition levels of TST have been correlated to average power output[8,9] and jumping height.[8] In addition, both power and work performed during 60s continuous jumping protocols were positively related to changes in TST levels.[8] These correlations are thought to be due to TST's significant effects on motor neurons,[91] and serves to highlight the importance of increased TST concentrations and the significance of TST-nervous interactions within sports performance.

Resting concentrations of TST (and concomitant increases in FSH and LH) have been reported to increase only after ≥2 year's resistance training experience.[43,45,54] As previously discussed, this may represent an advanced strategy for force production,[45] and may augment the neural adaptations that are required for additional strength gains in highly trained power athletes when protein accretion is no longer possible or desirable.

Manipulating Training Variables

Large muscle group exercises such as squats, deadlifts,[22] Olympic lifts[54] and jump squats,[93] significantly increase TST concentrations, whereas little or no change has been reported with bench presses and exercises involving smaller musculature.[23,65] It may be concluded therefore, that within a training session, large muscle group exercises are performed before small muscle group exercises in order to expose the smaller musculature to the increased concentrations of TST.[61,64] This is supported by research from Hansen *et al*.,[46] who measured strength changes in elbow flexors following 9 weeks of strength training. Two groups performed elbow flexion exercises, however, one group preceded these with lower body exercise. Only this group significantly increased acute TST concentrations with concomitant increases in the strength of the elbow flexors.

The optimised training strategy for the release of TST has been reported as 3–5 sets of 5–10 repetitions performed close to repetition-maximum loads.[51,53,70] For example, significant elevations were observed following 8 exercises using 5RM and 3min rest periods as well as 10RM and 1min rest periods between sets and exercises.[53] This is in agreement with Hakkinen and Pakarinen,[44] who reported increases following 10 sets of 10 repetitions at 70% 1RM but no significant changes following 20 sets of 1RM. Further, Bosco *et al*.,[10] reported no change following 10 sets of 2 to 3 repetitions, but when the volume increased to 20 sets of 2 to 4 repetitions, increases in TST were noted. Finally, Volek *et al*.,[93] noted increased TST levels following 15 sets of 10 repetitions at 30% 1RM, using jump squats and following 5 sets to failure with a 10RM protocol using the bench press. To the contrary however, Guezennec *et al*.,[35] reported no change following 6 sets of bench press at 70% 1RM, which is a finding in agreement with Kraemer *et al*.[65] The subtle and oftentimes contradictory results reported within the subject of neuroendocrinology and resistance training can often be explained by limitations in research design, as discussed later in this text.

A moderate to high volume of exercise, achieved with multiple sets, repetitions or exercises may be required as the release of TST may be correlated with lactate accumulation.[68,69,71] This was corroborated by Gotshalk *et al*.,[34] who reported greater increases in 3-set heavy resistance protocols, when compared with single-set heavy resistance protocols and by investigators who note the significance of short rest periods.[23,65] Kraemer *et al*.,.[51,53] and Beaven *et al*.,[7] summarise that bodybuilding (hypertrophy) programmes, utilising moderate load, high volume training, with short rest periods, are most effective for stimulating acute TST increases. Research examining the acute response of TST to various exercise protocols is illustrated in Table 1.

The use of high volumes and short rest intervals likely enhances the accumulation and maintenance of lactate and hydrogen ions (H^+), which may act to stimulate the acute release of TST (as is the case with growth hormone as described later in this text). When performed in combination with large muscle group exercises, this likely provides for the optimal stimulation of TST release. Moreover, these findings may also suggest that athletes should limit local dynamic activity during the rest periods, as this is likely to dissipate these by-products. In addition, the valsalva manoeuvre, as well as increasing the safety of an exercise, may also serve to create intramuscular hypoxia and therefore further increase lactate and H+ and subsequent TST concentrations.[18]

Finally, acyclic high-resistance efforts are too brief to affect TST concentrations of the same effort (91). When repeated, the concentration of hormones increases and may therefore affect subsequent repetitions and sets.[91] This may explain why on occasion, the second or third attempt is better. In addition, this delayed response may also act as a contributing factor to the enhanced force achieved following post activation potentiation (PAP) protocols. For example, one of the explanations for the PAP phenomenon is increased neuromuscular drive.[1] As explained above, increased TST release would stimulate the nervous system leading to such a consequence. Moreover, accelerated hormonal responses following endocrine adaptations may further explain why PAP enhancement may only be seen in experienced athletes.[21] Finally, following ≥2years strength training, the increase in resting concentrations of TST may facilitate the PAP phenomenon further. These theories however, are only speculative based on the reviewed research and require further investigation.

TST Concentrations

TST concentrations during training sessions have been reported to remain elevated for up to 45 – 60 minutes and decrease from then on.[97] Viru *et al*.,[91] further suggested that following training sessions of 1 hour duration, the TST-to-cortisol ratio (discussed later in this article), may decrease as a fatigue phenomenon. It may be prudent therefore to limit exercise sessions to ≤60 minutes, as beyond this duration, the session may begin to progress towards catabolism, whereby more

Table 1. The effects of exercise, intensity and volume on the acute response of TST and GH (Table adapted from Crewther et al.,[16] and Kraemer and Ratamess[64]).

Reference	Protocol	TST	GH
Craig *et al.*,[14]	7 ex, 3 x 8-10 at 75% 1RM		Sig ↑ (520 fold)
Gotshalk *et al.*,[34]	8 ex, 1 x 10 at 10RM 8 ex, 3 x 10 at 10RM	Sig ↑ (≈ 14%) Sig ↑ (≈ 32%)	Sig ↑ (≈ 400%) Sig ↑ (≈ 700%)
Hakkinen and Pakarinen[44]	20 sets of 1RM SQ 10 x 10 at 70% 1RM	NC Sig ↑	
Kraemer *et al.*,[51,52]	8 ex, 3-5 x 5RM vs. 10RM with 1 and 3 min rest	Sig ↑:↓ as load ↓ and rest ↑	
Pullien *et al.*,[77]	1 ex, 10 x 6 at 50% 1RM, 4min rest 1 ex, 10 x 6 at 50% 1RM, 1min rest	Sig ↑ 16% Sig ↑ 18%	
Raastad *et al.*,[78]	3-6RM vs. 70% 3-6RM	Sig ↑: 100% > 70%	
Ratamess *et al.*,[80]	1 x 10 SQ, 80-85% 1RM 6 x 10 SQ, 80-85% 1RM	NC Sig ↑	
Rubin *et al.*,[81]	1 ex, 6 x 10 at 10RM, T vs. UT		Sig ↑ (T = 13 fold, UT = 9 fold)
Samilios *et al.*,[83]	4 ex, 2 x 10 at 75%1RM 4 ex, 4 x 10 at 75%1RM 4 ex, 6 x 10 at 75%1RM		Sig ↑ (≈ 400%) Sig ↑ (11 fold) Sig ↑ (19 fold)
Volek *et al.*,[93]	BP, 5 sets to failure at 10RM, 2min rest Jump SQ, 5 x 10 at 30% 1RM, 2min rest	Sig ↑ (7%) Sig ↑ (15%)	
Weiss *et al.*,[94]	4 ex, 3 sets to failure, 80% 1RM	Sig ↑	
Zafeiridis *et al.*,[96]	4 ex, 4 x 10 at 75% 1RM		Sig ↑ (13-fold)

NC = no change; Sig = significant; ex = exercise; SQ = squats; BP = bench press; T = trained; UT = untrained; ↑ = increase; ↓ = decrease

receptors become responsive to cortisol interactions. Moreover, training sessions are often defined by a break of ≥30minutes as this is reported to be enough time to restore TST levels.[97]

TST exhibits diurnal variations, whereby concentrations are typically higher in the morning and drop throughout the day. The reader should note that this is also the case for cortisol.[31,67] The question emerges of whether it is better to exercise in the morning, when concentrations are highest, or to exercise in the evening, to maintain increased concentrations throughout the day.[65] This awareness may allow for several deductions and further advocates the use of split sessions. Split sessions essentially divide one session into two parts to avoid neuromuscular fatigue and concomitant loss of intensity. This may also assist in the maintenance of anabolic hormone concentrations as described above. Furthermore, it may be beneficial to split the session so as the majority of larger muscle group, i.e. lower-body exercises, are performed in the evening when resting concentrations are lower and the majority of the smaller muscle group, i.e. upper-body exercises, are performed in the morning when higher resting concentrations may compensate for the reduced stimulus of these exercises. It should be noted however, that all sessions should commence with large muscle group exercises to optimally initiate the release of TST.[22,54,93] Again, the above suggestion is only speculative and due to the mirroring changes in TST and cortisol, it is equally likely that the same training benefit will be experienced throughout the day.

Methodological Considerations: Measuring the TST Response

To limit the effects of diurnal variations, TST should be sampled immediately post exercise. After 4 hours, these fluctuations, along with recovery mechanisms, can affect the magnitude.[20,57] Because of the high correlation between serum and salivary measures of TST and due to its convenience, being a low-stress and non-invasive method,[7] the latter is commonly used as a marker of gonadial function.[70] Moreover, Beaven *et al.*,[7] found that functional strength gains in sub-elite athletes were strongly related to resistance training protocols that induced maximal free salivary TST responses, and suggested this as a marker to evaluate the efficacy of a strength training protocol. If available, this assessment could be carried out at regular time intervals to ensure optimum benefits, as it is unlikely that an individual would continue to maximally respond to one type of programme.[7] This observation could prove beneficial for periodisation strategies and exercise prescription.

Limitations of Research

The non-standardised protocols, exhibiting variations in volume load, population and subject resistance training experience, have no doubt contributed to the relatively large variation in results. For example, Bosco *et al.*,[8] noted the most significant increases in TST in subjects with higher jumping performance. In addition, untrained men may require several workouts before

exercise-induced increases in TST are noted.[58,64] For example, Kraemer *et al*.,[60] reported significant elevations in serum free TST only after 10 weeks of periodised strength training.

Growth Hormone

Growth hormone (GH), also called samotropin, is secreted by the anterior pituitary gland and is classed as a non-steroid/peptide hormone and as such, (unlike TST), cannot cross the cell membrane. Consequently, its receptors are located on the membrane. Hormone-receptor binding leads to the formation of an intracellular second messenger known as cyclic adenosine monophosphate (cAMP), which then produces the hormone specific physiological response.[95] Specific to resistance training, GH causes hypertrophy through enhanced protein synthesis and amino acid uptake in skeletal muscle.[65] Further, its release following resistance training has been correlated to muscle fibre hypertrophy in type I and II fibres.[72] In addition to these direct effects, GH is also mediated through the production of Insulin like Growth Factors (discussed later in this article), at the autocrine (fat cells), paracrine (muscle cells) and endocrine (liver secretion – where the majority of IGF is released) level of the cell.[25]

GH Response to Exercise

GH release and concentration is highly correlated with glycolytic metabolism and is reported to rise with increased concentrations of lactate and H^+.[32,44] Moreover, H^+ accumulation produced via lactic acidosis may be the primary factor stimulating GH release.[55] In addition to acid base shifts, breath holding, hyperventilation[18] hypoxia[87] and protein catabolism may also influence GH release.[55] This data may therefore further support the use of the valsalva manoeuvre for the stimulation of anabolic hormones as previously discussed.

With respect to increasing peripheral serum concentrations, GH and TST share similar characteristics in acute training variables. Kraemer *et al*.,[51] reported highest GH values following 3 sets of 10 repetitions, at 10RM loads with short (1 min) rest periods. Hakkinen and Pakarinen[44] also reported increases following 10 sets of 10 repetitions at 70% 1RM, but no significant changes following 20 sets of 1RM. Vanhelder *et al*.,[89] reported a significant rise in GH concentrations following 7 sets of 7 repetitions of squats at 80% 1RM, but not following 7 sets of 21 repetitions of squats performed at 30% 1RM. According to Linnamo *et al*.,[69] the load should be near the 8-12RM load, with each set performed towards failure. Moreover, several studies[15,34,74] report the superiority of multiple sets versus single set programmes. Samilios *et al*.,[83] also noted that 4 sets induced more GH (and cortisol) than 2 sets. However, when 6 sets were performed, hormonal responses were not increased further. Finally, Goto *et al*.,[33] examined the effect of adding a down set, (lighter set), after 5 sets of repetitions performed to muscular fatigue at 90% 1RM. They reported that a 50% 1RM down set, performed for as many repetitions as possible, resulted in significant increases in GH and concluded that athletes may be able to magnify the anabolic response of a training session by simply adding a down set.

Based on the significant relationship between lactate and H^+ accumulation and GH response, several points may be deduced. Firstly, if hypertrophy is the goal of a training programme, then resistance training sessions should have a muscle group focus in order to accumulate and maintain localised concentrations of these metabolic by-products. Programmes that alternate between body parts may allow for their dissipation, and therefore reduce the GH response. This may further suggest, that athletes should limit local dynamic activity during the rest periods, as this is further likely to dissipate lactate and H^+. Moreover, muscle group focus and performing sets towards failure may recruit additional motor units due to motor unit fatigue.[82,97] This will therefore subject additional muscle fibres to mechanical loading and consequently increase those that undergo the remodelling process. Finally, a slow-continuous method e.g. 4s concentric – 4s eccentric, would also be beneficial, as this would increase time under tension (facilitating the accumulation of lactate and H^+), reduce local blood circulation (with total occlusion occurring at loads >45% 1RM[84]) and promote venous pooling. The consequent promotion of blood pooling and fluid volume shifts in order to maintain osmotic pressure may then increase the concentration of hormones, time available for interaction and therefore the probability of hormone-receptor interactions.

Insulin like Growth Factors

Many of the effects of GH are mediated through Insulin like growth factors (IGF), also called samatomedins.[27,24] IGF's have been shown to increase protein synthesis in muscles through increasing glucose and amino acid uptake and stimulating myoblast proliferation and differentiation.[25] It has been theorised that the starting levels of IGF will determine if an increase is observed following exercise training, i.e. IGF will only increase if starting levels are low, as high starting levels result in no change.[58]

As well as via GH stimulation, IGF production and release may also occur through direct factors such as mechanical loading and stretch, and these may have greater significance to local IGF concentration levels.[2] For example, the eccentric phase of resistance training appears to be a potent stimulus for the production and release of local growth factors.[6] This may suggest that increases in load during the eccentric phase, or additional negative repetitions at the end of a set may augment IGF release and further increase the hypertrophy seen with GH/bodybuilding type programmes.

Finally, IGFs are structurally related to insulin and can therefore bind with insulin receptors.[64] It may be further speculated that by increasing insulin receptors, for example through a carbohydrate rich meal or exercise training, IGF receptor binding probability may be enhanced (the effect of nutrient manipulation however, is beyond the scope of this article).

Cortisol

Cortisol, a steroid hormone, is secreted from the adrenal cortex following stimulation from adrenocorticotrophic hormone (released by the anterior pituitary gland). The primary pathway for cortisol secretion is through stimulation of the hypothalamus by the central nervous system as a result of hypoglycaemia, the flight or fight response, or exercise.[65] Cortisol is considered a catabolic hormone to skeletal muscle tissue and is released in response to low levels of glycogen, when proteins need to be

catabolised and converted into carbohydrates (gluconeogenesis).[70] TST and insulin can counter the catabolic effects of cortisol by blocking the genetic element in the DNA for cortisol.[65] However, this can only be achieved if they are bound to a greater number of receptors than cortisol. After a period of training and endocrine adaptation, the effects of cortisol may become less dramatic, due to disinhibition of cortisol by TST.[65] Resistance training experience of ≥2 years has been shown to be accompanied by increases in the TST-to-cortisol ratio[45] and may be indicative of enhanced strength and training tolerance.[30]

Circulating cortisol levels reflect tissue remodelling and concurrent inflammatory responses.[56] High levels of cortisol (>800mmol/L) may signify an overtrained state[27,29] and have been highly correlated to serum creatine kinase concentration, which is a marker of muscle damage.[55] In addition, the ratio of TST-to-cortisol may provide a gross estimation (as both hormones have multiple functions across multiple tissue organs) of the anabolic/catabolic state of the body.[28,30] This has been positively related to performance,[3] overreaching[41] and overtraining.[86]

Cortisol release response is similar to GH, whereby anaerobic metabolism acts as a potent stimulus.[80] Therefore, despite chronically high levels of cortisol reflecting adverse affects and progression towards overtraining, acute responses may be an essential part of the remodelling process, whereby the muscle must first be disrupted before it can adapt.[64] It is however, suggested that these acute training variables are varied to allow the adrenal gland to recover, (secrete less cortisol), and prevent overtraining. Continued stress causes delayed recovery, due to the over release of cortisol and its negative effects exerted through gluconeogenesis and immune system depression.[65]

Finally, the rise in GH and cortisol concentrations may contribute to the regulation of glucose and glycogen metabolism.[83] Therefore, in strength-endurance type protocols (low load, high repetitions), the low tension applied for an extended period of time may cause hormonal responses in response to the activation of the anaerobic metabolism and the need for restoration of energy substrates.[83] It should be noted however, that although bodybuilding type programmes evoke concurrent adaptations in both hormones, the magnitude of GH is greater than cortisol, which may compensate for the negative effects.[83]

Catecholamines

Upon stimulation from the sympathetic nervous system, the adrenal medulla and sympathetic neurons produce and release adrenaline, noradrenalin and dopamine. These catecholamine's stimulate the central motor system, enhance muscular enzyme activity, augment the secretion of other hormones (e.g. TST, GH, IGF), promote energy availability, act as peripheral vascular dilators (modulating blood pressure and the redistribution of blood), and ultimately facilitate the contractile characteristics of skeletal muscle.[52,63,88] Plasma catecholamine's are therefore significant mediators of force output and are consequently likely the first endocrine mechanism to respond to the psychophysiological stress associated with resistance training programmes.[26,65]

Anticipatory Response

French *et al*.,[26] reported a significant rise in catecholamine levels before the onset of high-intensity exercise in trained men. This is termed the "anticipatory" response, which occurs via sympathoadrenal activity and the magnitude of rise is determined by the anticipated intensity. This psychologically induced drive occurs at ≤15mins before the start of exercise and by ≤10mins is significantly higher than pre-exercise levels.[26] These rapid hormonal influences are thought to effect force expression through their significant effects on intracellular calcium shifts in muscle fibres or neurons,[91] and are therefore considered critical for optimal force production at the commencement of exercise.[26] This data highlights the significance of pre-exercise "psyching-up" interventions for the development of optimal force,[88] and further highlights the importance of differentiating between training maximums and competition maximums during exercise sessions.[97] If the latter was used during training programmes, whereby competition arousal can increase the athlete's 1RM by up to 12%,[97] the resultant may be unsuccessful lifts and therefore reduce the effectiveness of training. Finally, French *et al*.,[26] also reported elevated catecholamine concentrations 5 mins into the recovery period, and suggested that this was a means to facilitate the homeostatic process.

Neurohormone Effects on Power and Power-Endurance

Repetitive muscular efforts result in decreased membrane excitability, through a disruption of ionic balance across the muscle membrane (sarcolemma).[5] Catecholamine's are able to attenuate this by stimulating the Na^+-K^+ pump, which restores ionic balance across the sarcolemma, enhancing actin-myosin interactions and muscle force generation and maintenance.[13] Moreover, this quality may be trainable. McKenna *et al*., (73) reported that sprint training increases human skeletal muscle Na^+-K^+ - ATPase concentration and improves K^+ regulation. Therefore, concentrations of catecholamine's are essential for preconditioning the neuromuscular system to enable maximal performance,[91] and exhibit an essential role for achieving the maximal rate of glycogenolysis.[90] This can be evidenced by French *et al*.,[26] who report that subjects who are better able to maintain force production throughout an exercise protocol tend to have the highest catecholamine concentrations. Significantly, heavy resistance training increases the ability of athletes to secret adrenaline during maximal exercises.[50]

Optimal Release Strategy

To optimise the secretion of catecholamine's, it is recommended that programmes use high volume, large muscle groups and short rest periods (similar to TST and GH programmes).[65] In addition, plasma TST concentrations are correlated with catecholamine concentrations,[26] and can be evidenced by data suggesting that catecholamine's may enhance blood flow to the testis and enhance secretion.[27,62] Catecholamines and TST may therefore stimulate each other, ensuring optimal force expression. French *et al*.,[26] and Podolin *et al*.,[76] also reported that exercise induced changes in catecholamine concentrations were highly correlated with circulating lactate and glucose

Table 2. Example hypertrophy programme based on 3 sessions per week.

Muscle Groups	Unless otherwise stated, all exercises should be performed at 3 sets of 10 reps, at or near 10RM loads, with ≤60s rest between sets and exercises. Down sets, slow continuous training, negatives and increased eccentric loading may also be utilised but the programme should not progress beyond one hour in duration.					
Chest, Shoulders and Triceps	Snatch or derivative (3 sets x 5 reps, with >2mins rest, @ variable loads)	Bench Press (flat bench; progress to dumbbell [DB])	Bench Press (incline or decline bench; progress to DB)	DB Fly (flat, incline or decline bench)	Shoulder Press (progress from barbell to DB)	Triceps pull down or any variation (not relevant to athlete populations)
Back and biceps	Clean and Jerk or derivative (format as per snatch)	Lat-pull down (progress to chin-ups)	Diagonal pull-down20 sets of 1RM SQ 10 x 10 at 70% 1RM	Bent-over row, inverse row or one arm row	Shoulder shrugs	Bicep curls or any variation (not relevant to athlete populations)
Legs	8 ex, 3-5 x 5RM vs. 10RM with 1 and 3 min rest		Split squat or BB step-ups (alternate between the two)	Stiff leg deadlift	Nordic curls	Calf raises

Table 3. Example strength session emphasising neural development with no hypertrophy.

The following exercises are 4 sets of 4 reps, performed at 4-5RM loads, with >3 minutes rest between sets and exercises. Athletes can progress from one exercise to the other following the completion of each exercise or at the completion of each set (similar to a circuit format).				
Back squat	Bench press	Chins	Stiff leg deadlift	Bent-over row or seated row

during exercise (indicative of enhanced glycogenolysis). It may be concluded therefore, that catecholamines are likely to respond favourably to a hypoxic environment, which again supports the use of the valsalva manoeuvre for maximal force generation and maximal increases in rate of force development. In addition, and in alignment with the general adaptation theory, it is suggested that these acute training variables are varied to allow the adrenal gland to recover and prevent overtraining.

Summary and Conclusion

For muscle hypertrophy, training programmes should utilise 3 sets of 10 repetitions at, or near, 10RM loads, with short rest periods of no longer than 1 minute. This also appears to optimally release all aforementioned hormones (with the exception of IGF). To increase the number of fibres that undergo the remodelling process and therefore optimally increase total cross-sectional area, each session should target specific muscle groups ensuring that each group works through its full range of motion and should be performed to, or near to, failure. These suggestions, along with the valsalva manoeuvre and relatively inactive rest periods, will assist in the accumulation and maintenance of lactate and H^+, thus further enhancing hormonal concentrations.

To optimally evoke TST concentrations and further enhance hypertrophy within the smaller musculature, that may otherwise only be under the influence of GH and IGF, sessions should be initiated with large muscle group exercises. These could be squats or deadlifts for example, however these may negatively affect subsequent sessions that target the lower-body musculature. Alternatively, any of the Olympic lifts or their derivatives could be used and therefore assist the transition into the subsequent periodisation phases such as strength and power. It is worth noting, however, that high repetitions, with no inter-repetition rest period, result in a noticeable decrease in power output during each set,[36,66] and are therefore sub-optimal if the goal of training is to maximise power or velocity.

To further enhance the hypertrophic effect of resistance training programmes, exercises may utilise increased eccentric loading and negative repetitions and/or the addition of down sets. These will increase IGF and GH concentrations respectively. The athlete should also be mentally ready to train, thereby increasing concentrations of catecholamines, as they may exert their positive effects on TST release and nervous stimulation. A 'psyching-up' intervention may be achieved by motivation provided by the S&C coach or training partner. Finally, training programmes should be limited to one hour in duration, to prevent the negative effects of cortisol as it progressively becomes the dominant hormone. Split sessions are advocated when sessions may be in excess of this and these should be separated by at least 30 mins to restore TST levels.

Acute training variables can also be manipulated to bring about strength adaptations without concomitant increases in hypertrophy. This is especially important for athletes that compete in weight regulated sports such as martial arts. For this purpose, S&C coaches should increase the training load and the length of rest periods. In addition, body parts should be rotated to further ensure the dissipation of lactate and H^+ and reduce the release of anabolic hormones (namely TST and GH). Moreover, such adjustments will enable a higher intensity (%1RM) to be utilised and maintained throughout the session to further facilitate neural adaptations and strength gains.

The above tables illustrate a hypertrophy programme based on 3 sessions per week (Table 2) and an example strength session (Table 3).

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