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Acute and Chronic Testosterone Responses to Physical Exercise and Training

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1. Introduction

High-intensity physical training is a powerful stimulus to acute increases in blood steroid hormone levels (Ahtiainen et al., 2005; Cadore et al., 2008a, 2008b, 2009a; Häkkinen & Pakarinen, 1994a, 1995; Staron et al., 1994). Moreover, strength training (ST) has been shown to stimulate greater increases in testosterone levels when compared to aerobic training (Copeland, et al., 2002; Tremblay et al., 2003), which can be explained by the powerful influence of the anaerobic glycolytic pathway in stimulating acute hormonal increases in response to exercise (Kraemer & Ratamess, 2005). This stimulus features control mechanisms independent from luteinising hormone (LH) stimulation (Fahrner & Hackney, 1998; Lu et al., 1997), and some factors related to the training session are directly associated with this response (Cadore et al., 2008c; Häkkinen et al., 1988; Häkkinen & Pakarinen, 1995, Kraemer et al., 1993; Smilios et al., 2003, 2006).

Despite the well-known acute hormonal response to physical exercise (Kraemer et al., 1990; Hansen et al., 2001; Cadore et al., 2009c), data on resting concentrations remain controversial. Some studies demonstrate increased resting testosterone levels following ST (Ahtiainen et al., 2003; Häkkinen et al., 1998; Izquierdo et al., 2006; Kraemer et al., 1993; Kraemer et al., 1999; Marx et al., 2001; Staron et al., 1994), leading authors to suggest this type of training as a form of intervention for maintaining testosterone levels during ageing (Kraemer et al., 1999). However, increases in resting testosterone levels were not observed in middle-aged (Cadore et al. 2008a) or elderly people (Häkkinen et al., 2000, 2001a; Kraemer et al., 1999). Chronic adaptations to ST apparently occur at the level of cellular androgen receptors (ARs), given that ARs present on muscle cells seem to increase in number in response to this type of training (Inoue et al., 1994; Willoughby & Taylor, 2004), and this adaptation may result in improved hormone-receptor interaction (Bamman et al., 2001; Willoughby & Taylor, 2004).

Conversely, reductions in testosterone levels associated with increases in cortisol levels have been observed in response to aerobic training in athletes subjected to high-volume training (Maïmoun et al., 2003). These alterations may be associated with the overtraining process and consequent suppression of the hypothalamic-pituitary-gonadal and adrenocortical axis (Bell et al., 2001; Hu et al., 1999; Kraemer et al., 1995). Nevertheless, reductions in testosterone levels may occur without overtraining. This alteration may be transient, reflecting the variation in volume and training intensity, and it may be explained by changes in plasma volume (Hu et al., 1999; Kraemer & Ratamess, 2005; Maïmoun et al., 2003).

It has been suggested that the neuromuscular adaptations observed during ST (Häkkinen et al., 2000, 2001b; Tsolakiis et al., 2004) are partly mediated by acute responses to circulating testosterone levels resulting from the training session (Kraemer & Ratamess, 2005) as well as modifications in the cellular receptors present on muscle cells (Ahtiainen et al., 2011; Kadi et al., 2000). Besides the known effects exerted by these hormones on muscle metabolism (Bhasin et al., 2001), the magnitude of increase in muscular strength in individuals subjected to strength training has been associated with testosterone concentration and other hormone-related parameters (i.e., the testosterone:cortisol ratio and testosterone:sex hormone-binding globulin (SHBG)) (Cadore et al., 2010; Häkkinen et al., 1988, 2000; Häkkinen & Pakarinen, 1993a, 1993b; Izquierdo et al., 2001). The figure 1 shows a Schematic diagram of the mechanism of training adaptations: anabolic process as adaptation to strength training and chronic catabolic process resulting from excessive volume of both strength and aerobic training.
Due to the possible importance of acute hormone responses, as well as chronic adaptation of androgen receptors due to strength training, it may be important to determine which aspects of training influence these responses to establish an optimum anabolic environment during a training session or period. Therefore, the objective of this chapter is to review existing data on the influence of testosterone levels on physical training and to determine which factors related to the training session are associated with the acute and chronic hormone responses the endocrine system to exercise.

2. Testosterone and strength trainability

Testosterone is a powerful stimulator of protein synthesis, specifically in the context of muscle metabolism (Griggs et al., 1989). Its effects are exerted through the interaction between the hormone and its specific receptor located on the muscle cell (Bamman et al., 2001). The main mechanism through which testosterone induces protein synthesis is the activation and induction of the proliferation of satellite cells, which subsequently incorporate into muscle fibres, resulting in an increase in myonuclear number (Kadi et al., 2000). Moreover, this hormone is capable of influencing strength production by stimulating the transition of type II fibres to a more glycolytic profile (Ramos et al., 1998), increasing the secretion of insulin-like growth factor I (IGF-I), mediated by its influence on the amplitude of growth hormone (GH) pulses (Bross et al., 1999) as well as its influence on the production of neurotransmitters that are important for muscle contraction (Kraemer et al., 1999).
Several studies demonstrate that among individuals subjected to the same volume and intensity of ST, those presenting higher testosterone levels achieve greater muscular strength and/or power following training (Ahtiainen et al., 2003; Cadore et al., 2010; Häkkinen et al., 1988; Häkkinen & Pakarinen, 1993a). This suggests that the trainability of individuals is related to testosterone and hormonal parameters associated with this hormone, such as the testosterone:sex hormone-binding globulin (SHBG) ratio and the testosterone:cortisol ratio, as demonstrated by results obtained in published studies (Ahtiainen et al., 2003; Cadore et al., 2010; Häkkinen et al., 1988; Häkkinen & Pakarinen, 1993a). Furthermore, in transversal studies that investigated middle-aged and elderly individuals, strength production was correlated to serum testosterone levels (Häkkinen & Pakarinen, 1993a; Cadore et al., 2008a).
Häkkinen et al. (1988) investigated male weightlifting athletes (22.3 ± 2.1 years) and observed a correlation between testosterone:cortisol and testosterone:SHBG ratios and the variations in maximum strength and rate of force development (RFD), respectively, as a result of ST (r =

0.77 and r = 0.84, P < 0.05). In an investigation of the relationship between the endocrine system and strength production, Häkkinen & Pakarinen (1993a) observed a correlation between maximum strength and testosterone levels as well as testosterone:SHBG ratio (r = 0.62 and 0.68, respectively, P < 0.01). A study by Izquierdo et al. (2001) revealed that individuals with greater increases in isometric strength following ST also showed higher total (r = 0.78, P < 0.01) and free (r = 0.71, P < 0.05) testosterone levels. The same trend was observed in data obtained by Ahtiainen et al. (2003) when evaluating highly trained men subjected to a 21-week ST program, where positive correlations were observed between the changes in isometric strength and total testosterone (r = 0.84, P < 0.01), the testosterone:cortisol ratio (r = 0.88, P < 0.01) and the isometric strength development and free testosterone (pre-training values: r = 0.78, P < 0.05 and post-training values: r = 0.82, P < 0.05). A study conducted in our laboratory by Cadore et al. (2008a) showed significant correlations between testosterone:SHBG ratios and DHEA concentrations as well as strength production in bench press, leg press and squat exercises (r = 0.55 to 0.82, P < 0.05 to P < 0.001) in trained and untrained middle-aged men. In another study, Cadore et al. (2010) showed significant correlations between increases in the strength of knee extensors and average basal total testosterone levels throughout the training period (3 measurements in 12 weeks of training) (r = 0.94, P < 0.01) and the average total testosterone:cortisol ratio (r = 0.93, P < 0.01). Table 1 shows the results obtained from studies where correlations between hormonal parameters and variables related to muscle strength were identified. One aspect that must be emphasised is that other structural factors, such as pennation angle and fibre type composition, may interfere with strength production (Ramos et al., 1998), just as the volume and intensity of ST largely influence the increase in strength resulting from training (Marx et al., 2001).

Author	Strength performance vs. sex hormonal parameters
Ahtianinen *et al*., 2003	↑ MVC after ST with TT and TT:COR ratio (r=0.84 and 0.88, P<0.01), and MVC after ST with FT post training (r=0.82, p<0.05)
Cadore *et al*., 2008a	Squat 1 RM values with TT:SHBG and DHEA before (r=0.71 and 0.65, respectively) and after ST bout (r=0.76 and 0.82, respectively) (p<0.01 to 0.05).
Cadore et al., 2010	↑ knee extension 1 RM values after ST with TT and TT:COR ratio (r=0.94 and 0.93, respectively, p<0.01)
Häkkinen & Pakarinen, 1993a	TT and TT:SHBG with MVC and RFD (r = 0.66 to 0.69, p<0.01)
Häkkinen & Pakarinen, 1993b	↑ MVC after ST with TT and TT:COR (r=0.57 and 0.61, respectively, p<0.05)
Häkkinen *et al*., 1988	Annual average of TT:COR ratio and MVC (r=0.77, p<0.05); and, annual average of TT:SHBG ratio and ↑ RFD after ST (r=0.84, p<0.05)
Häkkinen *et al*., 2000	↑ 1 RM values after ST with FT and TT (r=0.55 and 0.43, respectively, p<0.05)
Izquierdo *et al*., 2001	↑ MVC after ST with TT and FT (r = 0.78 and 0.71, respectively, p<0.01).

Table 1. Relatioship between sex hormonal parameters and strength performance. TT: total testosterone; FT: free testosterone; COR: cortisol; DHEA, dehidroepiandrosterone; SHBG: sex hormone binding globuline; ↑: increases; MVC: maximal voluntary contraction (maximal isometric strength); and, 1 RM: one-maximum repetition (maximal dynamic strength); ST: strength training.

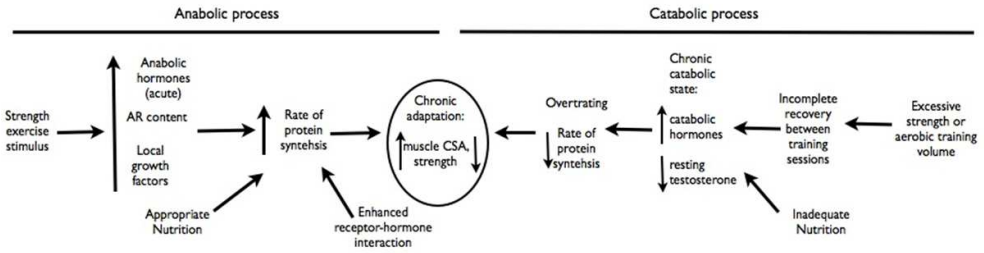

Fig. 1. Schematic diagram of the mechanism of training adaptations: anabolic process as adaptation to strength training and chronic catabolic process resulting from excessive volume of both strength and aerobic training. AR, androgen receptor; CSA, cross-sectional area.

3. Acute testosterone responses to physical exercise

Acute testosterone responses to ST exhibit plasticity, and their pattern depends on factors related to the training session, such as volume, intensity, method (i.e., single or multiple sets) (Cadore et al., 2009a; Häkkinen & Pakarinen, 1993a; McCaulley et al., 2009), type of muscle contraction (Durand et al., 2003; Kraemer et al., 2006) and muscle mass involved (Häkkinen et al., 1998), as well as factors such as age (Kraemer et al., 1999; Cadore et al., 2009a) and the individual's level of training (Ahtiainen et al., 2003; Cadore et al., 2008a, 2009; Kraemer et al., 1999). The response of testosterone levels to ST may expose the skeletal musculature to an elevated peripheral hormonal concentration, which may improve the interaction between the hormone and its cellular receptors (Hoffman et al., 2003, Willoughby & Taylor, 2004). Regarding aerobic training, even though the importance of the anabolic hormone response remains unclear, it seems that testosterone is more responsive to higher intensity exercises (Enea et al., 2009) and a longer duration of exercises (Harris et al., 1989; Trembley et al., 2005).

3.1 Possibles physiological mechanisms for the stimulus

The response of testosterone levels to exercise sessions may reflect certain regulatory mechanisms in addition to the processes that regulate the secretion of this hormone at rest (Fahrner and Hackney, 1998; Lu et al., 1997). A study conducted by Lu et al. (1997) demonstrated that exercise-induced increase in testosterone levels in male rats correlated with an increase in blood lactate levels. Following this observation, the authors proceeded to conduct an *in vitro* study where lactate was infused into the rats' testes, and a dose-dependent increase in testosterone was observed. Methods of ST aimed at achieving muscle hypertrophy or resistance have been shown to cause high lactate production (McCaulley et al., 2009; Smilios et al., 2003), suggesting a strong relationship between the mechanism of testosterone increase and lactate stimulation in the testes (Lu et al., 1997).

Other mechanisms may be responsible for the exercise-induced increase in testosterone levels, among which are increased sympathetic activity in response to exercise (Fahrner and Hackney, 1998) and blood flow and vasodilation related to the release of nitric oxide, which increases hormone secretion (Meskaitis et al., 1997). Even though various studies have used different strength training exercise protocols, Kraemer et al. (1999) and Ahtiainen et al.

(2003) suggest that these mechanisms may also be mediators of testosterone increase in response to this type of training.

3.2 The influence of strength training variables on testosterone responses

It may be stated that the hormonal response to exercise is connected to certain characteristics inherent to the training session, such as the number of sets and repetitions, the relative intensity (percentage of 1 maximum repetition - 1 RM) and time intervals (McCaulley et al., 2009). The amount of work done during ST may be a determining factor in the acute hormone response, leading to an optimum combination of anabolic and catabolic hormone stimulation. This, in turn, may result in a more favourable environment for neuromuscular adaptations to training, resulting in increases in muscle strength and mass (Smilios et al., 2003).

The preponderant influence of volume on the hormonal response to different training methods was observed by Häkkinen and Pakarinen (1993b), who compared the hormonal response to a session involving 20 sets of 1 RM with a session composed by 10 sets of 10 repetitions at 70% of 1 RM; both ST bout were conducted with 3-minute intervals between sets. The authors observed a significant increase ($P<0.05$) in total (22%) and free (23%) testosterone in response to the high-volume training and no increase in the training session that included a higher load and fewer repetitions. Smilios et al. (2003) showed that the hormone response observed in young men increased as the number of sets in each session increased, approaching maximum strength, muscle hypertrophy and resistance. These authors observed that when the number of sets was increased from 4 to 6, the anabolic hormone levels stabilised, while cortisol levels continued to increase. Their results suggest that modifying the volume of a ST session causes alterations in the balance between anabolic and catabolic hormones. When considering different training methods, McCaulley et al. (2009) observed a higher total testosterone response to muscle hypertrophy protocols when compared to protocols that aimed at developing maximum strength and power, despite equalisation of the total work load for each session (load x sets x repetitions). When investigating the influence of the total muscle mass involved in training, Häkkinen et al. (1998b) demonstrated a greater testosterone response in young and elderly men using protocols involving simultaneous use of the lower and upper limbs (27%). However, an increase in hormone levels was also observed for protocols that involved the upper and lower limbs separately ($P<0.01$), indicating that the greater the amount of muscle mass involved, the greater the acute total testosterone response.

Regarding the influence of resting intervals on the acute hormone response, the smaller the interval between sets, the greater the stimulus (Kraemer et al., 1990). Nevertheless, when the sets are performed with maximum repetitions, the interval appears to have no influence within a certain intensity range, as demonstrated by Ahtiainen et al. (2005), who showed that there was no difference in acute hormone response between two protocols of 10 RM with 2- and 5-minute intervals. Notwithstanding, sessions with moderate to high intensity that involve multiple sets and short time intervals, during which energy is derived mainly from glycolytic lactate metabolism, appear to be the greatest stimulus for the steroid hormone response to ST.

Little is known about the influence of combined aerobic and strength training (i.e., concurrent training) on the acute testosterone response. A study by Goto et al. (2005) demonstrated that the GH response to strength training was found to be suppressed by prior aerobic training. However, no differences were observed in strength training-induced

testosterone concentrations, with or without prior aerobic training, possibly due to the low-volume protocol used in the study. However, unpublished data from our laboratory show that the manipulation of the order of modalities (strength and aerobic) may, in fact, influence the testosterone response produced by concurrent training sessions. A significant increase was observed in both protocols following the first modality, though levels remained high only at the end of the training session when the protocol involved aerobic training followed by strength training; the same response was not observed when strength training was performed before aerobic training. When comparing acute testosterone responses to strength and aerobic training, some studies show that strength training appears to stimulate greater increases in testosterone levels when compared to aerobic training (Copeland et al., 2002, Tremblay et al., 2003). In a previous study (Cadore et al., 2009a), we showed significantly higher salivary free testosterone responses to water-based resistance exercise compared with water-based aerobic exercise in both young and elderly healthy men. These results can be explained by the powerful influence of the anaerobic glycolytic pathway on acute hormonal increases in response to exercise (Kraemer & Ratamess, 2005).

3.3 The Influence of age and training status

The profile of the population subjected to training sessions is one of the factors that influence hormone response to ST. Studies aimed at investigating this response in different age groups have mainly observed lower responses in elderly individuals as demonstrated by Kraemer et al. (1999). For instance, when comparing acute total and free testosterone responses in groups of men aged 30 ± 5 and 62 ± 3 years, Kraemer et al. (1999) demonstrated that even though both groups showed an increase in free testosterone, a lower testosterone response was observed in the elderly group. According to these authors, the reduced response is associated with andropause, which is characterised by a smaller number and decreased secretory capacity of Leydig cells due to ageing. Similar results were observed by Cadore et al. (2009a), where elderly individuals showed significantly lower free-testosterone responses to water-based resistance exercise than young men.

The individual's training status may also influence the response of the endocrine system to ST (Cadore et al., 2008a, 2009b; Kraemer et al., 1992), given that different anabolic responses may occur before and after a period of ST. Kraemer et al. (1992) investigated weightlifting male athletes aged 17 ± 2 years and observed that individuals with more than 2 years of training presented higher acute testosterone responses. A study by Cadore et al. (2008a) observed different patterns of hormone response in trained and untrained middle-aged men (40 ± 4 years) after a ST protocol. Significant increases in free testosterone (27%) were observed in members of the trained group, whereas significant increases in both total (28%) and free (22%) testosterone as well as DHEA (127%) were observed in the untrained group ($P<0.05$). These results may suggest a higher capacity for testosterone dissociation from carrier proteins, increasing the bioactivity of the hormone without the need for an increase in production. A study by Kraemer et al. (1999) showed higher free testosterone responses in young and elderly individuals following 10 weeks of periodic strength training. Some results suggest the existence of specific responses to certain types of training, as demonstrated in a study developed by Tremblay et al. (2003). The study showed greater increases in anabolic hormone levels in response to strength training in strength-trained subjects when compared to aerobically trained individuals, whereas aerobically trained subjects produced higher hormonal responses to aerobic exercise when compared to strength-trained individuals.

However, the influence of the training status on the testosterone response to ST was not found in many studies. When submitting untrained and previously strength-trained individuals to a 21-week strength training protocol, Ahtiainen et al. (2003) observed similar alterations in total and free testosterone for both groups before and after training. This discrepancy may be due to factors such as sample profile, exercise protocol or potential changes in plasma volume. Moreover, testosterone responses to this type of exercise may be influenced by the relationship of the hormone with its cellular receptors, given that this interaction appears to be greater in trained individuals and that they thus may not require the same magnitude of an acute response in order to obtain an optimum hormone-receptor interaction (Ahtiainen et al., 2011; Willoughby & Taylor, 2004).

4. Chronic endocrine adaptations to physical training

4.1 Basal testosterone adaptations induced by strength training

While some studies demonstrate an increase in the resting levels of testosterone as an adjustment to ST (Izquierdo et al., 2006; Kraemer et al., 1995; Nicklas et al., 1995; Raastad et al., 2003; Tsolakis et al., 2004), other studies have observed no significant differences in this parameter (Ahtiainen et al., 2003; Hansen et al., 2001; Hickson et al., 1994). So far, available data indicate that only young individuals are capable of altering their resting hormone concentrations (Häkkinen et al., 1988; Staron et al., 1994; Tsolakis et al., 2004), whereas middle-aged and elderly individuals show no significant changes in such parameters (Häkkinen & Pakarinen, 1994; Häkkinen et al., 2000; 2001a; Izquierdo et al., 2006; Ryan et al., 1994). Increases in resting levels of testosterone seem to occur during periods of high-volume (Kramer et al., 1995; Marx et al., 2001) and high-intensity training (Staron et al., 1994; Kraemer et al., 1998; Raastad et al., 2003). These changes may occur in men (Häkkinen et al., 1988) and women (Marx et al., 2001) in response to long (Häkkinen et al., 1988; Marx et al., 2001) or short training periods (Staron et al., 1994; Kraemer et al., 1998).

The influence of training volume on chronic adaptations of basal testosterone was described in a study by Marx et al. (2001), which 34 women (22 ± 5 years) were evaluated before and after performing a 24-week ST protocol. In this study, resting levels of testosterone were measured in order to compare groups of ST performing single vs. multiple sets. The results showed an increase in testosterone in both training groups, and the first adaptations took place after 12 weeks of training. However, after 24 weeks of training, only the multiple sets group had further increases in their resting testosterone levels, which was higher in this point than after 12 weeks, and higher than the single set ST group. Even though the performance of high-volume ST sessions may induce higher acute increases in catabolic hormones (Smilios et al., 2003), the study developed by Marx et al. (2001) demonstrated that high-volume and high-intensity ST may lead to higher chronic increases in anabolic hormones compared to low-volume ST. This may contribute to the greater strength production observed in individuals who trained with multiple sets when compared to those who trained with simple sets (Kemmler et al., 2004).

The Table 2 shows subjects characteristics, training protocol and results from some of the studies that have investigated resting hormone adaptations to ST. Nevertheless, modifications in resting concentrations appear to be transient, resulting from the increase or decrease in intensity and, mainly, in volume (Ahtiainen et al., 2003). However, the precise role of resting testosterone concentrations in neuromuscular adaptation to training is yet to be determined.

Author	Subjects	Training protocol	Results
Ahtiainen *et al.*, 2003	Young previously trained and untrained men	21 weeks, 2 times/week, 8-10 RM	↑ FT in trained after 14 weeks; no modifications after 21 weeks
Cadore *et al.*, 2008a	Middle-aged long-term trained and untrained men	10 ± 5 years of strength training, 4 times a week, 8-12 RM	No difference at rest, different responses to exercise
Häkkinen *et al.*, 2001a	Elderly men	21 weeks, 40-80% of 1 RM	No modifications
Häkkinen *et al.*, 1988	Elite powerlifters young men	Two years, 5 times/week	↑ TT
Staron et al., 1994	Young men and women	8 weeks, 3 times/week, 6-12 RM	↑ TT in men
Häkkinen & Pakarinen, 1994	Middle-aged and elderly men and women	12 weeks, 3 times/week, 40-80% of 1 RM	No modifications
Izquierdo *et al., 2001*	Young men and women	16 weeks, 3 times/week, 50-80% of 1 RM	No modifications
Kraemer *et al.*, 1995	Military young men	12 weeks, 4 times/week, 3-10 RM, strength vs. concurrent training	↑ TT after concurrent training
Kraemer *et al.*, 1999	Young and elderly men	10 weeks, 3 times/week, 3-15 RM	↑ FT jovens
Marx *et al.*, 2001	Young women	24 weeks, 3-15 RM, single vs. 3 sets per exercise	↑ TT in both groups after 12 weeks, higher after 3 sets training group
Nicklas *et al.*, 1995	Middle-aged and elderly men	16 weeks, 3 times/week, 5-15 RM	No modifications
Ryan *et al.*, 1994	Elderly men	16 weeks, 3 times/week, 5-15 RM	No modifications

Table 2. Testosterone modifications at rest after strength training: TT: total testosterone; FT: free testosterone; ↑: increases; RM: maximal repetitions.

4.2 Changes in circulating testosterone in response to aerobic training

With regards to aerobic training, studies have demonstrated that endurance athletes have lower testosterone levels when compared to sedentary individuals (Strüder et al., 1998; Maïmoun et al., 2003). However, Strüder et al. (1998) showed that although testosterone levels were indeed lower in elderly male runners compared to age-matched sedentary subjects, the same was not true for previously sedentary subjects who performed aerobic training for 20 weeks 3 times per week with an intensity of 50 to 65% of aerobic power. A previous study conducted in our laboratory (Cadore et al., 2010) demonstrated a significant reduction in free testosterone in elderly men following 12 weeks of aerobic training on a

cycle ergometer 3 times per week with intensity varying between 55 and 85% of aerobic power (9.7 ± 2.8 vs. 7.9 ± 3.0 pg/mL, $P < 0.01$). Possible discrepancies between the results of different studies may reflect the different intensities and volumes used, given that the intensity used by Strüder et al. (1999), for instance, was lower than the intensity used in our study (Cadore et al., 2010). However, in animal models, Hu et al. (1999) observed a significant reduction in testosterone levels in rats submitted to continuous swimming for 3 weeks. Levels were restored to normal following 6 weeks of training, suggesting an adjustment to training on LH secretion in the endocrine system that was associated with negative feedback. Even though testosterone reduction during aerobic training has not been clearly demonstrated, it is possible that a certain amount of time is necessary for the endocrine system to adapt to the volume and intensity of training when these factors exceed a certain stimulus threshold (Calbet et al., 1993; Kraemer & Ratamess, 2005; Maïmoun et al., 2003).

Though high-volume physical training may result in the suppression of testosterone via direct inhibition due to the effect of cortisol on the testes (Brownlee et al., 2005), this does not completely explain the occurrence of testosterone reduction with aerobic training, given that increases in basal cortisol levels and the consequent testicular suppression are most commonly related to overtraining. In fact, testosterone levels have been shown to be reduced in endurance athletes with no alterations in cortisol levels (Maïmoun et al., 2003) as well as in non-athletes subjected to aerobic training (Cadore et al. 2010). Furthermore, other mechanisms, such as hypervolemia, increased utilisation of the hormone by muscle tissue and increased hepatic degradation of the hormone, may be responsible for the decrease in testosterone levels that result from endurance training (Izquierdo et al., 2004).

4.3 Changes in muscle cell androgen receptors

Evidence shows that cell adjustments may be key factors for training-induced hypertrophy (Ahtiainen et al., 2011; Deschenes et al., 1994; Inoue et al., 1993, 1994; Kadi et al., 2000; Bamman et al., 2001; Willoughby & Taylor, 2004; Ratamess et al., 2005). Some of these adjustments correspond to an increase in the number of androgen receptors (AR) in the muscle, and they are apparently dependent on the pattern of acute testosterone response to exercise (Willoughby & Taylor, 2004). A greater number of ARs and an increased sensitivity of these receptors to the hormone may improve the trophic effects of testosterone on target cells (Kadi et al., 2000). Inoue et al. (1993) subjected male rats to training using electrical stimulation and demonstrated that muscle hypertrophy occurred in parallel with a significant increase in cellular ARs. In a different study, Inoue et al. (1994) observed that the suppression of androgen receptors by receptor antagonists reduced the increase in muscle mass obtained with electrical stimulation.

Kadi et al. (2000) measured the number of ARs per area of muscle fibre in the superior trapezius and the *vastus lateralis* muscle of high-performance weightlifting men. The sample was composed of trained individuals with (31 ± 3 years) and without (28 ± 8 years) the use of exogenous anabolic steroids as well as untrained individuals (23 ± 3 years). Results showed higher numbers of ARs per area of muscle fibre in the superior trapezius of individuals from both trained groups when compared with untrained individuals. Moreover, individuals using exogenous anabolic steroids presented higher values than those that were only training ($P<0.05$). Surprisingly, these differences were observed only in the superior trapezius. The proportion between the different types of muscle fibres present in each of the evaluated muscle groups (i.e., type I and type II) may have influenced the different behaviours of the

androgen receptors in response to training. In fact, while studying rats subjected to strength training, Deschenes et al. (1994) were only able to observe an increase in the number of ARs in muscles with a predominance of fast glycolytic fibres, whereas muscles with a predominance of slow oxidative fibres showed decreased numbers of ARs.

Willoughby & Taylor (2004) conducted a study where 18 young men were subjected to 3 sessions of ST with 3 sets of 8 to 10 RM. Results showed a significant increase in protein synthesis, AR expression and AR messenger RNA following the training sessions, where values increased up to 202% 48 hours after the third session. Furthermore, the study revealed a correlation between testosterone increase (significant following all sessions) and an increase in the number of receptors ($r = 0.89$, $P < 0.05$). These results suggest that the hormone-receptor complex constitutes an important element in the mechanism responsible for mediating the adjustments to strength training, such as an increase in muscle strength and exercise-induced hypertrophy (Inoue et al., 1993; Kadi et al., 2000; Willoughby & Taylor 2004).

5. Conclusion

As noted in the studies presented in this chapter, there is a connection between the trainability of individuals subjected to ST and their levels of circulating testosterone. However, factors related to the training sessions and population profile seem to influence acute and chronic hormone responses, which result in a plasticity in the pattern of testosterone response to physical exercise, particularly strength training. Among the types of strength training sessions, high-volume protocols with moderate to high intensity (70-85% 1 RM), which are typically used to achieve muscle hypertrophy and predominantly rely on the glycolytic lactate metabolic pathway, appear to stimulate greater responses. Moreover, the increase in the number of ARs appears to have a key role in muscle hypertrophy observed with ST. However, determining which factors might be related to the hormone response to ST may be of great importance for the prescription of a training session and determination of the optimum period that will optimise the anabolic environment determined by testosterone and thus maximise the neuromuscular adaptations resulting from strength training.

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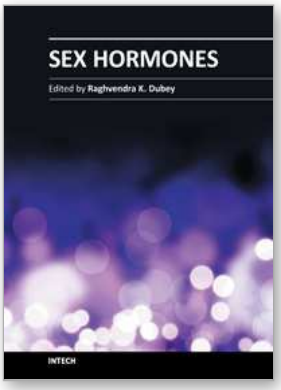

Sex Hormones

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Sex Hormones not only regulate reproductive function, but they also play a prominent role in the biology and physiology of several organs/tissues and in the pathophysiology of several diseases. During the last two decades, the information on the mechanisms of action of sex hormones, such as estrogens and androgens, has rapidly evolved from the conventional nuclear receptor dependent mechanisms to include additional non-nuclear, non-genomic and receptor-independent mechanisms. This highlights the need to update the current knowledge on sex hormones and their mode of action. Increasing evidence that exogenous/epigenetic factors can influence sex hormone production and action highlights the need to update our knowledge on the mechanisms involved. This book provides a systematic and updated overview of the male/female sex-hormones and their impact in the biology and physiology of various organs. Additionally, the book discusses their positive and negative association with the pathophysiology of various diseases (e.g. osteoporosis, cardiovascular-disease, hypogonadism, reproduction, cancer) and their therapeutic potential.

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