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Endogenous Anabolic Hormone Responses to Endurance Versus Resistance Exercise and Training in Women

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Abstract

Research in exercise endocrinology has flourished over the past few decades. In general, research examining short- and long-term hormone responses to endurance exercise preceded studies on resistance exercise, and research on women lagged behind research on men. Sufficient data are now available to allow a comparison of endogenous anabolic hormone responses to endurance versus resistance exercise and training in women. Circulating levels of testosterone, dehydroepiandrosterone, dehydroepiandrosterone sulphate, estradiol, growth hormone and cortisol have been shown to increase in response to an acute bout of endurance exercise in women. However, only growth hormone, estradiol and cortisol have been reported to increase following resistance exercise. Hormone changes following training, either endurance or resistance, have been variable, probably because of differences in experimental design and major differences in the length, intensity and volume of training programmes. With the notable exception of growth hormone, the anabolic hormones reviewed here appear to decline with endurance training. Resistance training has little effect on resting hormone levels, except insulin-like growth factor-I, which has been shown to increase following a training programme. These hormone changes potentially have both metabolic and hypertrophic implications, and future research needs to focus on the biological significance of these adaptations.

Early research in exercise endocrinology focused on exercise responses in males, predominantly the response to endurance exercise. In the 1970s it became increasingly evident that female athletes were experiencing higher rates of menstrual cycle disturbances than their sedentary counterparts.[1,2] Endocrine disturbances to the anterior pituitary and ovary were believed to play a significant role in these menstrual irregularities. These concerns attracted the attention of scientists to examine the female endocrine response to exercise. Initial studies documented endocrine differences between amenorrhoeic and normal cycling athletes[3,4] and have been reviewed elsewhere.[2,5,6] Gender differences in basal hormone levels, hormone production and hormone metabolism have reinforced the importance of endocrine research on the female population. Although exercise-induced amenorrhoea is still a concern, other areas of interest have surfaced.

Aging has been associated with the decline of many anabolic hormones in both males and females, and these decreases have been associated with disease and disability such as osteoporosis[7] and postmenopausal complaints in women.[8] Attempts to delay the decline of these hormones have involved the use of costly, synthetic hormone replacement therapies. Currently, endocrinologists are investigating the use of exercise, as a possible method of increasing anabolic hormones naturally.

In recent years, exercise endocrinological research on women has started to accumulate. For the purpose of this review 'anabolic' will be defined as a hormone with a growth promoting action and we will focus specifically on five anabolic hormones that have been prominent in the exercise endocri-

nology literature to date. We will examine the research documenting the short- and long-term anabolic hormone responses to endurance versus resistance exercise in women and identify where further research is required.

1. Endogenous Anabolic Hormones

The primary anabolic hormones believed to be involved in muscle tissue growth and remodelling are testosterone, growth hormone and insulin-like growth factor-I (IGF-1).[9] Other anabolic hormones such as estradiol, dehydroepiandrosterone (DHEA; prasterone) and dehydroepiandrosterone sulphate (DHEAS) are also believed to have critical roles in the maintenance of bone density and overall health. Hormones are transported in the blood with a portion being bound to protein carriers and a portion that is unbound. The ratio of bound to unbound hormone as well as the specific protein carrier differs depending on the hormone. There is still controversy over whether the unbound or 'free' hormone is the portion that is available to the target tissues and therefore the biologically active portion of the hormone. If this is the case, then exercise-induced changes in the level of protein carriers may also play a significant role in anabolism.

The primary source of testosterone in women comes from the peripheral conversion of androstenedione and DHEA, with the remainder secreted from the ovaries and the adrenal glands.[10,11] The anabolic effects of testosterone are realised through an increase in protein synthesis and a decrease in the rate of protein catabolism in the muscle fibre.[12] This process involves testosterone binding with cytosol receptors in a target cell, resulting in an increased transcription of genes responsible for the synthesis of contractile proteins.[11,13,14]

DHEAS is the predominant adrenal steroid for both genders.[15] Peak levels of DHEA and DHEAS occur at age 19 to 20 years in women after which these levels decline.[15] The precise biological function of DHEA and its sulphate ester, DHEAS, remain unclear at present.[16] However, previous research indicates that optimal DHEA and DHEAS levels may be related to the preservation of bone mass,[17] protection against cardiovascular disease,[18-20] and the prevention of problems associated with menopause.[8] It is still unclear through what mechanism(s), if any, DHEA and DHEAS may influence anabolic actions at the muscular or skeletal level.

Estradiol is secreted primarily from the ovaries, and is produced to a lesser extent from extraglandular conversion in women.[21] Estradiol levels are important for the maintenance of bone density,[22] and low levels of estradiol appear to be associated with amenorrhoea[6] and osteoporosis.[7] Indirect effects on bone occur through the suppression of parathyroid hormone mediated bone resorption and/or through stimulating the synthesis of 1,25-dihydroxyvitamin D which assists in calcium absorption.[23] Eriksen et al.[24] speculated that estradiol may have direct actions on the bone as well and in fact there are two estrogen receptor subtypes found in human bone.[25]

Growth hormone is the most abundant hormone released from the anterior pituitary gland.[26] The secretion of this polypeptide hormone is episodic and is under hypothalamic control, stimulated by the release of growth hormone–releasing hormone and inhibited by the release of somatostatin.[27] The growth hormone axis is complex, involving binding proteins, neuropeptides and neurotransmitters. While an understanding of this axis is important for endocrine research, it is beyond the scope of this review. A comprehensive review of this hormone is examined elsewhere.[28]

Growth hormone has been reported to play a critical role in the growth and development of bone, connective, visceral, adipose and muscle tissue.[12] While some research suggests that growth hormone may exert its anabolic effect by binding directly to receptors on the muscle, other data indicate that the anabolic effects are indirect and carried out by somatomedins, commonly referred to as insulin-like growth factors (IGF).[9,12]

IGF are a group of polypeptides that are synthesised primarily in the liver and are released into the blood after stimulation of the liver by growth hormone.[14] Many of the actions of growth hormone are mediated by locally produced IGF-1 acting in an autocrine or paracrine manner.[29] Extrahepatic

sites of IGF-1 production include muscle, bone and adipose tissue.[29] Local production of IGF-1 is under the influence of different regulators dependent on the tissue type.[30] Age, gender, nutritional status and growth hormone all appear to affect levels of IGF-1.[30] Evidence that IGF-1 levels can stimulate both DNA and protein synthesis, lends support to the anabolic role of IGF-1.[31,32]

It was originally believed that the anabolic role of insulin was purely indirect, through the preservation of muscle fibres.[33] However, it is currently believed that insulin acts in a direct anabolic fashion by increasing the rate of amino acid uptake into the muscle and other tissues.[12] A detailed examination of the response of this hormone to exercise is not provided in the present review but has been examined elsewhere.[14]

When examining the anabolic hormone response to exercise, it is important to consider the main catabolic hormone that is involved in counteracting these hypertrophic hormones. Cortisol is a glucocorticoid produced from the adrenal cortex.[14] The catabolic effects of cortisol include the breakdown of cellular proteins, allowing amino acids to be used for gluconeogenesis.[12] An elevated resting cortisol level, which causes a decrease in the testosterone to cortisol ratio, has been previously used as an indicator of overtraining in athletes.[34]

2. Confounding Variables

Applied exercise endocrinology research on humans may be contaminated by many factors (table I). Difficulty in standardising all potential confounding variables likely contributes to the contradictory research findings often observed. While factors such as research design and method of specimen collection are important to any endocrine research, of particular relevance to this review are the effects of age and menstrual status. Comprehensive reviews on the impact of other potential confounding variables are available elsewhere.[35,36]

2.1 Age

It has been well documented that there are significant changes in endocrine function that occur throughout the lifespan. For example, DHEAS is the most abundant steroid hormone in the body, and levels of this hormone have been shown to be age-

Table I. Summary of potential confounding or interfering variables for exercise endocrinology research (from Tremblay and Chu,[36] with permission)

Potential confounding variable	Relative impact on hormone measurement
Participant profile	
Species	+
Gender	+
Age and maturation status	+++
Racial background	+
Pregnancy	+
Body composition	+
Mental health	+
Disease, surgery, medications	+++
Standardised conditions	
Temperature and humidity	+
Altitude and hypoxia	+
Substance use	+
Nutritional status	+
Hydration	+
Acid/base balance	+
Emotional stress	+
Sleep deprivation	+
Menstrual cycle	+
Previous activity	+++
Specimen collection	
Posture	+
Rhythmical variations	+++
Specimen collection	+
Specimen storage	+
Choice of specimen	+
Haemolysis	+
Experimental intrusion	+
Analytical procedure	
Assay type	+
Specificity and cross reactivity	+++
Exercise variables	
Exercise intensity	+++
Exercise duration	+++
Exercise volume	+
Exercise type	+
Initial training status	+
Overtraining	+
Length of training intervention	+

dependent in both males and females.[15] The circulating level of DHEAS increases during adrenarche and peaks around 15 to 20 years of age, after which it declines steadily through adulthood. Levels of other anabolic hormones such as testosterone and growth hormone have also been shown to decrease with increasing age.[37-40] Clearly the age range of a study population can have a significant impact on endocrine research in humans.

In addition to age-related changes in basal levels of anabolic hormones, age also influences the hormonal response to exercise. Children demonstrate a significant growth hormone response to short-term exercise; however, the peak growth hormone response increases as pubertal development increases.[41,42] There is very little information available on the impact of long-term exercise training on the endocrine physiology of children and adolescents. In young women, however, the activation of the gonadal axis and the timing of puberty can be delayed by exercise training.[43]

Hormonal responses to exercise are also modified as we progress into older adulthood. In general, it has been shown that increasing age diminishes or blunts the short-term hormone response to physical work. Malarkey et al.[44] reported a decreased hormonal response to a short-term bout of ultra-endurance exercise in older males compared with young males. Unfortunately, there are no data available to confirm this result in females. Similar results have been found with resistance exercise. The growth hormone response to resistance exercise is typically diminished in older women when compared with young individuals.[37,45,46] These findings highlight the importance of taking the age of a population sample into consideration when interpreting the anabolic hormone responses to both short- and long-term exercise.

2.2 Menstrual Status

To effectively examine exercise-induced hormone changes in women, it is important to control for the changes that occur throughout the development of the reproductive system. As discussed in the previous section, there can be significant differences in exercise responses before and after the onset of puberty, a time when sex steroid levels increase significantly. After puberty, there are cyclical changes in gonadotropins, estrogen and progesterone throughout the menstrual cycle. It seems intuitive that the changing levels of these hormones in circulation throughout the menstrual cycle would have a significant impact on endocrine responses to exercise, although this would depend on the hormone. Interestingly, while some research has shown a significant difference in exercise-induced hormone changes during different phases of the menstrual cycle,[47,48] others have demonstrated no significant phase effect.[49-51] However, these studies used very different exercise protocols, and the training status of the participants ranged from highly trained[51] to untrained,[48] which could influence the results.

The impact of menstrual phase may be influenced by other factors as well. For example, Bonen et al.[49] reported that growth hormone responses to exercise depend on an interaction between nutritional status and menstrual phase. In light of the conflicting results from these investigations, it seems prudent to control for phase of menstrual cycle, particularly when comparing different modes or intensities of exercise. It is also important to note when studying athletic female populations, that strenuous training has been associated with menstrual irregularities including oligomenorrhoea and amenorrhoea.[52] To properly control for phase of menstrual cycle individuals must be eumenorrhoeic.

Finally, as women approach the end of their reproductive years, there is a dramatic decline in the synthesis and secretion of estrogen, progesterone and androgens, known as the climacteric. During this time span there are many changes that are associated with the down-regulation of ovarian steroidogenesis and there can be significant interindividual variability in the pattern of these changes.[53] There is little research available that compares exercise-induced hormone changes before, during and after the menopause; however, the variability that can occur during this time could be a potential confounding variable to endocrine research in older females.

3. Short-Term Hormone Responses

The documentation of short-term hormonal responses to exercise is important to determine the immediate effect of exercise on the endocrine system. Short-term endocrine changes usually occur in an attempt to maintain homeostasis. The investigation of short-term responses may assist exercise physiologists in explaining the physiological adaptations that occur during long-term exercise training.

To the authors' knowledge only one study has attempted to compare the short-term anabolic hormone responses between an endurance and resistance session, in the same group of premenopausal women. Consitt et al.[54] reported that 40 minutes of cycling (75% of maximal heart rate) was capable of increasing serum levels of testosterone and estradiol, compared with a resting session. Although increases were observed in these hormones after a resistance-exercise session including three sets of eight exercises at 10 repetition maximum (RM) intensity, they did not reach statistical significance when compared with the resting session. Growth hormone levels after the endurance and resistance sessions were both increased over the resting session. However, these increases were not significantly different between the two types of exercise. Neither DHEA nor cortisol demonstrated significant exercise induced changes in this study.

The following sections provide a summary of previous literature examining the response of these hormones to each type of exercise separately.

3.1 Endurance Exercise

3.1.1 Testosterone

Several studies indicate that testosterone levels rise in women after a short-term bout of endurance exercise.[55-60] These acute increases appear to be short-term and have been reported to return to resting levels within hours of exercise cessation.[55-57] Increases in testosterone have been found to be linearly related to both exercise intensity[57] and duration.[56,60] In less controlled field studies, increases in testosterone have been observed in women after 10-mile and marathon runs.[55,56] While beneficial in supplying preliminary information, caution must be used when forming conclusions based on field studies. Studies utilising racing conditions[55,56] enter many uncontrolled variables into the study, including the exercise intensity, duration and anxiety. Anticipatory increases in this hormone before endurance exercise have been observed,[56,57] while others have found no such increases.[58]

The majority of early research documenting testosterone responses to endurance exercise focused on trained women, hoping to identify a cause for exercise-induced amenorrhoea. While short-term increases have been reported in both trained and untrained women,[57] studies comparing these two groups have resulted in inconsistent findings.[57,58] While both of these studies[57,58] involved exercise to exhaustion, the timing of the intensity increments differed greatly, and may account for the discrepancy between their findings. It should also be noted that Keizer et al.[58] utilised different exercise modes for the trained and untrained participants. Trained participants were tested on a treadmill test, while untrained individuals were tested on a cycling ergometer. The results from this study must be viewed with caution because of the lack of research on the anabolic response to exercise of differing muscular involvement and postural position.

As with other steroid hormones, increased hormone levels may also occur from decreased metabolic clearance rate (MCR) caused by the reduction in hepatic blood flow during endurance exercise.[61,62] Cadoux-Hudson et al.[62] determined that the increase in testosterone observed in males was attributed solely to a reduction in clearance rate, mainly caused by decreases in hepatic plasma flow. This has not been confirmed in females.

3.1.2 Dehydroepiandrosterone (DHEA) and Dehydroepiandrosterone Sulphate (DHEAS)

The impact of endurance exercise on DHEA and DHEAS levels in women has been relatively understudied. Those studies that have been conducted, indicate that both DHEA and DHEAS are elevated after a short-term bout of endurance exercise.[55,56,58,63-65] Unfortunately, as discussed previously, many of these studies utilised racing conditions,[55,56,64] and therefore, exercise intensity and duration were not con-

trolled. In addition, the competitive nature of these runs may produce anxiety in some participants and consequently have an impact on the adrenal secretion of DHEA and DHEAS.

In contrast to DHEA, DHEAS has been reported to remain elevated for hours or even days after an intense bout of endurance exercise.[55,56,64] It is because of this finding that investigators are considering the use of DHEAS levels to replace or assist cortisol levels as an indicator of stress in athletes.[64]

Similar to the testosterone response to endurance exercise, both DHEAS and DHEA levels after a 10-mile run,[55] and DHEAS levels in the last 10km of a 40km marathon[56] were reported to be elevated. Increases in DHEA have also been reported in both trained and untrained women after 15 minutes of incremental cycling to exhaustion.[63]

While most of these studies measured increases in premenopausal individuals, Johnson et al.[65] reported that both DHEA and DHEAS levels were elevated after 30 minutes of treadmill exercise [at 80% maximal oxygen uptake ($\dot{V}O_{2max}$)] in postmenopausal women. Of interest to these investigators was that estrogen therapy appeared to enhance the DHEA response. This may be of importance because resting levels of DHEA and DHEAS decrease after age 25 years,[66] and these decreases are later associated with disease and disability.[67]

3.1.3 Estrogen

Increases in estradiol after a short-term endurance bout have been previously reported.[47,56,68] These increases appear to be dependent on both intensity of exercise[47] and menstrual cycle phase.[46,68,69] At similar exercise intensities, increases in estradiol levels are greater during the luteal phase compared with the follicular phase, suggesting that the ovary may be more sensitive during the former.[47,68] Jurkowski et al.[47] tested healthy, premenopausal women during 20 minutes of cycling at light (30 to 35% $\dot{V}O_{2max}$), heavy (60 to 66% $\dot{V}O_{2max}$) and exhaustive intensity (85 to 95% $\dot{V}O_{2max}$). Testing sessions occurred during both midfollicular and midluteal phases of the menstrual cycle. In the luteal phase, a significant increase in estradiol occurred after both heavy and exhaustive exercise. In contrast, during the follicular phase, significant estradiol increases were only noted after exhaustive exercise. The impact the menstrual phase has on the estradiol response is not surprising since most of this hormone is secreted from the ovary.[21] Therefore, studies such as Bonen and Keizer,[56] which included women of varying menstrual status and menstrual cycle phase, must be viewed with caution.

Decreases in MCR can not be ruled out as playing a role in these increases. Keizer et al.[70] found a significant decrease in the MCR of estradiol in young women after a brief bout of exercise. In contrast, Montagnani et al.[71] reported an increase in the MCR of estradiol and a corresponding decrease in the plasma estradiol level. These two studies used very different exercise protocols (10 minutes of exercise[70] *vs* 2 hours of exercise[71]) which may provide some explanation for this discrepancy.

3.1.4 Growth Hormone

Exercise is a known stimulant of growth hormone secretion[72] and the magnitude of elevation has been found to be related to both the duration and intensity of the exercise.[73] It is apparent however, that exercise must be at least 10 minutes in duration to stimulate a growth hormone response.[74] Increases in growth hormone levels after endurance exercise have been well documented in both females and males.[22,51,75-78]

Females have higher resting growth hormone levels than their male counterparts.[22,78-80] These increased levels may be related to higher estradiol levels in females, believed to stimulate growth hormone-releasing factor.[81] Wideman et al.[78] reported that despite premenopausal women having higher growth hormone levels at rest, incremental exercise to fatigue elicited a similar magnitude of increase between the two genders.

The role the menstrual cycle phase has on growth hormone release is inconclusive to date. Kanaley at al.[51] reported that in young women, a 90-minute treadmill run at 60% $\dot{V}O_{2max}$ was capable of eliciting a dramatic increase in growth hormone at 20 minutes of exercise, followed by a gradual decline. This pattern of increase was independent of both

menstrual phase and status (eumenorrhoeic and amenorrhoeic). Others have reported higher growth hormone levels during the periovulatory phase than the follicular phase at rest, and in response to short duration (10-minute) exercise.[48]

Sensitivity issues are a concern when measuring growth hormone. Levels of this hormone at rest and in the elderly can reach as low as 0.10 μg/L, which is below the detection level of some assays. The use of an ultrasensitive chemiluminescence assay (Nichols, San Juan Capistrana, CA, USA), by some of the studies discussed in this section,[76,78] strengthens the validity of their results.

It is still unclear what specific mechanism(s) are involved in the stimulation of growth hormone during exercise. Early research indicated that lactate or hypoxia may be the main stimulant of growth hormone release during exercise.[82] This belief has been questioned with research indicating that significant increases in growth hormone can occur below the lactate threshold.[76] In addition, a lack of growth hormone response has been reported after lactic acid infusion.[83] A recent study by Weltman et al.[77] examined the relationship between sympathetic outflow and the subsequent growth hormone response to short-term exercise in men. Results indicated that increases in both noradrenaline and adrenaline always preceded peak growth hormone response. Growth hormone release may also be affected by β-endorphins.[14] During intense exercise it has been shown that β-endorphins are released into the blood and that these hormones may be able to offset the inhibitory effect of somatostatin on growth hormone release.[84]

3.1.5 Insulin-Like Growth Factor-I (IGF-1)

Significant correlations between resting IGF-1 levels and physical fitness ($\dot{V}O_{2max}$) have been reported.[85,86] Short-term increases in IGF-1 have been reported in both men and women after 10 minutes of cycling.[48,87,88]

Hornum et al.[48] compared the IGF-1 response to 10 minutes of high-intensity cycling in untrained eumenorrhoeic women at different menstrual cycle phases (follicular and periovulatory). IGF-1 levels increased significantly at the end of exercise, and these increases did not appear to be dependent on menstrual cycle phase. Bang et al.[87] studied the IGF-1 response to 30 minutes of cycling (at 83% maximal heart rate) in untrained men and women. A limitation of this study was the small sample size (n = 6). However, similar to Hornum et al.,[48] IGF-1 levels significantly increased 10 minutes into cycling. These levels then decreased to resting levels through the remainder of the exercise session. Therefore, changes in IGF-1 may be transient and only observed during the onset of exercise. IGF-1 levels appear to be independent of growth hormone responses during exercise and recovery.[86,88]

It is possible that local muscular production of IGF-1 may be increased during short-term endurance exercise and therefore undetected in some studies. Five days of endurance training in rats has resulted in increases in muscular IGF-1 without changes in circulating IGF-1.[89] More research at the cellular level needs to be performed in the future. Unfortunately, no studies to date have investigated the IGF-1 response to prolonged endurance exercise in women. A study involving men, reported that IGF-1 levels decreased after 7.5 hours of a 75km cross-country ski race.[90] Based on evidence that physical fitness may affect resting levels of IGF-1,[85,86] additional information is needed to explore the role of physical fitness on the response of this hormone.

3.1.6 Cortisol

Increases in plasma cortisol levels after short-term endurance exercise have been documented in both pre- and postmenopausal women.[55,63,65] During endurance exercise cortisol increases appear to be related to both exercise intensity and duration.[91,92] During short duration (<15-minute) high-intensity exercise, cortisol levels have been reported to peak after exercise cessation in women.[63] Short-term increases in cortisol are reported to return to normal within hours after exercise of long duration such as a 10-mile race.[55]

3.2 Resistance Exercise

3.2.1 Testosterone

Most studies investigating the response to short-term resistance exercise in women have found no significant changes in testosterone.[45,93-98] Cumming et al.[99] were the only group that reported an increase in testosterone after a short-term bout of resistance exercise and their protocol used isokinetic exercises. They noted that both total and free testosterone increased and that the increases were greater than the observed increase in hematocrit, which rules out haemoconcentration as the mechanism responsible for the testosterone response.

Although the majority of studies reported no change in testosterone, women are capable of achieving significant muscle mass gains from resistance training.[100] A number of possibilities could account for this apparent discrepancy between testosterone response and muscle hypertrophy. First, it is unclear the impact short-term hormonal responses have on the long-term adaptations that are observed during repeated exercise sessions such as training. Other anabolic responses such as increases in growth hormone can not be ruled out as being responsible for the hypertrophic events that occur at the muscular level after repeated resistance sessions. Since it has been suggested that increases in anabolic hormones must be maintained for muscle anabolism to occur,[101] further research is needed to document these hormones during recovery and during repeated exercise sessions.

3.2.2 DHEA and DHEAS

There is minimal information available on the short-term response of DHEA and DHEAS to resistance exercise in women. Häkkinen et al.[37] reported no short-term changes in DHEA or DHEAS in middle-aged or elderly women, either before or after a 6-month training programme. Clearly, more research is required in this area.

3.2.3 Estrogen

The response of estrogen to resistance exercise has been relatively understudied. Kraemer et al.[96] have demonstrated that young, untrained women experienced an increase in estradiol after three sets of four resistive exercises at 10RM. This response was observed during both the follicular and luteal phases and similar to endurance findings,[47,68] a more pronounced increase in this hormone was noted in the latter.

Walberg-Rankin et al.[102] investigated the response of estradiol to resistance exercise during energy balance and energy restriction in young females who were familiar with resistance exercise. Despite post-exercise increases under both dietary conditions, increases were only significant during the hypocaloric diet. These findings suggest that diet may play a role in the release of this hormone during exercise. Walberg-Rankin et al.[102] cautioned that because estradiol is known to have a negative feedback effect on the hypothalamus to reduce gonadotropin-releasing hormone, it may be possible that repeated elevations of this hormone during weightlifting, along with energy restriction, could contribute to disruptions in the menstrual cycle.

3.2.4 Growth Hormone

Previous research indicates that strenuous resistance exercise can elevate growth hormone levels in both genders.[94-96,103] Increases appear to be related to exercise intensity[46,94,95,103] and may be blunted with age.[45,46]

Kraemer et al.[94] compared the growth hormone responses between a protocol utilising a 5RM load and 3-minute rest periods, with a more anaerobic protocol which consisted of a 10RM load and 1-minute rest periods. Significant increases in growth hormone were only observed during the more anaerobic workout. In a follow-up study, Kraemer et al.[95] determined that when extending the length of the rest period (10RM with 3 minutes rest), or reducing the duration of the set by increasing the resistance (5RM with 1 minute rest), growth hormone showed no significant increase above resting levels. Increases have also been greater in women utilising a multiple-set protocol compared with women using a single-set protocol.[103] It appears that exercise that places the greatest demand on anaerobic glycolysis may have the greatest effect on elevating serum growth hormone.[95]

It is possible that these increases in growth hormone may be dependent on age. Pyka et al.[46] investigated the impact of age on the growth hormone response after short-term resistance exercise. Results indicated no significant increases in growth hormone in older individuals (mean age 72 years), despite observing significant increases in younger individuals (mean age 28 years).

Because of the pulsatile release of growth hormone, frequent sampling is recommended when studying this hormone. Sampling throughout the exercise session is commonly performed during endurance studies,[51,75,76,78] but is less common in resistance protocols. Studies such as Häkkinen and Pakarinen[45] and Mulligan et al.[103] that only included measurements before and after the exercise session, do not allow for a detailed analysis of the secretion pattern of the hormone.

Another methodological issue (that is not limited to resistance exercise) is that different immunoassay techniques can produce varying levels of growth hormone from the same sample.[104,105] This variability is likely caused by factors including antibody specificity and the molecular heterogeneity.[104,105] Therefore, caution must be used when comparing data from studies using different assay techniques. The studies discussed in this section were all believed to utilise radioimmunoassays as the method of measuring growth hormone levels.

3.2.5 IGF-1

Previous investigations documenting the IGF-1 response to resistance exercise in women have produced equivocal results.[94,95] Kraemer et al.[94] observed increases in IGF-1 mid-exercise and immediately after completion of a 10RM protocol with 1-minute rest periods. In the same study, a 5RM load with 3 minutes rest only produced elevated IGF-1 levels after 60 minutes of recovery. The validity of these increases was later questioned when Kraemer et al.,[95] using different heavy resistance protocols (including those used by Kraemer et al.[94]), did not observe changes in IGF-1 either during or after resistance exercise. The different results from these two studies can not be attributed to methodological differences in correcting for plasma volume changes.

The inconsistency in these findings may be related to the large interindividual variability that is observed in IGF-1 responses. Kraemer et al.[95] have suggested that inconsistencies in results may also be due to a number of different physiological factors. These include a concentrating mechanism in the blood (e.g. different metabolic clearance rates), increases in transporter proteins, and/or the release of IGF-1 from other nonhepatic cells (e.g. fat, muscle and connective tissue cells) caused by tissue disruption from exercise.[95] Differences in the physical fitness of the individuals in the previously mentioned studies may account for some of the inconsistent findings.[85,86] Similar to endurance exercise, it is unclear what role local muscular production of IGF-1 has during resistance exercise. In addition, investigators have recently attempted to investigate the response of the individual components of the IGF-1 system to exercise. Nindl et al.[106] reported that in males no changes occurred to either free or total IGF-1 levels during 13 hours of recovery after a resistance session. However, IGF binding protein-2 and the acid-labile subunit increased and decreased, respectively. Therefore, the impact of resistance exercise may not be in the alteration of IGF-1 levels, but instead in alterations to individual components of the IGF-1 system, that may result in subsequent anabolic events.

When evaluating the measurement of IGF-1, one must consider the methodological problems associated with the binding proteins in the assays. There is no apparent universal procedure in which IGF-1 is separated from its binding protein. Therefore, further research is needed to distinguish between the bound and free levels of this hormone.

3.2.6 Cortisol

Resistance exercise of high intensity is capable of eliciting short-term cortisol increases in women.[95,103] These increases are likely dependent on the volume of exercise performed. Mulligan et al.[103] reported increases in cortisol levels in women after both single and multiple set protocols; however, they did not correct for changes in plasma volume, therefore

these increases could be explained by haemoconcentration. Higher levels were observed in the multiple set compared with the single-set protocol immediately after exercise and up to 15 minutes into recovery. In contrast, Häkkinen and Pakarinen[45] reported no change in cortisol levels in women between the ages of 30 and 70 years after resistance exercise. The use of longer rest periods, and older, less experienced individuals in the Häkkinen and Pakarinen[45] study may explain these conflicting findings.

4. Training Adaptations

It has been established that there are changes in levels of anabolic hormones in response to short-term bouts of exercise; however, the biological impact of these transient increases in hormones has not been determined. Repeated bouts of exercise or long-term training may influence the hormonal milieu and long-term training programmes may affect both basal levels of anabolic hormones and the short-term response to exercise.

4.1 Endurance Training

4.1.1 Testosterone

Resting testosterone levels in females are typically quite low, and some longitudinal studies have shown that they can decrease to even lower levels following endurance training. These results have been consistently demonstrated in men;[107-109] however, the data for women are less consistent. Keizer et al.[110] found that testosterone decreased in women after 3 months of endurance training, although this decrease was observed only in the luteal phase of the menstrual cycle. Urhausen et al.[111] also reported a decrease in testosterone with training; however, their study population consisted of only three female participants. These results are supported by cross-sectional studies that found elite athletes had lower resting free testosterone levels when compared with sedentary controls.[58,112] Other research has found no significant change in testosterone levels in women as a result of endurance training,[64,113,114] or found no difference in testosterone levels between female athletes and sedentary controls.[115] Krahenbuhl et al.[116] did note that although there was no change in testosterone level with training, improvement in aerobic power was significantly correlated with pre-training testosterone levels in women. They suggested that individual levels of testosterone play an important role in the trainability of women.

Endurance training can also influence the short-term exercise-induced response of testosterone, but again the results are conflicting. Keizer et al.[110] found that the short-term increase in testosterone following a bout of exercise in the follicular phase was significantly greater in women after a 3-month endurance training programme. However, subsequent research from Keizer et al.[64] found no variation in the short-term testosterone responses to exercise in women preparing for a marathon, although these participants were tested in the middle of their menstrual cycle and this may provide an explanation for their inconsistent findings.[107]

4.1.2 DHEA and DHEAS

Women who engage in endurance training tend to have a decreased resting level of plasma DHEAS[64,110] and also saliva DHEA.[112] The mechanism responsible for this adaptation is unknown. It is interesting that although resting DHEA and DHEAS levels appear to decline with training, the short-term increase in DHEA and DHEAS with exercise of the same relative intensity is significantly greater after training,[110] and is greater in trained individuals compared with untrained individuals.[58] This may indicate that the absolute workload of exercise is more important for eliciting increases in adrenal androgens since the absolute workload of an exercise session would be greater after training.

In contrast to these results, Milani et al.[117] reported no change in DHEAS following 12 weeks of training in male and female cardiac patients, and Hersey et al.[118] found no change in DHEAS following 6 months of training in elderly men and women. These differing results could be explained by the age differences of the participant populations since DHEA and DHEAS declines with age.[15] Circulating levels of DHEAS would already be low in older or elderly individuals and this may explain

why younger participants showed a response to training[64,110] and older participants did not.[117,118] Bonnefoy et al.[119] found that a lower level of physical activity in elderly people was associated with lower levels of DHEAS. Abbasi et al.[120] found a significant positive relationship between aerobic power and DHEAS in elderly men but not women. These results indicate that regular physical activity may slow the age-related decline in synthesis of adrenal androgens.

4.1.3 Estrogen

The influence of strenuous endurance training on the female reproductive system has been well studied. A comprehensive review of athletic amenorrhoea is beyond the scope of this review, however, DeCree[2] provides an excellent summary of the literature on this topic.

Significant menstrual disturbances have been demonstrated in female participants after only 8 weeks of intense endurance training,[121] and several studies have reported lower levels of both urinary and blood estrogen (estradiol, estrone, estriol or free estradiol) in endurance-trained women.[107,113,122-124] It is interesting that changes in ovarian hormone production do not always lead to amenorrhoea,[113,123] so even eumenorrhoeic individuals could experience small but potentially significant decreases in estrogen throughout the menstrual cycle as a result of endurance training. Data from Winters et al.[123] suggest that a delay in estrogen production in the early follicular phase may be responsible for the lower bone mineral density they observed in trained runners. A decrease in the level of estrogen may suggest impairment in the development of the ovarian follicle.[6] Possible mechanisms of this altered reproductive function in trained females include impaired release of pituitary gonadotropins or decreased sensitivity of the ovaries to gonadotropins.[125]

4.1.4 Growth Hormone

Long-term endurance training in women has been shown to significantly increase growth hormone release.[126] Weltman et al.[126] had female participants complete a 1-year running programme and found that individuals who regularly trained above their lactate threshold had an increased 24-hour growth hormone release. The growth hormone response was not significant in individuals who trained *at* their lactate threshold. Therefore, Weltman et al.[126] suggested that there is a threshold training intensity that is required to stimulate a change in growth hormone with long-term training. This relationship is supported by Eliakim et al.[127] who found a positive correlation between fitness and overnight growth hormone levels in adolescent females. Sidney and Shephard[128] reported that resting growth hormone levels were significantly elevated after endurance training in elderly men, but not in elderly women. Similarly, Bell et al.[129] reported no change in growth hormone levels in women following 12 weeks of training. Unfortunately, it is difficult to compare these results to those of Weltman et al.[126] because of the major differences in the length and intensity of the training programme and the age of the participants.

Endurance training may also have an influence on the short-term growth hormone response to exercise. Weltman et al.[130] demonstrated a blunted growth hormone response in men to an exercise test after training. The exercise test was set at the same absolute power output before and after training, therefore the relative intensity would have been lower after training and this may explain the blunted response. The results from this study suggested that the decrease in growth hormone level was related to both reduced secretion and enhanced clearance of growth hormone as a result of training. Weltman et al.[130] pointed out that a decrease in the short-term growth hormone response does not necessarily mean that the 24-hour growth hormone level was decreased by training. In contrast to this study, Sidney and Shephard[128] found that the short-term growth hormone response to exercise in elderly men and women was significantly *greater* after 10 weeks of training. They hypothesised that conditioning restored hypothalamic sensitivity in elderly individuals and subsequently increased growth hormone release. If this is the case, then the growth hormone response to training may be significantly different depending on the age of an individual.

4.1.5 IGF-1

It has been suggested that IGF-1 may be a more useful indicator of the status of the growth hormone-IGF-1 pathway, because of its low diurnal variability compared with growth hormone.[131] Several investigations have found that endurance training results in increased circulating levels of IGF-1 in young men and women.[132,133] Other research using older participants has found no change in IGF-1 with training.[134-136] These differing results could be caused by age differences in the study populations but they could also reflect differences in training programmes since the studies using younger individuals[132,133] used high-volume and high-intensity training. Although Vitiello et al.[136] found no change in IGF-1 in older men and women who trained for 6 months, they did find that levels of IGF-1 were significantly correlated to fat-free mass (FFM) and concluded that the lack of change in FFM in their participants may explain the lack of change in IGF-1. However, Poehlman et al.[134] noted increases in IGF-1 following a training programme even though there were no changes in FFM in their participants. Clearly, body composition changes do not always coincide with changes in IGF-1.

Poehlman et al.[134] did find a significant change in aerobic power with training and also noted a significant correlation between changes in IGF-1 and changes in $\dot{V}O_{2max}$; however, the changes in IGF-1 were significant in men, but not in women. The women who participated in this study were postmenopausal, and low estrogen levels may explain the blunted exercise response of the growth hormone-IGF-1 axis, since estrogen is believed to stimulate growth hormone-releasing factor.[81] Haydar et al.[137] found that aerobic power was related to IGF-1 levels in a sample of men and women of various ages, but this relationship may be explained by chronological age since both $\dot{V}O_{2max}$ and IGF-1 decrease with age. However, Bonnefoy et al.[119] found that lower levels of physical activity were significantly correlated with lower levels of IGF-1 in elderly men and women, independent of age. This may indicate that physical activity provides a stimulus for IGF-1 production regardless of physical fitness or age.

4.1.6 Cortisol

Most studies have found very little change in resting cortisol levels in women in response to endurance training.[64,111,128,138,139] Cortisol is a stress hormone and elevated levels of this hormone have been associated with overtraining;[34] however, Hooper et al.[140] reported no change in cortisol levels in female swimmers during 6 months of intense training, even though some participants demonstrated other symptoms of overtraining. Tsai et al.[114] found an increase in cortisol levels in elite female endurance athletes during their competition season and they also noted that female athletes had significantly greater levels of cortisol than male athletes. Cross-sectional research by Tegelman et al.[115] found that elite female athletes had significantly greater cortisol levels compared with sedentary controls.

Cortisol is the primary catabolic hormone and the ratio of catabolic to anabolic hormones can provide information about the balance of anabolism and catabolism over prolonged periods of endurance training.[111] Some studies have found a decreased ratio of testosterone to cortisol (T/C) in elite female athletes, which suggests an increase in catabolic activity.[111,115] In contrast, Filaire et al.[138] reported an increase in the ratio of DHEA to cortisol (DHEA/C) over 16 weeks of training; however, of their two groups of participants, the group that performed the greatest amount of training had the lowest DHEA/C ratio. It may be important to note that Filaire et al.[138] measured DHEA and cortisol levels in saliva in contrast to other studies discussed here which measured cortisol levels in blood. Kiilavuori et al.[139] found no change in the ratios of T/C or DHEA/C after 12 weeks of training in cardiac patients, but the intensity and frequency of the training was lower than in other studies of elite athletes. Based on the current literature it is difficult to conclude whether any androgen to cortisol ratio is a useful indicator of training status in female endurance athletes.

4.2 Resistance Training

Resistance training results in significant gains in muscular strength and hypertrophy in both men and women. Although women have lower absolute strength than men, the relative increases in strength following a training programme are similar between genders.[37,141,142] Early increases in strength are thought to result from neural adaptations and improvements in coordination while later strength increases arise from increased muscle hypertrophy.[142-144] Anabolic hormones are known to play an important role in muscle hypertrophy by stimulating muscle protein synthesis, therefore, it seems logical that increases in muscle mass could result from hormonal adaptations to training.

4.2.1 Testosterone

Circulating levels of testosterone have been shown to correlate with training-induced increases in muscular strength in women.[37,100,141,145] There is a high degree of variability in testosterone levels in women, which may influence individual trainability. However, while the levels of testosterone may influence training adaptations, increases in strength and muscle mass do not always coincide with increases in levels of testosterone.

Westerlind et al.[146] found no change in testosterone in female participants after a 10-week resistance training programme and they also showed no difference in strength gains between the training group and a control group. Hickson et al.[147] found increases in strength and muscle fibre area in men and women who participated in 16 weeks of training, but there were no changes in testosterone levels. Similar results have been reported in middle-aged[141] and elderly women.[37] Häkkinen and Pakarinen[141] actually found a decrease in testosterone in elderly women in the final weeks of a training programme. However, Marx et al.[148] reported a significant increase in resting testosterone levels in young women after 12 and 24 weeks of resistance training, and the increase was significantly greater in participants who completed high-volume training compared with low-volume training. They suggested that previous studies with conflicting results did not use a long enough training period to see changes in testosterone.[141,146,147] Marx et al.[148] reported significant strength increases, particularly in the last 3 months of the 6-month programme.

Häkkinen et al.[100] found no change in testosterone levels following a 3-week intensive strength-training programme even though the participants demonstrated increases in muscle cross-sectional area and maximal force. The authors noted that their results support the concept that initial increases in strength are primarily a result of neural adaptations rather than muscle hypertrophy and this was supported by Marx et al.[148] who only found changes in testosterone levels after a long-term (6-month) training programme. Contrary to this, Kraemer et al.[97] found increases in testosterone following a relatively short training period of 6 weeks and also at the completion of the 8-week programme. They concluded that there are hormonal adaptations in the early phase of resistance training, a period that has been typically associated with neural adaptations; however, there is currently little research to support this claim. Kraemer et al.[97] measured hormone levels after 6 and 8 weeks of training but it does not appear that they controlled for phase of menstrual cycle in their participants. The mechanism responsible for increases in testosterone levels in women after resistance training is not clearly understood; however, it has been suggested that a significant contribution is made from the adrenal gland.[148]

Häkkinen and colleagues[100] found a strong correlation between the ratio of testosterone to its primary binding globulin (sex-hormone binding globulin) and individual changes in muscle cross-sectional area. This relationship between the amount of bioavailable testosterone and strength gains has been supported by subsequent research from this group.[37,141] Häkkinen et al.[37] also found a significant correlation between the T/C ratio and strength development in women during a resistance training programme, and this was supported by other research.[141,148] This relationship was only significant in women and Häkkinen et al.[37] concluded that testosterone levels may be an important indicator of trainability

in women specifically. However, other research has shown comparable results in elderly men so it may be that testosterone is predictive of strength gains only in individuals with a diminished anabolic environment.[141,149] Long-term resistance exercise may also result in changes in the short-term testosterone response to resistance exercise. Häkkinen et al.[37] showed a significant post-exercise increase in free testosterone in women, but only after a 6-month training programme. This finding is similar to the results from Hickson et al.[147] and comparable to results found in men.[97] This suggests that a certain amount of experience with the training stimuli is necessary before exercise will induce a hormonal response.[97] In contrast, Kraemer et al.[97] found no increase in testosterone following a short-term bout of resistance exercise in women, before or after training, although the training period was relatively short.

4.2.2 DHEA and DHEAS

Research on changes in DHEA or DHEAS in response to long-term resistance training in women is lacking. This is interesting considering that DHEA may be a more important androgen than testosterone in women.

Häkkinen et al.[37] measured DHEA and DHEAS before and after a 6-month resistance training programme in middle aged and elderly women. They found no significant changes in DHEA or DHEAS following training, and no training effect on the short-term DHEA and DHEAS response to exercise. However, Kostka et al.[150] found a significant correlation between circulating levels of DHEAS and quadriceps muscle function in elderly women, but not in men. These results suggest that DHEA and DHEAS may be more important predictors of muscle function in older women when testosterone levels are drastically reduced.

4.2.3 Estrogen

There is currently very little research examining the estrogen response to long-term resistance training. Häkkinen et al.[145] found no change in estradiol following 16 weeks of power type strength training, although it is important to note the small sample size (n = 7). The emphasis in the literature regarding estrogen has been on the effects of endurance training, since it is known to be associated with menstrual disturbances in female athletes. It is unclear if long-term resistance training could have similar effects on the female reproductive system since some of the proposed mechanisms of athletic amenorrhoea (low body fat, psychological stress) could apply to highly trained resistance athletes as well.[151]

4.2.4 Growth Hormone

Although growth hormone has been found to reliably increase following a short-term bout of resistance exercise of sufficient intensity (see section 3.2.4), long-term resistance training appears to have little influence on resting levels of growth hormone in women. Pyka et al.[152] found no change in resting levels of growth hormone following 1 year of strength training in elderly women, despite significant increases in muscle strength. Other research has found a similar lack of change in growth hormone in both middle aged and elderly women.[37] Pyka et al.[152] concluded that deficits in growth hormone levels that occur as a result of age[40] explain the lack of responsiveness to resistance training. However, others have reported no change in growth hormone even in young women following resistance training.[97,148] Craig et al.[153] found that 12 weeks of resistance training in young men increased the growth hormone response to a single bout of resistance exercise; however, most other research has found no differences following training in older women[37,152] or young women.[97]

4.2.5 IGF-1

There has been an increased interest in recent years in the relationship between IGF-1 and exercise, particularly in the older population. It has been suggested that low levels of testosterone in women may result in stimulation of the growth hormone-IGF-1 axis following training to enhance muscle protein synthesis.[119] Kostka et al.[150] demonstrated that in older women there was a significant correlation between quadriceps muscle function and IGF-1. However, there has been limited research on the influence of long-term resistance training on circulating levels of IGF-1. Pyka et al.[152] found no

change in IGF-1 in older men and women following 1 year of resistance training; however, other more recent studies have reported significant increases in IGF-1 in women following resistance training.[131,147] Marx et al.[148] found that low-volume resistance training resulted in increased IGF-1 levels following 24 weeks of training, while high-volume resistance training induced significant increases in IGF-1 after only 12 weeks of training. Borst et al.[131] reported a similar increase in IGF-1 in men and women after 13 and 25 weeks of resistance training. Borst et al.[131] also found a significant decrease in one of the major binding proteins of IGF (IGF binding protein-3) in individuals who completed high-volume training, but not in individuals who completed low-volume training. A decrease in IGF binding protein-3 would increase the level of free IGF-1, and therefore may contribute to even greater strength gains. This is supported by the greater strength increases that were observed in the high-volume training group.[131] Together, these studies indicate that there may be a certain volume of resistance training necessary to elicit changes in the amount of bioavailable IGF-1. Marx et al.[148] found that the changes in IGF-1 occurred with no coinciding change in growth hormone, which lends further support to the idea that IGF-1 changes occur independently of changes in growth hormone.[87,88] In addition, none of these studies rule out the possibility of increases in IGF-1 produced in the muscle, which would not be detected in serum measurements.

4.2.6 Cortisol

Kraemer et al.[97] reported that resting cortisol levels decreased in female participants after 8 weeks of training, which they concluded would improve the anabolic environment. Marx et al.[148] also reported a decrease in cortisol levels in female participants following resistance training, although the decrease was only significant in those who completed high-volume training and not in individuals who completed low-volume training. The high-volume training group also demonstrated the greatest increases in strength and lean body mass, which supports the concept that lower cortisol levels are more conducive to anabolism and strength gains. Other studies have shown increases in strength with no coinciding changes in resting cortisol levels,[100,141,147] so a decrease in catabolic stimulus is not necessary for strength and muscle mass development with training.

Although elevated cortisol levels are often an indication of overtraining in endurance athletes, it appears that resistance exercise overtraining does not have a similar effect on cortsiol levels, at least in male participants.[154]

5. Cross-Training

The American College of Sports Medicine[155] recommends that an individual participate in a combination of both endurance and resistance exercises. Many sports consist of both endurance and strength components and therefore many athletes include cross-training to their athletic preparation. Despite the popularity of concurrent endurance and strength training, the majority of research in exercise endocrinology has investigated the hormonal response to these two types of exercise in isolation.

The influence of combined endurance and resistance training on endurance performance is still under debate. Authors have reported both detrimental effects[156,157] and no effect[129,158,159] on performance. Most research indicates that concurrent endurance and resistance training is antagonistic to strength gains.[129,158-161] This compromise appears to be directly related to muscular contractions at high velocities.[158]

Decreased strength gains during combined training are likely attributed to differences in muscle fibre morphology that occur during combined training compared with strength training alone.[129,161] This may be related to an altered anabolic/catabolic state caused by concurrent training.[161] Previous research indicates that concurrent training may be responsible for increased cortisol levels[129,160,161] and either no[129,160] or minimal changes[161] in anabolic hormones.

Kraemer et al.[161] investigated the hormonal adaptations that occurred during 12 weeks of high-intensity strength and/or endurance training in healthy

young men. Combining the two training types diminished the muscle fibre hypertrophy that was observed during strength training alone. This may have, in part, been attributed to the different hormonal milieus that were produced by each protocol. Strength training resulted in decreases in cortisol and no change in testosterone levels, producing an overall anabolic environment, promoting muscle hypertrophy. In contrast, concurrent endurance and strength training produced a dramatic and progressive increase in cortisol throughout the training period, producing an overall catabolic environment, conducive to muscle breakdown.

Recently, Bell et al.[129] reported similar results in young women. After 12 weeks of combined training, increases in knee extension strength and muscle hypertrophy were less pronounced than those produced by strength training alone. Women involved in concurrent training had significantly higher levels of urinary cortisol at rest compared with those participating in strength or endurance training alone. In addition, no change in the anabolic hormones, testosterone or growth hormone, were reported. Therefore, an overall catabolic state was achieved in these women and may have accounted for diminished strength gains and muscle hypertrophy. These findings are consistent with earlier research by Bell et al.[160] who investigated 16 weeks of concurrent training versus strength training alone in healthy young women. Those individuals undergoing concurrent strength and endurance training had delayed and less pronounced strength gains than women involved in strength training alone. This compromised strength gain during concurrent training was associated with elevated urinary cortisol levels during the final 8 weeks of training.

It appears that concurrent endurance and resistance training is capable of creating a catabolic environment that may affect strength development. However, caution must be used when examining these results. In most studies, endurance or strength training alone was compared with concurrent training with a disproportionately higher training volume. Therefore, the incompatibility of endurance and resistance training may be the result of overtraining caused by the increased training volume.

6. Conclusion

Despite the initial attention given to female exercise endocrinology approximately 30 years ago, the field is still not completely understood. There are many contradictory results that make it difficult to draw conclusions about endocrine responses to exercise and training. This may be largely caused by the significant interindividual variability that exists in endocrine data. This variability limits the utility of cross-sectional study designs, especially those with a small sample size. Future research needs to focus on longitudinal or prospective studies that could clarify some of these conflicting results. Table II provides a summary of the findings to date, involving both endurance and resistance exercise and training in women. Similar to early studies completed on their male counterparts, preliminary research on females has focused on endurance exercise. Therefore, comparisons between these two types of exercises are difficult because of the shortage of information regarding the endocrine response to resistance exercise.

Changes in hormone levels may have metabolic and hypertrophic implications with each type of exercise. For example, increases in testosterone during a resistance exercise may have anabolic signif-

Table II. Summary of research on anabolic and catabolic hormone responses to endurance and resistance exercise and training in women[a]

	Endurance		Resistance	
	short-term exercise	training	short-term exercise	training
Testosterone	↑↑	↓↔	↔	↑↔
DHEA(S)	↑↑	↓	↔	↔
Estradiol	↑↑	↓	↑	↔
Growth hormone	↑↑↑	↑↔	↑↑	↔
IGF-1	↔	↑↔	↔	↑↔
Cortisol	↑↑	↔↔	↑	↓↔

a Number of arrows indicates the strength of research.

DHEA(S) = dehydroepiandrosterone and/or dehydroepiandrosterone sulphate; **IGF-1** = insulin-like growth factor-I; ↑ indicates increases; ↓ indicates decreases; ↔ indicates no change or equivocal results.

icance. However, increases in testosterone during endurance exercise may be important for glycogen compensation.[162] Further research is necessary to clarify the biological significance of endocrine increases with each type of exercise.

Future research needs to expand on the preliminary data gathered on resistance exercise. It is surprising the lack of attention cross training has received in endocrine research based on its popularity both in the competitive and recreational settings. Future studies involving concurrent endurance and resistance exercise must carefully control for exercise volume.

The physiological mechanisms underlying changes in hormone levels need to be further investigated. Gender differences in the receptor and binding protein response to exercise needs to be examined. The local production of hormones, in particular IGF-1, has recently been a source of interest and deserves further attention. Finally, research needs to continue to document the biological significance that both short- and long-term changes in anabolic hormones have on the human body.

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