

Testosterone Responses After Resistance Exercise in Women: Influence of Regional Fat Distribution

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Regional fat distribution (RFD) has been associated with metabolic derangements in populations with obesity. For example, upper body fat patterning is associated with higher levels of free testosterone (FT) and lower levels of sex-hormone binding globulin (SHBG). We sought to determine the extent to which this relationship was true in a healthy (i.e., non-obese) female population and whether RFD influenced androgen responses to resistance exercise. This study examined the effects of RFD on total testosterone (TT), FT, and SHBG responses to an acute resistance exercise test (ARET) among 47 women (22 ± 3 years; 165 ± 6 cm; 62 ± 8 kg; 25 ± 5 %BF; 23 ± 3 BMI). RFD was characterized by 3 separate indices: waist-to-hip ratio (WHR), ratio of upper arm fat to mid-thigh fat assessed with magnetic resonance imaging (MRI ratio), and ratio of subscapular to triceps ratio (SB/TRi ratio). Skinfolds were measured for the triceps, chest, subscapular, mid-axillary, suprailiac, abdomen, and thigh regions. The ARET consisted of 6 sets of 10 RM squats separated by 2-min rest periods. Blood was obtained pre- and post- ARET. TT, FT, and SHBG concentrations were determined by radioimmunoassay. Subjects were divided into tertiles from the indices of RFD, and statistical analyses were performed by an ANOVA with repeated measures (RFD and exercise as main effects). Significant ($p \leq .05$) increases following the AHRET were observed for TT (~25%), FT (~25%), and SHBG (4%). With multiple regression analysis, anthropometric measures significantly predicted pre-concentrations of FT, post-concentrations of TT, and pre-concentrations of SHBG. The SB/TRi and MRI ratios but not the WHR, were discriminant for hormonal concentrations among the tertiles. In young, healthy women, resistance exercise can induce transient increases in testosterone, and anthropometric markers of adiposity correlate with testosterone concentrations.

Key Words: androgen responses, sex steroids, adiposity, strength training

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Introduction

Regional fat distribution (RFD) has been associated with metabolic derangements mainly in populations with obesity (5, 27). Women with upper-body obesity (as assessed via the waist-to-hip ratio) express a metabolic profile more similar to men than women with lower-body obesity (e.g., higher resting concentrations of free testosterone (FT), and lower concentrations of sex-hormone binding globulin (SHBG; 3, 11, 14, 15). Static measurements of blood concentrations of these sex steroids have been identified as playing a potential role in the regulation of fat distribution (14). The mechanisms of action have not been fully elucidated, but alterations in hepatic metabolism and/or direct effects on adipocyte volume and size have been suggested (15, 26). The vast majority of the previous literature that has established the link between concentrations of sex hormones and RFD has been performed in obese populations. However, two reports have suggested that this association is also present in non-obese populations (21, 29).

A relationship between the free androgen index, calculated by dividing total testosterone by sex-hormone binding globulin, and RFD has recently been reported in young (mean age = 24 years), healthy men exhibiting homogeneity with regard to fitness levels and overall percentage body fat (~15%) (19). This study reported significant correlations between the free androgen index and dual-energy X-ray absorptiometry (DEXA) assessed regional fat mass of the trunk ($r = 0.37$) and of the leg ($r = -0.35$). In addition, in a group of non-obese women averaging 38 years, Seidell et al. (29) used multiple regression analysis to demonstrate that fat distribution was related to the degree of androgenicity (i.e., testosterone concentrations). However, whether or not the relationship between RFD and sex hormones observed in obese populations is robust enough to also be observed in young, healthy (i.e., non-obese) female populations, where the extremes of differing adipose phenotypes are not found, and whether RFD influences androgen responses to exercise—remains to be studied.

As in men, androgen levels in women have been implicated in exerting positive effects on muscular hypertrophy and strength primarily through its anabolic actions. These actions enhance muscle protein metabolism, protein accretion, and neuromuscular transmitter concentrations (10, 17). Because of the potential therapeutic benefit of resistance training in combating osteoporosis and the fact that resistance training is becoming more popular among women, a better understanding of the endocrine response is needed concerning resistance exercise in women. While exercise-induced elevations in testosterone have been frequently reported for men, the findings in women have been conflicting, with studies showing either an increase (2, 30) or no change (6, 16, 17, 34) in testosterone concentrations after exercise. In women, these conflicting results could be attributed to differences in mode of exercise, age, health, or fitness. But more importantly, it could be because an association also exists between RFD and sex steroid concentrations in young, healthy women.

We had hypothesized that, similar to the results found in obese women, healthy women would also show a relationship between upper-body fatness and FT and SHBG, and that within a sample of healthy women, those possessing a greater degree of upper-body fatness would exhibit accentuated testosterone responses to acute resistance exercise, thus possibly offering a partial explanation to reconcile previous contrasting findings concerning female testosterone responses to exercise.

The primary purpose of the present study, therefore, was to examine the effects of RFD on androgen responses to an acute resistance exercise test among young, healthy women. A secondary purpose was to evaluate and compare three different indices for classifying RFD and to determine differences in testosterone concentrations when the subjects were broken into tertiles of these RFD indices.

Methods

Experimental Approach

In this study, we employed a large sample of women ($n = 47$) in a cross-sectional design to address what effect RFD has on androgen responses after resistance exercise in women. Because RFD can be assessed through a variety of measures, and it is likely that these various measures reflect different aspects of RFD (15), we employed the following indices that have been used previously in the literature: waist-to-hip ratio (WHR), ratio of upper arm fat to mid-thigh fat (A/L_{FAT}) assessed via magnetic resonance imaging, and the subscapular-to-triceps ratio (SB/TRi).

Subjects

Forty-seven women volunteered for this investigation. Prior to inclusion in the study, all women read and signed an institutionally approved informed consent form and were medically screened and approved by a physician. This indicated that all subjects were healthy (i.e., free from any orthopedic, endocrine, or medical problems), not taking oral contraceptives, and menstruating regularly. The women were determined to be eumenorrheic, according to methods described previously (16) and defined as "regular" 28–32-day menstrual day cycles over the previous year. Women were tested in the early follicular phase (days 1 to 7) of their cycle. All testing was performed in the morning after an overnight fast. No subjects had performed structured resistance training for 6 months prior to the study.

Body Composition

Height and weight were measured with a physician's scale. (All subjects were weighed on the same scale.) Seven skinfold thicknesses (chest, mid-axillary, triceps, subscapular, abdominal, suprailiac, thigh) were measured with a Lange skinfold caliper and circumferences by a single experienced and trained observer. In addition, circumferences of the waist (midway between lower rib margin and iliac crest) and hip (widest circumference in the trochanter area) were also measured. Measurements were made on the dominant side of the body. Percentage body fat was then calculated with the Jackson and Pollock 7-site skinfold equation for women (12), and fat-free mass was subsequently calculated by subtracting fat mass from body mass.

Characterization of Regional Fat Distribution

Various indices have been utilized in the literature to characterize RFD (7, 10, 11). It is likely that these indices all measure different dimensions of RFD (11). Thus, for this study we employed three indices to operationally define RFD: (a) the waist-to-hip ratio (WHR; the waist circumference [cm] divided by the hip circumference [cm]); (b) the subscapular-to-triceps ratio (SB/TRi; the subscapular skinfold [mm]

divided by the triceps skinfold [mm]); and (c) the ratio of upper arm fat to mid-thigh fat (A/L_{FAT}) assessed via magnetic resonance imaging. The WHR reflects abdominal versus gluteal fat distribution, the SB/TRI reflects upper-body subcutaneous fat distribution, and the A/L_{FAT} ratio reflects upper limb versus lower limb adiposity.

Magnetic Resonance Imaging

Thigh muscle cross-sectional area (TMCSA) and upper-arm muscle cross-sectional area (UAMSCA) of the dominant leg and arm were assessed using a MRI 0.5-Tesla super conduction magnet (Picker International, Inc., Highland Heights, OH) with MR6B software. Images were obtained by alteration of the spin-lattice or longitudinal relaxation time (T1). Weighting of T1 was with repeat time (TR): 500 ms; echo time (TE): 13 ms; and radio frequency (RF, at 90°) power absorption was 0.028 W/kg. Analysis of the CSAs was determined from the MRI scans using a gradient echo technique that allows the greatest delineation and distinction between muscle and fat tissue and has been shown to be more sensitive than CT scans (4). Sagittal images of the thigh and upper arm were obtained, and a 15-slice grid was placed over the sagittal images, and the trans-axial images were obtained. Fifteen trans-axial images of 1-cm slices were obtained equidistantly between the base of the femoral head and mid-knee joint of the thigh and the superior head of the humerus and mid-elbow joint of the upper arm. All MR images were then ported to a Macintosh computer for calculation of muscle CSA using a modified NIH image software package. For the TMCSA, slice 8 was used (slice 1 being at the superior humerus). For UAMSCA, slice 9 was used (slice 1 being the base of the femoral head). Tissue CSA was obtained by displaying the images through a Maxitron displayer and an Adobe program, and using the NIH 1.55.20A Image Analysis pixel counting program. The same investigator performed all tracings; the test-retest reliability was $R = 0.99$.

One Repetition Squat Assessment (1 RM) and Acute Heavy Resistance Exercise Test (AHRET)

At least 48 hours prior to the AHRET, subjects had their 1 repetition maximum (1RM) in the squat position assessed using a concentric only 1 RM squat test on the Plyometric Power System (PPS) (Norsearch, Ltd., Lismore, Australia) from a 90° knee angle. The AHRET consisted of performing 6 sets of 10 RM squats with a 2-min rest between sets. The initial 10 RM load was calculated as ~75% of the squat 1RM. If due to fatigue on any given set, the subject failed to perform 10 repetitions, the load was subsequently adjusted (i.e., lightened) to allow the completion of 10 repetitions on the following set. The squats were performed with the Plyometric Power System (PPS). Blood was obtained via venipuncture pre- and immediately (within 2 min) after completion of the 6th set (see Figure 1).

Hormonal and Biochemical Analyses

Blood was allowed to clot and then centrifuged at $1500 \times g$ at -4°C for 15 min. All serum samples were then distributed to appropriate preservative tubes and stored at -84°C until analysis. All samples were run in duplicate and were decoded after analyses were completed (blinded analyses). Serum total and free testosterone were measured using an ^{125}I solid-phase radioimmunoassay (Diagnostics Products Corp.,

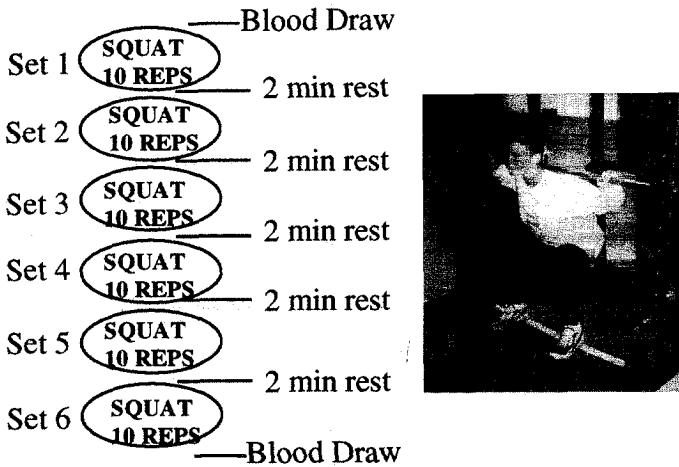


Figure 1 — Schematic of the acute heavy resistance exercise test (AHRET). The test consisted of performing 6 sets of 10 repetition maximum squats. Two-minute rest intervals were given between sets. All squats were performed on the Plyometric Power System. Blood was drawn before and after the test.

Los Angeles, CA) with a sensitivity of 0.14 nmol/L for total testosterone and 0.15 pg/ml for free testosterone. Sex-hormone binding globulin was measured using an ^{125}I immunoradiometric assay (Diagnostic Systems Laboratories, Webster, TX) with a sensitivity of 3 nmol/L. Intra- and interassay variances were calculated to be < 5% and 10%, respectively. A LKB Model 1272 Cline gamma counter with on-line data reduction capabilities (Pharmacia LKB Nuclear, Gaithersburg, MD) was used to determine immunoreactivity. Blood lactate concentrations were measured in duplicate using a lactate analyzer (model 1500, Yellow Springs, OH).

Statistical Analysis

Regional fat distribution was characterized by the three indices outlined above: WHR, SB/TRi, and A/L_{FAT} . Based on each of these indices, the intact cohort of subjects was divided into tertiles and subsequently evaluated by analysis of variance for differences in physical characteristics. Three separate repeated measures analyses of variance then determined the effects of exercise and RFD on the hormonal (TT, FT, and SHBG) responses to the AHRET. When appropriate, a Tukey post hoc test was used to compare effect differences. Bivariate relationships were evaluated with Pearson product moment correlations. Multiple regression analyses determined the relationship between body morphology and testosterone concentrations and responses. An alpha level of $p \leq .05$ was used for all tests.

Results

Table 1 lists the physical characteristics of all subjects grouped by tertiles of the regional fat distribution indices (i.e., WHR, A/L_{FAT} , SB/TRi). For all 47 subjects, the mean physical characteristics follows: 22.4 ± 3.5 years, 164.5 ± 6.3 cm, 61.7 ± 7.7

Table 1 Physical Characteristics of Subjects Divided By WHR, MRL, and SB/TRI Tertiles

Subjects	Age	Height	Weight	%BF	BMI	Σ 7 SF	WHR	A/L _{FAT} Ratio	SB/TRI
All subjects	22.4 ± 3.5	164.5 ± 6.3	61.7 ± 7.7	24.6 ± 5.3	22.8 ± 2.6	130.1 ± 37.8	0.78 ± 0.05	0.36 ± 0.08	0.72 ± 0.20
WHR									
Tertile 1	22.3 ± 3.7	161.4 ± 5.7	60.8 ± 7.9	25.0 ± 6.4	23.3 ± 2.9	134.7 ± 47.0	0.73 ± 0.02	0.35 ± 0.07	0.68 ± 0.19
Tertile 2	22.5 ± 4.2	166.9 ± 6.1	63.1 ± 7.4	25.4 ± 4.8	22.6 ± 2.4	135.9 ± 33.4	0.78 ± 0.01	0.37 ± 0.07	0.67 ± 0.13
Tertile 3	22.4 ± 2.6	165.4 ± 6.1	61.3 ± 8.1	23.1 ± 4.6	22.4 ± 2.5	119.6 ± 30.7	0.83 ± 0.03	0.37 ± 0.09	0.81 ± 0.25
A/L_{FAT}									
Tertile 1	22.8 ± 3.4	164.4 ± 4.5	61.0 ± 6.3	23.1 ± 5.8	22.6 ± 2.5	120.9 ± 42.0	0.76 ± 0.05	0.28 ± 0.02	0.70 ± 0.16
Tertile 2	21.9 ± 3.1	164.4 ± 7.7	60.8 ± 8.1	23.5 ± 4.6	22.4 ± 2.3	122.9 ± 32.0	0.79 ± 0.05	0.37 ± 0.02	0.69 ± 0.13
Tertile 3	22.5 ± 4.3	165.1 ± 6.6	64.1 ± 9.0	27.3 ± 5.0	23.5 ± 3.1	149.4 ± 36.2	0.79 ± 0.03	0.46 ± 0.05	0.80 ± 0.31
SB/TRI									
Tertile 1	23.5 ± 4.4	162.9 ± 6.7	57.0 ± 7.1	22.5 ± 4.8	21.4 ± 1.6	115.6 ± 31.7	0.77 ± 0.04	0.37 ± 0.08	0.53 ± 0.07
Tertile 2	21.4 ± 2.5	166.1 ± 6.4	61.1 ± 6.1	23.1 ± 4.2	22.1 ± 1.6	119.8 ± 28.5	0.78 ± 0.04	0.35 ± 0.06	0.69 ± 0.03
Tertile 3	22.4 ± 3.3	164.4 ± 5.9	66.9 ± 6.9	28.0 ± 5.5	24.8 ± 3.0	154.6 ± 40.7	0.79 ± 0.05	0.38 ± 0.09	0.93 ± 0.20

Note. Sample sizes: Tertile 1 = 16, Tertile 2 = 16, Tertile 3 = 15.

kg, 24.6 ± 5.3 %BF, 22.8 ± 2.6 body mass index (BMI), and 130.1 ± 37.8 sum of seven skinfolds. When subjects were grouped into WHR tertiles, significant differences were found for height between tertile 1 and tertile 2. No significant differences were found for any of the physical characteristics when the subjects were divided by A/L_{FAT} tertiles. When subjects were grouped by SB/TRi tertiles, significant differences were found for body mass between tertiles 2 and 3, for %BF between tertile 1 and 3 and tertiles 2 and 3, for BMI between tertiles 2 and 3, and for sum of seven skinfolds between tertiles 1 and 3 and between tertiles 2 and 3.

Main Exercise Effects on Hormonal Concentrations

Main exercise effects were observed, demonstrating that the AHRET was a potent stimulus for acute increases in TT (1.24 vs. 1.55 nmol/L; ~25% increase), FT (7.18 vs. 9.0 pg/ml; ~25% increase), and SHBG (145.4 vs. 150.9 nmol/L; ~4% increase). Also, lactate was significantly elevated after AHRET (1.4 vs. 10.9 nmol/L; see Figure 2). The plasma volume loss after exercise was $8.2 \pm 5.6\%$.

Main Regional Fat Distribution Effects on Hormonal Concentrations

Figures 3–5 depict the hormonal concentrations for the regional fat distribution groupings: WHR, A/L_{FAT} ratio, and SB/TRi ratio. In Figure 3, no group effects were observed for any of the hormonal concentrations when the population was tertiled according to WHR. In Figure 4, when the groups were divided into tertiles of A/L_{FAT} , significant effects were observed only for FT. Free testosterone concentrations were higher in tertile 3 than either tertiles 1 or 2. For SB/TRi ratio in Figure 5, tertile 2 had

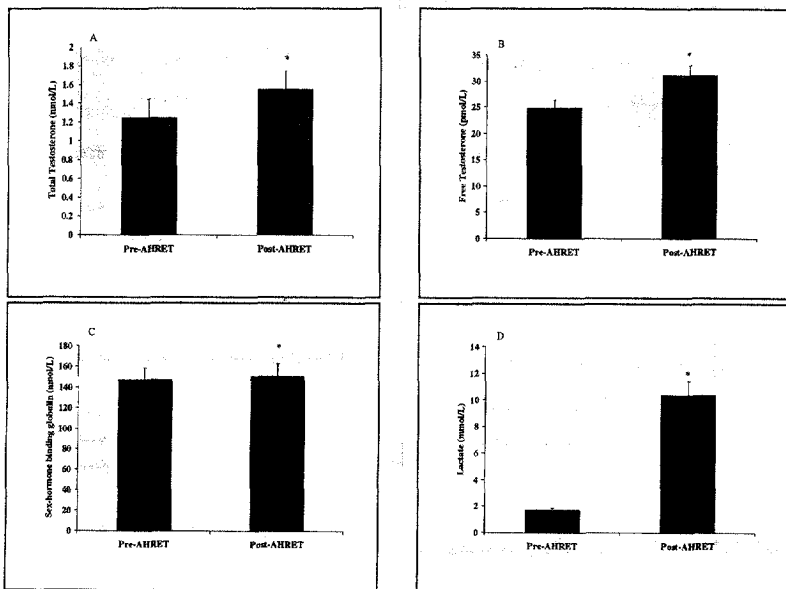


Figure 2—Hormonal responses after acute heavy resistance exercise for total testosterone (Figure 1A), free testosterone (Figure 1B), and sex hormone binding globulin (Figure 1C). Lactate responses are given in Figure 1D. * $p \leq .05$.

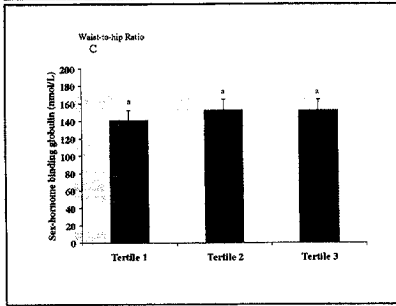
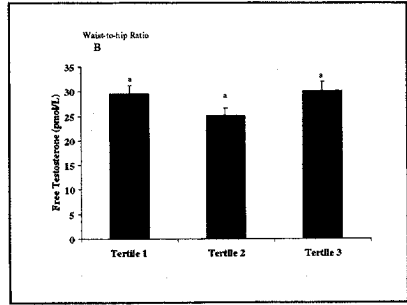
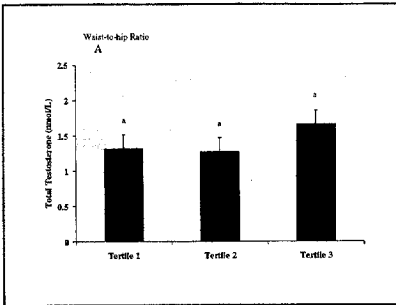


Figure 3— Main effects due to grouping by waist-to-hip ratio for total testosterone (Figure 2A), free testosterone (Figure 2B), and sex hormone binding globulin (Figure 2C) concentrations. Similar letters denote statistical similarity between groups, while different letters denote statistical difference.

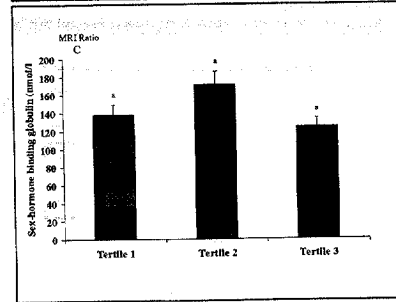
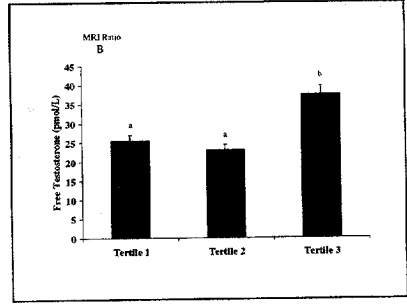
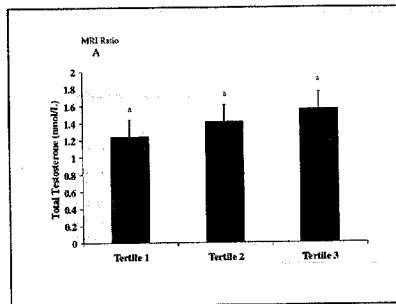


Figure 4— Main effects due to grouping by A/L_{FAT} ratio for total testosterone (Figure 2A), free testosterone (Figure 2B), and sex hormone binding globulin (Figure 2C) concentrations. Similar letters denote statistical similarity between groups, while different letters denote statistical difference.

higher concentrations than tertile 1 for TT and higher SHBG concentrations than tertile 3. Also, tertile 2 had higher concentrations of TT than tertile 1 and higher SHBG concentrations than tertile 3.

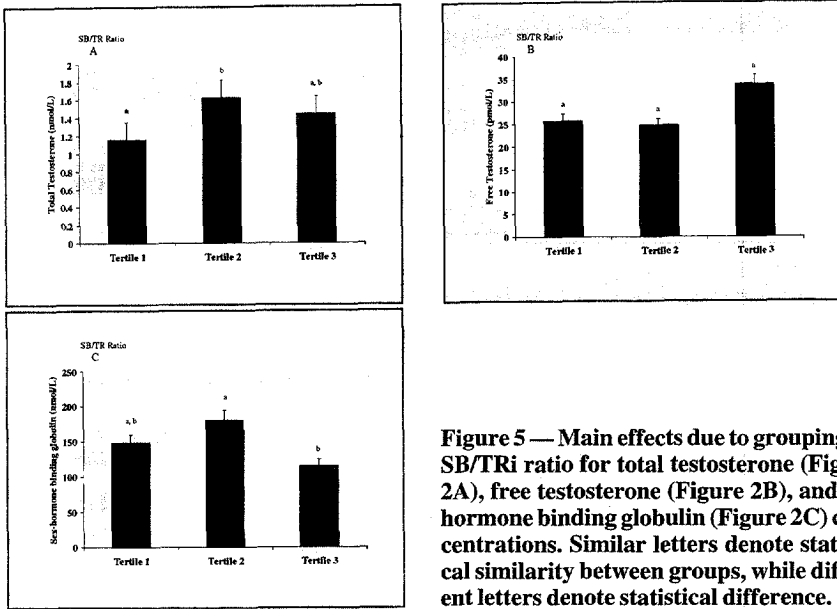


Figure 5 — Main effects due to grouping by SB/TRi ratio for total testosterone (Figure 2A), free testosterone (Figure 2B), and sex hormone binding globulin (Figure 2C) concentrations. Similar letters denote statistical similarity between groups, while different letters denote statistical difference.

Relationship Between Regional Fat Distribution and Hormonal Concentrations

Table 2 lists correlation coefficients between all skinfold and skinfold proportion measurements and pre-, post-, and percent change in TT, FT, and SHBG. The chest skinfold significantly correlated ($r = 0.34$) with percent change after AHRET in SHBG. The subscapular skinfold was significantly correlated with post-concentrations of TT ($r = 0.30$), pre- ($r = 0.37$), and post- ($r = 0.34$) concentrations of FT and percent change after AHRET in SHBG ($r = 0.30$). The abdomen skinfold correlated with pre- ($r = 0.47$) and post- ($r = 0.38$) concentrations of FT. No other significant correlations were found.

The proportion of the subscapular skinfold divided by the sum total of all skinfolds was correlated with post-concentrations of TT ($r = 0.37$) and FT ($r = 0.29$). The proportion of the abdominal skinfold to the sum total of all skinfolds correlated with pre-concentrations of FT. The proportion of the thigh skinfold to the sum total of all skinfolds was correlated with pre-concentrations of FT ($r = -0.30$). The ratio of subscapular/triceps correlated with post-concentrations of TT.

Table 3 shows the results of multiple regression analysis. Skinfold and skinfold proportions were used as predictor variables, and pre-, post-, and percent change values for TT, FT, and SHBG were used as outcome variables. There was no significant prediction equation for either pre-concentrations of TT or post-concentrations of SHBG. A significant regression equation was obtained for post-concentrations of TT, accounting for 14% of the variance. Significant equations were also obtained for both pre- and post-concentrations of FT, accounting for 34% and 38% of the variance, respectively. A prediction equation for pre-concentrations of SHBG accounted for 25% of the variance.

Table 2 Correlations Between Skinfold Measures, Skinfold Proportions, WHR, A/L_{FAT} , and SB/TRI and Hormonal Measures of TT, FT, and SHBG

Variable	Pre TT	Post TT	% Δ TT	Pre FT	Post FT	% Δ FT	Pre SHBG	Post SHBG	% Δ SHBG
Skinfolds									
Tricep	0.01	0.12	0.04	0.21	0.14	-0.18	-0.03	0.00	0.14
Chest	0.10	0.14	-0.01	0.28	0.20	-0.09	-0.10	-0.06	0.34*
Subscapular	0.15	0.30*	0.15	0.37*	0.34*	-0.10	-0.21	-0.18	0.30*
Mid-axillary	0.05	0.17	0.11	0.28	0.28	-0.06	-0.18	-0.15	0.27
Suprailiac	0.06	0.14	0.08	0.23	0.16	-0.16	-0.08	-0.06	0.17
Abdomen	0.20	0.29	-0.02	0.47*	0.38*	-0.21	-0.28	-0.25	0.17
Thigh	-0.01	0.09	0.18	0.19	0.15	-0.18	-0.08	-0.04	0.20
Skinfold proportion									
Tricep	0.00	-0.13	-0.03	-0.21	-0.20	-0.01	0.21	0.20	-0.28
Chest	0.07	0.02	-0.12	0.07	0.04	0.09	0.03	0.05	0.26
Subscapular	0.21	0.37*	0.17	0.27	0.29*	0.04	-0.16	-0.15	0.24
Mid-axillary	-0.06	0.07	0.13	0.12	0.17	0.09	-0.12	-0.11	0.27
Suprailiac	-0.07	-0.06	-0.03	-0.06	-0.16	-0.13	0.16	0.15	-0.05
Abdomen	0.15	0.17	-0.28	0.29*	0.20	-0.11	-0.26	-0.28	-0.12
Thigh	-0.23	-0.26	0.14	-0.30*	-0.23	0.02	0.11	0.12	-0.16
WHR	0.16	0.16	-0.15	0.04	0.03	0.07	0.11	0.08	-0.05
MRI	0.16	0.23	0.13	0.26	0.21	-0.03	-0.04	-0.03	0.02
SB/TRI	0.10	0.31	0.20	0.27	0.28	0.00	-0.21	-0.20	0.26

Note. WHR = waist-to-hip ratio; MRI = ratio of upper arm fat to mid-thigh fat as assessed via magnetic resonance imaging; SB/TRI = ratio of subscapular skinfold to tricep skinfold.

* $p \leq .05$.

Table 3 Multiple Linear Regression Results By Using Anthropometric Variables in Table 2 As Independent Variables and Hormonal Concentrations As Dependent Variables ($p \leq .05$)

Dependent variables	Equation	R	r ²	SEE
Pre TT	None			
Post TT	= 0.09 + 13.76 (A)	0.37	0.14	0.66
Pre FT	= 7.40 - 85.55 (B) + 0.43 (C)	0.58	0.34	3.73
Post FT	= 5.66 + 77.16 (A) - 123.48 (B) + 0.41 (C)	0.62	0.38	4.11
Pre SHBG	= 82.74 + 1575.39 (B) - 5.38 (C)	0.50	0.25	61.89
Post SHBG	None			

Note. None = no regression equation significantly predicted hormonal concentration; A = ratio of subscapular skinfold to sum total of skinfolds; B = ratio of suprailiac skinfold to sum total of skinfolds; C = abdominal skinfold.

Discussion

Effects of Exercise

This study has demonstrated significant elevations (~25%) above baseline concentrations for TT and FT after acute heavy resistance exercise in women. Whereas increases in TT in women after aerobic exercise have been reported after a marathon (18) and a 10-mile run (2), studies utilizing resistance exercise models have consistently reported no changes in TT (13, 31). In men, it has been suggested that there may be a threshold intensity, volume, and muscle mass necessary to produce increases in TT concentrations (16, 17). It is likely the same for females, and those prior studies have employed exercise models below this theoretical threshold. The lactate concentrations (10.4 mmol/L) after the AHRET in the present study (6 sets of 10 RM squats) are higher than reported by Kraemer et al. (16, 17) in two previous studies (7.6 and 7.9 mmol/L, respectively), thus attesting to the intensity of the AHRET used in this study. These previous studies utilized an exercise protocol that consisted of eight separate exercises (each performed at 10 RM for 3 sets with a 1-min rest between exercises) but did not include the squat exercise. The fact that this previous protocol involved more isolation-type exercises (i.e., exercises of local muscle groups), and required a longer duration and more total work to complete, strongly suggests that TT responses in women are mostly associated with the amount of total muscle mass recruited and activated and the intensity at which the exercise is performed.

The role of the exercise-induced rise in TT and FT is unclear. Testosterone is a steroid hormone whose classical signal transduction involves high-affinity binding to the cell nucleus and whose subsequent genomic stimulatory longer-term effects on gene transcription and muscle protein synthesis in men and women are well known (24, 32). It is tempting to speculate that the elevated TT and FT concentrations after exercise interact with the neuromuscular interface to mediate the

metabolic and cellular processes of muscle adaptation to resistance exercise and training (8, 23, 25, 28, 33). However, the results from our study should be interpreted with caution, as it has yet to be demonstrated whether increases in testosterone after exercise have any functional implications. Additionally, our results are based on a single point. The potential biological significance of acute exercise-induced testosterone increases in women warrants further study (16, 20).

The mechanism/origin of acute rises in testosterone in women must also be considered. Changes in plasma volume (i.e., hemoconcentration) account for some but not all of the increases observed for hormonal responses after exercise. Decreased hepatic clearance of total and free testosterone also cannot be excluded. It is probable that hemoconcentration and decreased clearance explain most, if not all, of the observed increase in testosterone for the women in this study. If any secretory mechanisms are operational, the likely source in women is the adrenal gland. Regardless of the mechanism, after the AHRET, the body's tissues were potentially exposed to greater concentrations of testosterone, notably free or "bioavailable" testosterone. This finding is of particular significance, as two separate studies have demonstrated that concentrations of testosterone in women are predictive of the trainability of their neuromuscular system (10, 18). Additionally, the transporter protein SHBG may become increasingly more important in modulating the androgenic milieu with repetitive exercise sessions, as we have previously shown increases at rest for circulating SHBG in women after 6 weeks of resistance training (17).

Effects of Regional Fat Distribution

For the women in this study, significant correlations were evident between anthropometric measures of adiposity and serum concentrations of androgens. Multiple regression analyses also predicted hormonal concentrations from anthropometric measures. The highest relationships were noted between the subscapular and abdominal skinfolds and free testosterone, both before and after the AHRET (r values ranging from 0.34 to 0.47). Additionally, the proportion of the subscapular skinfold to the sum total of all skinfolds was correlated with post-AHRET concentrations of both total and free testosterone ($r = 0.30$ and 0.34 , respectively). Nearly 40% of the variance in FT concentrations for the women in this study were accounted for by skinfold measures (viz. ratio of suprailliac to sum total of skinfolds, abdominal skinfold, and ratio of subscapular ratio to sum total of skinfolds). In a population ($n = 25$) that ranged in body mass index from 19.3 to 48.1 kg/m², Pederson et al. (22) also reported a significant correlation ($r = 0.64$) between abdominal fat (assessed with DEXA) and FT. In another study using a heterogeneous population of ranging adiposity, Kissebah et al. (15) reported that the subscapular and suprailliac skinfolds correlated significantly with impaired glucose tolerance and hyperinsulinemia during an oral glucose tolerance test suggesting a link to metabolic aberrations (i.e., diminished insulin sensitivity). Whether or not the association of RFD and metabolic parameters also existed in this population could not be determined from the present experimental paradigm. To our knowledge, the clinical significance of the association between skinfolds and androgenicity in non-obese population has not yet been established. Our data extend previous work by demonstrating that the relationship between markers of central adiposity and androgenicity also occur in a healthy non-obese population more homogenous with respect to body mass index and percentage body fat. However, due to the large number of correlations that were

calculated, the data should be viewed with caution due to the possible presence of a type I error.

This study did not find any significant differences in TT, FT, or SHBG concentrations when subjects were divided into WHR tertiles. These findings are in contrast to those of Kissebah et al. (15) and others who reported increased FT and decreased SHBG as WHR rose. While the WHR measure is "field expedient" and convenient to use in a clinical setting, the ability of the WHR to delineate androgen concentrations in healthy, non-obese women is questionable. Haffner et al. (9) suggested that the WHR ratio and SB/TRi ratio measure different aspects of regional fat variation. For example, Seidell et al. (29) reported different relationships between WHR and SB/TRi and cardiovascular risk factors. In the current study, differences in hormonal concentrations were observed when the subjects were divided into tertiles of SB/TRi ratio and the A/L_{FAT} ratio. The WHR reflects abdominal versus gluteal fat distribution, the SB/TRi reflects upper-body subcutaneous fat distribution, and the A/L_{FAT} ratio reflects upper limb versus lower limb adiposity. Thus, as previous studies have reported that different anthropometric indices of regional fat distribution convey different information concerning cardiovascular health risk, this study has also demonstrated that different anthropometric indices also demonstrate different relationships with testosterone concentrations. These findings are likely attributable to the various indices assessing divergent aspects of subcutaneous/intramuscular fat levels.

It is interesting to note that both the SB/TRi and A/L_{FAT} ratios involved a measure of arm adiposity. Fat tissue of the arm is known to serve as a reservoir for fat storage in women and to exhibit greater relative losses after physical training in women (21). One provocative hypothesis may be that in young, non-obese women, elevated arm adiposity is predictive of future metabolic derangements that are eventually manifested as more central fat distribution. A longitudinal study tracking the life spans of young, non-obese women who later become obese would be particularly useful in providing insight into the etiology of obesity.

Despite the observed relationship between indices of adiposity and androgen concentrations, women in this study who differed with respect to indices of RFD (viz. WHR, A/L_{FAT} , and SB/TRi ratios) responded similarly to the stress of resistance exercise with regard to TT, FT, and SHBG concentrations (positive main exercise effects were evident as discussed previously). Contrary to our original hypothesis, women with higher relative amounts of upper body adiposity did not show an accentuated hormonal response following exercise. Therefore, previous negative findings of testosterone concentrations after exercise are likely due to differences in exercise protocols and not in regional fat distribution among subjects, at least in a young, non-obese population.

In summary, an acute, heavy resistance exercise bout elicited significant rises (~25%) in total and free testosterone in young, healthy, non-obese women. Hemoconcentration and reduced hepatic clearance, not secretion, are probable mechanisms accounting for this increase among women. Of particular physiological relevance is the increase in free or "bioavailable" testosterone. The role of this increase is presently unclear but could serve to augment neuromuscular performance and metabolism. Additionally, associations were observed between skinfold measures and testosterone concentrations. Regression analyses explained 34% and 38%, respectively, of the variance in pre- and post-exercise concentrations of FT. When the subjects were grouped according to tertiles of A/L_{FAT} and SB/TRi ratios but not the

WHR ratios, differences in hormonal concentrations were observed. Interestingly, the indices that were discriminatory for hormonal concentrations involved a measure of upper arm adiposity. In conclusion, resistance exercise can induce transient increases in androgen concentrations in young, healthy women.

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