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Resistance training increases SHBG in overweight/obese, young men

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Abstract

Objective—Evidence suggests that SHBG affects glycemic control, predicts both T2D and metabolic syndrome, and is low in obese subjects. We sought to determine if resistance exercise training (RT) can increase sex hormone-binding globulin (SHBG) and ameliorate levels of related steroid hormones in overweight/obese, sedentary young men.

Materials/Methods—36 participants (BMI 31.4 kg/m^2, age 22 years) were randomized into an RT (12 weeks of training, 3/week) or control group (C, 12 weeks no training), and assessed for changes in SHBG, cortisol, testosterone, free testosterone (FT) and free androgen index (FAI). In addition, body composition and oral glucose tolerance testing was performed.

Results—12 weeks of RT increased SHBG (P=0.01) and decreased FAI (P<0.05) and cortisol (P<0.05) compared to C. FT decreased in RT (P=0.01). Total testosterone did not change in either group. These changes were noted without weight loss, and in concert with increases in lean body mass (P=0.0002 vs C) and decreases in glucose area under the curve (AUC) (*P*= 0.004), insulin AUC (*P*=0.03), and total (*P*=0.002) and trunk (*P*=0.003) fat mass in RT.

Conclusion—In overweight/obese young men, RT increases SHBG and lowers FAI in obese young adult men.

Keywords

Steroid hormone; Cortisol; Testosterone; Insulin sensitivity; Strength training; Exercise

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Author contributions

C.K.R. conception and design of research; D.M.C. led training intervention; N.A. performed experiments; D.M.C. analyzed data. C.K.R., D.M.C. interpreted results of experiments; C.K.R. drafted manuscript; C.K.R., D.M.C., N.A., A.W.B, and C.C.L. edited manuscript; C.K.R., D.M.C., N.A., A.W.B, and C.C.L. approved final version of manuscript.

Conflict of interest

The authors declare no conflict of interest.

1. Introduction

Type 2 diabetes (T2D) has progressed into a major cause of preventable death over the past half century, increasing from just over one million diagnosed in 1958 to nearly 21 million in 2010 [1]. An additional ~7 million are undiagnosed with T2D and approximately 80 million exhibit prediabetes [2]. Thus, prevention of future T2D in present day young adults is a major public health challenge. Resistance training (RT) may represent a preventive strategy for T2D [3], as it improves insulin sensitivity and glucose tolerance, independent of weight loss [4–7]).

Independent of traditional risk factors, biochemical markers have been identified that may be associated with increased risk of T2D, such as sex-steroid hormones and sex hormone-binding globulin (SHBG). The function of SHBG has classically been ascribed to the binding of steroid hormones in circulation to regulate their bioavailability. Because SHBG is decreased with obesity, it was thought that SHBG may be a marker for obesity in relation to T2D risk. However, evidence suggests that SHBG independently affects glycemic control [8,9] and predicts both T2D [10–12] and metabolic syndrome [13]. In addition, it is known that insulin [14,15] and glucose [16] also have reciprocal action on SHBG to regulate SHBG production in the liver.

To date the studies that have investigated the effects of RT on SHBG [17–20] have generally noted that RT does not affect SHBG. However, these studies were performed in healthy young [17,20] and middle-aged men [18] or older men and women [19]. The effect of RT on SHBG in obese young subjects is unknown.

We investigated if an RT intervention (12 weeks, 3 sessions/week) can ameliorate low levels of SHBG as well as levels of related steroid hormones in sedentary, overweight/ obese young men. Our primary hypothesis was that RT would increase SHBG in concert with improved glucose tolerance and body composition, independent of weight loss.

2. Methods

2.1. Study participants

Of the 94 participants who attended the first screening visit, 75 were consented and qualified for the study, 49 attended their pre-test visit and were subsequently randomized to either RT or C. 36 young adult (ages 18–35) males who were overweight/ obese (BMI≥27 kg/m^2) completed the study (Fig. 1).At baseline, participants were sedentary (participated in light-intensity physical activities ≤2 times/wk) and did not exhibit any other overt chronic disease symptoms, as indicated by a screening comprehensive history and physical examination. Potential participants who had: 1) documented CVD, cardiac surgery, or any heart arrhythmia found on an electrocardiogram (ECG) reading, 2) participated in a structured exercise, nutrition, or weight loss program within the previous 6 months, or 3) used tobacco products or medications that influence cardiovascular function, body composition or insulin indices in the prior 6 months, were excluded from the study. Participants were instructed to maintain their normal *ad-libitum* diet and normal activities of daily living. Pre- and post-intervention assessments were made at weeks 0 and 13, respectively. All of the study protocols were approved by the University of California, Los Angeles Institutional Review Board and were performed according to the Declaration of Helsinki.

2.2. Randomization

Following their pre-intervention assessment, participants were randomized into one of two groups at a 1:3 control (C, n=8) to resistance training (RT, n=28) ratio. Both groups were reminded to maintain their normal *ad-libitum* diet and normal activities of daily living.

Participants randomized to the C group completed a 12-week control period without RT. Pre-and post-intervention assessments were made at weeks 0 and 13, respectively.

2.3. Resistance training intervention and muscular strength testing

All training occurred at the John Wooden Recreation Center at UCLA. Participants in the RT group completed 12 weeks of RT at three supervised sessions/week, with each session lasting approximately one hour. The training overload was modified using a linear periodization model with 3 phases. During phase 1 (weeks 1–2), participants completed two sets of 12–15 repetitions for each exercise at 100% of their approximated 12–15 repetition maximum (RM). In phase 2 (weeks 3–7) participants completed three sets of 8–12 repetitions at 100% of their 8–12 RM, and in phase 3 (weeks 8–12) participants completed 6–8 repetitions at 100% of their 6–8 RM. As participants adapted to the training overload, the weight was increased to maintain the prescribed training intensity. All participants trained on 3 non-consecutive days/week, rotating between two daily workout regimens. Workout I consisted of dumbbell (DB) squat, cable row, DB front lunge, DB row, barbell (BB) deadlift, DB triceps extension, and DB curl. Workout II was DB step-up, BB chest press, machine squat, DB overhead press, DB incline chest press, DB side raise, DB reverse fly, and abdominal crunches. A certified personal trainer led all training sessions with a maximum 3:1 participant to trainer ratio.

Maximal strength testing consisted of 1-RM lifts for the barbell bench press, 45° incline leg press (Hammer Strength Linear Leg Press), and machine-seated row (Life Fitness Pro2 series; all Life Fitness products, Schiller Park, IL, USA). Participants first warmed up each muscle group by performing 8–10 repetitions with weight equivalent to 40%–60% of their estimated 1-RM. The weight was progressively increased while decreasing the repetitions until participants could safely attempt an estimated 1-RM for each exercise. A successful 1-RM occurred on the penultimate set, having failed their last set. Participants were allowed 3–4 min of rest between all sets. All participants performed a total of 2 maximal strength tests: the RT group participants performed one immediately preceding the first training session and the second immediately preceding their penultimate training session, while the C group participants performed the tests at weeks 0 and 13 after their outpatient visits to prevent any acute exercise effects. Relative strength measures were calculated by dividing each measure by participant body weight.

2.4. Outpatient visit procedures

Measurements were taken from participants at baseline (pre-test) and on week 13 (post-test). While selecting the precise time from the last bout of training to evaluate primary outcome variables is debatable, to assess predominately chronic adaptations of the training program, the outpatient visit occurred approximately 72 h after the last training session. Before each visit, participants were reminded to: 1) avoid all moderate to vigorous physical activity 24 hours prior to testing and 2) to abstain from all food and drink (except water) for approximately 12 h prior to each visit. Verbal confirmation of adherence to the aforementioned criteria was obtained immediately prior to all testing.

The outpatient procedures at the Clinical and Translational Research Center (CTRC) began at 7:30 am and typically lasted 3.5 h.A 12-lead ECG was administered as a safety measure and checked by a physician before allowing any participation in exercise testing/ intervention. Height, weight and waist circumference were measured in duplicate in all participants. Fasting blood samples were collected and serum was separated and stored at −80 °C until assayed. Subsequently, a 2-h oral glucose tolerance test (OGTT) was performed.

2.5. Body composition

Body composition was determined by dual energy x-ray absorptiometry (DXA) scan (Hologic QDR4500 Fan Beam X-ray Densitometer, Hologic, Waltham, MA).

2.6. Steroid hormone assays

Plasma levels of SHBG, cortisol and testosterone were measured by an electrochemiluminescent immunoassay (ECLIA) on an Elecsys 2010 autoanalyzer (Roche Diagnostics, Indianapolis, IN) at the UCLA Clinical and Translational Research Laboratory (CTRL). The coefficients of variation for SHBG, cortisol and testosterone test results from blinded quality-control samples were 4.3%, 3.8%, and 6.2% respectively. Free testosterone (FT) was calculated via the Sodergard method [21]. FAI was calculated by 100*(Total Testosterone/SHBG).

2.7. Oral glucose tolerance test

The participants completed a 2-h OGTT using 75 g of anhydrous glucose dissolved in water. Venous blood samples were obtained at baseline and every 30 min (−30, 0, 30, 60, 90 and 120, relative to glucose ingestion), and were assayed for glucose and insulin. UCLA CTRL analyzed serum glucose via in vitro hexokinase method (Olympus AU400 Chemistry Analyzer, Beckman Coulter, North America Commercial Operations, Irving, TX 75063, USA). Serum insulin was measured by solid-phase, enzyme-labeled chemiluminescent immunometric assay (Immulite® 2000, Diagnostic Products, Los Angeles, CA) by the UCLA Clinical Laboratories.

Total area under the glucose and insulin curves (AUC) was calculated by trapezoidal rule. AUC from 0 to 120 min was calculated for glucose ($G_{AUC(0–120)}$) and insulin ($I_{AUC(0–120)}$). Glucose and insulin at time points 0 (fasting) and 120 min (2-h), and AUC measures were used as indicators of glucose tolerance and insulin resistance. Glycated hemoglobin (HbA1c) was measured via DCA Vantage® Analyzer (Siemens Medical Solutions Diagnostics, New York, USA).

2.8. Statistical analyses

Non-parametric analyses were chosen for statistical inference due to the presence of non-normally distributed data, unequal samples sizes, and heteroscedasticity. The overall effects of the intervention were tested for evaluable participants. Significance was calculated using Wilcoxon signed-rank test. Confidence intervals were derived from bias-corrected bootstrap methods (100,000 permutations). Data are reported as median (interquartile range) unless stated otherwise. Statistical analyses were performed with the use of Stata 11.2 statistical software (StataCorp LP., College Station, TX). A p-value of <0.05 was considered statistically significant.

3. Results

A total of 36 participants, 28 in the RT group and 8 in the C group, finished the pre- and post-test visits (Fig. 1). At baseline, there were no significant differences between groups. Additionally, comparing baseline anthropometrics, there were no differences between the 13 participants who did not continue following their pre-test visit and the 36 participants who completed the entire study (all $P>0.2$).

3.1. Body composition, strength and OGTT

In total, participants in the RT group attended 99.7% of their training sessions. Table 1 illustrates changes in anthropometric data for between- and within-group effects. LBM

(*P*=0.0002, Fig. 2) and 1RM strength in chest, leg, and row significantly increased in the RT compared to C (all *P*<0.05). Total (*P*<0.002) and trunk (*P*<0.003) fat mass decreased significantly in the RT group (Fig. 2). There was no change in BMI, waist circumference (WC), or body weight between groups, although body weight (*P*=0.07) and BMI (*P*=0.06) trended to increase in RT vs. C and BMI increased within the RT group (*P*=0.03). There was no change in HbA1c within- or between-groups. Both fasting insulin and glucose trended to increase in RT vs. C (*P*=0.054, *P*=0.05, respectively). However, glucose AUC and 2-h glucose trended to decrease in RT vs. C (*P*=0.07, *P*=0.05, respectively). Glucose AUC (*P*=0.004), insulin AUC (*P*= 0.03), 2-h glucose (*P*=0.007), and 2-h insulin (*P*=0.002) all decreased significantly in RT.

3.2. Steroid hormones

Table 2 indicates that compared to C, SHBG increased (*P*= 0.005) and FAI (*P*<0.05) and cortisol (*P*<0.05) decreased in RT. FT decreased in RT (*P*=0.01) and total testosterone did not change in either group. Percent changes in steroid hormone indices are depicted in Fig. 3.

3.3. Individual responses

Fig. 4 represents the percent change from pre-test to post-test in each individual for SHBG sorted by LBM (4A), strength score (4B) and total fat mass (4C). Notably, although nearly all subjects in RT exhibited an increase in SHBG, the effect was highly variable. The individualized effect of RT on SHBG was generally unrelated to the effects on these outcomes.

4. Discussion

Recently, there is evidence to suggest that steroid hormone biology plays a role in metabolic disease. For example, although the function of SHBG has classically been ascribed to the binding of steroid hormones in circulation to regulate their bioavailability, SHBG has been demonstrated to affect glycemic control [8,9] and to predict both T2D [10–12] and metabolic syndrome [13].

We investigated the effects of an RT intervention on SHBG, cortisol, testosterone and indices of free androgens in sedentary, overweight/obese young men. We noted that: 1) RT increased SHBG, and decreased cortisol and FAI, compared to C; 2) FT decreased in RT; 3) these changes occurred in conjunction with improvements in glucose tolerance, strength, LBM, and decreases in total and trunk fat mass, but in the absence of weight loss; and 4) the effects of RT on SHBG exhibited significant individual variability. These results supported our primary hypothesis, that RT would increase SHBG, independent of weight loss.

Our findings are in contrast to studies that have demonstrated SHBG does not change in young men with RT [17,20,22], and these findings were also noted in middle-aged [18] or older men and women [19]. However, Daly et al. [23] noted that in older overweight adults with T2D, despite no change in SHBG with weight loss (low calorie diet) compared with weight loss+RT, there was a within group increase in SHBG after 6 months in the group performing RT. To our knowledge, ours is the first study to determine the effects of RT alone on SHBG in young obese men. We noted an increase in SHBG of ~25% relative to the change in the control group (Fig. 3), and it is likely that the main difference in the results of the aforementioned studies and our study was the baseline phenotypes of the subjects. The implications of an increase in SHBG is unknown, however it has been suggested that low SHBG is associated with higher rates of obesity [24] and T2D [10–12]. Interestingly, the increase in SHBG that we noted occurred in the absence of weight loss. In fact, due to

greater increases in LBM than decreases in fat mass, body weight of these overweight/obese young men increased with RT. It is apparent that the relationships between metabolic disease risk factors (body weight, fat mass, SHBG, etc) is complex and not simply a function of weight loss improving metabolic risk profile and weight gain resulting in a more deleterious profile. The increase in SHBG was accompanied by improvements in other metabolic risk factors associated with risk of T2D, metabolic syndrome or mortality including LBM, strength, fat mass and glucose tolerance. The increase in SHBG may be related to the change in glucose tolerance as it is known that both insulin [14,15] and glucose [16] regulate SHBG production in the liver. Selva et al. [16] elegantly demonstrated that elevated glucose (and fructose), rather than insulin might be the primary stimulus to lower SHBG. In this study, transgenic mice expressing different SHBG transgenes exposed to diets with elevated monosaccharides led to large decreases in SHBG.

Levels of testosterone have also been found to be associated with insulin resistance [25,26], visceral adiposity [27], diabetes [28,29] and metabolic syndrome [30]. However, we did not note a change in testosterone after RT, although FT did exhibit a small but statistically significant decrease. The implications of this finding are unknown, but the improvements in body composition (increased LBM, decreased total and trunk fat mass), strength, and insulin dynamics occurred without increases in total or FT. The decrease in FAI, indicative of biologically active testosterone, is likely due to the increase in SHBG since total testosterone did not change. This suggests that increased levels of bioavailable androgens are not required for the aforementioned improvements in metabolic risk variables with RT intervention. Similarly, the improvement in strength and increase in LBM noted are likely independent of change in androgen levels [31].

Historically, it has been demonstrated that higher cortisol levels are noted in obesity, and thus may be noted in obese patients with T2D and/or metabolic syndrome. Indeed, several studies have reported increased cortisol in subjects with T2D and metabolic syndrome [32,33]. However, in a recent study by DeSantis et al. [34], salivary cortisol was not related to metabolic syndrome. We noted that RT decreased cortisol compared to C. This would be consistent with the expected change in cortisol associated with an improved metabolic profile. Given that the effects of cortisol on metabolic syndrome and T2D are controversial, the implications of this finding remain unclear.

What also has been underappreciated is the individual responsiveness to different forms of exercise training. It has previously been demonstrated that aerobic exercise training responses are highly variable (see Fig. G.2.4 in Refs. [3] and [35]). Determination of individual responses with RT is important as we move toward individualizing exercise training programs. We noted strength increases of 2%–60% and LBM of <1% to 9%, with decreases in fat mass of −10% (a 10% gain to 30% decrease). This occurred in conjunction with highly variable responsiveness for SHBG (35% decrease to 130% increase). Thus, the improvements in SHBG and other phenotypic outcomes – across subject LBM, strength and fat mass improvements – were highly variable, and suggest the existence of a complex relationship, whereby genetic variation, training motivation and other factors likely account for the spectrum of responses noted.

In summary, in overweight/obese young men, RT increases SHBG and decreases both cortisol and FAI.The primary strength of this study is the novel investigation of the effects of RT on SHBG and steroid hormones in an early risk population of young obese men. Limitations of this study include the small sample size and gender-specific population. The implications of the effects of RT on SHBG and its relationship with T2D and metabolic syndrome warrant further study.

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Abbreviations

T2D	Type 2 diabetes
RT	Resistance training
C	Control
SHBG	Sex hormone-binding globulin
FT	Free testosterone
FAI	Free androgen index
AUC	Area under the curve
CVD	Cardiovascular disease
ECG	Electrocardiogram
RM	Repetition maximum
DB	Dumbbell
BB	Barbell
CTRC	Clinical and Translational Research Center
OGTT	Oral glucose tolerance test
DXA	Dual energy x-ray absorptiometry
CTRL	Clinical and Translational Research Laboratory
LBM	Lean body mass
BMI	Body mass index
WC	Waist circumference
MAD	Median absolute deviation

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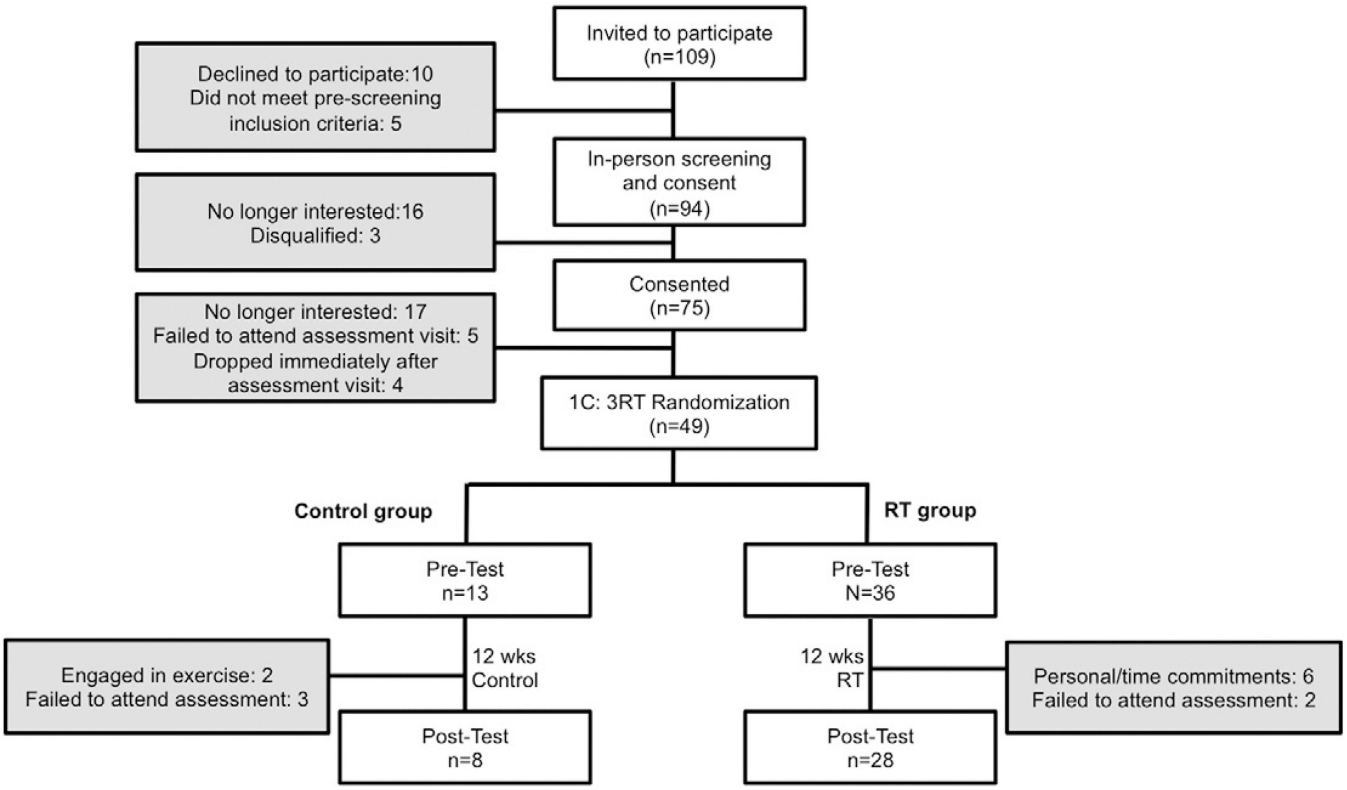

Fig. 1.
Participant Flow Diagram.

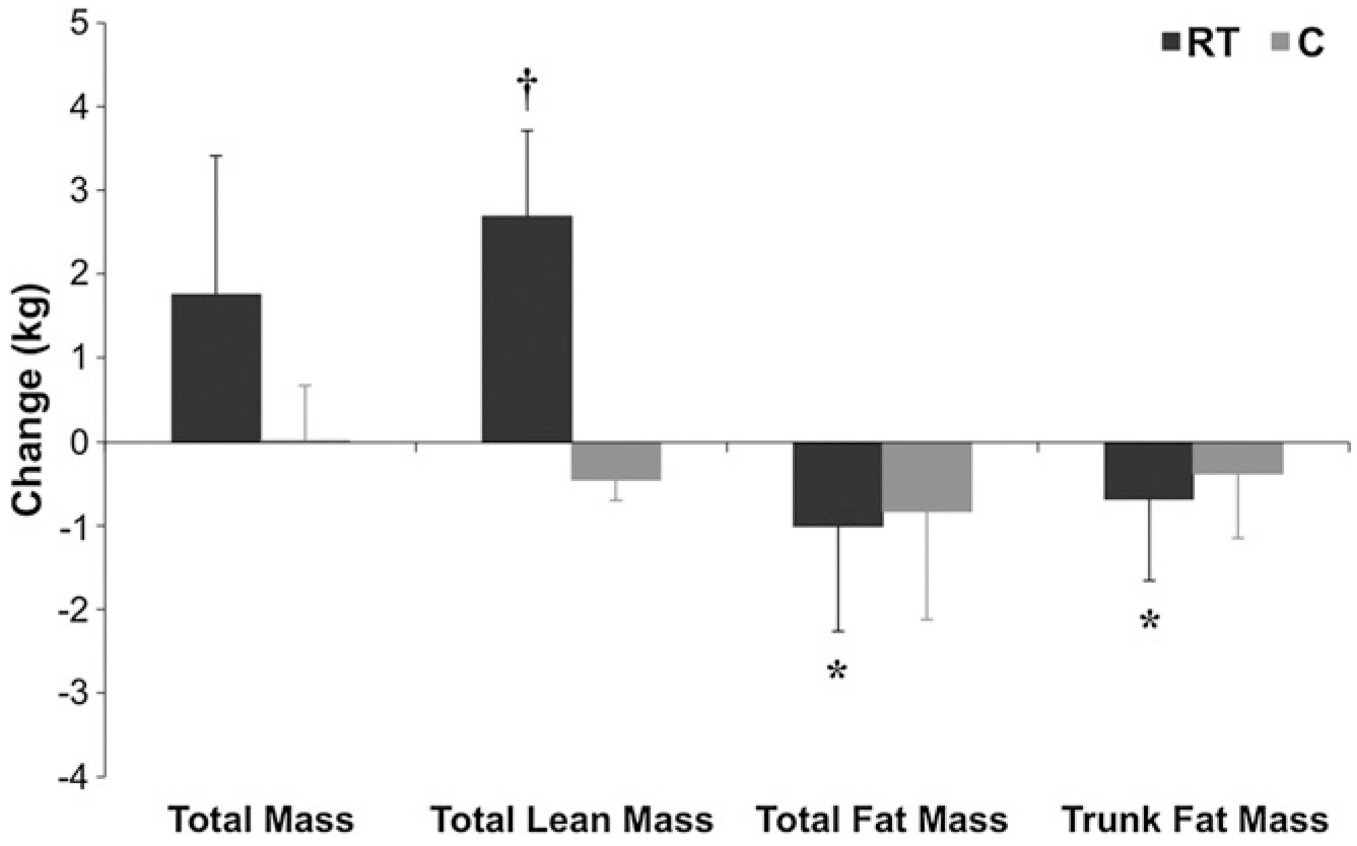

Fig. 2.

Effects of RT on Body Composition. Bar graphs present median and median absolute deviation (MAD) for pre- and post-test. *P<0.01 within RT; †P=0.0002 between RT (n=28) and C (n=8).

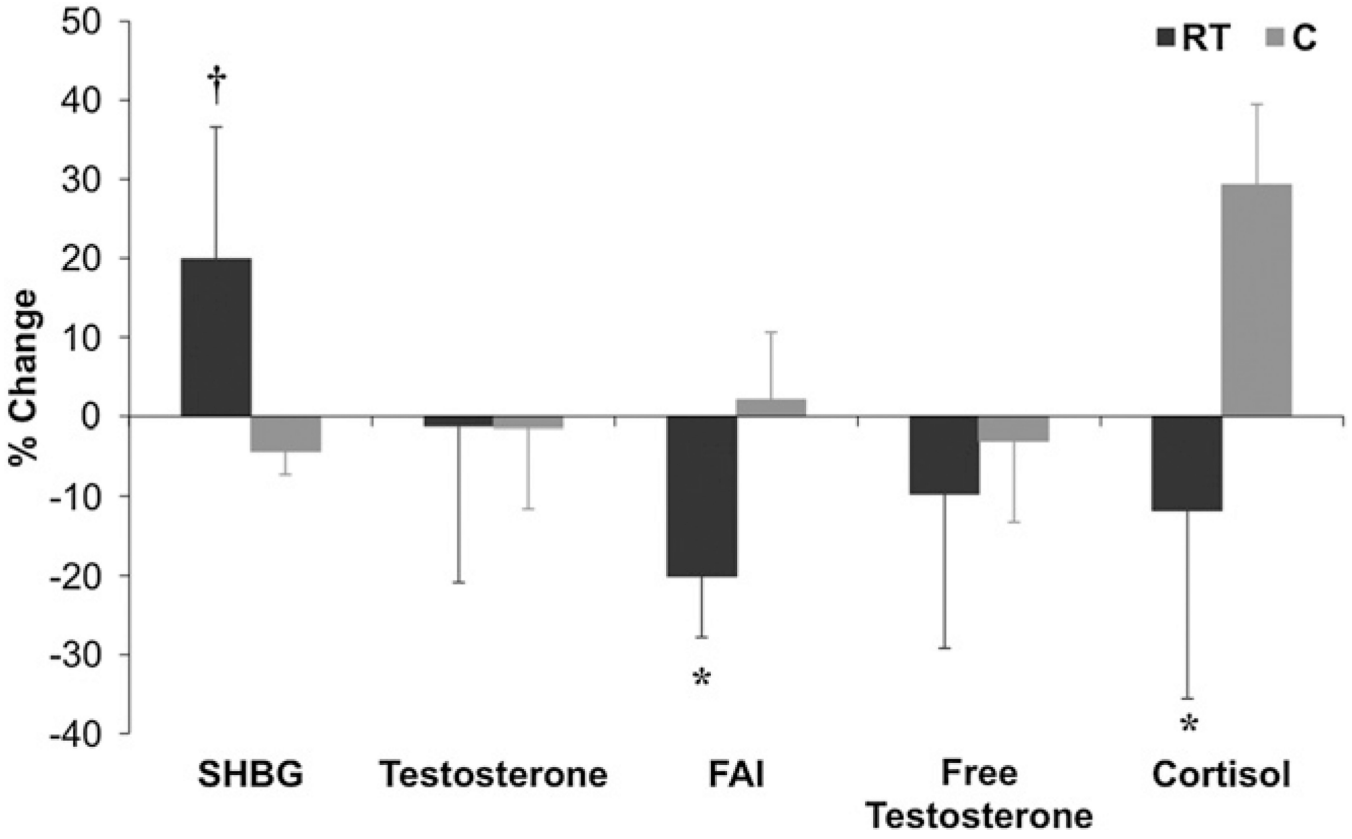

Fig. 3.

Effects of RT on Steroid Hormone Changes. Bar graphs present median and median absolute deviation (MAD) for pre- and post-test. **P*<0.05 between RT and C; †*P*=0.005 between RT (n=27) and C (n=7).

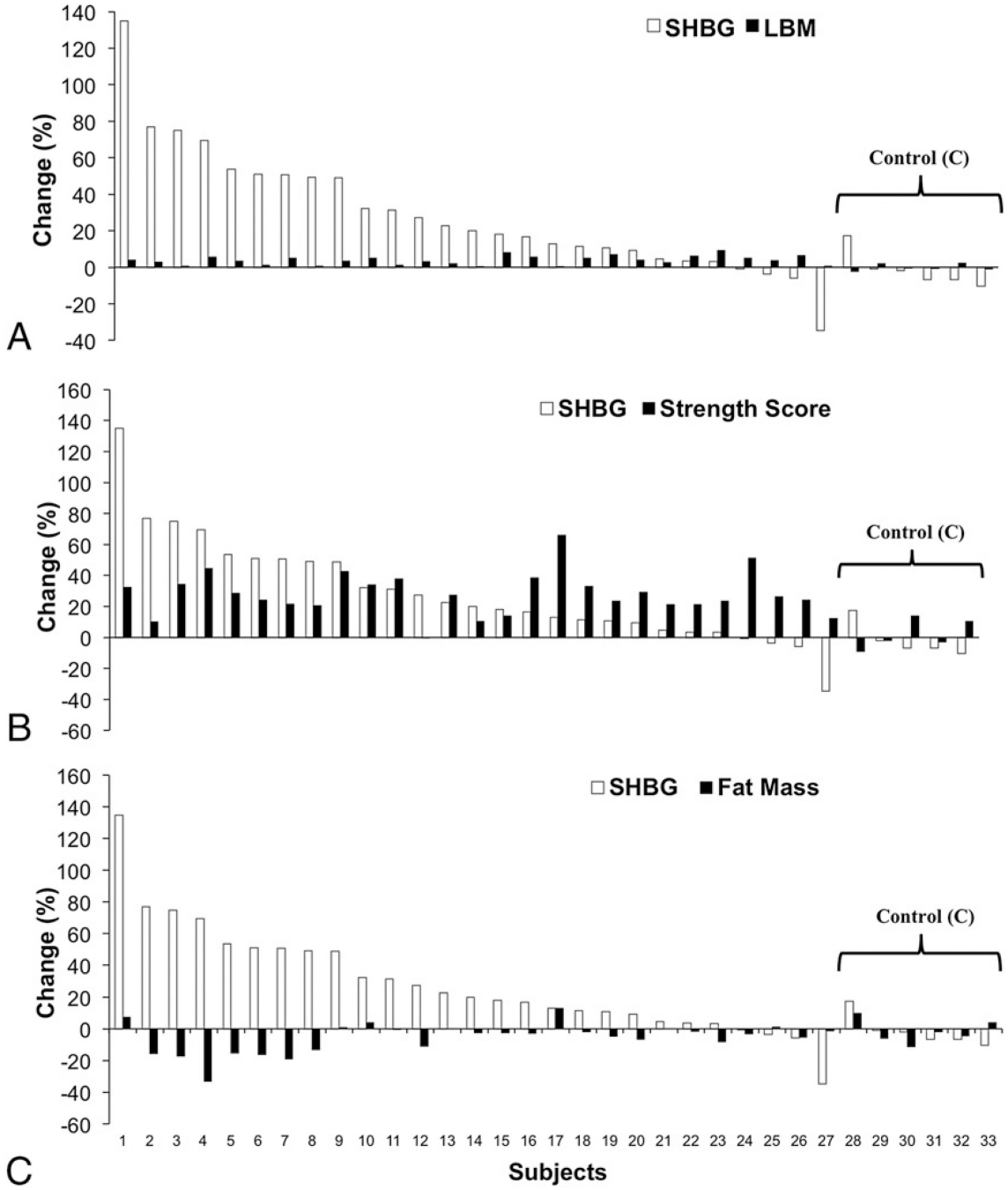

Fig. 4.

Individual Responses. Individual responsiveness to 12-week RT intervention presented as a percent change from pre-test values and sorted from greatest to least training response for SHBG sorted by LBM (A), strength score (B) and total fat mass (C). RT (n=27) C (n=6).

Table 1

Effects of RT on Body Composition, Strength, and OGTT.

Outcomes	Median (25th–75th percentile)		Median (95% CI)		P value
	Pre-test	Post-test	Within-Group Changes	Between-Group Changes	
Age (years)				–	_*b*
Control	22.0 (20.8–22.8)		–		_*w*
RT	21.5 (20.0–23.0)		–		_*w*
Height (m)				–	–
Control	1.74 (1.70–1.77)		–		–
RT	1.77 (1.73–1.81)		–		–
Weight (kg)				1.7 (−0.56 to 3.7)	0.07
Control	98.5 (91.6–106.2)	98.0 (90.9–105.9)	0.02 (−1.4 to 0.57)		0.58
RT	96.6 (90.0–103.5)	97.1 (91.2–105.4)	1.8 (0.00 to 3.0)		0.06
BMI (kg/m2)				0.58 (−0.22 to 1.4)	0.06
Control	33.6 (31.2–34.7)	33.2 (31.3–34.6)	−0.19 (−0.57 to 0.19)		0.16
RT	30.9 (29.7–32.7)	31.2 (30.3–32.7)	0.39 (−0.18 to 0.96)		**0.03**
WC (cm)				−0.27 (−3.4 to 2.9)	0.77
Control	106.5 (96.1–110.8)	106.7 (93.6–112.0)	−0.28 (−2.0 to 1.9)		0.83
RT	103.3 (99.4–111.3)	101.4 (96.8–108.7)	−0.55 (−2.4 to 0.85)		0.24
Total Fat Mass (kg)				−0.18 (−2.7 to 1.1)	0.25
Control	25.0 (22.3–32.3)	26.1 (22.1–31.0)	−0.82 (−1.9 to 1.3)		0.58
RT	27.9 (23.7–32.6)	26.2 (20.5–30.1)	−1.0 (−2.9 to −0.44)		**0.002**
Trunk Fat Mass (kg)				−0.30 (−1.5 to 0.78)	0.36
Control	12.7 (11.5–17.2)	12.7 (11.2–16.4)	−0.38 (−1.3 to 0.90)		0.48
RT	13.6 (11.6–16.9)	12.8 (10.4–15.8)	−0.68 (−1.5 to −0.33)		**0.003**
Lean Mass (kg)				3.1 (1.2 to 4.1)	**0.0002**
Control	69.3 (68.0–71.9)	70.2 (68.6–71.4)	−0.45 (−0.70 to 1.4)		0.58
RT	69.5 (64.7–72.8)	70.9 (66.5–76.2)	2.7 (2.0 to 3.4)		**<0.0001**
1RM Chest (kg)				15.9 (10.2 to 21.6)	**<0.001**
Control	70.3 (65.8–96.4)	70.3 (65.8–99.8)	0.00 (0.00 to 13.6)		0.45

Outcomes	Median (25th–75th percentile)		Median (95% CI)		P value
	Pre-test	Post-test	Within-Group Changes	Between-Group Changes	
RT	70.3 (56.7–80.5)	86.2 (70.3–94.1)	15.9 (13.6 to 19.3)		**<0.0001**
1RM Leg (kg)				59.0 (15.9 to 95.3)	**<0.001**
Control	272.2 (272.2–347.0)	281.2 (260.8–349.3)	9.1 (−9.1 to 54.4)		0.45
RT	251.7 (237.0–293.7)	331.1 (311.9–365.1)	68.0 (56.7 to 90.7)		**<0.0001**
1RM Row (kg)				18.1 (4.5 to 29.5)	**0.015**
Control	81.7 (70.3–95.3)	88.5 (82.8–89.6)	−2.3 (−4.5 to 17.0)		0.67
RT	79.4 (72.0–90.7)	94.1 (90.2–104.9)	15.9 (13.6 to 20.4)		**<0.0001**
Strength Score (kg)				77.1 (28.4 to 124.7)	**<0.001**
Control	455.9 (399.2–511.4)	437.7 (411.6–526.2)	22.7 (−11.3 to 77.1)		0.40
RT	409.4 (373.7–470.6)	517.1 (479.1–560.8)	99.8 (88.5 to 129.3)		**<0.0001**
Fasting Glucose (mg/dL)				8.5 (0.75 to 17.0)	0.05
Control	95.3 (92.0–98.5)	87.5 (85.6–89.4)	−7.8 (−13.5 to 2.0)		0.05
RT	88.0 (85.5–93.9)	90.0 (84.9–94.8)	0.75 (−2.5 to 3.5)		0.89
Fasting Insulin (μIU/mL)				2.8 (−0.50 to 7.5)	0.054
Control	6.5 (5.8–8.0)	4.3 (3.6–4.9)	−3.3 (−6.5 to 1.0)		0.05
RT	9.0 (5.5–12.0)	9.0 (6.0–11.5)	−0.50 (−2.0 to 2.0)		0.88
HbA1c (%)				0.00 (−0.20 to 0.70)	0.79
Control	5.6 (5.2–5.6)	5.5 (5.3–5.5)	0.00 (0.00 to 0.40)		1.00
RT	5.3 (5.2–5.6)	5.3 (5.2–5.4)	0.00 (−0.05 to 0.20)		0.99
Glucose AUC (mg/dL)*min				−2137.5 (−3420.0 to 315.0)	0.07
Control	15,461.3 (13,680.0–15,656.3)	14,118.8 (13,621.9–15,673.1)	780.0 (−1357.5 to 1762.5)		0.83
RT	14,812.5 (13,665.0–16,155.0)	13,357.5 (12,195.0–1475.0)	−1357.5 (−2062.5 to −352.5)		**0.004**
Insulin AUC (μIU/mL)*min				−258.8 (−2408.8 to 2197.5)	0.80
Control	4200.0 (3791.3–5486.3)	3903.8 (3671.3–4524.4)	−788.8 (−2418.8 to 1260.0)		0.25
RT	7008.8 (5381.3–9543.7)	6180.0 (4657.5–9255.0)	−1042.5 (−1252.5 to −438.8)		**0.03**
2-h Glucose (mg/dL)				−22.5 (−37.0 to −1.0)	0.05
Control	110.5 (104.8–114.8)	112.5 (104.5–123.5)	9.0 (9.0 to 23.0)		0.46
RT	111.0 (100.3–121.8)	97.0 (87.8–113.0)	−13.5 (−19.0 to −3.0)		**0.007**
2-h Insulin (μIU/mL)				−15.0 (−27.5 to 21.0)	0.27
Control	29.0 (16.8–55.5)	24.5 (19.3–29.0)	−6 (−24.5 to 14.0)		0.25

Outcomes	Median (25th–75th percentile)		Median (95% CI)		P value
	Pre-test	Post-test	Within-Group Changes	Between-Group Changes	
RT	54.0 (36.0–82.0)	35.0 (25.0–48.0)	−21.0 (−22.0 to −1.0)		**0.002**

Abbreviations: BMI: body mass index; WC: waist circumference; 1RM: 1 repetition maximum; Strength Score: sum of all 3 strength measures. Relative strength measures were calculated by dividing each measure by participant body weight. HbA1c: Glycated hemoglobin. AUC: area under the curve. RT (n=28) C (n=8).

Bold data denotes $P<0.05$.

[b] between group significance was calculated using Wilcoxon rank-sum test on post-test -- pre-test change scores.

[w] within group significance was calculated using Wilcoxon signed-rank test.

Table 2

Effects of RT on Steroid Hormone Indices.

Outcomes	Median (25th–75th percentile)		Median (95% CI)		*P* value
	Pre-test	Post-test	Within-Group Changes	Between-Group Changes	
Cortisol (μg/dL)				−4.6 (−7.2 to −0.78)	**0.04**[b]
Control	13.5 (13.4–14.6)	17.5 (17.2–18.0)	2.9 (1.2 to 4.6)		**0.03**[w]
RT	14.8 (12.5–19.1)	12.7 (11.2–14.9)	−1.7 (−3.5 to 1.1)		0.17[w]
SHBG (nmol/L)				4.0 (1.7 to 7.7)	**0.005**
Control	15.2 (11.8–17.9)	16.7 (11.7–17.8)	−0.73 (−1.2 to 2.6)		0.35
RT	14.3 (11.4–19.7)	19.4 (13.7–24.3)	3.3 (1.6 to 7.0)		**0.0001**
Testosterone (ng/mL)				0.06 (−0.76 to 0.86)	1.0
Control	4.1 (4.1–5.3)	4.2 (3.5–5.7)	−0.10 (−0.60 to 0.68)		0.75
RT	4.6 (4.1–5.6)	4.5 (3.8–5.4)	−0.05 (−0.69 to 0.29)		0.64
Free Testosterone (ng/dL)				−0.57 (−3.1 to 1.2)	0.40
Control	11.9 (9.6–12.5)	10.3 (10.2–13.8)	−0.49 (−1.8 to 2.0)		0.60
RT	11.6 (9.8–14.0)	10.4 (8.6–12.3)	−1.1 (−2.6 to −0.10)		**0.01**
FAI				−23.3 (−47.4 to −4.1)	**0.04**
Control	99.4 (79.9–135)	99.8 (88.8–115)	0.61 (−9.5 to 21)		0.75
RT	113.0 (77.3–134)	80.2 (64.9–105)	−22.7 (−41.6 to −9.0)		**0.0005**

[b] between group significance was calculated using Wilcoxon rank-sum test on post-test - pre-test change scores.

[w] within group significance was calculated using Wilcoxon signed-rank test. RT (n=28) C (n=8).