Strength Training Does Not Alter the Effects of Testosterone Propionate Injections on High-Density Lipoprotein Cholesterol Concentrations

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The purpose of the study was to examine the long-term effects of a high-volume strength training program (vertical ladder climbing) and testosterone propionate injections (intraperitoneal) on serum lipid and lipoprotein concentrations in male Sprague-Dawley rats. The animals were randomly divided into a testosterone (T)-treated group (dose per injection, 2.5 mg/kg testosterone propionate solubilized in 1 mL safflower oil) and a control (C) group (injected with an isovolumic amount of safflower oil alone). Animals were further divided into a strength-trained group (E) and a sedentary group (S). The 10-week resistance training program consisted of weights (100% of body mass) appended to the tail as the animal climbed an 85-cm ladder to volitional fatigue. Following 10 weeks of strength training and testosterone injections, body weight was not significantly different between the main effects of strength training exercise (TE + CE *v* TS + CS) and testosterone injections (TE + TS *v* CE + CS) or between groups. Testicular mass (mean ± SE) was measured as a relative indicator of testosterone effects. Both TE and TS had significantly reduced testicular mass (2.56 ± 0.04 and 2.38 ± 0.03 g, respectively) compared with CE and CS (3.49 ± 0.03 and 3.49 ± 0.04 g, respectively). No significant differences were observed between groups for total serum cholesterol, serum triglycerides, or serum low-density lipoprotein cholesterol (LDL-C). In contrast, significant decreases in high-density lipoprotein cholesterol (HDL-C) were observed for both TE (26.7 ± 1.6 mg/dL) and TS (27.5 ± 1.3 mg/dL) compared with CE (48.7 ± 2.9 mg/dL) and CS (43.5 ± 2.6 mg/dL). As a result, the total cholesterol to HDL-C ratio was significantly greater for TS + TE (4.7 ± 0.1) compared with CS + CE (2.9 ± 0.2). These observations suggest that in animals, a 10-week program of high-volume strength training does not elicit any beneficial effect on the lipid or lipoprotein status, nor does it attenuate the altered lipoprotein profile induced by testosterone propionate injections.

THE EFFECTS of strength training on the serum lipid and lipoprotein profiles have yielded contrasting results. A majority of the reports have suggested that strength training results in no alteration of the lipid and lipoprotein profiles[1-4] and may in fact produce an adverse effect on the lipoprotein status.[5-7] In the latter studies, the lack of control for age, loss of body fat, training regimen, and possible use of androgenic anabolic steroids (AAS) may have compromised the results, leading to the contradictory observations. In contrast, several studies have reported that resistance training can increase the serum concentration of high-density lipoprotein cholesterol (HDL-C) and concomitantly decrease the concentration of both low-density lipoprotein cholesterol (LDL-C) and triglycerides.[8-11] The outcome is an improved lipid and lipoprotein status, thereby reducing the risk of cardiovascular disease. However, the results of these studies are confounded by the potential lack of control for factors that can impact lipid and lipoprotein concentrations such as gender, diet, residual effects of the last bout of exercise, and, in some instances, the absence of a control group.[10,11]

High-volume strength training, typically used by bodybuilders, uses moderate resistance and a high number of repetitions. The use of high-volume strength training was initially proposed as a potential factor to explain the discrepancies pertaining to the impact of resistance exercise on the lipid and lipoprotein profiles.[12,13] This factor has been implicated in recent observations in both short- and long-term resistance training reports in which a high-volume strength training stimulus was cited as the factor responsible for the positive alterations in the lipid and lipoprotein profiles.[14,15] High-volume strength training regimens that use more aerobic metabolism are hypothesized to elicit effects similar to endurance training activities.[1,16] Specifically, aerobic exercise training enhances the use of fatty acids. This results in an elevation of lipoprotein lipase activity.[1,16] The augmentation in lipoprotein lipase is associated with increases in HDL-C and decreases in plasma triglycerides. Consequently, high-volume strength training protocols have been hypothesized to elicit a beneficial effect on the lipid and lipoprotein status similar in direction and magnitude to that of endurance training regimens.[16]

Injectable forms of AAS and testosterone preparations are intended to promote anabolic effects on the skeletal muscle when used in combination with resistance training.[17] In this regard, these substances have been abused by athletes and nonathletes to improve muscular development and enhance athletic performance.[18] However, the effects of injectable testosterone preparations and other injectable forms of AAS on the lipid and lipoprotein profiles are poorly defined. The use of injectable testosterone preparations in humans has produced conflicting results. Some reports have observed no alterations in HDL-C or LDL-C,[19,20] while other studies have reported modest yet significant decreases in HDL-C[21-23] and either no change in LDL-C[22,23] or a concomitant decrease in LDL-C.[21] However, in animal studies, significant decreases in HDL-C of 30% or greater were observed for injections of testosterone propionate.[24,25] While these animal studies suggest that injectable testosterone preparations elicit greater changes in HDL-C concentrations than human studies, animal models allow for greater control of the factors that can impact the outcome of the study.

With the use of rats as an animal model to control for gender, diet, age, residual effects of the last bout of exercise, and AAS use, the purpose of the present investigation was twofold. The

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first purpose was to determine if high-volume strength training results in an alteration of the lipid and/or lipoprotein profile, specifically an elevation in the serum concentration of HDL-C and/or a decrease in LDL-C or triglycerides. If strength training elicits a beneficial effect on the lipid and lipoprotein status, the second purpose was to determine if strength training can attenuate the adverse effects on the lipoprotein profile normally associated with testosterone injections.

MATERIALS AND METHODS

The experimental protocol for the study was preapproved by the Chapman University Institutional Review Board and conformed with the policy statement on the humane care and use of laboratory animals. Experiments were performed on male Sprague-Dawley rats (initially 200 to 225 g) housed in pairs in a temperature-controlled room, given food and water ad libitum, and maintained on a 12-hour light/dark cycle. The animals were allowed 1 week to acclimate to these conditions following their arrival, were not exposed to exercise, and remained inactive. The animals were randomly divided into control (n = 16) or testosterone (n = 16) groups and received two intraperitoneal injections per week for 10 total weeks. The testosterone-treated group was injected with testosterone propionate (dose per injection, 2.5 mg/kg solubilized in 1 mL safflower oil) while the control group received isovolumic injections of safflower oil. The animals were weighed every week to determine the adjusted amount of testosterone propionate to be injected for that specific week.

The control (C) and testosterone (T)-treated animals were further divided into a strength training group (E) or a sedentary group (S). Strength training consisted of a vertical ladder-climbing task in which tail weights were appended as the animals climbed an 85-cm ladder to volitional fatigue. There were 24 steps across the 85-cm ladder, and they were positioned to ensure that the animal took each sequential step (ie, they could not skip a step). As such, one repetition along the length of the vertical ladder required 24 total lifts by the animal (or 12 lifts per limb). Resistance was provided by tail weights (initially 10% of body weight) that were gradually increased for the following 6 weeks until the animals were able to carry 100% of their body mass for the remaining 4 weeks of the training regimen. The animals trained 5 d/wk and were operantly conditioned to climb to avoid a vat of cold water beneath them. Volitional fatigue was assessed by the inability to continue the stair-climbing task on three successive attempts to motivate the animal. To minimize the residual effects of the last bout of exercise, animals were weighed, anesthetized with ether, and decapitated 48 hours following the last injection and the last bout of resistance training exercise. Blood samples were collected via exsanguination, and the right and left testes were quickly removed, weighed, and recorded as testicular mass. Finally, the heart was dissected and rapidly frozen in liquid nitrogen. The blood samples were placed on ice, allowed to clot, and later centrifuged to separate the serum component. The serum and tissue samples were then frozen at −80°C for subsequent analyses.

All assays were performed by spectrophotometry at 37°C using previously described methods.[26-28] Briefly, total serum triglycerides were determined enzymatically by measuring glycerol release at an absorbance of 540 nm.[28] Total serum cholesterol was determined by the hydrolysis of cholesterol to hydrogen peroxide and the formation of quinoneimine dye measured at an absorbance of 500 nm.[26] Serum HDL-C was determined following the precipitation of LDL-C and very–low-density lipoprotein cholesterol from the solution using phosphotungstic acid in conjunction with $MgCl_2$.[27] Following centrifugation, the precipitate was separated from the solution and then discarded. The remaining HDL-C in the solution was then measured as described for total serum cholesterol. Total cholesterol was then divided by HDL-C to determine the ratio. LDL-C was calculated using the formula validated by Friedewald et al,[29] LDL-C = total cholesterol − [HDL-C + (triglycerides/5)].

To assess the effectiveness of the strength training regimen, the frozen hearts were later weighed on an analytical balance. An elevated heart weight provided indirect support for the cardiac hypertrophy previously observed as a result of exercise training.[30,31]

The data were analyzed using a two-factor ANOVA to test for the main effects of exercise and testosterone, as well as all possible interactions. Statistical significance was set at a P level less than .05.

RESULTS

The body weight at completion of the 10-week period was not significantly different for strength-trained animals (TE + CE), 371 ± 4 g, compared with sedentary controls (TS + CS), 359 ± 5 g. Nor were significant differences observed for testosterone-treated animals compared with controls or between groups (Table 1). In contrast, testicular mass was significantly lower in TE + TS (2.47 ± 0.02 g) compared with CE + CS (3.49 ± 0.02 g). Despite similar body weight, the volume of resistance training completed per exercise bout during the last 4 weeks of the strength training regimen was significantly higher for TE (12.5 ± 0.5 ladder-climbing repetitions; 300 ± 12 body lifts) compared with CE (8.4 ± 0.3 ladder-climbing repetitions; 202 ± 7 body lifts). To support a training effect, heart weight was significantly elevated in TE + CE (1.47 ± 0.04 g) compared with TS + CS (1.28 ± 0.03 g). In contrast, there was no significant difference for TS + TE versus CS + CE. Despite the lack of difference between the main effect of testosterone-treated groups compared with the controls, TS + TE (1.42 ± 0.03 g) were significantly different compared with the sedentary group not exposed to a strength training regimen (CS, 1.23 ± 0.05 g; Table 1).

Total serum cholesterol was not significantly different between the main effects of strength training and testosterone following the 10-week period (126.7 ± 2.4 mg/dL for TS + CS, 131.6 ± 2.1 mg/dL for TE + CE, 126.3 ± 2.2 mg/dL for TE + TS, and 131.8 ± 2.5 mg/dL for CE + CS). Nor was the total cholesterol level different between any of the groups (Table 2). Similarly, no significant difference in the HDL-C concentration was noted on the main effect of strength training. However, serum HDL-C was 41% lower in TE + TS (27.1 ± 1.0 mg/dL) compared with CE + CS (46.1 ± 2.0 mg/dL; Fig 1). As a result of the lower HDL-C in the testosterone-treated groups, the ratio of total cholesterol to HDL-C was significantly higher for testosterone-injected animals (4.7 ± 0.1) compared with controls (2.9 ± 0.2; Fig 2). Despite the reduction in HDL-C, no

Table 1. Effects of Testosterone and Strength Training on Body and Organ Weights

Group	Body Weight (g)	Testicular Weight (g)	Heart Weight (g)
TE (n = 8)	371 ± 5	2.56 ± 0.04*	1.50 ± 0.04†
TS (n = 8)	357 ± 8	2.38 ± 0.03*	1.34 ± 0.04
CE (n = 8)	371 ± 8	3.49 ± 0.03	1.43 ± 0.06†
CS (n = 8)	361 ± 7	3.49 ± 0.04	1.23 ± 0.03

NOTE. Values are the mean ± SE.

*Significant difference between the main effect of testosterone injections and placebo injections.

†Significant difference between the main effect of exercise and sedentary conditions.

Table 2. Effects of Testosterone and Strength Training on Serum Lipids and Lipoproteins

Group	Total Cholesterol (mg/dL)	LDL-C (mg/dL)	Triacylglycerol (mg/dL)
TE (n = 8)	128.1 ± 1.8	70.7 ± 4.3	141.7 ± 5.8
TS (n = 8)	124.6 ± 3.8	71.9 ± 6.1	135.3 ± 6.2
CE (n = 8)	134.9 ± 3.6	58.2 ± 6.0	139.0 ± 4.1
CS (n = 8)	128.7 ± 3.4	59.3 ± 5.2	143.8 ± 5.5

NOTE. Values are the mean ± SE. There was no significant difference between the main effect of testosterone injections and placebo injections or between any group.

significant differences were observed for LDL-C and triacylglycerol concentrations (Table 2) between any of the main effects or between any of the groups. While the LDL-C concentration showed a 21% increase for the testosterone-treated groups compared with the controls, it failed to reach statistical significance ($P = .063$).

DISCUSSION

The current results demonstrate that in the laboratory rat, testosterone propionate injections elicit a significant reduction in the HDL-C concentration and a significant increase in the total cholesterol to HDL-C ratio. Further, the high-volume strength training regimen did not result in any alteration of the lipid and lipoprotein status. As such, the strength training regimen did not mitigate the detrimental decline in HDL-C observed with testosterone propionate injections. Thus, under the current conditions using rats, high-volume strength training does not elicit any protective effect on the lipid and lipoprotein status, nor does it attenuate the detrimental effects of testosterone propionate injections on the lipid-lipoprotein profile.

Aerobic training regimens have consistently been advocated as a prophylactic against coronary artery disease.[1,4,7,13,16,32] The improvements in the lipid and lipoprotein status that can reduce the risk of cardiovascular disease include an increase in HDL-C, a decrease in triglycerides, and a decrease in the total cholesterol to HDL-C ratio.[1,4,7,13,16,32] However, the effects of strength

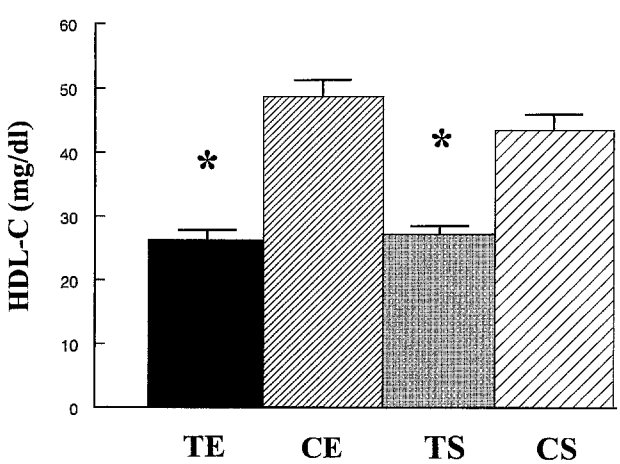

Fig 1. Effects of strength training and testosterone propionate injections on serum HDL-C. Values are the mean ± SE (n = 8 per group). Significant difference between the main effect of testosterone injections and placebo injections ($P < .05$).

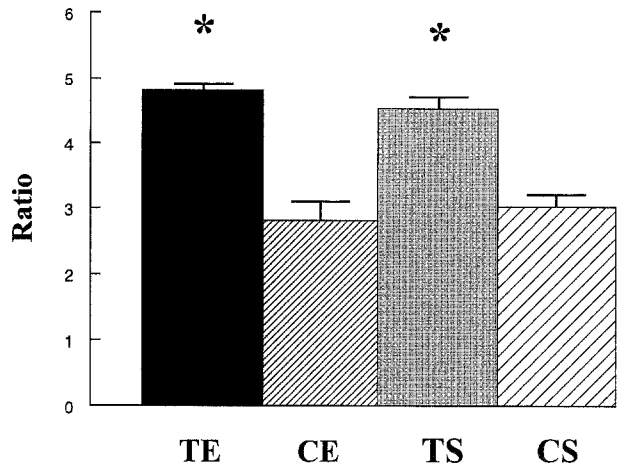

Fig 2. Effects of strength training and testosterone propionate injections on the total serum cholesterol to HDL-C ratio. Values are the mean ± SE (n = 8 per group). Significant difference between the main effect of testosterone injections and placebo injections ($P < .05$).

training on the lipid and lipoprotein profile have produced equivocal results.[1-11] Recent reports further confound the interpretations from these prior studies, suggesting that strength training volume may be a factor in the favorable alterations of serum lipid and lipoprotein concentrations.[12-15] As such, strength training regimens that involve a larger total number of repetitions and thus use more aerobic metabolic pathways have produced alterations in the lipid and lipoprotein status similar in direction and magnitude to the adaptations elicited from endurance training.[1,8,11]

While the use of animals allows for the control of various confounding factors, the implementation of a high-volume strength training protocol in animals has previously limited such examinations. Previous attempts using strength training regimens in animals involved clambering up a slope of 80°[33] and jumping with a weighted vest.[34] In the current study, the vertical stair-climbing task with weights appended to the tail provides an alternate high-volume strength training stimulus. The enhanced skeletal muscle growth with the use of AAS and/or testosterone preparations in combination with resistance training is well established.[17] While the current study did not quantify skeletal muscle hypertrophy in the animals, several observations indirectly support an improvement in skeletal muscle development resulting from the strength training regimen and testosterone injections. First, the strength-trained groups had a significantly greater heart weight than the controls. In humans, cardiac hypertrophy following endurance training can result from an elevation in the preload, whereas hypertrophy following resistance training is attributable to an increase in the afterload (ie, the Valsalva maneuver). While the high-volume strength training protocol in the current study appears to have elicited cardiac hypertrophy, an explanation for the current observation remains to be determined. The use of the high-volume strength training protocol may have resulted in cardiovascular adaptations similar to endurance training regimens. Alternatively, the use of the forelimbs could result in an increase of intrathoracic pressure, which may elevate the afterload, thereby providing the stimulus for cardiac hypertrophy. Second,

the significant increase in repetitions performed by the TE group (300 ± 12 body lifts) compared with CE rats (202 ± 7 body lifts) suggests an ergogenic effect on the skeletal muscle mass elicited by the combination of testosterone injections and resistance training. This increase subsequently resulted in greater absolute workloads with each exercise bout such that the testosterone-treated animals performed 49% more lifts than the resistance-trained control animals.

Interestingly, the testosterone-treated groups demonstrated a significantly greater heart weight than the nonexercised controls. This observation is consistent with previous animal studies that reported cardiac hypertrophy in rats injected for 5 to 6 weeks with testosterone[35] or nandrolone decanoate.[36] As with these other reports, the mechanisms responsible for the elevations in heart weight are unknown but provide indirect support for an androgenic effect attributable to the testosterone treatment. The measurement of testicular mass, on the other hand, was used as a relative indicator of an androgenic effect attributable to the testosterone propionate injections. As such, the current results are consistent with previous reports of testicular atrophy with the use of testosterone preparations,[37,38] as well as our previous report of a 34% decrease in testicular mass.[31] Thus, the current observations of a 32% decrease in testicular mass and a concomitant elevation in heart weight are consistent with an androgenic effect attributable to the testosterone propionate injections.

With the use of a high-volume strength training stimulus in animals, we failed to observe any alteration in the lipid or lipoprotein profile. These results are in contrast to reports suggesting beneficial training adaptations in the lipid and lipoprotein profiles derived from high-volume resistance training.[8-11,15,34] The reports demonstrating a beneficial effect may have been limited by a lack of control for factors that can impact lipid and lipoprotein concentrations such as age, gender, diet, residual effects of the last bout of exercise, and, in some instances, the absence of a control group. In addition, several of these studies[8,9,12] involved a cross-sectional examination in which selection bias may be the factor resulting in the observations noted. However, in a well-designed study by Leeds et al,[34] an 8-week anaerobic jumping program with a weighted vest in male rats resulted in a significant increase in HDL-C and a significant decrease in LDL-C. There is no apparent explanation for the discrepancy between the current study and the observation by Leeds et al,[34] although the amount of safflower oil (1 mL) used to administer testosterone propionate in the current study was significantly higher than the amount of sesame seed oil used by Leeds et al[34] (0.1 mL) to administer durabolin. Further, they administered the injections during the last 3 weeks of their 8-week training program, whereas we administered the injections throughout the entire 10-week regimen. While safflower oil contains only a small percentage of saturated fat, it is conceivable that the longer duration and larger amount of vegetable oil used as the carrier for testosterone in the current study may have affected the results.

Ostensibly, the rats used in the current study were young healthy animals in which the lipid and lipoprotein concentrations may not have been altered irrespective of the intervention. Despite this potential limitation, our results are consistent with previous human studies that demonstrated no beneficial effect on the lipid and lipoprotein profiles resulting from strength training regimens.[1-4] Kokkinos et al[2] reported no alterations in the lipid or lipoprotein profiles using a high-repetition resistance training program in men. While these subjects were not at risk for coronary heart disease, Kokkinos et al[3] subsequently observed no alterations in the lipid or lipoprotein status in men at risk for heart disease following 20 weeks of resistance training. Further, no strength training–induced elevations in either postheparin lipoprotein lipase activity or hepatic lipase activity were noted.[3] Thus, consistent with the observations in humans, 10 weeks of high-volume resistance training in animals does not bestow any beneficial effect on the lipid and lipoprotein profiles.

The deleterious effects of parenteral testosterone preparations and other injectable forms of AAS remain equivocal. Several reports[19,20] observed no changes in lipoprotein concentrations in men after 10 to 12 weeks of injections of testosterone enanthate. It has been suggested that since injectable testosterone preparations and other injectable forms of AAS lack the 17α-alkylated group, it readily aromatizes to 17β-estradiol.[20,21] Since estrogens decrease hepatic triglyceride lipase activity, this would mitigate an increase induced by exogenous androgens, which would result in no impact on the HDL-C concentration.[19,20] As such, the observations in the current study are in contrast to these prior reports. However, other human reports[21-23] have demonstrated statistically significant, albeit marginal, decreases in HDL-C of 9% to 16% in men receiving intramuscular injections of testosterone preparations. In monkeys, Weyrich et al[24] reported a 31% decrease in HDL-C after biweekly injections of testosterone propionate (2 mg/kg per injection) for 10 weeks with an additional injection (2 mg/kg) of testosterone cypionate every other week. In addition, we have previously reported[25] a significant decrease of 32% in HDL-C concentrations using the same testosterone preparations and a slightly higher dose (3 mg/kg biweekly). In the previous animal studies and the current study, the testosterone dose used to elicit the dramatic decrease in HDL-C was significantly greater than the dose in previous observations in humans, suggesting a species-specific effect. As such, the use of animal models to study the effects of injectable steroid preparations may exaggerate the actual impact in humans. Nevertheless, while the mechanism remains to be determined, the current results support the few animal reports demonstrating that parenteral testosterone administration elicits a deleterious effect on the lipoprotein profile.

In summary, a long-term regimen of high-volume strength training in animals does not elicit any alteration in the lipid or lipoprotein status, nor does a long-term program of strength training mitigate the decrements associated with testosterone propionate injections. The results are consistent with previous reports on the detrimental effects of testosterone propionate injections on the lipoprotein profile resulting in a decrease in HDL-C concentrations. Thus, these observations suggest that in rats, testosterone propionate injections result in an atherogenic lipoprotein profile that is not attenuated by a program of high-volume resistance training.

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