Cardiovascular Topics

Relationship between resistance training and lipoprotein profiles in sedentary male smokers

INA SHAW, BRANDON S SHAW

Summary

Epidemiological studies have found plasma lipid and lipoprotein levels to be predictive of cardiovascular disease in adults. To date, regular aerobic modes of exercise have been associated with favourable alterations in lipid and lipoprotein levels. However, the effect of resistance training on lipid and lipoprotein levels is inconclusive and conflicting. Therefore, the aim of this study was to provide some clarity on whether resistance training could be used to improve sedentary male smokers' lipoprotein profiles.

The study made use of a pre-test, a treatment period and a post-test. Subjects were placed into one of two groups, namely, a resistance-training (RES) group (n = 13) or a control (CON) group (n = 12). Throughout the 16-week experimental period the CON group received no treatment whatsoever. After resistance training, serum triglyceride levels were significantly decreased by 18.42% from 1.162 mmol/l (± 0.476) to 0.831 mmol/l (± 0.058) (p = 0.038) in the RES group. However, resistance training was found to have no impact on any of the other measured lipid and lipoprotein measures.

In conclusion, these findings indicate that resistance training appears to have no significant effect on lipid and lipoprotein profiles in sedentary male smokers and therefore cannot prevent the advance of CAD.

Submitted 22/11/07, accepted 30/3/08

Cardiovasc J Afr 2008; **19**: 194–197 www.cvja.co.za

Atherosclerosis is the leading cause of coronary artery disease (CAD) in most Western populations and is associated with an accumulation of cholesterol in the muscular walls of arteries.[1] This finding is substantiated by the World Health Organisation which indicated that nearly half of the variance in CAD rates is due to differences in blood cholesterol levels.[2] In South Africa, 80% of 'westernised' South Africans have an elevated cholesterol level, with the other 20% having cholesterol levels that place them at risk of developing hypercholesterolaemia.[3]

Vaal University of Technology, Department of Marketing and Sport Management, Vanderbijlpark

INA SHAW, PhD (Biokinetics)

Tshwane University of Technology, Department of Sport, Rehabilitation and Dental Sciences, Pretoria

BRANDON S SHAW, PhD (Biokinetics); shawbs@tut.ac.za

The terms 'hyperlipidaemia' and/or 'hypercholesterolaemia' should be considered misnomers since an increase in high-density lipoprotein cholesterol (HDL-C) (hyperlipidaemia) must be seen as a negative risk factor and so the term dyslipidaemia should rather be utilised to describe this risk factor for CAD. Dyslipidaemia is generally characterised by hypertriglyceridaemia, an increased low-density lipoprotein cholesterol (LDL-C) and a decreased HDL-C.[4]

Exercise training plays an important role in improving lipid and lipoprotein levels, since dietary or caloric restriction alone has been shown to be an ineffective method of improving lipid and lipoprotein profiles in the long term.[5-7] Exercise provides an additional benefit in that it decreases appetite in men (but not in women), effectively reducing caloric and lipid intake and accumulation of fat mass, which all result in favourable lipid and lipoprotein profiles.[8] Conversely, intensive exercise training in the presence of high serum lipid levels may even aggravate the development of atheromatosis by releasing catecholamines which damage the vascular walls, leading to their susceptibility to lipid deposition.[9]

To date, regular aerobic modes of exercise have been associated with favourable alterations in lipid and lipoprotein levels.[10-12] However, few studies have generated conclusive data on the effects of resistance training on lipid and lipoprotein levels. The effects of this mode of training are as yet not well documented and when such data are forthcoming, the findings are not consistent.[6,13]

Resistance training could be beneficial in improving lipid and lipoprotein levels since it results in an increased muscle lipoprotein lipase (LPL) and disparate decrease in hepatic LPL, which might result in favourable lipid and lipoprotein alterations.[14] The increase in muscle LPL and the decrease in hepatic LPL following resistance training may result in increased very low-density lipoprotein cholesterol catabolism and decreased HDL-C breakdown, respectively.[14]

The aim of this study was therefore to investigate whether resistance training could improve a sedentary male smoker's lipid and lipoprotein profile.

Methods

The study made use of a quantitative, pre-test/post-test research design. Twenty-five apparently healthy, sedentary male smokers between the ages of 20 and 35 years (mean age was 28 years and 7 months) volunteered to participate in the study. The subjects were informed about the potential risk of participation and gave their signed consent. Subjects were placed in either a control group (CON) (n = 12) or resistance-training group

(RES) (n = 13). Sedentary male smokers were selected as the target sample group since cigarette smoking and physical inactivity are two of the major CAD risk factors and both place the sedentary smoker at risk for developing an unfavourable lipid and lipoprotein profile.

Each subject's total cholesterol (TC), triglyceride (TG), LDL-C and HDL-C concentrations and TC:HDL-C, LDL-C:HDL-C and non-HDL-C (n-HDL-C) ratios/indexes were measured and/or calculated[6,15-17] following a nine- to 12-hour fasting period and prior to any exercise.[6,17]

The exercise intensity for each subject in the RES group was calculated subsequent to each subject undergoing a 10-repetition maximum evaluation for each prescribed exercise. The results of each subject were recorded in terms of the weight lifted and the amount of repetitions, which had to be less than 10, to reach fatigue. Each subject's one-repetition maximum (1-RM) was then calculated according to the following formula: 1-RM = (weight lifted) ÷ [1.0278 − (repetitions to fatigue × 0.0278)].[18] The number of crunches prescribed to each subject was determined by the maximum number of repetitions that a subject could perform in 60 seconds.

Members of the CON group were instructed to maintain their usual activities and not to take part in any form of structured exercise during the experimental period. As with the experimental group, they were also not given any advice on their diet, alcohol intake and smoking behaviour over the experimental period.

The RES group subjects were instructed to exercise three times weekly for a period of 16 weeks. All exercise sessions were preceded by five minutes of easy cycling at a heart rate of less than 100 beats per minute (bpm), followed by eight stretching exercises performed for two sets of 30 seconds. Exercise sessions were concluded with five minutes of easy cycling at a heart rate of less than 100 bpm.[6,13] Each RES group subject performed three sets of 15 repetitions using shoulder press, latissimus dorsi pull-downs, seated chest press, seated rows, crunches, unilateral leg press, unilateral knee extensions and unilateral prone leg curls at an intensity of 60% 1-RM.[13] The subjects were allowed a 60- to 90-second rest period between each set.[19] For crunches, each subject was required to perform three sets of 60% of the maximum number of repetitions that he had performed during the initial evaluation. Each individual's 1-RM was re-evaluated every four weeks and his exercise programme adjusted accordingly.[13]

Descriptive statistics and dependant t-tests were calculated by the Rand Afrikaans University's Statistical Consultation Service (STATKON) using the Statistical Package for Social Sciences (SPSS) II. A 95% confidence level ($p \leq 0.05$) was used for determining a statistically significant change from the pre-test to the post-test.

Results

With regard to the CON group, a non-significant 0.350% decrease in TC levels was found at the conclusion of the 16-week experimental period (p = 1.000) (Table 1). Even though the CON group had a 2.300% increase in LDL-C levels, this change was found to be non-significant (p = 0.754). Similarly, the increase in HDL-C levels was found to be non-significant (p = 0.754). With regard to the calculated indexes, the n-HDL-C and TC:HDL-C ratio in the CON group decreased non-significantly by 0.780% (p = 0.530) and 0.260% (p = 0.638), respectively. The CON group's LDL-C:HDL-C ratio increased non-significantly by 2.870% from the pre- to the post-test (p = 0.875). The CON group's TG levels increased significantly (albeit unfavourably) by 9.480% from 1.621 mmol/l to 1.761 mmol/l (p = 0.026).

Similar to the CON group, no significant change in mean TC concentration was observed following 16 weeks of RES training, with only a 1.570% increase being found at the post-test (p = 0.807). Despite the RES group demonstrating a 7.000% increase in LDL-C levels, this change was found to be non-significant (p = 0.116). Similarly, the increase in HDL-C levels was found to be non-significant following RES training (from 1.052 mmol/l to 1.169 mmol/l) (p = 0.069) as was the 5.640% decrease in n-HDL-C levels (from 2.625 to 2.456) (p = 0.152). Although the RES group was found to have an 8.500% reduction in the TC:HDL-C ratio, this reduction was found to be non-significant (p = 0.173). Moreover, the 16 weeks of RES training resulted in a large mean (albeit non-significant) 13.420% decrease in their

TABLE 1. PRE- AND POST-TEST LIPOPROTEIN LIPID RESPONSES TO 16 WEEKS OF RESISTANCE TRAINING

Group	*Pre-test (SD)*	*Post-test (SD)*	*Difference (SD)*	*% change (significance)*
Total cholesterol (TC) (mmol/l)				
CON	5.062 (± 0.272)	5.043 (± 0.310)	0.019 (± 0.198)	0.350% decrease (p = 1.000)
RES	3.614 (± 0.819)	3.625 (± 0.666)	0.011 (± 0.477)	1.570% increase (p = 0.807)
Low-density lipoprotein cholesterol (LDL-C) (mmol/l)				
CON	4.121 (± 0.274)	4.209 (± 0.415)	0.088 (± 0.386)	2.300% increase (p = 0.754)
RES	2.859 (± 0.914)	2.621 (± 0.765)	0.238 (± 0.486)	7.000% decrease (p = 0.116)
High-density lipoprotein cholesterol (HDL-C) (mmol/l)				
CON	1.276 (± 0.165)	1.292 (± 0.231)	0.016 (± 0.170)	1.350% increase (p = 0.754)
RES	1.052 (± 0.264)	1.169 (± 0.299)	0.117 (± 0.211)	12.230% increase (p = 0.069)
Non-high-density lipoprotein cholesterol (n-HDL-C)				
CON	3.786 (± 0.288)	3.751 (± 0.286)	0.035 (± 0.211)	0.780% decrease (p = 0.530)
RES	2.625 (± 0.865)	2.456 (± 0.767)	0.169 (± 0.433)	5.640% decrease (p = 0.152)
Total cholesterol: high-density lipoprotein cholesterol ratio (TC:HDL-C ratio)				
CON	4.031 (± 0.582)	4.003 (± 0.637)	0.028 (± 0.494)	0.260% decrease (p = 0.638)
RES	3.689 (± 1.242)	3.323 (± 1.105)	0.366 (± 0.824)	8.500% decrease (p = 0.173)
Low-density lipoprotein cholesterol:high-density lipoprotein cholesterol ratio (LDL-C:HDL-C ratio)				
CON	3.297 (± 0.599)	3.359 (± 0.683)	0.063 (± 0.596)	2.870% increase (p = 0.875)
RES	2.932 (± 1.340)	2.472 (± 1.140)	0.460 (± 0.912)	13.420% decrease (p = 0.152)
Triglycerides (TG) (mmol/l)				
CON	1.621 (± 0.377)	1.761 (± 0.391)	0.140 (± 0.201)	9.480% increase (p = 0.026*)
RES	1.162 (± 0.476)	0.831 (± 0.058)	0.332 (± 0.494)	18.420% decrease (p = 0.038*)

RES: resistance training group; CON: control group; SD: standard deviation, *significant.

LDL-C:HDL-C ratio ($p = 0.152$). In contrast, the RES training did result in a significant 18.420% decrease in TG levels from 1.162 mmol/l to 0.831 mmol/l ($p = 0.038$).

Discussion

There is considerable consensus from existing literature and the findings of the present study that resistance training has no effect on TC levels.[6,13,15,17,20,21] The present study's finding that resistance training had no effect on LDL-C levels is also consistent with several studies.[6,13,16,17,20,22,23] However, these findings are not unequivocal since several other studies have demonstrated that resistance training may indeed decrease LDL-C levels following a period of resistance training.[7,14,15,22,24,25-27] What is perplexing is that there is no evident difference in study design (i.e. in terms of samples and exercise variables) between those studies that did not significantly alter LDL-C levels, implying that there are no definite variables ensuring success in lowering LDL-C levels.

The findings of several studies, including those of the present investigation, have found that resistance training had no effect on HDL-C levels,[7,13,16,17,20-23,26] whereas in some instances, resistance training has been found to increase HDL-C levels.[6,14,15,22,25,27] A possible explanation for this is that the studies that found increases in HDL-C made use of samples with lower baseline levels of HDL-C and shorter study periods.

Even though the determination of concentrations of small, dense LDLs, either by direct laboratory methods or via the calculation of n-HDL-C (TC minus HDL-C), is essential in determining CAD risk, few studies have focused on the effect of resistance training on this risk factor.[10] There are almost no other studies therefore to which these findings can be compared.

The present study and other studies[13,17] found that resistance training had no effect on the TC:HDL-C ratio. Most previous studies, however, have found that resistance training decreased this ratio,[6,7,15,22,26] especially in men.[6,26] As in the case of the present study, several studies have demonstrated a decrease in TG levels.[7,13,14,22] However, resistance training has also been shown to have no effect[20] or to unfavourably increase TG levels.[17,28]

The findings of the present study demonstrated that resistance training was not associated with favourable changes in lipid and lipoprotein levels in sedentary male smokers. Also, the sheer diversity of the different methods of applying resistance training is in itself a source of complication when attempting to determine if resistance training can indeed favourably alter lipid and lipoprotein profiles. Therefore, an optimal combination of resistance-training workload, intensity and number of repetitions still eludes researchers in this field. The contradictory findings in the literature, including the present findings, suggest that such an 'optimal combination' probably does not exist. It is therefore difficult to make a case for the use of resistance training to favourably alter the lipid and lipoprotein profiles of sedentary male smokers, and thus to reduce their risk of CAD.

The authors are grateful to the Rand Afrikaans University, Johannesburg, for the use of the Centre for Sport Science and Biokinetics and the Rand Afrikaans University's Statistical Consultation Service (STATKON).

References

1. Buist R. *The Cholesterol Myth*. Cape Town: Struik, 1995.
2. Simons LI. Interrelations of lipids and lipoproteins with coronary artery disease mortality in 19 countries. *Am J Cardiol* 1986; **57**(14): 5G−10G.
3. Medical Research Council (MRC). Unpublished statistics of cardiovascular disease in South Africa, 1996.
4. Ginsberg HN. Treatment for patients with the metabolic syndrome. *Am J Cardiol* 2003; **91**(suppl): 29E−39E.
5. American College of Sports Medicine (ACSM). *ACSM's Guidelines for Exercise Testing and Prescription*. 6th edn. Baltimore: Lippincott Williams & Wilkins, 2000.
6. Joseph LJ, Davey SL, Evans WJ, Campbell WW. Differential effect of resistance training on the body composition and lipoprotein-lipid profile in older men and women. *Metabolism* 1999; **48**(11): 1474−1480.
7. Prabhakaran B, Dowling EA, Branch JD, Swain DP, Leutholtz BC. Effects of 14 weeks of resistance training on lipid profiles and body fat percentage in premenopausal women. *Br J Sports Med* 1999; **33**(3): 190−195.
8. Byrne HK, Wilmore JH. The Effects of a 20-week exercise training program on resting metabolic rate in previously sedentary, moderately obese women. Int *J Sport Nutr Exer Metab* 2001; **11**(1): 15−31.
9. Kraus H, Raab W, White PD. *Hypokinetic Disease: Diseases Produced by Lack of Exercise*. Springfield, IL: Charles C Thomas, 1961.
10. American Association of Clinical Endocrinologists (AACE). The American Association of Clinical Endocrinologists medical guidelines for clinical practice for the diagnosis and treatment of dyslipidemia and prevention of athergenesis. *Endocrin Pract* 2000; **6**(2): 162−213.
11. LeMura LM, von Duvillard SP, Andreacci J, Klebez JM, Chelland S, Russo J. Lipid and lipoprotein profiles, cardiovascular fitness, body composition and diet during and after resistance, aerobic and combination training in young women. *Eur J Appl Physiol* 2000; **82**(5−6): 451−458.
12. Mosher PE, Nash MS, Perry AC, LaPerriere AR, Goldberg RB. Aerobic circuit exercise training: effect on adolescents with well-controlled insulin-dependent diabetes mellitus. *Arch Phys Med Rehabil* 1998; **79**(6): 652−7.
13. Shaw BS, Lategan L, Loots JM. Effects of 8 weeks resistance training on serum lipids in sedentary male smokers. *Afr J Phys Hlth Ed Recr Dance* 2003; Oct(Suppl): 148−157.
14. Ullrich IH, Reid CM, Yeater RA. Increased HDL-cholesterol levels with a weight lifting programme. *South Med J* 1987; **80**(3): 328−331.
15. Hurley BF, Hagberg JM, Goldberg AP, Seals DR, Ehsani AA, Brennan RE, *et al*. Resistive training can reduce coronary risk factors without altering VO_{2max} or percent body fat. *Med Sci Sports Exer* 1988; **20**(2): 150−154.
16. Kokkinos PF, Hurley BF, Smutok MA, Farmer C, Reece C, Shulman R, *et al*. Strength training does not improve lipoprotein-lipid profiles in men at risk for CAD. *Med Sci Sports Exer* 1991; **23**(10): 1134−1139.
17. Manning JM, Dooly-Manning CR, White K, Kampa I, Silas S, Kesselhaut M, *et al*. Effects of a resistive training programme on lipoprotein-lipid levels in obese women. *Med Sci Sports Exer* 1991; **23**(11): 1222−1223.
18. Heyward VH. *Advanced Fitness Assessment and Exercise Prescription*. Champaign, IL: Human Kinetics, 1997.
19. Wallace MB, Mills BD, Browning CL. Effects of cross-training on markers of insulin resistance/hyperinsulinemia. *Med Sci Sports Exer* 1997; **29**(9): 1170−1176.
20. Banz WJ, Maher MA, Thompson WG, Basset DR, Moore W, Ashraf M, *et al*. Effects of resistance versus aerobic training on coronary artery disease risk factors. *Exp Biol Med (Maywood)* 2003; **228**(4): 434−440.
21. Morgan DW, Cruise RJ, Giradin BW, Lutz-Schneider V, Morgan DH, Qi WM. HDL-C concentrations in weight-trained, endurance-trained and sedentary females. *Phys Sportsmed* 1986; **14**(3): 166−181.
22. Boyden TW, Pameter RW, Going SB, Lohman TG, Hall MC, Houtkooper LB, *et al*. Resistance training is associated with decreases in serum low-density lipoprotein cholesterol levels in pre-menopausal women. *Arch Intern Med* 1993; **153**(1): 97−100.
23. Kokkinos PF, Hurley BF, Vaccaro P, Patterson JC, Gardner LB, Ostgrove SM, *et al*. Effects of low- and high repetition resistive training on lipoprotein-lipid profiles. *Med Sci Sports Exer* 1988; **20**(1): 50−54.
24. Fripp RR, Hodgson JL. Effect of resistive training on plasma lipid and lipoprotein levels in male adolescents. *J Pediatr* 1987; **111**(6 Pt 1):

926–931.
25. Fleck SJ, Kraemer WJ. Resistance training: physiological responses and adaptations (Part 3 of 4). *Phys Sportsmed* 1988; **16**(5): 63–76.
26. Goldberg L, Elliot DL, Schutz RW, Kloster FE. Changes in lipid and lipoprotein levels after weight training. *J Am Med Assoc* 1984; **252**(4): 504–506.
27. Johnson CC, Stone MH, Byrd RJ, Lope SA. The response of serum lipids and plasma androgens to weight training exercise in sedentary males. *J Sports Med Phys Fitness* 1983; **23**(1): 39–44.
28. Lehman R, Kaplan V, Bingisser R, Bloch KE, Spinas GA. Impact of physical activity on cardiovascular risk factors in IDDM. *Diabetes Care* 1997; **20**(10): 1603–1611.

Book Review

Cardiac Imaging
Direct Diagnosis in Radiology Series

Authors: CD Claussen, S Miller, M Frenchel, U Kramer, R Riessen
ISBN 978-3-13-145111-8
Publishers: Thieme
Stuttgart, New York, 2008
300 pages

Radiologists have until recently seen the heart as just an oblong shape on the chest X-ray, which is either enlarged or not, or have ignored it altogether as that blurry object in the middle of the chest CT field of view. Change is afoot. The recent rapid expansion in the field of cardiac imaging necessitates the general radiologist to familiarise him/herself with the often-alien (to the radiologist) field of cardiology. As a result, numerous texts are being published almost monthly on the subject, most of them overwhelming in their size and content, with endless time needed to extract the most pertinent information. As a department just starting to perform modern cardiac imaging, we have found Claussen *et al's Cardiac Imaging* volume of the *Direct Diagnosis in Radiology* series an extremely useful tool in guiding us through this rapidly expanding process. Our copy is published in soft cover by Thieme publishers.

The pocket-sized 300-page volume contains short notes on all the important diseases likely to be encountered when imaging the heart. The contents cover a wide range of pathologies, from the familiar coronary artery stenosis and valvular disease, to lesser-known entities such as ventricular non-compaction and arrhythmogenic right ventricular dysplasia. Each chapter has concise paragraphs, usually in bulleted format, starting with definitions, epidemiology and pathogenesis with the most important clinical aspect being highlighted, including course and prognosis. The most significant findings on each imaging modality are listed and the modality of choice is specified for each condition. At the end of each chapter, useful tips are given and pitfalls to be avoided are emphasised. Differential diagnoses are listed and what the clinician wants to gain from the imaging study is discussed. The text is supplemented with good-quality annotated images and diagrams.

The final chapters have very valuable additional information in an easily accessible format. This covers the standard views of the heart, normal measurements and values, myocardial segments, coronary arteries and their variants, classification of cardiomyopathies and other necessary information not usually part of the radiologist's gambit.

Although this text is primarily aimed at radiologist and radiology registrars, it will certainly be an invaluable aid to clinicians requesting cardiac imaging, giving guidance to which imaging modality is most appropriate for each clinical situation and to what information can be gained from each modality. It is an invaluable aid to the rapid and effective imaging assessment of the heart.

H VILJOEN, JW LOTZ

Department of Radiology, Tygerberg Hospital, University of Stellenbosch