The effects of a university fitness programme on health-related variables in previously sedentary males

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This study reports on the effects of a 10-week university fitness programme on health-related fitness variables. Twenty-one male exercisers, aged 37.0(10.3) years (mean(s.d.); range 21–58), and 22 male controls, aged 38.6(7.9) years (mean(s.d.); range 17–54), volunteered to take part. Two sample t-tests and 95% confidence intervals were used to determine if the exercise group demonstrated a greater average improvement than the control group and the average improvement in both groups separately. The exercise group showed a greater average improvement over the controls from Test 1 (before fitness programme) to Test 2 (after the following): steady-state heart rate (beats min⁻¹) 95% confidence intervals (–7.8, –16.2); predicted VO₂ max (ml kg⁻¹ min⁻¹): 95% confidence intervals (3.2, 6.6); sit-ups (repetitions): 95% confidence intervals (3.1, 7.0); flexibility (cm): 95% confidence intervals (3.3, 6.9). There was no significant difference between the exercise group and control group in body weight, percentage body fat, blood pressure, total plasma cholesterol, high-density lipoprotein and triglycerides. The exercise programme improved aerobic fitness, local muscular endurance and flexibility. However, the increase in aerobic fitness did not coincide with beneficial changes in the coronary risk profile.

Keywords: Aerobic training, body fat, blood pressure, lipids, local muscular endurance, flexibility

Epidemiological studies have revealed that those who are physically active gain some protection from coronary heart disease (CHD)¹⁻³. Other studies have attempted to ascertain the effect of exercise on CHD risk factors. The results of those studies have been equivocal, with differences in initial baseline levels, and changes in body weight or body fat, dietary intake, alcohol consumption and tobacco usage affecting the final results⁴⁻⁵.

Some aerobic training programmes have been criticized for excessive demands on the time of participants despite improvements in the coronary risk factors. A programme involving limited time commitment but with a prophylactic effect on coronary risk factors has advantages over pharmacological treatment. Drug treatment of CHD risk factors is expensive, may have side-effects, and previously asymptomatic subjects could become 'medically dependent'.

Low levels of strength, local muscular endurance and flexibility can impose limitations on individuals. Faulkner et al.⁶ note that reduced strength and endurance in the abdominal muscles and lack of hamstring and iliopsoas extensibility have been reported in patients with back pain compared with controls. Hall et al.⁷ and Faulkner et al.⁸ suggest that these characteristics may promote a forward pelvic tilt, increasing lumbar lordosis. Quality of life is often reduced in people who suffer low back pain (80% of the population at some stage of life)⁹. Increased abdominal muscle endurance and/or improvement in the flexibility of the lower back and hamstrings can decrease the incidence of low back pain⁹.

The Department of Physical Education and Sports Science of the University of Glasgow offers a wide range of mass fitness sessions to a large section of students and staff in the university and to the local community. Such fitness sessions are thought to improve aerobic fitness, local muscular endurance and flexibility and may influence CHD risk factors. Studies of the physiological and health-related changes in a structured training programme help in evaluating potential benefits and should provide guidance on specific exercise prescriptions for promoting and maintaining health.

This study assessed the effects on CHD risk factors and other health-related variables of an exercise session carried out three times per week, 30 min per session over 10 weeks.

Methods

Twenty-one male exercisers, mean(s.d.) age 37.0(10.2) years, and 22 male controls, mean(s.d.) age 38.6(7.9) years, were studied to assess the effects of a 10-week training programme. Advertisements were placed in a variety of locations in the university. Sedentary male staff and students were invited to participate or act as controls. The subjects elected to
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be in the exercise or control group. Twenty-six exercisers and 23 controls entered the study. 'Tune-up' sessions were conducted in a gymnasium by physical education specialists. Subjects were asked to carry out three sessions per week for 10 weeks. The training programme consisted of 20 min aerobic activity, 5 min muscle conditioning exercises and 5 min flexibility exercises. Training heart rates were measured by the subjects palpating the radial artery and were entered into their training diaries. PE 3000 heart rate recorders (Polar Electric, Kempele, Finland) were also used to monitor exercise heart rate and check the accuracy of the subjects' palpation. No systematic comparison of the heart rates as determined by palpation and the heart rate recorders was made, but all exercisers were given the opportunity to compare these values.

The exercisers were asked to complete a weekly training diary which included frequency of training and intensity (as assessed by heart rate) of training sessions. The controls were asked to include any physical activity in a weekly training diary.

Assessment was carried out before training (Test 1) and 10 weeks later (Test 2) at the conclusion of training. Blood pressure measurements were taken using a Copal digital sphygmomanometer UA-251 (Andrew Stephens, Blackpool, UK). On arrival in the laboratory the subjects sat on a chair for 5 min and then measurements were taken at 5, 7 and 9 min. The mean of the three readings was recorded. The subject was weighed (wearing only shorts) and body composition was estimated by skinfold measurement using the method described by Durnin and Womersley. Aerobic fitness was assessed on a Monark 818 bicycle ergometer (Monark Crescent, Varberg, Sweden), with heart rates recorded using a Tunturi cardiosteter (Tunturipyra, Turku, Finland). A workload of 60 W was given for 3 min to each subject. The heart rate response during the first 3 min provided guidance for the second 3 min workload, which in turn provided information for the selection of the third and final workload lasting 5 min. This procedure resulted in subjects working between 75% and 85% of predicted maximum heart rate for 5 min. The last workload steady-state heart rate was used to predict maximum oxygen uptake (VO2max) by the method outlined by Astrand and Ryhming.

Local muscular endurance was measured by a modified version of the sit-up test outlined by the Canadian Standardized Test of Fitness. In this study subjects held their earlobes with their fingers whereas in the Canadian Standardized Test of Fitness the hands were placed at the side of the head. The degree of knee flexion was determined by placing the subject's foot as close to the buttock as possible and marking the extremity of the big toe. The heel was then placed at the 'big toe' mark. The test was conducted on a mat. A sit-up consisted of the subject rising from a back lying position and touching his knees with outstretched elbows before returning to a back lying position. The ankles of the subject were held throughout the test by the experimenter to ensure that the heels were in constant contact with the mat. Flexibility was measured using the Sit and Reach apparatus of Wells and Dillon.

All subjects were asked to report following a 12-h fast to provide a blood sample for the measurement of total plasma cholesterol, high-density lipoprotein cholesterol and triglycerides. Subjects sat for 5 min before 10 ml of blood was drawn from an antecubital vein. Total plasma cholesterol and triglyceride concentrations were measured on a Cobas-Bio centrifugal analyser (Roche Diagnostics, Welwyn Garden City, UK) employing fully enzymatic methods, cholesterol oxidase and aminoantipyrine (CHOD-PAP) for cholesterol (Boehringer Mannheim Diagnostics, Lewes, UK) and glycerol kinase/pyruvate for triglycerides – triglycerides were hydrolysed to glycerol and free fatty acids by a special lipase preparation (Merekdiagnostics, Darmstadt, Germany). Following precipitation of low and very low-density lipoprotein (LDL + VLDL) using managanous chloride–heparin reagent, high-density lipoprotein (HDL) cholesterol was estimated in the plasma supernatant, using the same cholesterol reagents as above. Aliquots of Test 1 blood samples were stored at −20°C and re-analysed 10 weeks later using the same reagent as for the Test 2 samples.

Statistical methods

The analysis investigated whether each of the variables showed a significant average difference from Test 1 and Test 2 for the exercisers and controls separately and between these groups. Paired t-tests were used to assess if a particular group (exercise and control) had shown an average improvement from Test 1 to Test 2. Two sample t-tests were used to determine if the exercisers demonstrated a greater average improvement from Test 1 to Test 2 than the controls. The 95% confidence intervals indicate the range of average improvement.

Results

The training heart rates (as recorded in the training diaries) are shown in Figure 1. The mean(s.d.) number of 'tune-ups' attended for the 10-week period was 26.7(2.9) sessions. The median number of tune-ups for the 10-week period for the exercise group was 28 (range 21–31). Five subjects from the original number of 26 exercisers failed to complete a minimum of 20 tune-ups and were eliminated from the study. Twenty-three control subjects entered the study but one control subject did not undergo a second test.

Test 1 and Test 2 data are shown in Table 1 for both exercise and control groups. Confidence intervals are given for the average improvements (i.e. Test 1 minus Test 2 values) for each group separately as well as for the average difference in improvements between the groups.

Exercisers showed significant improvement in total plasma cholesterol, high-density lipoprotein, steady-state heart rates (all three workloads), estimated VO2 max local muscular endurance and flexibility. The controls showed significant improvement in total plasma cholesterol, high-density lipoprotein, steady-state heart rates (second and third stages) and
### Table 1. Body weight, percentage body fat, systolic blood pressure, diastolic blood pressure, total plasma cholesterol, high-density lipoprotein, triglycerides, submaximal exercise heart rates, predicted VO$_{2\text{max}}$, local muscular endurance and flexibility at Test 1 and Test 2.

<table>
<thead>
<tr>
<th>Attribute</th>
<th>Test 1*</th>
<th>Test 2*</th>
<th>Mean difference</th>
<th>95% confidence interval</th>
<th>95% confidence interval of average improvement (exercisers over controls)†</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Body weight (kg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercisers</td>
<td>77.6 (11.4)</td>
<td>76.7 (10.3)</td>
<td>(+0.1, −2.0)</td>
<td>(+0.2, −2.0)</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>75.0 (8.9)</td>
<td>74.9 (8.8)</td>
<td>n.s.</td>
<td>n.s.</td>
<td></td>
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<tr>
<td><strong>Percentage body fat</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercisers</td>
<td>22.2 (5.0)</td>
<td>21.6 (5.0)</td>
<td>(+0.1, −1.3)</td>
<td>(+0.3, −1.1)</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>22.4 (5.2)</td>
<td>22.2 (5.4)</td>
<td>n.s.</td>
<td>n.s.</td>
<td></td>
</tr>
<tr>
<td><strong>Systolic blood pressure (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Exercisers</td>
<td>129.8 (14.7)</td>
<td>131.1 (14.2)</td>
<td>(+5.5, −2.8)</td>
<td>(+6.8, −4.6)</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>125.9 (11.2)</td>
<td>126.2 (10.2)</td>
<td>n.s.</td>
<td>n.s.</td>
<td></td>
</tr>
<tr>
<td><strong>Diastolic blood pressure (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercisers</td>
<td>85.3 (8.1)</td>
<td>85.3 (11.7)</td>
<td>(+4.0, −3.9)</td>
<td>(+3.1, −5.4)</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>80.1 (8.3)</td>
<td>82.1 (7.8)</td>
<td>n.s.</td>
<td>n.s.</td>
<td></td>
</tr>
<tr>
<td><strong>Total plasma cholesterol (mmol l$^{-1}$)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercisers</td>
<td>6.5 (1.2)</td>
<td>5.6 (1.1)</td>
<td>(−0.54, −1.27)</td>
<td>P &lt; 0.0001</td>
<td>(+0.27, −0.62)</td>
</tr>
<tr>
<td>Controls</td>
<td>6.2 (1.2)</td>
<td>5.5 (0.8)</td>
<td>(−0.46, −0.01)</td>
<td>P &lt; 0.0001</td>
<td></td>
</tr>
<tr>
<td><strong>High-density lipoprotein (mmol l$^{-1}$)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercisers</td>
<td>1.12 (0.22)</td>
<td>1.29 (0.24)</td>
<td>(+0.86, +0.26)</td>
<td>(0.05, −0.21)</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>1.16 (0.20)</td>
<td>1.26 (0.27)</td>
<td>(−0.68, −0.28)</td>
<td>&lt; 0.0001</td>
<td></td>
</tr>
<tr>
<td><strong>Triglycerides (mmol l$^{-1}$)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercisers</td>
<td>1.38 (0.33)</td>
<td>1.36 (0.54)</td>
<td>(+0.24, −0.28)</td>
<td>(+0.31, −0.15)</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>1.34 (0.54)</td>
<td>1.42 (0.70)</td>
<td>n.s.</td>
<td>n.s.</td>
<td></td>
</tr>
<tr>
<td><strong>First workload steady-state heart rate (beats min$^{-1}$)</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Exercisers</td>
<td>105 (12)</td>
<td>93 (10)</td>
<td>(−8.5, −15.2)</td>
<td>(−2.3, −13.6)</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>105 (12)</td>
<td>101 (10)</td>
<td>(−0.8, −8.6)</td>
<td>P &lt; 0.01</td>
<td></td>
</tr>
<tr>
<td><strong>Second workload steady-state heart rate (beats min$^{-1}$)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercisers</td>
<td>125 (10)</td>
<td>113 (7)</td>
<td>(−7.9, −15.8)</td>
<td>(−8.0, −10.7)</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>124 (9)</td>
<td>118 (8)</td>
<td>(−2.7, −9.4)</td>
<td>P &lt; 0.05</td>
<td></td>
</tr>
<tr>
<td><strong>Third workload steady-state heart rate (beats min$^{-1}$)</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Exercisers</td>
<td>149 (10)</td>
<td>132 (8)</td>
<td>(−13.5, −19.9)</td>
<td>(−7.8, −16.2)</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>148 (9)</td>
<td>143 (12)</td>
<td>(−1.8, −7.6)</td>
<td>P &lt; 0.0001</td>
<td></td>
</tr>
<tr>
<td><strong>Estimated VO$_{2\text{max}}$ (ml kg$^{-1}$ min$^{-1}$)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercisers</td>
<td>33.0 (5.7)</td>
<td>39.6 (6.6)</td>
<td>(+5.4, +7.8)</td>
<td>(+3.2, +6.6)</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>32.1 (5.9)</td>
<td>33.6 (6.1)</td>
<td>(+0.37, +2.9)</td>
<td>P &lt; 0.0001</td>
<td></td>
</tr>
<tr>
<td><strong>Sit-ups (repetitions)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercisers</td>
<td>27.6 (6.3)</td>
<td>34.4 (4.9)</td>
<td>(+5.2, +8.5)</td>
<td>(+3.1, +7.0)</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>27.0 (7.4)</td>
<td>29.9 (8.4)</td>
<td>(+0.7, +2.9)</td>
<td>P &lt; 0.0001</td>
<td></td>
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<tr>
<td><strong>Flexibility (cm)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercisers</td>
<td>28.0 (9.1)</td>
<td>34.1 (6.8)</td>
<td>(+4.0, +6.8)</td>
<td>(+3.3, +6.9)</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>28.2 (6.9)</td>
<td>28.5 (6.8)</td>
<td>(+1.5, −1.0)</td>
<td>P &lt; 0.0001</td>
<td></td>
</tr>
</tbody>
</table>

* Values are mean (s.d.); † for 95% confidence intervals P is indicated if significant, otherwise the result is shown to be n.s. (not significant)
evaluation

Figure 1. Mean training heart rate and standard deviation for the exercise group (21 men) over the 10-week training period

Estimated $V_{O_{2\max}}$ and local muscular endurance. However, the average improvement for the exercisers was significantly greater than that for the controls for all steady-state heart rates, estimated $V_{O_{2\max}}$, local muscular endurance and flexibility.

Table 2 gives the mean values for total plasma cholesterol for the Test 1 blood samples carried out immediately after Test 1 and 10 weeks later. A paired t-test ($P < 0.87$) and 95% confidence intervals show no significant difference between the samples analysed immediately after Test 1 and samples analysed 10 weeks later.

### Discussion

#### Aerobic fitness

This 10-week study produced a 20% increase in predicted $V_{O_{2\max}}$ which compares favourably with other studies with a similar exercise prescription and initial values. The mean training heart rate of 88% of predicted maximum heart rate (which approximates to 80% $V_{O_{2\max}}$) could be one reason why the training programme resulted in such a large increase in $V_{O_{2\max}}$. Wenger and Bell show that $V_{O_{2\max}}$ improvements are greater as intensity of training increases from 50% to 100% of $V_{O_{2\max}}$. Thus, a mean frequency of 2.7 sessions per week including a 20 min session of aerobic training results in a marked improvement in aerobic fitness. The decrease in submaximal heart rate is also similar to other aerobic programmes with a comparable training prescription. That the control group demonstrated a significant fall in heart rate in the second and third work stages indicates that other variables unrelated to the training programme influenced the heart rates. Test 1 was carried out in the first week of the New Year. Most subjects had been extremely indolent over the holiday period whereas several became more active after the vacation. Activity diaries showed that several control subjects walked to and from work. This regular exercise could have influenced the aerobic fitness levels of some controls. Less apprehension in the test environment could be another explanation for the apparent improvement in aerobic fitness in the control group.

#### Body composition

A decrease in percentage body fat may have been expected in this study as energy expenditure increased during the 10-week training period. A recent study by Grant et al. investigated the responses of 10 women to a 'tune-up' in a laboratory. The mean(s.d.) energy expenditure for the women (mean(s.d.) body weight of 54.0(5) kg was 254(35) kcal per session. Similar relative exercise heart rates were found between the subjects in this study and the female group. The energy expenditure in a heavier male group might be expected to be higher but this 10-week tune-up programme did not result in a decrease in percentage body fat.

#### Blood pressure

At Test 1 the mean blood pressure values for the exercise group and the control group were both normotensive. Other studies with similar initial blood pressure levels to those of the present study have suggested that a fall in blood pressure with aerobic training would be unlikely as it is not possible to reduce blood pressure in the normal range by a large amount. In their review of the effects of aerobic training in normotensives and hypertensives Fagard et al. stressed that the pretraining blood pressure is an important determinant of the pressure response to training but conceded that an increase in physical work capacity is linked to a blood pressure lowering effect. In most aerobic exercise/blood pressure studies the exercise groups demonstrated an improvement in fitness. Our study supported these findings. However, Seals and Hagberg indicate that it is not essential to improve $V_{O_{2\max}}$ to elicit changes in cardiovascular factors at rest.

#### Lipids

Total plasma cholesterol and high-density lipoprotein cholesterol were altered in both groups. That both
groups demonstrated a similar ‘change’ suggests some factor other than exercise influenced these variables. Seasonal variation in total plasma cholesterol levels (peak in December, lowest in June) was found by Gordon et al.24 However, dietary and weight changes could only explain one-third of the seasonal variation of 0.20 mmol l⁻¹. This variation was much smaller than that seen in our study. It began in early January and lipid levels could have been influenced by an atypical diet over the Christmas period. The alteration in both groups in total plasma cholesterol and HDL cholesterol could not be attributed to methodological error. Blood sampling was standardized with the subject seated after a 12-h fast. All analyses were carried out on the same instrument and accuracy checks were performed. Furthermore, Test 1 total plasma cholesterol samples were analysed with the same reagents as Test 2 samples and no significant difference was found.

There is no consensus on effect of body weight changes in total plasma cholesterol levels after training. Vu Tran et al.25 in their meta-analysis showed that the subjects with the greatest reduction in body weight demonstrated the greatest decrease in total plasma cholesterol. Perhaps the non-significant fall in body weight in our study resulted in a non-significant decrease in total plasma cholesterol. Some researchers have suggested that improvements in fitness are insufficient in themselves to alter HDL cholesterol levels. Williams et al.26 failed to demonstrate changes in HDL cholesterol until the subjects had maintained 10 miles a week of running for at least 9 months. They also reported that, despite an earlier increase in aerobic fitness and a decrease in body weight the changes in HDL cholesterol did not manifest themselves until later. They suggested that this training threshold for HDL cholesterol increases might be ‘turned on’ by some physiological process, for example an increase in lipoprotein lipase activity. However, several studies of short duration have disputed the theory that relatively long training programmes are necessary to influence HDL cholesterol.27, 28

Both the exercise and control groups had initial triglyceride levels well within the guidelines of the European Atherosclerosis Society.29 While some studies have demonstrated a decrease in triglycerides with baseline levels similar to the present study or below, the literature indicates that aerobic training is unlikely to lower triglyceride levels under 1.35 mmol l⁻¹.

**Flexibility**

The Sit and Reach test has been used to evaluate changes in flexibility in a variety of training regimes with wide-ranging age groups and male and female subjects. The increase in our exercise group of 6 cm is large compared with Lucas and Koslow30 whose various flexibility groups showed a mean increase of around 3 cm, and the circuit programme of Wilmore et al.31 which failed to improve flexibility. Research32, 33 suggests that long-duration static stretching carried out with elevated body temperatures is optimal for improvement in flexibility. In our study the flexibility exercises were carried out at the end of the ‘tune-up’ session when body temperature would be raised. Furthermore, static stretching was emphasized and stretches were held for around 20 s.

**Local muscular endurance**

The tune-up sessions were effective in increasing the local muscular endurance in the abdominal area of the exercise group. Sit-ups were performed between 30 s and 1 min at or near maximal effort. Thus, the 1-min sit-up test not only mirrored the muscle movement but the predominant energy system. Anaerobic glycolysis would be emphasized during the maximal test. Our findings support those of other groups34, 35 using training programmes which have included abdominal exercises in the exercise programme and which have shown increased local muscular endurance as measured by sit-ups.35 In their comparison of myoelectrical activity in selected abdominal and hip flexor muscles across eight variations of the sit-up, Hall et al.7 reported considerable involvement of the rectus abdominis, external oblique and rectus femoris in a sit-up movement similar to the sit-up in this study.

**Conclusion**

Significant and substantial improvements in estimated VO₂max local muscle endurance and flexibility were achieved by the exercise group. However, an improvement in aerobic fitness does not necessarily produce beneficial changes in coronary risk factors.

**Acknowledgement**

We wish to thank Dr Rodney Taylor of St Loyes College, Exeter, UK for his constructive comments on this paper.

**References**

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