Randomized controlled trial of home-based exercise training to evaluate cardiac functional gains

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ABSTRACT

There is evidence that multiple benefits can be obtained through exercise training that leads to increases in peak oxygen consumption (\(V_\text{O}_2\max\)). It is unclear whether significant improvements can also be achieved through unsupervised low-budget home-based training regimes, especially in terms of cardiac functional gains. A randomized cross-over trial was conducted to investigate the effects of a home-based unsupervised exercise training programme of moderate intensity on aerobic capacity, cardiac reserve and peak cardiac power output in healthy middle-aged volunteers. Nine subjects with no known cardiovascular diseases performed symptom-limited treadmill cardiopulmonary exercise tests after an 8-week period of exercise training, and results were compared with those obtained after a similar ‘non-exercising’ control period. Cardiac output was measured non-invasively during exercise tests using the CO2-rebreathing method. With exercise training, resting heart rate decreased significantly from 88\(–\)3\(\pm\)3 to 78\(–\)7\(\pm\)2 beats\[\text{min}\]\(^{-1}\) (\(P<0.05\)), heart rate at a submaximal workload (\(V_\text{O}_2\)fl) decreased from 125.5\(\pm\)2.4 to 115.5\(\pm\)1.6 beats\[\text{min}\]\(^{-1}\), and peak \(V_\text{O}_2\) increased by 9\% from 2.62\(\pm\)0.19 to 2.85\(\pm\)0.18 litres\[\text{min}\]\(^{-1}\) (\(P<0.01\)). Baseline cardiac power output was 1.11\(\pm\)0.05 W, and this remained unchanged with training. Peak cardiac power output increased by 16\% from 4.1\(\pm\)0.3 to 4.7\(\pm\)0.3 W (\(P<0.001\)), and cardiac reserve increased by 21\% (\(P<0.01\)). A major contribution to these increases was from the 11\% increase in stroke volume, from 100.1\(\pm\)5.3 to 111.2\(\pm\)6.2 ml (\(P<0.001\)). All subjects reported more positive perceptions of their health (\(P<0.05\)), fitness (\(P<0.01\)) and levels of activity (\(P<0.01\)) after the training period. These results show that motivated subjects undergoing low-budget unsupervised home-based exercise training of moderate intensity can derive benefit in terms of symptoms, aerobic capacity and cardiac functional reserve.

INTRODUCTION

There is evidence from classic studies involving normal young and middle-aged adults [1–4], and from more recent studies particularly involving older adults [5–9], that benefits can be obtained through exercise training. Although specific to the intensity and type of training programme, exercise training can lead to an increase in peak oxygen consumption (\(V_\text{O}_2\max\)) [1–9], which may be achieved through cardiac adaptation resulting in increased peak cardiac output [10,11] and peripheral adaptations leading to greater oxygen utilization [12], or
both. Regular aerobic exercise has many cardiovascular effects in both men and women [13–15], and may even reduce the risk of fatal or non-fatal myocardial infarction, as well as other coronary events [16–18]. There is increasing evidence that regular aerobic exercise provides some degree of prognostic benefit in the prevention of coronary events [16–18]. The costs of joining exercise classes or clubs are often beyond the means of the majority of populations. Therefore, for motivated individuals, the possibility of performing a low-budget home-based exercise training regime is attractive, but it is unclear whether such a regime can confer sufficient benefit, especially in terms of cardiovascular function.

Exercise training has been shown to improve $V_{\text{o}2\max}$ [1–9] and skeletal muscle function [12,19,20]. The performance of each group of skeletal muscles can be readily measured [12,19,20]. The enhanced peak performance of all the exercising muscles contributes towards the higher $V_{\text{o}2\max}$. The performance of cardiac and vascular systems, however, is not as readily measured during assessments of the impact of exercise training. Part of the reason is that commonly measured clinical parameters of cardiac function, such as left ventricular ejection fraction, are unsatisfactory indicators of the efficacy of the effects of training on cardiac function [21]. Increases in left ventricular end-diastolic diameter at rest and at peak exercise [22–25] following exercise training have been demonstrated. Studies investigating changes in myocardial contractility have been inconsistent. Using the extent or velocity of circumferential fibre shortening as an index, increases [26] or decreases [27] in contractility have been reported. In endurance-trained older men, cardiac adaptations are characterized by volume-overloaded left ventricular hypertrophy and enhancement of left ventricular peak systolic performance [23]. It is also difficult to be certain that an observed improvement in a component function is not negated by a deterioration in another component function (e.g. more myocardial hypertrophy through power training in a subject with hypertrophic cardiomyopathy, thereby worsening ventricular diastolic dysfunction), giving an overall lack of improvement or even a deterioration. There is a need to use a reliable and representative variable to evaluate overall cardiac function.

The use of various indices to assess cardiac performance, particularly in relation to heart failure, has been re-examined, and an alternative conceptual approach of evaluating physiological cardiac reserve has been proposed [30,31]. We have also developed a non-invasive method of measuring such functional reserve [28] based on the conceptual basis formulated [29–31]. Recent investigations have shown that peak power cardiac output is not only a major determinant of exercise capacity [28,30], but is also the most powerful predictor of prognosis in cohorts of heart failure patients [31–34]. To our knowledge, this form of cardiac evaluation has not been applied in the assessment of the cardiac response to exercise training. The purpose of the present study was to use this method to investigate how much cardiac functional gain can be achieved through moderate-intensity exercise training in healthy volunteers.

**METHODS**

**Subjects**

Nine middle-aged subjects (eight male and one female), with a mean ±S.D. age of 58.1 ± 7.0 years and body weight of 79.6 ± 7.3 kg, who had no known history or physical evidence of cardiovascular or respiratory diseases, volunteered to participate in the study. The age and weight criteria were selected to reflect the age group and weights of patient populations presenting with coronary artery disease. All participants were fully informed about the study, which had been approved by the local hospital ethics committee, and all gave written consent.

**Study protocol**

This was a randomized cross-over study, during which each subject was tested following 8 weeks of exercise training (training phase) and 8 weeks of ‘non-exercising’. All participants underwent the tests before and after both the non-exercising control and training phases.

**Exercise testing**

All participants attended the laboratory before starting the study, to undergo a period of familiarization in order to improve the reproducibility of the exercise tests [35,36] and to confirm the absence of any cardiovascular disease. The subjects performed symptom-limited treadmill exercise tests, using a programmable treadmill (Quinton; model no. Q55). The participants performed a 1-min incremental test developed in our laboratory. Blood pressure was monitored every 3 min during exercise, and heart rate was monitored continuously. On-line breath-by-breath respiratory gas analysis was performed at rest, during the incremental test and during the recovery period, using the MedGraphics CardiO2 Cardiopulmonary Exercise testing system (Medical Graphics Corporation, St. Paul, MN, U.S.A.).

Following a rest period of at least 30 min, to ensure that $V_{\text{o}2}$ end-tidal partial pressure of CO$_2$, heart rate and blood pressure had returned to within 5% of the pre-incremental test values, cardiac output was measured at rest. Then, during a constant-load test, with the workload set to achieve $V_{\text{o}2}$ levels within 5% of the $V_{\text{o}2\max}$ attained during the incremental test, cardiac output was again measured. At least three estimates of cardiac output were taken both at rest and during the constant-load exercise test. At rest, estimates of cardiac output were
made using the equilibrium $\text{CO}_2$-rebreathing method, while during peak exercise the exponential $\text{CO}_2$-rebreathing method was employed [28].

**Exercise training protocol**

Participants trained for 8 weeks at home, on a static, upright cycle (Tunturi F220). They were instructed to exercise at 50 rev·min$^{-1}$ for a 20 min period on 5 days per week, with the resistance on the flywheel set to produce a heart rate that was consistent with a $\dot{V}_{\text{O}_2}$ at 75–80% of the $\dot{V}_{\text{O}_2}\text{max}$ achieved during the control incremental test. Heart rate was monitored using a commercial short-distance telemetry system (Polar Fitness Watch, Polar, Finland). Heart rate was used as there were no facilities to monitor $\dot{V}_{\text{O}_2}$ in the home. Compliance to the training protocol was assessed from a measure of the ‘distance covered’ during the training, based on a count of the number of cycle wheel revolutions. Poor compliance was judged as a training distance of less than 60% of a potential maximum distance. Participants also completed a daily exercise diary. Additional exercise, e.g. walking, cycling and swimming, was also allowed, but the extent of this was not quantified formally. Following 4 weeks of exercise, participants underwent an incremental test to assess for any training effect and to allow for the setting of a new target heart rate for the remainder of the training period.

**Non-exercising control phase**

During this period, all participants were instructed to carry out their pre-baseline activities and to avoid any additional exercise. Individuals who performed weekly activities, such as swimming and golf, were asked to avoid such activities until after the non-exercising control period.

**Self-assessment questionnaire**

During each visit a self-assessment questionnaire, based on a Likert scale (see Table 2), was completed.

**Calculations**

Treadmill workload was estimated using the following equation [37]:

$$\text{Workload} (W) = \frac{(\text{weight} \times \text{speed} \times \sin \beta)}{6.12}$$

where weight is in kg, speed is in m·min$^{-1}$ and $\beta$ is the angle of elevation.

Cardiac power output (CPO) was calculated from the averaged cardiac output and mean arterial pressure using the following equation [29]:

$$\text{CPO} = (Q \times \text{MAP}) \times K$$

where CPO is in W, Q is cardiac output in litres·min$^{-1}$, MAP is mean arterial pressure in mmHg, and $K$ is the conversion factor ($2.22 \times 10^{-3}$). Cardiac reserve is equal to the difference between baseline resting CPO and peak CPO.

Arteriovenous $\text{O}_2$ content difference, expressed as ml of $\text{O}_2$/dl, was calculated as $\left(\frac{\dot{V}_{\text{O}_2}}{Q}\right) \times 100$. The anaerobic threshold was calculated off-line on completion of the exercise test, using the V-slope method proposed by Beaver et al. [38].

**Statistical analysis**

For data with a normal distribution, comparisons between each period of the study were compared using ANOVA and Student’s $t$-test. The self-assessment questionnaires were analysed using the Mann–Whitney $U$ test. Statistical significance was taken to be $P < 0.05$ in all cases. Data are presented as means$\pm$S.E.M. unless otherwise indicated. The presence of a training effect was determined by analysing the heart rate–$\dot{V}_{\text{O}_2}$ relationship [39,40]. A statistically significant decrease in the elevation of the computed regression line relating heart rate to $\dot{V}_{\text{O}_2}$ after training was regarded as evidence of a training effect. In addition, the heart rates at a fixed submaximal $\dot{V}_{\text{O}_2}$ before and after training were also compared to confirm the presence of a training effect [41].

**RESULTS**

Following the training period, when compared with the non-exercise control period, all subjects had a lower heart rate at rest ($78.7 \pm 3.2$ and $88.3 \pm 3.4$ beats·min$^{-1}$ respectively; $P < 0.05$). In addition, at a submaximal workload ($\dot{V}_{\text{O}_2} 1.5$ litres·min$^{-1}$) the mean heart rate was lower after training ($115.5 \pm 1.6$ beats·min$^{-1}$) than after the ‘non-exercising control’ phase ($125.5 \pm 2.4$ beats·min$^{-1}$; $P < 0.001$). Only three of the nine sub-jects showed a significant decrease in the heart rate–$\dot{V}_{\text{O}_2}$ relationship.

Following training, $\dot{V}_{\text{O}_2}\text{max}$ was increased significantly by 9%, to $2.85 \pm 0.18$ litres·min$^{-1}$, compared with values of $2.62 \pm 0.19$ litres·min$^{-1}$ after the non-exercising control phase ($P < 0.01$) and $2.64 \pm 0.17$ litres·min$^{-1}$ at baseline ($P < 0.05$). Mean body weight did not alter significantly throughout the study. Baseline mean body weight was 80.11 kg, compared with 79.89 kg following the non-exercising control period and 79.67 kg following exercise training. After training, subjects exercised for significantly longer ($762 \pm 34$ s, compared with $679 \pm 27$ s following the non-exercising control phase ($P < 0.001$) and $642 \pm 29$ s at baseline ($P < 0.001$)). They also achieved a higher peak workload after training ($333 \pm 25$ W, compared with $272 \pm 22$ W following the non-exercising control phase ($P < 0.001$) and $252 \pm 26$ W at baseline ($P < 0.001$)).
Table 1  Summary of haemodynamic, respiratory and exercise data for normal subjects

Values are presented as means (S.E.M.) (n = 9). Abbreviations: HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; Q, cardiac output; SV, stroke volume; SVR, systemic vascular resistance; CPO, cardiac power output; a-vdO2, arteriovenous O2 content difference; AT, anaerobic threshold; AT (%), anaerobic threshold as a percentage of \( V_\text{O2} \) max; ET, exercise time; WLmax, maximum workload. Significance of differences: **\( P < 0.01 \), ***\( P < 0.001 \) post-exercise training compared with baseline; †\( P < 0.05 \), ††\( P < 0.01 \) and †††\( P < 0.001 \) post-exercise training compared with non-exercising period.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Non-exercise control</th>
<th>Post-exercise training</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats min(^{-1}))</td>
<td>86.67 (5.21)</td>
<td>88.33 (3.44)</td>
<td>78.67 (2.19)‡</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>125.56 (4.12)</td>
<td>130.60 (5.92)</td>
<td>128.33 (4.17)</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>76.67 (2.36)</td>
<td>78.89 (2.99)</td>
<td>81.11 (2.17)</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>92.96 (2.48)</td>
<td>96.12 (3.77)</td>
<td>96.82 (2.71)</td>
</tr>
<tr>
<td>( Q (l \text{ min}^{-1}) )</td>
<td>5.39 (0.17)</td>
<td>5.40 (0.16)</td>
<td>5.44 (0.18)</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>64.23 (4.66)</td>
<td>61.92 (3.15)</td>
<td>70.28 (4.14)</td>
</tr>
<tr>
<td>CPO (W)</td>
<td>1.11 (0.05)</td>
<td>1.15 (0.06)</td>
<td>1.18 (0.05)</td>
</tr>
<tr>
<td>a-vdO2 (ml of O2/dl)</td>
<td>15 –</td>
<td>18 –</td>
<td>20 –</td>
</tr>
<tr>
<td>SVR (dynes s cm(^{-5}))</td>
<td>1397 (59.00)</td>
<td>1462 (77.60)</td>
<td>1436 (52.40)</td>
</tr>
<tr>
<td>( V_\text{O2} ) (l kg(^{-1}))</td>
<td>0.37 (0.03)</td>
<td>0.42 (0.03)</td>
<td>0.40 (0.03)</td>
</tr>
<tr>
<td>( V_\text{O2} ) (l min(^{-1}))</td>
<td>4.66 (0.31)</td>
<td>5.22 (0.34)</td>
<td>5.11 (0.40)</td>
</tr>
<tr>
<td>AT (l min(^{-1}))</td>
<td>1.74 (0.08)</td>
<td>1.72 (0.09)</td>
<td>1.74 (0.12)</td>
</tr>
<tr>
<td>AT (%)</td>
<td>66.63 (2.60)</td>
<td>66.12 (3.13)</td>
<td>61.41 (3.81)</td>
</tr>
<tr>
<td>ET (s)</td>
<td>642 (29)</td>
<td>679 (27)</td>
<td>762 (34)†††</td>
</tr>
<tr>
<td>WLmax (W)</td>
<td>252 (26)</td>
<td>272 (22)</td>
<td>333 (25)†††</td>
</tr>
</tbody>
</table>

Figure 1  Mean peak CPO and individual CPO estimates for normal subjects in the various phases of the study

Post-training values were significantly greater than the baseline (\( P < 0.001 \)) and non-exercise control (\( P < 0.001 \)) values. Individual data are presented, along with the mean ± S.E.M. The * symbol denotes the female participant.

Table 1 shows measured and derived values obtained at the baseline study and following the non-training and training phases. Apart from heart rate showing a significantly lower value (\( P < 0.05 \)) after the training compared with the non-training period, comparison of other values obtained while the subjects were at rest showed no other significant differences after the training compared with the non-exercising control period.

Peak heart rate and anaerobic threshold did not alter with training status. However, CPO, stroke volume and cardiac output at peak exercise were significantly greater following exercise training (Table 1 and Figures 1 and 2) compared with baseline and non-exercising control values. Peak CPO increased by 16% from 4.05 ± 0.3 W following the non-exercising control period to 4.70 ± 0.35 W after exercise training (\( P < 0.001 \)), and cardiac reserve increased by 21% (\( P < 0.01 \)). A major
contribution to these improvements was observed in the significant 11.2% rise in peak stroke volume, from 100.1 ± 5.3 to 111.2 ± 6.2 ml (P < 0.001). In the absence of significant changes in peak heart rate, this increase was similar to the 12% increase in peak cardiac output (from 16.9 ± 0.95 to 18.9 ± 1.1 litres·min⁻¹; P < 0.01). There were no-significant increases in mean arterial pressure and decreases in systemic vascular resistance and arteriovenous \( O_2 \) content difference.

With regard to the responses to the self-assessment questionnaire, as shown in Table 2, subjects were significantly more positive in their perceptions of their health (P < 0.05), fitness (P < 0.01) and levels of activity (P < 0.01).

<table>
<thead>
<tr>
<th>Question</th>
<th>Baseline</th>
<th>Non-exercise control</th>
<th>Post-training</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Over the past 2 weeks how do you feel about:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Your health</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Your level of fitness</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Your outlook on life in general</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>NS</td>
</tr>
<tr>
<td>Your ability to perform your daily activities</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>NS</td>
</tr>
<tr>
<td>Your ability to sleep</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>NS</td>
</tr>
<tr>
<td>Your level of energy</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>NS</td>
</tr>
<tr>
<td>Your social life, going out and seeing friends</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>NS</td>
</tr>
<tr>
<td>Your sexual activity</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>NS</td>
</tr>
<tr>
<td>Since your last visit have you noticed any difference in your:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quality of life</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>NS</td>
</tr>
<tr>
<td>Outlook on life</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>NS</td>
</tr>
<tr>
<td>Level of fitness</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Level of activity</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>&lt; 0.01</td>
</tr>
</tbody>
</table>

DISCUSSION

This randomized controlled study has demonstrated that, in motivated volunteers, a home-based supervised 8-week exercise training programme of moderate intensity significantly increased exercise capacity, \( V_{O_2} \)max and exercise duration, and led to a subjective improvement in quality of life. The increase in \( V_{O_2} \)max of 9% was due to a large extent to the improvements in overall cardiac function, as shown by a 16% increase in peak CPO and a 21% increase in cardiac reserve. The largest contribution to the enhanced cardiac function was through the 11% increase in peak exercise stroke volume, with smaller (non-statistically significant) increases in peak exercise heart rate and mean arterial pressure.

These results are notable for two reasons. First, CPO at rest and during peak exercise has not been characterized non-invasively in healthy middle-aged volunteers, and the effect of exercise training on peak CPO has also never been assessed in this category of human subjects. Consistent with a previous estimation [30], the resting CPO of an average European adult was found to be between 1.1 and 1.2 W (Table 1). The peak exercise CPO of the cohort of healthy volunteers before training was approx. 4.1 W, and this increased to 4.7 W after a period of moderate unsupervised training. Secondly, the present study has demonstrated that, in appropriately motivated individuals, low-budget home-based unsupervised exercise training programmes can provide significant functional and cardiac benefits. Whether the improvement could be greater with supervised exercise training programmes in healthy middle-aged individuals has not been evaluated systematically.

The observed 9% increase in \( V_{O_2} \)max was not as high as the increases of 18% reported by Saltin et al. [1], 16% by Ekblom et al. [2] or 17–21% by Stratton et al. [25]. The differences in the magnitude of the increment may be explained by differences in the age range of the subjects and in the mode and intensity of the exercise training programmes. In all the quoted cases, the training was supervised in training centres and lasted longer. Ekblom et al. [2] studied young men aged between 19 and 27 years and used a 16-week programme involving cross-country running; \( V_{O_2} \)max increased by 16%. The mean baseline peak CPO of these subjects was 5.7 W, which was 1.6 W greater than that in our study population, suggesting that these subjects also had superior cardiac function. After training, their power output increased to 6.8 W, an increment of 17%, which was similar in magnitude to that found in our study population. Stratton et al. [25]
studied younger and older subjects, and adopted a 6-month training programme, incorporating walking, jogging and cycling. In their older age group (mean age 68 ± 6 years), $V_{\text{O}_2\text{max}}$ increased by 21 %, compared with an increase of 17 % in the younger group (age 28 ± 3 years). In our study the duration of training was shorter. Saltin et al. [1] showed that the largest increases in $V_{\text{O}_2\text{max}}$ occurred in the students who were less fit at the start of the study. Peak $V_{\text{O}_2}$ only increased by 4 % in their active subjects [1]. Some of the volunteers in our study were reasonably fit at the start, and this could explain the relatively modest degree of improvement.

In the present study we have demonstrated that central haemodynamic adaptations to training can occur in healthy middle-aged subjects. We did not set out to address the effects of training on skeletal muscle physiology, which has been extensively investigated and reported [12,19,20]. We have shown that moderate-intensity exercise training does have beneficial effects on the performance of the heart itself. Consistent with previous studies [2,3,25] there was a significant fall in resting heart rate and a 12 % increase in cardiac output. Compared with our study, Ekblom et al. [2] reported a smaller increase in peak cardiac output (8 %), whereas the increase reported by Stratton et al. [25] was larger (16 %). The differences quoted in the literature may be explained by the fact that different exercise testing protocols were used, along with different training protocols. Exercise testing on a bicycle ergometer imposes a greater demand on the contractile power of the exercising leg muscles, thereby putting a different strain on the cardiopulmonary system than testing on a treadmill. Ekblom et al. [2] used cycle ergometry, whereas Stratton et al. [25] used treadmill testing. The degree of central haemodynamic adaptation may be directly related to the intensity and duration of training. As shown in patients with chronic heart failure [42] and in patients following myocardial infarction [43], high-intensity training is required to produce central training effects.

Spina et al. [44] demonstrated that gender differences may also affect changes in cardiac output following exercise training. They demonstrated that, in older women (60–69 years), the increase in $V_{\text{O}_2}$ was accounted for by an increase in the arteriovenous oxygen content difference. In contrast with their findings, the one female participant in the present study did increase her cardiac output, by 13.0 %. Indeed, her 5.0 % increase in $V_{\text{O}_2}$ was achieved as a result of the increase in cardiac output, as her arteriovenous oxygen content difference was slightly lower following exercise training compared with values at baseline and after the non-exercise phase. However, our female participant was much younger (50 years old) than the female subjects in the study of Spina et al. [44].

It is worth mentioning that our investigation found that exercise training produced beneficial effects on cardiac function and, of all the indicators of cardiac function, the greatest percentage improvements were observed in peak exercise $V_{\text{O}_2}$ and cardiac reserve. This finding is consistent with the concept that the overall peak function of the heart is best represented by the peak $V_{\text{O}_2}$, which reflects the cumulative sum of the (positive and negative) changes in various component functions of the heart. The feasibility of achieving improvements in $V_{\text{O}_2}$ through physiological means, i.e. exercise training, may also have important implications, in the light of findings indicating that peak $V_{\text{O}_2}$ is the most powerful predictor of prognosis in cohorts of patients with heart failure [29,32–34,45]. Whether comparable improvements in peak $V_{\text{O}_2}$ can be achieved through pharmacological or surgical therapy requires further study.

In conclusion, we have shown that, in motivated participants, home-based unsupervised exercise training on cycle ergometers can improve exercise capacity, $V_{\text{O}_2\text{max}}$, exercise duration, cardiac functional gains, and perceived fitness and health. The results also suggest that evaluation of peak $V_{\text{O}_2}$ and cardiac reserve provides a useful index for directly quantifying the overall cardiac functional gain obtained through exercise training. Further research is required to investigate whether patients with cardiac disease and heart failure can acquire similar gains, and whether the functional gain is due to any improvement in the intrinsic function of the heart.

ACKNOWLEDGMENTS

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Exercise training improves cardiac reserve