Left Ventricular Adaptation to 12 Weeks of Indoor Cycling at the Gym in Untrained Females

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ABSTRACT
Cross-sectional studies provide evidence of larger cardiac dimensions and mass in endurance trained than in untrained females. Much less is known regarding adaptations in cardiac function following training in untrained subjects. We aimed to study left ventricular (LV) adaptation to indoor cycling in previously untrained females, in regard of LV dimensions, mass and function. 42 sedentary females were divided into 2 equally sized groups, either training indoor cycling at regular classes at a local gym for 12 weeks, in average 2.6 times per week, or maintaining their sedentary lifestyle. Echocardiography at rest and a maximal exercise test were performed before and after the intervention. Exercise capacity increased in average 16 % in the exercise group (p < 0.001), together with decreased heart rate at rest (p < 0.05) and at 120 watts steady-state (p < 0.001). There were no difference in systolic or diastolic function following the intervention and minimal increases in LV internal diameter in diastole (+ 1 mm, p < 0.01). LV mass was unchanged with training (137 ± 25 vs. 137 ± 28 g, p = 0.911). Our findings indicate that attending indoor cycling classes at a gym 2-3 times per week for 12 weeks is enough to improve exercise capacity, while a higher volume of training is required to elicit cardiac adaptations.

Introduction
Competitive endurance trained female athletes typically present with larger cardiac internal dimensions and slightly thicker left ventricular walls compared to sedentary subjects, resulting in a larger left ventricular mass [12, 18, 25]. In addition, we have previously found differences in cardiac function at rest between elite female endurance athletes and sedentary females [12]. In contrast to studies of elite athletes, the minimum weekly amount of leisure time exercise needed to elicit cardiac adaptations is largely unknown. A weekly volume of more than 3 h of exercise has been suggested [9], while interventional exercise studies report increases in cardiac dimensions with less than 2 h of endurance training per week [2]. As most studies comparing trained and untrained subjects are cross-sectional, drawing definitive conclusions about a training effect upon cardiac size and function is somewhat difficult. The limited number of longitudinal studies investigating effects on cardiac size and function with endurance training in females often apply vigorous exercise regimens [2, 11, 20, 22], making the applicability and feasibility somewhat low.

The purpose of the current longitudinal, controlled trial was to determine the effects of attending regular indoor cycling classes 2-3 times per week, for 12 weeks at a local gym on exercise capacity and left ventricular dimensions, mass and function at rest in untrained females.

Methods
Subjects
53 healthy, non-smoking women (age 21–45 years) without regular participation in aerobic exercise training within the last year were recruited and allocated into either an exercise (EX) or control (CON) group. A majority of subjects were students or hospital employees. All were non-pregnant, without hypertensive medication...
or any history of cardiovascular disease or diabetes and able to perform a maximal bicycle ergometer test.

4 subjects in the EX group and 7 CON dropped out during the 3 months between baseline and follow-up examinations, and the final study population included 21 subjects in each group (∗ Table 1). The regional ethical review board in Linköping, Sweden approved the study. All subjects gave their written consent to participate and the study meets recently published ethical standards in sport and exercise science as well as the standards of the IJSM [10].

Exercise training
While control subjects were instructed to maintain their sedentary lifestyle, participants in the EX group were instructed to perform indoor cycling 3 times per week for 12 weeks. To obtain high generalizability and feasibility, they attended regular instructor-led indoor cycling classes at a local gym. Participants recorded their attendance and possible comments following each exercise session and the exercise diaries were collected after the intervention.

Exercise intensity and duration of each session were not monitored in detail. In brief, each session lasted for 45–60 min and consisted of a warm-up, followed by aerobically more challenging phases where resistance and cadence were altered until a period of peak effort was reached, followed by a cool-down phase.

Procedures

Echocardiography
Echocardiographic examination was carried out on the same day, prior to the exercise test and subjects were instructed to refrain from heavy exercise and alcohol at least 12 h and caffeine for 3 h before examination. The same echocardiographer performed all examinations. A HDI 5000, Philip Medical Systems digital ultrasound system (ATL Ultrasound, Bothell, WA, USA) equipped with a phased array (P4-2) transducer was used.

Left ventricular internal diameter in diastole and systole (LVIDd and LVIDs) and septal and posterior wall thickness in diastole (SWT and PWT) were determined using M-mode registrations from the parasternal long-axis view. Left ventricular mass was calculated from M-mode registrations according to current recommendations [15]. Absolute dimensions were indexed by body surface area (BSA), calculated according to the Dubois and Dubois formula.

Systolic excursion of the mitral annular plane (MAPSE) was measured with M-mode echocardiography and calculated as the average of septal, lateral, inferior and anterior wall systolic displacement. Ejection fraction was calculated according to the Teichholz formula [23], and fractional shortening (%) was determined as 100 * (LVIDd − LVIDs)/LVIDd.

In the apical 4-chamber view, blood flow velocities at the tip of the mitral leaflets in early (E) and late (A) diastole were measured with pulsed-wave blood Doppler, and the E/A-ratio was calculated. In the same view, pulsed-wave tissue Doppler imaging was utilized to measure systolic (S’) as well as early and late diastolic (e’ and a’) peak myocardial velocities at the septal and lateral portions of the mitral annulus.

Exercise test
Body mass and height, as well as heart rate and blood pressure after 10 min of rest were determined prior to the exercise test. Maximal work capacity was determined on an electronically braked cycle ergometer (RE830, Rodby Electronic, Södertälje, Sweden), connected to an exercise ECG system (Marquette CASE 8000, GE Medical Systems, Milwaukee, WI, USA). Heart rate was continuously monitored from a 12-lead ECG, while brachial systolic blood pressure and rating of perceived exertion (RPE), determined with the Borg RPE-scale [5], were determined every third minute during the test. An exercise protocol starting at 80 Watts (W) with a 10 W/min continuous workload increment until volitional fatigue was used. At 120 W, the increment was halted at a steady-state plateau for 5 min in order to obtain submaximal heart rate, blood pressure and perceived exertion.

Statistical analyses
Between-group differences were tested with Student’s t-test for continuous variables, with Mann-Whitney U test for RPE-ratings and with Chi² for smoking status. Within group differences between baseline and follow-up measurements were tested with paired t-test for continuous variables and with Wilcoxon signed Rank test for RPE-ratings. Data presented as mean ± standard deviations or median with range. A p-value ≤ 0.05 was considered statistically significant. SPSS version 23 (IBM Software, Armonk, NY, USA) was used for statistical analysis.

Results
The median time between the first and last exercise session was 12 weeks (range 10–14). In average, the EX group trained 2.6 (1.8–4.1) times per week during the 12 weeks. A statistically non-significant trend toward lower body mass at follow-up in the EX group was seen, but not in CON (EX: -0.7 ± 1.6 kg, p = 0.074, CON: +0.3 ± 1.6 kg, p = 0.478).

At the follow-up exercise test, the mean maximal workload achieved had increased by 16 % in the exercise group (p < 0.001) while being unchanged in the control group (∗ Table 2). Submaximal, steady-state heart rate at 120 W was 10 % lower at the follow-up in the exercise group (p < 0.001) compared to baseline, while unchanged in the control group. Heart rate at rest decreased from 67 to 62 beats per minute in the exercise group (p < 0.05), while unchanged in controls (p = 0.468).

In EX, a statistically significant minimal decrease in septal wall thickness (−0.4 mm (4 %), p < 0.05) and an increase in LV internal
diameter in diastole (+1 mm (2%), p < 0.01) were seen, while LV mass did not change with training (Table 3). In addition, CON presented with an equally small but statistically significant decrease in posterior wall thickness (−0.4 mm (5%), p < 0.01) and in LV mass (−6 g (5%), p < 0.05) at follow-up compared to baseline measurements. There were no statistically significant differences between groups in wall thickness or internal diameters, when BSA was taken into account. Within group comparisons for BSA-indexed dimensions. There were no statistically significant differences between groups for any variable at baseline. BP, blood pressure; RPE, rating of perceived exertion. Data presented as mean ± standard deviation or median with range.

**Table 2** Exercise test data.

<table>
<thead>
<tr>
<th></th>
<th>Exercise (n = 21)</th>
<th>Control (n = 21)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Follow-up</td>
</tr>
<tr>
<td>Heart rate at 120W</td>
<td>154 ± 15</td>
<td>139 ± 11†</td>
</tr>
<tr>
<td>Systolic BP at 120W</td>
<td>174 ± 17</td>
<td>172 ± 16</td>
</tr>
<tr>
<td>RPE at 120W</td>
<td>14 (11-17)</td>
<td>12 (9-14)†</td>
</tr>
<tr>
<td>Watt at MAX</td>
<td>197 ± 31</td>
<td>229 ± 33†</td>
</tr>
<tr>
<td>Heart rate at MAX</td>
<td>187 ± 9</td>
<td>186 ± 7</td>
</tr>
<tr>
<td>RPE at MAX</td>
<td>19 (17-19)</td>
<td>19 (17-20)</td>
</tr>
</tbody>
</table>

† and bold style, p ≤ 0.05 for with-in group repeated measures significance testing; *, p ≤ 0.001 for between-group difference at follow-up testing. No statistically significant difference was evident between groups for any variable at baseline. BSA-indexed dimensions are presented as mean ± standard deviation or median with range.

**Table 3** Left ventricular dimensions and mass.

<table>
<thead>
<tr>
<th></th>
<th>Exercise (n = 21)</th>
<th>Control (n = 21)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Follow-up</td>
</tr>
<tr>
<td>LV dimensions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SWT (mm)</td>
<td>8.8 ± 0.9</td>
<td>8.4 ± 1.0†</td>
</tr>
<tr>
<td>PWT (mm)</td>
<td>8.3 ± 1.3</td>
<td>8.1 ± 1.2</td>
</tr>
<tr>
<td>LVIDs (mm)</td>
<td>48 ± 3</td>
<td>49 ± 3†</td>
</tr>
<tr>
<td>LVIDd (mm)</td>
<td>32 ± 3</td>
<td>33 ± 3†</td>
</tr>
<tr>
<td>LV mass (g)</td>
<td>137 ± 25</td>
<td>137 ± 28</td>
</tr>
<tr>
<td>LV mass INDEX (g x m²)</td>
<td>76 ± 14</td>
<td>76 ± 15</td>
</tr>
</tbody>
</table>

† and bold style, p ≤ 0.05 for with-in group repeated measures significance testing; *, p ≤ 0.05 for between-group difference at baseline or follow-up testing. LV, left ventricular; SWT and PWT, septal and posterior wall thickness; LVIDs and LVIDd, LV internal diameter in diastole and systole, respectively. INDEX denotes indexing by body surface area.

Discussion

The unique feature of the current study was the realistic setting, where untrained females started attending instructor-led indoor cycling classes 2–3 times per week. Whether this is enough to elicit changes in cardiac dimensions or function has not been studied previously. We found that attending indoor cycling classes at a regular gym for 12 weeks had minimal effects on LV size and function at rest in females, while several signs of improved physical capacity and fitness were observed.

Thus, our results suggest that the total training dose of the intervention (i.e., exercise intensity × frequency × duration) was enough to elicit improvements in physical capacity and fitness of subjects, but not enough to induce significant cardiac adaptations. Although there were small, statistically but doubtfully clinical significant increases in LV chamber diameter (+1 mm in both systole and diastole) and a decrease in septal wall thickness (−0.4 mm), LV mass and all measures of systolic and diastolic function at rest remained unchanged with the intervention. The small increases in LV internal diameter could be related to an increased diastolic filling time, as heart rate at rest decreased with training, leading to a slightly increased end-diastolic volume rather than representing a hypertrophic response. The small decrease in LV wall thickness between baseline and follow-up measurements, seen in both EX and CON, could possibly be related to inherent variations in echocardiographic measurements [13].

In well-trained endurance athletes, there is a typical pattern of eccentric cardiac hypertrophy with increased LV end-diastolic volume, internal diameter and wall thickness [12, 25]. This hypertrophy is proposed to arise from neurohumeral signalling and biomechanical stress, resulting from repeated bouts of volume load imposed upon the heart during training [8]. This, in turn, allow for larger stroke volume during exercise, explaining the increased maximal cardiac output in endurance athletes. The minimum requirement in exercise dose for eccentric hypertrophy to occur is not clearly defined, in part due to the scarcity in longitudinal studies upon sedentary subjects. Cox et al. (1986) applied a high-intensity, high-frequency exercise regimen in 11 young untrained subjects (6 females), 6 days per week for 7 weeks and found increases in septal wall thickness (+11%), LVIDd (+3%) and LV mass (+9%) [7]. Rojek et al. (2014) found increases in LVIDd, septal wall thick-
ness and LV mass following one year of vigorous triathlon training (12 ± 1 h/week) in 21 (5 female) young non-professional athletes [19]. In addition, Mier et al. (1997) reported small, but statistically significant increases in LVIDD with only 10 consecutive days of cycling exercise in 10 untrained subjects (5 female) [17]. In males, a few longitudinal studies with weekly training regimens of 4-5 sessions per week for 6–12 weeks showed an increase in LV mass [16, 21] or LVIDD [1]. In contrast, a more recent study failed to show any increase in LV mass or LVIDD with 3 sessions of indoor cycling per week for 12 weeks in elderly females [11]. Altogether, results from previous studies indicate that both the weekly training volume and exercise intensity are possible determinants of the cardiac adaptation to training, where the intensity may play a major role.

As far as we know, no study has investigated cardiac adaptations from a realistic, instructor-led indoor cycling class previously, although a few studies have investigated physiological effects and metabolic demands in this setting [3, 4, 6, 14]. In summary, these studies show that during a standard indoor cycling class, similar to that applied in the current study, there are relatively large variations in oxygen uptake and heart rate, with considerable time spent above the anaerobic threshold [3, 4, 14]. This has led to different conclusions about the feasibility and suitability for untrained indi-
In the current study, no signs of negative effects upon the heart were apparent, while there were several signs of improved fitness in the EX group.

The observed lower heart rate at rest and during steady state cycling at 120 W in the EX group following the intervention, implies that there were training-induced adaptations affecting the heart. According to the principle of Fick, oxygen uptake equals the product of heart rate and stroke volume (i.e., cardiac output) times the arterio-venous oxygen difference [24]. As oxygen demand and thus oxygen uptake at rest and at the same submaximal workload can be assumed to be very similar pre- and post intervention, this implies that either stroke volume, the arterio-venous oxygen difference or both had changed in order to perform the same oxygen uptake with lower heart rate post intervention. Wilmore et al. (2001) found that in 354 females of varying age following 20 weeks of 3 indoor cycling sessions per week at a submaximal workload, heart rate decreased 2%, while estimated stroke volume increased 11% and arterio-venous oxygen difference increased by 6% [26]. Thus, a plausible explanation to the lower submaximal heart rate after the intervention in the current study, was improved peripheral oxygen utilization (with larger arterio-venous oxygen difference) in combination with increased stroke volume. Moreover, alterations in autonomic tone and cardiovascular regulatory mechanisms could contribute. Furthermore, it is possible that the subtle differences in LV internal diameter at rest following training would be more pronounced if measured during work, or that differences in systolic or diastolic function not detectable at rest would appear and contribute to an increased stroke volume.

Some limitations of the current study need to be addressed. First, as we chose a realistic milieu, with regular classes, we had no influence over training intensity at each session and only have data on duration and frequency of training sessions, making it impossible to calculate the exact dose of training for each subject. Second, there was variability in how many sessions each subject performed to be assumed to be very similar pre- and post intervention, this implies that either stroke volume, the arterio-venous oxygen difference or both had changed in order to perform the same oxygen uptake with lower heart rate post intervention. Wilmore et al. (2001) found that in 354 females of varying age following 20 weeks of 3 indoor cycling sessions per week at a submaximal workload, heart rate decreased 2%, while estimated stroke volume increased 11% and arterio-venous oxygen difference increased by 6% [26]. Thus, a plausible explanation to the lower submaximal heart rate after the intervention in the current study, was improved peripheral oxygen utilization (with larger arterio-venous oxygen difference) in combination with increased stroke volume. Moreover, alterations in autonomic tone and cardiovascular regulatory mechanisms could contribute. Furthermore, it is possible that the subtle differences in LV internal diameter at rest following training would be more pronounced if measured during work, or that differences in systolic or diastolic function not detectable at rest would appear and contribute to an increased stroke volume.

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In conclusion, 12 weeks of participation in regular indoor cycling classes 3 times per week were effective in improving physical fitness in untrained females. In contrast, no clinically significant adaptations in cardiac size or function at rest were seen, suggesting that significant cardiac hypertrophy in a moderately trained female should not be dismissed as physiological without considering further investigations.

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Conflict of interest
The authors have no conflict of interest to declare.

References


