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Cardiac size of high-volume resistance trained female athletes: shaping the body but not the heart

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Introduction: Exercise training, besides many health benefits, may result in cardiac remodelling which is dependent on the type and amount of exercise performed. It is not clear, however, whether significant adaptation in cardiac structure is possible in females undergoing resistance type of exercise training. Rigorous high volume training of most muscle groups emphasising resistance exercises are being undertaken by athletes of some aesthetic sports such as female fitness (light bodybuilding). The impact of this type of training on cardiac adaptation has not been investigated until now. The aim of the current study was to disclose the effect of high volume resistance training on cardiac structure and function. *Methods:* 11 top-level female fitness athletes and 20 sedentary age-matched controls were recruited to undergo two-dimensional echocardiography. *Results:* Cardiac structure did not differ between elite female fitness athletes and controls ($p > 0.05$), and fitness athletes had a tendency for a smaller ($p = 0.07$) left ventricular (LV) mass indexed to lean body mass. Doppler diastolic function index (E/A ratio) and LV ejection fraction were similar between the groups ($p > 0.05$). *Conclusions:* Elite female fitness athletes have normal cardiac size and function that do not differ from matched sedentary controls. Consequently, as high volume resistance training has no easily observable effect on adaptation of cardiac structure, when cardiac hypertrophy is present in young resistance-trained lean female, other reasons such as inherited cardiac disease are to be considered carefully.

Keywords: left ventricle, myocardial hypertrophy, resistance exercise, cardiac remodelling

Regular exercise training is a recognized factor of changes in cardiac morphology (5, 18, 23, 27). On the other hand, endurance and non-endurance training induce different morphological adaptation due to different impact on cardiac pressure and volume load (12, 13, 15, 25, 27). Endurance training tends to cause eccentric whereas strength-power training concentric left ventricle hypertrophy (1, 13). However, both types of sports could be of limited impact on changes in cardiac morphology (9, 10, 21). Among other factors, the development of cardiac remodelling in response to exercise training has been shown to be influenced by gender (19, 20, 24), and it remains questionable whether females are capable of developing the same extent and type of cardiac morphological alterations when subjected to similar training as males (8, 12, 31). Fitness sport is a class of physique-exhibition event mostly for women, which has recently gained large popularity in many countries. While being similar to bodybuilding, the sport does not require very large muscle mass itself.

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Therefore, the training of fitness sport athletes includes relatively low weights used with more repetitions performed and large overall resistance training volume. Muscle bulk is not the primary aim of fitness training, incorporation of substantial volume of aerobic exercise into the fitness sport program is not avoided. Yet, it is not clear how fitness sport practice could affect cardiac morphology.

We have hypothesised that voluminous resistance training performed by female fitness athletes induces cardiac hypertrophy to the extent of endurance athletes performing similar amount of exercise training. Therefore, the present study was designed to evaluate cardiac morphology in female fitness athletes undergoing relatively high volume of resistance training.

Materials and Methods

Subjects

The permission to conduct the study was granted by the local institutional committee of bioethics. Fitness sport athletes (n = 11) and sedentary but otherwise healthy adult females (n = 20) were recruited into the study after their informed consent to participate was obtained. The athletes were contacted to voluntarily participate in the study via their coaches. In the athletes group, inclusion criteria were at least two years of regular training in fitness sport just before the study, female gender, and 18 to 30 years of age by the time of the study. Athletes from three regional sports clubs participated in the study; sample size was limited mainly because of a relatively small number of female fitness athletes practicing at elite level in the region. The control group was formed of healthy sedentary age-matched university students which were invited to participate in the study via social networks. All the subjects were Caucasians, and the data were collected between October and December 2012.

Athletes were at their regular training schedule during the time of investigation, and none of the subjects did exercise for at least 24 hours and did consume any food or drink caffeine-containing beverages at least two hours before the echocardiography. Recruited athletes had been training and competing in fitness sport for two to six years, and by the time of the study all had achieved the national to international competitive level. Athletes were training 4 to 10 times per week which comprised 5 to 12 hours per week of intense activity in the gym. The frequency of the sessions depended mainly on whether a split routine was used by the athletes, i.e. whether they had been dividing resistance training of different muscle groups between sessions (e.g. leg and abdominal muscles in the morning, and then shoulder and arm muscles in the evening). Resistance training occupied 60 to 80 percent of the total training sessions which also corresponded to a similar percentage of total training time. The rest of the training was aerobic endurance ("cardio") sessions involving mostly running, cycling and rowing aimed mainly at diminishing the fat reserves to obtain "ideally lean" body composition.

Sedentary subjects were not engaged in any planned physical training regimen and were not exercising or being otherwise vigorously active for more than 2 hours per week. All subjects had normal blood pressure, were non-smokers and not on any cardiac medications or anabolic steroids. General characterisation of the subjects is presented in Table I. All parameters except body composition, which was greater in fitness athletes ($p < 0.05$), were similar between the two groups ($p > 0.05$).

Table I. General characteristics of the subjects

Parameters	Fitness athletes (n = 11)	Sedentary controls (n = 20)
Age, years	24.7 (2.8)	23.0 (2.3)
Height, cm	169.3 (6.3)	168.6 (5.8)
Body mass, kg	57.1 (4.8)	60.0 (6.6)
Lean body mass, kg	52.4 (3.9) **	47.8 (4.2)
Body mass index	19.9 (1.5)	21.3 (2.2)
Body surface area, m^2	1.65 (0.09)	1.68 (0.10)

** $p < 0.01$. Data are presented as mean (SD).

Measurements

Body mass was measured and composition (via bioimpedance) estimated using the electronic scales (Tanita TBF-300, Japan). Harpenden Portable Stadiometer was used to measure body height. Body mass index (BMI) was calculated as body mass (kg) / height squared (m^2). Body surface area (BSA) for indexing purpose was calculated using the standard equation (4).

Standard transthoracic two-dimensionally guided M-mode and Doppler echocardiography was performed with the subjects resting in a left lateral position, by means of an ultrasound device. LV measurements were made using two-dimensional guiding in long axis parasternal view. Internal LV diameter (LVDi) as well as septal (IVS), posterior wall (PW) thickness, right ventricular internal diameter, were measured at end-diastole, left and right atrium diameters were taken at end-systole as recommended by the American Society of Echocardiography (11). The early (E) and late (A) diastolic peak filling velocities were measured using the Doppler effect from apical four-chamber view, E/A ratio was calculated. Left ventricle ejection fraction (LVEF) was calculated by the Simpson method. The same cardiologist made measurements on three consecutive cardiac cycles from which the average for each parameter was calculated.

The relative wall thickness (RWT) was calculated by dividing the sum of IVS and PWT by LVDi. LV mass was calculated applying the following equation (2): LV mass (in g) = $0.8 \{ 1.04 \times [(IVS + LVDi + PWT)^3 - (LVDi)^3] \} + 0.6$. To adjust for possible effects of differences in body mass and composition, LV mass was indexed in several ways (16, 32): by dividing LV mass to BSA; by dividing LV mass to square rooted and then cubed BSA ($BSA^{3/2}$); by dividing LV mass to kilograms of body mass; and by dividing LV mass to bioimpedance-estimated lean body mass.

Statistical analysis was performed with SPSS software (version 21, IBM). Data are presented as mean and standard deviation. To compare the means, unpaired *t*-test was applied with the level of significance set at $p < 0.05$.

Results

Any of the measured and calculated echocardiographic indices of cardiac structure did not differ between elite female fitness athletes and matched sedentary controls ($p > 0.05$, Table II). Compared to controls, fitness athletes had a tendency for a smaller ($p = 0.07$) LV mass indexed to lean body mass derived from bioelectric impedance (Table II).

Table II. Echocardiographic indices of the subjects

Parameter	Fitness athletes (n = 11)	Sedentary controls (n = 20)
Left ventricular mass, g	116.6 (17.1)	114.6 (22.2)
Left ventricular mass indexed to BSA	70.5 (9.5)	67.7 (11.1)
Left ventricular mass indexed to $BSA^{3/2}$	54.9 (7.6)	52.3 (8.1)
Left ventricular mass indexed to body mass	2.04 (0.24)	1.90 (0.25)
Left ventricular mass indexed to lean body mass	2.22 (0.21)	2.40 (0.26)
Relative wall thickness	0.370 (0.030)	0.358 (0.031)
Left ventricular internal diameter, mm	44.2 (2.5)	44.5 (3.0)
Interventricular septum, mm	8.1 (0.7)	7.9 (0.8)
Left ventricular posterior wall, mm	8.2 (0.7)	8.0 (0.9)
Right ventricular internal diameter, mm	27.6 (2.4)	28.9 (4.3)
Left atrium diameter, mm	33.4 (3.1)	33.1 (2.2)
Right atrium diameter, mm	35.0 (4.0)	35.4 (4.3)
E (cm/s)	85.5 (10.6) **	98.6 (10.5)
A (cm/s)	48.9 (7.9) *	56.3 (7.8)
E/A	1.74 (0.24)	1.79 (0.34)
Ejection fraction	57.1 (5.9)	53.8 (7.1)

* $p < 0.05$, ** $p < 0.01$%. Data are presented as mean (SD).

Doppler diastolic function index E/A ratio was similar between fitness athletes and sedentary controls, and LV ejection fraction did not differ between the two groups either ($p > 0.05$, Table II). None of the subjects in either control or athlete groups had interventricular septum or posterior left ventricular wall thickness larger than 10 mm or internal LV diastolic diameter larger than 50 mm.

Discussion

Our results provide evidence that despite regular intense training to increase muscle mass and maintain lean body composition female fitness athletes do not develop cardiac hypertrophy or remodelling. Also, no changes in global cardiac function parameters at rest were observed as compared to age-matched sedentary controls.

Fitness training encompasses different resistance exercises for the development of strength (resistance training for whole body muscle building) which are supplemented with endurance exercise ("cardio", or fat burning sessions involving mainly leg musculature). However, combination of these stimuli seems insufficient to trigger changes in cardiac structure. Thus, despite significant static and dynamic components (14), fitness sport is of low impact on cardiac adaptation, at least in young female athletes not abusing steroids.

Physiological cardiac hypertrophy is relatively easily induced by performing high volume of endurance exercise with sufficient intensity (26, 28). It has been estimated that at least two years regular arduous physical training on average is required to develop changes in cardiac morphology (17). Athletes in the present study had been training for at least two years and, to support the sufficient efforts invested in the process, all had achieved the national to international competitive level.

Total weekly volume of aerobic training in our subjects might not surpassed the threshold of about three hours which is supposed to be required to induce cardiac remodelling (5) or the endurance exercise intensity might not reached the needed level. Therefore, larger mechanical, hormonal and/or metabolic stimuli on cardiomyocytes are required for the induction of cardiac hypertrophy in response to athletic training.

Data on the effect of resistance training on cardiac morphology remains controversial. While some authors have found increased LV mass and wall thickening in athletes engaged in strenuous strength training (3, 6) others report no difference from untrained healthy controls (10, 30, 31). We have reported previously that power training in elite female athletes does not affect cardiac size (29). Others have found only mild LV wall thickening in top level strength and power trained male athletes (21). While elite female weight lifters were reported to have concentric LV hypertrophy in some studies (6), in the others the stimulus of resistance training performed by elite female strength athletes, in support with the results of the present study, was also found to be insufficient for LV structural changes (9, 31). In opposite to bodybuilding, there is a clear decrease in requirements for muscularity of female fitness athletes during the last few years. This has probably instigated a change of training regimens to more voluminous and also diminished abuse of steroids, which not only maximize gains in skeletal muscle mass but also stimulate cardiac hypertrophy (3).

Alpine skiers, whose training requires the development of both muscle strength and endurance, were shown to develop LV hypertrophy due to both cardiac chamber enlargement and wall thickening (7). A degree and type of cardiac adaptation was reported to be independent of gender in these athletes (7). Therefore, cardiac remodelling seems quite possible in mixed training Olympic sport elite female athletes, most likely because of large endurance training component. Fitness sport training, on the other hand, does not seem to cause this type of adaptation, at least in young healthy female athletes free of illicit substances such as anabolic steroids. Interestingly, in elite male weight lifters LV mass measured by magnetic resonance imaging was reported to be even lower than in controls when indexed to lean body mass (10), a finding which is supported by the results of the present study where female fitness athletes had a tendency for a smaller LV mass indexed to lean body mass (measured via dual-energy X-ray absorptiometry) when compared with healthy sedentaries. Given the lower accuracy of biompedance estimate of lean body mass when compared with dual-energy X-ray absorptiometry, the results of the present study cannot refute that elite female fitness sport athletes possess decreased LV mass to lean body mass ratio.

As expected, global systolic LV function at rest in fitness sport athletes was normal and similar to controls. Global diastolic LV function was normal as well, which in contrary to the report on Strongmen who perform very intense resistance training and avoid aerobic training (30). Global diastolic function (assessed as E/A ratio) has been reported to be normal in female athletes from other resistance type of sports (9).

Researchers have reported on right ventricular dilation and hypertrophy in elite female endurance athletes. When compared to sedentary controls and marathon runners, even more pronounced right ventricular dilatation in the anaerobic power athletes was reported (22). However, the data of the present study does not provide the evidence for increased female's right ventricular size in response to strenuous high volume resistance exercise training.

It has been measured by means of MRI that both the right and the left atriums become proportionally larger with endurance training in female athletes (15). This is consistent with the symmetrical/proportional remodelling of the heart to the voluminous exercise training where all chambers of the heart are affected to similar extent. There were no signs of increase in volume of the atriums in our female fitness athletes.

One of the major limitations of the current study is a relatively small sample size of athletes. This was because of the limited availability of female fitness athletes with sufficient training history and continuing their sports career with intense training.

To summarise, despite being effective in improving body composition and increasing lean body mass and probably adding other health benefits, fitness sport does not seem to trigger alteration in cardiac muscle, at least in young healthy females. That is not to say that high volume resistance training does not bring the reduction of cardiovascular disease risk, but aspects other than structural cardiac adaptation remained beyond the scope of the current study. To conclude, elite female fitness athletes have normal cardiac size and function that do not differ from sedentary controls. While sculpturing the skeletal muscles and probably having many other health-related impacts, high volume resistance exercise training has negligible if any effect on structural cardiac adaptation in young healthy females. Therefore, when cardiac hypertrophy is observed in young female resistance trained athlete with lean body composition, other reasons such as inherited cardiac disease are to be considered carefully first of all.

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Conflict of interest

None declared.

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