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Are exercise-induced changes of fatty acids associated with cardiac hypertrophy in athletes? A pilot study

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Abstract

Study aim: In this study, we evaluated the effects of acute and chronic exercise on the plasma FAs and their association with cardiac hypertrophy indices.
Material and methods: In this pilot study, 15 sedentary and 15 athlete women underwent acute and long-term water aerobic exercise and their plasma FA levels and a number of electrocardiographic parameters, such as left ventricular end-diastolic diameter index (LVEDDI), left ventricular ejection fraction (LVEF), left ventricular mass index (LVMI), and wall thickness were evaluated before and after the exercise program.
Results: The acute exercise significantly increased palmitic and oleic acid levels in non-athletes and stearic acid in both groups. However, the same type of exercise decreased linoleic acid only in non-athlete women ($p < 0.05$). The water aerobics training caused a significant decrease in the levels of palmitic, stearic, and arachidonic acid, SFA/UFA, and ω3/ ω6 ratios and also an increase in α-Linolenic acid and MUFA in non-athletes. We found positive and negative correlations between LVEF with ω3 and SFA/UFA ratio in both groups, respectively. In the non-athlete group, the ω3/ω6 ratio showed negative correlations with LVMI and LVEDDI.
Conclusions: The study indicated that the 12-week exercise by sedentary women could make their plasma FAs composition similar to athlete women. Moreover, the plasma FA levels were associated with cardiac hypertrophy indices, showing the importance of FAs in physiological hypertrophy.

Keywords: Fatty acids – Exercise – Hypertrophy – Athlete – Diet

Introduction

Cardiac hypertrophy can be a result of elevated volume or pressure overload following physical activity or pathological conditions [10]. Physical activities such as isotonic and isometric exercises can induce cardiac structural and functional changes, especially in the left ventricle; known as athletic cardiac hypertrophy [29, 35]. Although there are some similarities between pathological and physiological cardiac hypertrophy, the complete underlying mechanism in athletic hypertrophy is not fully understood [23, 44].

Fatty acids (FAs) are the primary source of energy during basal metabolic state [43], but during physical activities, glucose is more important [19]. Although using glucose has an advantage of diminished oxygen demand in the hypertrophied myocardium, relying on glucose with less ATP production per mole in comparison to FAs may cause energy deficiency over time and subsequently inefficient cardiac performance [6]. To confirm the importance of FAs in exercise, it has been demonstrated that physical activity spanning from minutes to weeks could increase blood levels of free FAs [1, 27, 32]. Moreover, it has been shown that chronic exercise could decrease FAs levels, showing their consumption to supply energy [26]. The dietary FAs including saturated (SFAs) and unsaturated FAs (UFAs) can affect cardiac function as it has been indicated that the UFAs-enriched diet is associated with low risk of cardiovascular disease [22]. Interestingly, studies have shown that changing in FAs profile even for a short

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time could significantly affect cardiac function. In this regard, it has been reported that a short-term supplementation of 3-PUFAs improved right ventricular function and end-diastolic pressure in patients with chronic heart failure [2]. Furthermore, four weeks diet with a high level of SFAs induced cardiac systolic dysfunction in mice [27]. It also has been documented that omega-3 (ω3) FAs could improve cardiac hypertrophy indices [10]. Interestingly, cardiac hypertrophy was observed in mice with a fat-rich diet and was revealed that diastolic dysfunction was associated with serum lipid levels, showing the importance of blood lipid profiles [25]. Given the role of blood FAs in supplying energy of cardiac cells, especially in chronic exercises and on the other hand association of FAs with athletic hypertrophy, in the present study, we investigated effects of acute and chronic exercises on the plasma levels of FAs and their association with cardiac hypertrophy indices in athlete and non-athlete women.

Materials and methods

Study population

In the present study, 15 sedentary women (33 ± 5 years old) without a specific physical exercise and 15 athlete women (32 ± 7 years) were recruited. The athletes all were professional runners (average professional experience of 9 ± 2) with regular exercise of at least five two-hour sessions per week. The inclusion criteria for all participants were being in premenopausal age, having no history of medication (especially lipid-lowering drugs) use at least five weeks before the study, being nonsmoker, having no diabetes, cardiovascular, liver, thyroid, and kidney diseases. We asked the participants to not change their routine dietary habits. The study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and all the procedure was approved by the Ethical Committee of Urmia University. Moreover, a written informed consent was obtained from all participants before enrollment into the study.

Exercise procedures and intervention program

The following electrocardiographic parameters were evaluated in all participants before entering the exercise programs: heartbeat, Posterior wall thickness (PWT), Septal thickness, left ventricular end-diastolic diameter index (LVEDDI), left ventricular ejection fraction (LVEF), end-systolic stress (ESS), left ventricular mass index (LVMI), and relative wall thickness (RWT). The RWT was calculated based on WT = (2 × PWT)/LVDD formula, in which LVDD stands for the left ventricular internal diastolic dimension [33]. LVEF and ESS were also evaluated based on previously reported methods [37]. Furthermore, the Devereux formula was used for calculating LVMI [7].

The Bruce test was used for acute exercise and the blood samples were taken before and after exercise and the plasma was isolated and then kept at –80°C. For chronic exercise induction, a 12-week water aerobics training session with three days/week training sessions was used. Each session started with the intensity of 60% maximum heart rate for 30 minutes and by improving the condition of women, the duration of sessions gradually increased up to 40–60 minutes with high density (70–80% of maximum heart rate). The blood samples were collected 24 h after the end of the last session to minimize the interference of acute exercise. To investigate the long-term effect of exercise, another blood sample was collected three days later, while women had no physical activity during this time. The same electrocardiographic parameters were evaluated at the end of the experiments for all participants.

Lipid profile analysis

Fasting blood samples were collected to evaluate lipid profile of the participants before entering the exercise program. The serum levels of cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and triglyceride (TG) were measured using Pars Azmoon kits (Tehran, Iran) in auto-analyzer (Hitachi®, model 911, Japan).

Gas chromatography analysis of FAs

To extract plasma total lipids we applied the Bligh-Dyer method [11] and then the lipids were esterified with methanol during catalysis with acetyl chloride. We used Tridecanoic acid (13:0) as the internal standard and also known FAs standards (Sigma-Aldrich, USA) to identify peak retention times. Then, the composition of FAs was evaluated using the previously described method [14]. For this purpose, the extracted fatty acid methyl ester derivatives were separated on a 60 × 0.25-mm Teknokroma TR CN100 column using a Buck Scientific model 610 gas chromatograph (SRI Instruments, Torrance, USA) which equipped with a split injector and a flame ionization detector and helium as a carrier gas. The oven temperature was increased from 170ºC to 210ºC at the rate of 1ºC/min and then maintained stable for 45 minutes. The levels of FAs were calculated as the percentage of each fatty acid of the total.

Statistical analysis

Given the small sample size, non-parametric statistics were used in this study. To compare the data of various time points within one group and between the athlete and non-athlete groups, we used Friedman and Mann-Whitney tests, respectively. The Spearman test was used to evaluate the correlation between FAs and cardiac hypertrophy indices. *p*-values lower than 0.05 were considered significant. The statistical analysis was done using SPSS V.16.

Results

We found no significant difference ($p > 0.05$) between the athlete and non-athlete groups in the term of age, body mass index (23 ± 1.4 vs. 24.6 ± 3.5 kg/m^2, respectively), and body surface area (1.64 ± 0.31 vs. 1.72 ± 0.41 m^2, respectively). The lipid profile of participants is shown in Table 1. We found significantly higher levels of LDL in sedentary women compared to athlete group ($p = 0.026$). Moreover, the mean levels of maximum heart rate and maximal oxygen uptake (VO_2 max) were 182 ± 4 vs. 185 ± 6 bpm and 29 ± 5 vs. 52 ± 9 ml/kg/min in athlete and non-athlete groups, respectively.

Analysis of echocardiogram data demonstrated that LVEDDI and LVMI were significantly higher in athletes than non-athletes at the baseline level (before exercise). A comparison of these parameters before and after a 12-week aerobic exercise in the study groups revealed no significant change. We found that there was no significant difference in LVEDDI levels between the groups after the 12-week exercise; however, LVMI was still higher in athletes compared to non-athletes (Table 2).

The composition of plasma FAs following acute and long-term water aerobics exercises in athlete and non-athlete women are shown in Table 3. We found that at the baseline, athlete women had significantly higher levels of ω3 and ω3/ω6 ratio in comparison with the non-athletes ($p < 0.05$).

The acute exercises significantly increased levels of palmitic acid, stearic acid, oleic acid, as well as saturated fatty acids (SFA) and mono-unsaturated fatty acids (MUFA) levels in the non-athlete group ($p < 0.05$). However, the levels of linoleic acid were decreased following the acute exercise in this group. Interestingly, acute exercises only increased stearic acid and total SFA levels in the athlete group ($p < 0.05$). Nevertheless, the levels of palmitoleic acid, α-linolenic acid, arachidonic acid, eicosapentaenoic acid, docosahexaenoic acid, total ω3, and ω3/ω6 ratio were significantly different between the groups after the acute exercises.

Chronic exercises caused a significant decrease in the levels of palmitic acid, stearic acid, arachidonic acid, as well as SFA/UFA and ω3/ω6 ratios in the non-athlete group. However, the levels of α-Linolenic acid and MUFA were increased after a 12-week exercise in this group ($p < 0.05$). In athletes, only α-Linolenic acid levels were

Table 1. Lipid profile before in non-athlete and athlete women

	Non-athlete (n = 15)	Athlete (n = 15)	*p*-value
Cholesterol [mg/dl]	192.9 ± 44.3	171.1 ± 32.9	0.137
Triglyceride [mg/dl]	172.2 ± 52.1	153.3 ± 37.2	0.262
HDL [mg/dl]	44.7 ± 9.6	50.9 ± 7.6	0.059
LDL [mg/dl]	110.3 ± 11.2	98.7 ± 15.5	0.026

HDL – high-density lipoprotein; LDL – low-density lipoprotein.

Table 2. Echocardiogram parameters before and after a 12-week aerobic exercise in non-athlete and athlete women

	Non-athlete (n = 15)		Athlete (n = 15)	
	Before	After	Before	After
Septal thickness [mm]	8.2 ± 0.9	8.4 ± 1.1	8.8 ± 0.7	9.0 ± 0.9
Posterior wall thickness [mm]	8.5 ± 0.9	8.7 ± 1.0	9.4 ± 0.8	9.3 ± 0.9
RWT [%]	37.8 ± 7.1	37.2 ± 5.9	38.1 ± 6.1	38.9 ± 7.2
LVEDDI [mm/m^2]	24.1 ± 4.8	25.9 ± 3.4	28.1 ± 4.4*	28.4 ± 5.1
LVMI [g/m^2]	73.6 ± 8.2	75.5 ± 8.3	82.4 ± 9.1*	83.0 ± 9.2*
ESS [kdyne/cm^2]	63 ± 6	68 ± 8	70 ± 10	67 ± 7
LVEF [%]	58.9 ± 9.1	62.3 ± 10.4	65.1 ± 8.2	65.6 ± 9.1

RWT – Relative wall thickness; LVEDDI – left ventricular end-diastolic diameter index; LVMI – left ventricular mass index; ESS – end-systolic stress; LVEF – left ventricular ejection fraction; * – Significant difference ($p < 0.05$) between the non-athlete and athlete groups.

Table 3. Composition of serum fatty acids following acute and chronic exercises in non-athlete (n = 12) and athlete (n = 12) women

% of total fatty acids	Before exercise (S1)		Immediately after acute exercise (S2)		24 h after a 12-weeks chronic exercise (S3)		3 days after a 12-weeks chronic exercise (S4)	
	Non-athlete	Athlete	Non-athlete	Athlete	Non-athlete	Athlete	Non-athlete	Athlete
14:0 (Myristic acid)	0.64 ± 0.26	0.61 ± 0.24	0.58 ± 0.19	0.67 ± 0.21	0.54 ± 0.12	0.58 ± 0.20	0.56 ± 0.21	0.66 ± 0.18
16:0 (Palmitic acid)	17.36 ± 4.22	16.40 ± 4.60	21.93 ± 5.12[a]	19.43 ± 6.41	14.21 ± 2.8[a,b]	15.78 ± 4.71	16.92 ± 3.87[b]	17.04 ± 4.08
16:1 n-7 (Palmitoleic acid)	3.53 ± 1.08	4.54 ± 1.13	2.64 ± 1.60	5.48 ± 1.50*	4.74 ± 1.75[b]	5.32 ± 1.75	4.41 ± 1.7[b]	5.01 ± 1.82
18:0 (Stearic acid)	5.55 ± 1.83	5.20 ± 1.23	7.06 ± 1.42[a]	7.33 ± 1.93[a]	3.83 ± 1.27[a,b]	4.43 ± 1.76[b]	4.63 ± 1.52[b]	4.90 ± 1.36[b]
18:1 n-9 (Oleic acid)	19.41 ± 7.06	22.36 ± .6.43	25.28 ± 6.02[a]	23.06 ± 5.18	24.32 ± 6.41	23.84 ± 5.74	23.71 ± 8.03	22.65 ± 7.2
18:2 n-6 (Linoleic acid)	40.70 ± 10.48	37.55 ± 8.94	31.46 ± 8.51[a]	33.16 ± 6.49	39.80 ± 8.96[b]	36.91 ± 7.66	37.75 ± 9.21	37.01 ± 8.41
18:3 n-3 (α-Linolenic acid)	0.67 ± 0.18	0.87 ± 0.27	0.54 ± 0.21	0.82 ± 0.30*	0.95 ± 0.17[a,b]	1.06 ± 0.11[a,b]	0.89 ± 0.12[a,b]	0.96 ± 0.13
20:4 n-6 (Arachidonic acid)	6.54 ± 2.31	5.20 ± 1.74	6.47 ± 1.92	4.15 ± 1.338*	4.62 ± 1.56[a,b]	4.68 ± 1.70	4.78 ± 1.4[a,b]	4.76 ± 1.79
20:5 n-3 (Eicosapentaenoic acid)	1.63 ± 0.60	2.29 ± 0.86	1.16 ± 0.42	1.91 ± 0.71*	2.02 ± 0.84[b]	2.42 ± 0.73	1.99 ± 0.92[b]	2.16 ± 1.02
22:6 n-3 (docosahexaenoic acid)	3.97 ± 1.72	4.98 ± 1.67	2.88 ± 1.18	4.29 ± 1.29*	4.97 ± 1.66[b]	5.03 ± 1.10	4.36 ± 1.83	4.86 ± 1.29
SFA	23.55 ± 7.01	22.21 ± 6.44	29.57 ± 6.12[a]	27.43 ± 5.14[a]	18.58 ± 5.93[b]	20.79 ± 6.11[b]	22.11 ± 7.30[b]	22.60 ± 6.11[b]
MUFA	22.94 ± 5.21	26.90 ± 6.28	27.92 ± 6.08[a]	28.54 ± 6.08	29.06 ± 7.12[a]	29.16 ± 6.30	28.12 ± 6.92	27.66 ± 7.72
ω3	6.27 ± 1.97	8.14 ± 1.32*	4.58 ± .1.59	7.02 ± 1.48*	7.94 ± 2.1[b]	8.51 ± 1.84[b]	7.24 ± 1.86[b]	7.98 ± 2.12
ω6	47.24 ± 10.90	42.75 ± 7.56	37.93 ± 9.92	37.31 ± 6.38	44.42 ± 8.52	41.59 ± 7.25	42.53 ± 9.33	41.77 ± 8.34
SFA/UFA	0.31 ± 0.12	0.29 ± 0.10	0.42 ± 0.14	0.38 ± 0.13	0.22 ± 0.08[a,b]	0.26 ± 0.09[b]	0.28 ± 0.14[b]	0.29 ± 0.16
ω6/ ω3	7.53 ± 1.34	5.25 ± 1.34*	8.28 ± 1.21	5.31 ± 1.70*	5.59 ± 1.32[a,b]	4.89 ± 1.51	5.87 ± 1.52[a,b]	5.23 ± 1.66

Data are shown as Mean ± S.D; Levene's test confirmed homoscedasticity of variances and two-way ANOVA was used to evaluate the interaction between groups and days; Significant difference ($p < 0.05$) in comparison with [a] S1, [b] S2 in the same group. * – Significant difference in comparison with the non-athlete group on the same time course; SFA – saturated fatty acids; MUFA – monounsaturated fatty acids; UFA – unsaturated fatty acids; ω3 – omega 3 fatty acids; ω6 – omega 6 fatty acids; SFA/UFA – the ratio of saturated to unsaturated fatty.

significantly increased after 12-week exercises. To evaluate the long-term effect of exercise on FAs composition, we measured the FAs levels 3 days after the last session of the chronic exercise and found that the α-Linolenic acid level was higher and arachidonic acid level and ω3/ω6 ratio were lower in compared with the baseline levels in non-athletes.

The Spearman correlation analysis was used to address the possible association between echocardiogram parameters and plasma levels of FAs. We found positive correlations between LVEF and ω3 in both athlete and non-athlete women ($r = 0.52$ and $r = 0.41$, respectively; $p < 0.05$). Besides, a negative correlation was found between LVEF and the SFA/UFA ratio in both groups ($r = –0.39$ in athletes and $r = –0.46$ in non-athletes; $p < 0.05$). In the non-athlete group, the ω3/ω6 ratio showed a positive correlation with LVEF ($r = 0.5$, $p < 0.05$) and negative correlations with LVMI and LVEDDI ($r = –0.46$ and $r = –0.51$, respectively; $p < 0.05$). A positive correlation between LVMI and the SFA/UFA ratio was also observed in non-athlete women ($r = 0.38$, $p < 0.05$).

Discussion

In this study, we investigated the effects of acute and 12-week water aerobics training on the plasma fatty acid concentrations and their association with cardiac hypertrophy indices in female athletes and non-athletes.

In the present study, the athletes had a significantly higher LVMI and LVEDDI in this group. These two parameters imply the presence of mild cardiac hypertrophy in athletes which seems normal. Physiological hypertrophy, in particular in athletes, occurs as a result of volume and/or pressure overload following training which is dependent on the time and type of exercise [36]. Although the comparison of the cardiac hypertrophy parameters before and after a 12-week aerobic exercise showed no significant change in the study groups, there was no significant difference in the LVEDDI level between the groups at the end of the training course showing a positive effect of long-term exercise on cardiac hypertrophy. In supporting our finding, Evangelista et al. [9] also reported stimulatory effects of swimming exercise on cardiac hypertrophy in mice. Furthermore, the association of physical activity with left ventricular mass has been previously demonstrated [46]. Lack of significant increase in other hypertrophy indices could be due to relatively short (12 weeks) time of exercise and further studies with longer training courses and larger number of participants are required to demonstrate exercise-induced cardiac hypertrophy.

Our results showed that the plasma level of ω3 and ω3/ω6 ratio at baseline were significantly higher in athletes compared to non-athlete women which imply that repeated physical activity in athletes can keep the concentrations of ω3 FAs at the high levels. Owing to the positive effects of ω3 on cardiovascular functioning and adaptation as well as recovery in athletes [34], and also our findings regarding the elevated levels of ω3 in athletes, it can be suggested that ω3 is one of the mediators of exercise beneficial effects. Although studies have shown an association between fatty acids with physiological and pathological cardiac hypertrophy, the underlining mechanisms are still unclear [23, 44] and several mechanisms have only been proposed. Omega-3 fatty acids such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) can improve cardiac function by exerting anti-inflammatory effects to some extent by competition with omega-6 fatty acids [13]. It has been demonstrated that these effects may be mediated by different mechanisms, including 1) reducing the production of pro-inflammatory eicosanoids or generating resolvins, which play anti-inflammatory roles in the COX-2 pathway [15, 24], 2) activating peroxisome proliferator-activated receptors (PPARs) that suppresses NF-kB-regulated pro-inflammatory cytokines, IL-1, TNFa, and IL-6 [18], and 3) producing adiponectin via PPAR-gamma-dependent mechanism [15]. Omega-3 fatty acids can also prevent hypertension-induced hypertrophy by lowering the nitric oxide (NO) production [20]. Furthermore, EPA and DHA can improve mitochondrial function, impairment of which has been demonstrated in the pathogenesis of cardiac hypertrophy [28, 48]. It has been also suggested that fish oil rich in omega-3 fatty acids could prevent cardiac hypertrophy through DAG–PKC pathway [41]. Moreover, Siddiqui et al. [38] reported that the attenuating effects of DHA on cardiac hypertrophy are mediated through inhibition of down-stream kinases of the ERK pathway. Moreover, another mechanism regarding the effects of a saturated fat diet on cardiac hypertrophy in rats may be related to the enhanced activation of PKC β2 in the heart [16].

We found that acute exercise could increase plasma concentrations of total SFA such as stearic acid in both groups compared to pre-exercise values, demonstrating FAs mobilization and metabolism. Moreover, the levels of palmitic acid, oleic acid, and total MUFA were increased and the levels of linoleic acid were decreased following the acute exercise in the non-athlete women. In accordance with our findings, several studies reported that acute exercise could increase MUFA and UFA/SFA ratio in both animals and humans [12]. In a similar study, Conquer et al. also reported an increase in the levels of SFA after a course of acute exercise in sedentary women [3]. Two reasons can be considered for FAs increase after acute exercise including, 1) elevated endogenous synthesis of FAs in muscles [42], 2) adipose tissue lipolysis, and mobilization of FAs from adipose tissue [47]. It should be

mentioned that differences in the intensity and type of exercises can explain the possible controversial effects of exercise on FAs levels reported in previous studies. Interestingly, the athletes had significantly higher levels of α-linolenic acid, arachidonic acid, eicosapentaenoic acid, and docosahexaenoic acid than non-athletes after the acute exercise, while the pre-exercise levels of these FAs were not significantly different between the groups. Since α-linolenic, arachidonic, and eicosapentaenoic acids can be act as precursors of eicosanoids, especially prostaglandins [5], it can be postulated that the elevation of these FAs in athletes following the acute exercise is a physiological strategy to increase prostaglandins synthesis in athletes; we did not found such strategy in untrained individuals. On the other hand, it has been demonstrated that prostaglandins are involved in exercise-induced vasodilation of skeletal muscle arterioles [45]. Moreover, eicosanoids are involved in immune system regulation and cardiovascular responses [39].

The water aerobics training was used in the present study since it is a safe exercise for sedentary women who participated in our research. Mitigation of gravity makes water aerobics training suitable and safe for susceptible groups such as sedentary individuals and obese people [4]. We reported that, 12-week water aerobics training decreased levels of palmitic acid, stearic acid, and arachidonic acid as well as SFA/UFA and ω3/ω6 ratios, while concentrations of α-linolenic acid and MUFA were significantly increased when compared to pre-exercise values in non-athletes. In athletes, only α-linolenic acid levels were increased compared to pre-exercise levels. The reduction in SFA/UFA ratio observed in our study was in line with the findings of Mougios et al. [21] who evaluated FAs composition in male athletes following prolonged moderate exercise. These findings can be explained by the shift of energy source from FAs to glucose after long-term exercise due to diminishing oxygen demand in the hypertrophied myocardium. However, to prevent cardiac dysfunction over time because of less ATP yield of glucose per mole in comparison to FAs [6], supplying the essential FAs requirements for athletes seems to be essential. More interestingly, the FAs composition of plasma was not significantly different between athletes and non-athletes after a 12-week exercise, showing that a long-term exercise could make the composition of non-athletic plasma FAs similar to that of athletes. Long term exercise also had some beneficial effects in non-athletes when compared to acute exercise, such as reduction of n-6 PUFAs and SFAs and elevation of n-3 PUFA levels that shows the cardioprotective role of long-term training in non-athletes.

In the non-athlete group, there was a negative correlation between LVEDDI and ω3/ω6 ratio. In accordance with our findings, Rupp et al. [31] documented that reduction of ω3 FAs (in particular docosahexaenoic acid) was associated with high left ventricular end-diastolic diameter. Moreover, it has been reported that fish oil (ω3 PUFA) could reduce the dilatation of pressure overloaded rat left ventricle [30]. It can be hypothesized that ω3 FAs are involved in the reduction of LVDDI via suppressing pro-inflammatory factors such as TNF-α and peroxisome proliferator-activated receptors (PPARs)[8]. On the other hand, we found positive and negative correlations respectively between LVEF with ω3 levels and SFA/UFA ratio in both athletes and non-athletes.

Moreover, positive and negative correlations were found between LVMI with respectively SFA/UFA and ω3/ω6 ratios in non-athletes. These findings imply a possible role of SFA/UFA and ω3/ω6 ratios in the development of cardiac hypertrophy. In accordance, it has been indicated that cardiomyocyte diameters in left ventricular were significantly correlated with SFA [17]. Sundström et al. [40] also showed that a high intake of SFA could cause cardiac hypertrophy. Moreover, a significant reduction of heart weight indices following ω3 FAs treatment has been observed in rats [10]. In contrast, Duda et al. [8] introduced ω3 PUFA as protective factors for cardiac hypertrophy. To explain this controversy, it should be mentioned that Duda and colleagues considered pathological hypertrophy and ventricular dysfunction rather than physiological hypertrophy. Furthermore, the cardiac indices of our study were in the normal range in both groups and there was no sign of pathological hypertrophy. Nevertheless, the absence of any correlation between FAs and hypertrophy indices (LVMI and LVEDDI) in athletes, may diminish the role of ω3 in physiological hypertrophy; however, it does not ignore the role of ω3 in left ventricular function improvement and we believe that our findings should be confirmed in large-scale studies.

In conclusion, the present data indicated that acute and chronic exercises could significantly influence the plasma FAs profile in athlete and non-athlete women. However, the 12-week exercise by sedentary women could reduce their plasma levels of SFA and increase α-linolenic acid and MUFA levels and therefore made their plasma FAs composition similar to the athlete women. Moreover, we found that levels of plasma FAs especially ω3 FAs and SFA were associated with cardiac hypertrophy indices in particular LVEF and LVMI. Considering the plasma composition of FAs in the athlete women and its association with cardiac hypertrophy indices, it can be postulated that supplying the essential FAs especially ω3 FAs is required for physiological hypertrophy, however further studies are needed to confirm this.

Conflict of interest: Authors state no conflict of interest.

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