

Research article

The Effects of Aerobic Exercise on Plasma Adiponectin Level and Adiponectin-related Protein Expression in Myocardial Tissue of ApoE^{-/-} Mice

Xiao-Juan Zhu¹, Li-Hui Chen¹ and Jiang-Hua Li^{1,2}✉

¹ Key Laboratory of Training Monitoring and Intervention on the Sports in Water, State Sports General Administration, Institute of Physical Education, Jiangxi Normal University, Nanchang, China; ² Key Laboratory of Functional Small Organic Molecule, Ministry of Education, Jiangxi Normal University, Nanchang, China

Abstract

Numerous reports have confirmed the effect of ApoE knockout in the induction of cardiovascular diseases and the protective effect of adiponectin against the progression of cardiovascular diseases. The aim of this study was to reveal the roles of adiponectin signaling in the progression of cardiovascular diseases induced by ApoE knockout and to analyze the healthy effects of aerobic exercise on ApoE knockout mice (ApoE^{-/-} mice) through observing the changes of adiponectin signaling caused by ApoE knockout and aerobic exercise. A twelve-week aerobic exercise program was carried out on the male ApoE^{-/-} mice and the C57BL / 6J mice (C57 mice) of the same strain. Results show that the body weights, blood lipid level, plasma adiponectin level and adiponectin-related proteins in myocardial tissue were all significantly changed by ApoE knockout. A twelve-week aerobic exercise program exerted only minimal effects on the body weights, blood lipid levels, and plasma adiponectin levels of ApoE^{-/-} mice, but increased the expressions of four adiponectin-related proteins, AdipoR1, PPAR α , AMPK and P-AMPK, in the myocardial tissue of the ApoE^{-/-} mice. In summary, adiponectin signaling may play an important role in the progression of cardiovascular diseases induced by ApoE knockout, and the beneficial health effects of aerobic exercise on ApoE^{-/-} mice may be mainly from the increased adiponectin-related protein expression in myocardial tissue.

Key words: Aerobic exercise, adiponectin, myocardial tissue, ApoE^{-/-} mice, atherosclerosis.

Introduction

Adiponectin is an active peptide secreted by white adipose tissue with many biological functions, including the regulation of fatty acid and glucose metabolism, and the attenuation of inflammation and atherosclerosis. Numerous reports have shown the protective effects of adiponectin against various cardiovascular diseases. Liao et al. (2005) reported that adiponectin deficiency results in progressive cardiac remodeling in the setting of pressure overload, a process mediated via decreased AMPK signaling and impaired glucose metabolism. Shibata et al. (2004) observed that pressure overload in adiponectin-deficient mice resulted in both enhanced concentric cardiac hypertrophy and increased mortality and that adiponectin supplementation attenuated these symptoms. Kumada et al. (2003) demonstrated that male patients with hypoadiponectinemia had a 2-fold increase in coronary artery disease (CAD) prevalence, independent of well-known

CAD risk factors. Pischon et al. (2004) also observed that high plasma adiponectin concentration is associated with a lower risk of myocardial infarction among men. Via both the ligation of the left anterior descending coronary artery and reperfusion, Shibata et al. (2007) observed that the sizes of the myocardial infarcts that occurred following ischemia-reperfusion injury in adiponectin-knockout (APN-KO) mice were significantly expanded compared with those of wild-type mice, and that adiponectin supplementation significantly reduced the sizes of the infarcts in both APN-KO mice and wild-type mice.

By combining with AdipoR1, which is abundantly expressed in myocardial tissue, adiponectin exerts its biological functions in myocardial tissue primarily in the following two pathways: the peroxisome proliferator activated receptor (PPAR) and the AMPK signaling pathways. Impairment of these signaling pathways results in abnormal lipid metabolism and in the development of a variety of metabolic disorders (Chen et al., 2012; Lee and Kwak, 2014a). Yamauchi et al. (2003) showed that PPAR α is an important messenger in the adiponectin signaling pathway. Many PPAR α target genes are involved in lipid metabolism, such as in fatty acid uptake, binding, transport, oxidation and lipoprotein synthesis. By binding to the receptor and activating p38MAPK, adiponectin activates the PPAR α signaling pathway and subsequently plays a role in the regulation of insulin resistance, and anti-inflammatory and anti-atherosclerotic processes (Chinetti et al., 2004). AMPK activation is another means for adiponectin to exert its biological functions. Yamauchi et al. (2003) demonstrated that following binding to AdipoR1, adiponectin can activate AMPK, a protein kinase existing in most mammalian tissues, to increase glucose metabolism, fatty acid oxidation and insulin sensitivity.

Apolipoprotein E (ApoE) is one of the primary apolipoproteins found in human blood and plays a key role in maintaining both lipid metabolism and cholesterol balance. At the age of three months, ApoE knockout mice (ApoE^{-/-} mice) begin to present arterial fat accumulation, and various cardiovascular disease symptoms then gradually appear. As ApoE^{-/-} mice exhibit the symptoms of high cholesterol, atherosclerosis, coronary heart disease and other cardiovascular disease-related disorders, they are widely used in cardiovascular disease research. Recent studies reported an impact of ApoE knockout on the expression of adiponectin in adipose tissue and the protective effects of adiponectin against the damage to cardio-

vascular system induced by ApoE knockout (Lasrich et al., 2015; Li et al., 2015). However, the role of ApoE knockout on the adiponectin signaling pathway, especially the adiponectin-related proteins expression in myocardial tissue, remains unclear. Our hypothesis is that modulation of adiponectin signaling pathway may be an important way for ApoE knockout to induce the damage of cardiovascular system, while aerobic exercise may exert some protective effects against this damage through improving adiponectin signaling pathway. To test this hypothesis, a 12-week aerobic exercise program was carried out in ApoE^{-/-} mice and normal mice of the same strain to observe the changes of plasma adiponectin level and adiponectin-related protein expression in myocardial tissue induced by ApoE knockout and by aerobic exercise.

Methods

Animals

Twenty 8-week-old male ApoE^{-/-} mice and twenty C57BL/6J mice (C57 mice) of the same age and strain were purchased from the Scientific Experimental Animal Department of Peking University, maintained at an ambient room temperature of 22–25 °C and a humidity of 34–40%, fed normal rodent chow and allowed free access to water. The ApoE^{-/-} mice and the C57 mice were randomly divided into the following four groups: an ApoE^{-/-} control group (AC, n = 10), an ApoE^{-/-} exercise group (AE, n = 10), a C57 control group (CC, n=10), and a C57 exercise group (CE, n = 10). The exercise groups (AE and CE) trained on treadmills for 12 weeks at a speed of 13 m·min⁻¹, once a day, and six days a week. The training duration was 30 min·day⁻¹ during the first week, 40 min/day during the second week, and 50 min/day during subsequent weeks. The control groups (AC and CC groups) were not trained. The experimental procedures were performed in accordance with the Guide for the Care and Use of Laboratory Animals (Ministry of Science and Technology of China, 2006) and were approved by the animal ethics committee of Jiangxi Normal University.

Sample collection and preparation

Forty-eight hours after the last training session, samples were collected from the animals. The animals were fasted overnight (8 hours) before being subjected to ether anesthesia, and then their body weights were measured, and blood samples were collected from the inferior vena cava and immediately centrifuged for 5 min at 3000 g. The isolated plasma was stored at -80 °C until needed for analysis. Following blood sample collection, the mice were bled to death, and myocardial tissue samples were collected from their left ventricular walls, dried with filter paper, packed in vials, and stored in liquid nitrogen.

Plasma adiponectin and blood lipids

Plasma adiponectin was measured using a mouse adiponectin enzyme-linked immunosorbent assay (ELISA) kit (Mouse Adiponectin/Acrp30 Quantikine ELISA Kit, sensitivity 0.007 ng·mL⁻¹, assay range: 0.16 – 10 ng·mL⁻¹, intra-assay variation: 5.8% - 6.7%, inter-assay variation:

5.0% - 6.4%, R & D Company, USA). Total cholesterol (TC), triglycerides (TG), high density lipoprotein cholesterol (HDL-C), and low density lipoprotein cholesterol (LDL-C) were measured via the colorimetric method using kits purchased from Beijing BIOSINO Biotechnology Corporation, China.

Adiponectin-related protein expression

Western-blots were performed to measure the protein expression of the adiponectin-related proteins adiponectin receptor 1 (AdipoR1), peroxisome proliferator-activated receptor alpha (PPAR α), adenosine monophosphate activated protein kinase (AMPK) and phosphorylated adenosine monophosphate activated protein kinase (P-AMPK), using glyceraldehyde-3-phosphate dehydrogenase (GAPDH) as internal reference. Myocardial tissue (100 mg) was homogenized and solubilized in ice-cold phosphate-buffered saline (PBS) containing protease inhibitors and non-ionic detergent (NP-40). The total protein concentration was measured using a bicinchoninic acid (BCA) protein assay kit (Beyotime Institute of Biotechnology, Haimen, China). The extracted proteins were separated via sodium dodecyl sulfate–polyacrylamide gel electrophoresis (SDS-PAGE) and transferred onto polyvinylidene difluoride (PVDF) membranes (Millipore, Billerica, MA). The membranes were blocked in 5% nonfat dry milk in Tris-buffered saline-Tween-20 (TBST) solution for 60 min at room temperature. Anti-AdipoR1, anti-PPAR α , anti-P-AMPK and anti-GAPDH antibodies were used as the primary antibodies. The membranes were incubated with the primary antibodies overnight at 4 °C, followed by incubation with a horseradish peroxidase-conjugated secondary antibody for 1 hour at room temperature. Following the second incubation, each membrane was washed for 30 min, incubated for 5 min with an enhanced chemiluminescence (ECL) kit (Amersham, Piscataway, NJ), and exposed to Hyperfilm ECL film (Amersham, Pittsburgh, PA). Densitometric analysis was performed using Image J software (Version 1.43, Broken Symmetry Software, Bethesda, MD), and expression of the four adiponectin-related proteins was normalized to the densities of the respective GAPDH bands.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics software version 20.0 (SPSS Inc., Chicago, IL, USA). The data were expressed as the means \pm standard deviations (SDs). Two-way analysis of variance (ANOVA) was performed with the ApoE knockout and aerobic exercise as factors for the examinations of interactions and main effects. However, because each factor had only two groups (less than three), performing post-hoc tests for multiple comparisons was unpractical. To further explore the effects of aerobic exercise on ApoE^{-/-} mice and normal mice, independent T-tests were conducted to compare between any two groups. The significance level was set at 0.05.

Results

The effects of ApoE knockout and aerobic exercise on

body weight

As shown in Table 1, the weights of the mice of the four groups were very similar before the training program, and no significant differences were found between any of the groups ($p > 0.05$). However, after 12 weeks of training, the weights of all the mice had significantly increased. The results of two-way ANOVA revealed that the main effect of the ApoE knockout was significant ($p < 0.05$), but that the main effect of the aerobic exercise and the interaction (ApoE knockout and aerobic exercise) were not significant ($p > 0.05$). The mean comparisons showed that the mice of the AC and AE groups weighed significantly more than the mice of the CC and CE groups ($p < 0.05$). However, no significant differences were observed between the CC and CE groups, or between the AC and AE groups ($p > 0.05$). Therefore, the effect of the ApoE knockout on the body weights of the mice was clearly significant, resulting in the ApoE^{-/-} mice gaining significantly more weight than the C57 mice. However, the effects of the aerobic exercise and the interaction between the ApoE knockout and exercise were not significant.

Table 1. The body weights of the animals before and after the training program (n = 10).

Group	Before training (g)	After training (g)
C57 Control	21.11 (.88)	29.00 (1.39)
C57 Exercise	20.31 (.81)	28.14 (2.21)
ApoE ^{-/-} Control	21.14 (.99)	32.81 (1.36) *
ApoE ^{-/-} Exercise	20.20 (.85)	32.16 (2.21) #

* compared with the C57 control group, $P < 0.05$; # compared with the C57 exercise group, $P < 0.05$.

The effects of ApoE knockout and aerobic exercise on blood lipids

The data on the four of blood lipid indices are shown in Table 2. The results of two-way ANOVA indicated that the main effects of the ApoE knockout were significant on all four indices ($p < 0.05$), but that the main effects of the aerobic exercise and the interaction of these two factors were significant only on two indices, TG and HDL-C ($p < 0.05$), and not on TC and LDL-C ($p > 0.05$). As shown in table 2, compared with the C57 mice (CC and CE groups), the ApoE^{-/-} mice (AC and AE groups) exhibited significantly higher TC, TG and LDL-C levels ($p < 0.05$), and significantly lower HDL-C levels ($p < 0.05$). Compared with the CC group, TG level was significantly decreased ($p < 0.05$), and HDL-C level was significantly increased ($p < 0.05$) in the CE group. However, compared

Table 2. Blood lipid levels of the C57 and ApoE^{-/-} mice (n = 10).

Group	TC (mmol/L)	TG (mmol/L)	LDL-C (mmol/L)	HDL-C (mmol/L)
C57 Control	3.16 (.27)	.97 (.31)	1.03 (.78)	1.35 (.26)
C57 Exercise	3.13 (.30)	.54 (.12) *	.96 (.69)	1.68 (.13) *
ApoE ^{-/-} Control	18.79 (3.72) *	1.51 (.29) *	9.16 (1.76) *	.59 (.17) *
ApoE ^{-/-} Exercise	18.86 (3.88) #	1.36 (.27) #	9.19 (1.59) #	.66 (.21) #

TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; * compared with the C57 control group, $p < 0.05$; # compared with the C57 exercise group, $p < 0.05$.

Table 3. Plasma adiponectin levels in the C57 and ApoE^{-/-} mice (n = 10).

Group	C57 Control	C57 Exercise	ApoE ^{-/-} Control	ApoE ^{-/-} Exercise
Adiponectin ($\mu\text{g}\cdot\text{ml}^{-1}$)	18.96 (4.89)	19.11 (4.62)	12.41 (3.84) *	12.53 (3.99) #

* compared with the C57 control group, $p < 0.05$; # compared with the C57 exercise group, $p < 0.05$.

to those of the AC group, TC, TG, LDL-C and HDL-C levels of the AE group were not significantly different ($p > 0.05$). These results indicate that the ApoE knockout resulted in a significant increase in TC, TG and LDL-C levels, and a significant decrease in HDL-C levels. Additionally, although aerobic exercise may have significant health effects on the regulation of blood lipids in normal mice, the effects in ApoE^{-/-} mice were not significant.

The effects of ApoE knockout and aerobic exercise on plasma adiponectin level

As shown in Table 3, the plasma adiponectin levels of the two groups of ApoE^{-/-} mice were significantly lower than those of the groups of C57 mice ($p < 0.05$), but no significant differences were found between the CC and CE groups, and between the AC and AE groups. The results of two-way ANOVA demonstrated that the main effect of the ApoE knockout was significant ($p < 0.05$), but that the main effect of aerobic exercise and the interaction between the ApoE knockout and aerobic exercise were not significant ($p > 0.05$).

The effects of ApoE knockout and aerobic exercise on adiponectin-related protein expression in myocardial tissue

The Western blot results regarding the expression of the adiponectin-related proteins, AdipoR1, PPAR α , AMPK and P-AMPK, in myocardial tissue are presented in Figure 1, and the densitometric analysis results are included in Table 4. The results of two-way ANOVA showed that the main effects of both factors, the ApoE knockout and



Figure 1. Western blot results of the adiponectin-related protein expression. CC, C57 control group; CE, C57 exercise group; AC, ApoE^{-/-} control group; AE, ApoE^{-/-} exercise group; GAPDH, glyceraldehyde-3-phosphate dehydrogenase; AdipoR1, adiponectin receptor 1; PPAR α , peroxisome proliferator-activated receptor alpha; AMPK, adenosine monophosphate activated protein kinase; P-AMPK, phosphorylated adenosine monophosphate activated protein kinase.

Table 4. Densitometric analysis results of adiponectin-related protein expression based on Western-blot (n = 10).

Group	AdipoR1	PPAR α	AMPK	P-AMPK
C57 Control	.95 (.06)	.86 (.01)	.43 (.01)	.48 (.11)
C57 Exercise	1.17 (.05) *	.89 (.05)	.67 (.10) *	.64 (.31)
ApoE ^{-/-} Control	1.15 (.09) *	1.08 (.01) *	.62 (.07) *	.43 (.02)
ApoE ^{-/-} Exercise	1.43 (.07) @#	1.22 (.07) @#	.91 (.10) @#	.99 (.07) @##

AdipoR1, adiponectin receptor 1; PPAR α , peroxisome proliferator-activated receptor alpha; AMPK, adenosine monophosphate activated protein kinase; P-AMPK, phosphorylated adenosine monophosphate activated protein kinase; * compared with the C57 control group, $p < 0.05$; # compared with the ApoE^{-/-} control group, $p < 0.05$; ## compared with the ApoE^{-/-} control group, $p < 0.01$; @ compared with the C57 exercise group, $p < 0.05$.

aerobic exercise, on the expression of all four adiponectin-related proteins were significant ($p < 0.05$), but the interaction of these two factors had a significant effect on only P-AMPK expression ($p < 0.05$), and not on the expression of the other three proteins ($p > 0.05$). As shown in table 4, AdipoR1, PPAR α and AMPK expression levels were significantly higher in the groups of ApoE^{-/-} mice than in the groups of C57 mice respectively (AC vs. CC and AE vs. CE) ($p < 0.05$), but P-AMPK expression levels were significantly different only between the groups of AE and CE ($p < 0.05$), not between the groups of AC and CC ($p > 0.05$). Compared with the CC group, the levels of these 4 proteins in the CE group were all increased, but only AdipoR1 and AMPK were significantly increased ($p < 0.05$). Compared with the AC group, the levels of these 4 adiponectin-related proteins were significantly increased in the AE group ($p < 0.05$), particularly P-AMPK, which increased by more than 100%. Therefore the ApoE knockout and aerobic exercise were both significantly effective in stimulating increased expression of these 4 adiponectin-related proteins in myocardial tissue. However compared with the C57 mice, the effects of aerobic exercise appeared to be more significant in the ApoE^{-/-} mice. The increased amplitudes (IAs) of these proteins in the AE group (compared with the AC group) were larger than those in the CE group (compared with the CC group), except for AMPK (Table 4).

Discussion

The effects of ApoE knockout and aerobic exercise on body weight and blood lipid levels

As shown in Table 1, the weights of the 8-week-old ApoE^{-/-} mice were similar to those of the C57 mice. However, twelve weeks later, the ApoE knockout showed a significant effect on the body weights of the mice, regardless of whether aerobic exercise was performed. Therefore, without diet control, aerobic exercise do has little effect on body weight because the increased energy consumption by exercise may be quickly replenished via food intake; but ApoE knockout directly leads to a metabolic imbalance in lipid metabolism, which may explain why the ApoE^{-/-} mice became significantly heavier than the normal mice. As one of the primary apolipoproteins, ApoE plays a key role in maintaining both lipid metabolism and cholesterol homeostasis. As shown in table 2, compared with the normal mice, the TC, TG, LDL-C and HDL-C levels in the ApoE^{-/-} mice were significantly changed.

Regarding the effects of aerobic exercise on blood lipids, exercise was effective in regulating the TG and

HDL-C levels in the blood of the normal mice, but not in the ApoE^{-/-} mice. Although lipid mobilization may be accelerated with exercise, the ApoE knockout might cause impairment in plasma lipid residue removal, as well as other lipid metabolism disorders, thus impairing the effects of aerobic exercise in regulating the blood lipid levels in ApoE^{-/-} mice.

The effects of ApoE knockout and aerobic exercise on plasma adiponectin levels

Numerous reports have described the effects of exercise on plasma adiponectin, but the conclusions drawn by these studies were not consistent (Golbidi and Laher, 2014). Mahmoodi et al. (2014) reported that plasma adiponectin levels increased immediately following exercise but returned to normal within 30 minutes. Akbarpour et al. (2013) reported that 12 weeks of aerobic exercise significantly increased plasma adiponectin levels among females with coronary atherosclerosis. However, many studies (Bobbert et al., 2007; Lee and Kwak, 2014b) have also claimed that aerobic exercise exerts no significant effects on plasma adiponectin levels.

Based on the results shown in table 3, the plasma adiponectin levels of the mice were significantly affected by the ApoE gene knockout, but the effects of the aerobic exercise were negligible in both the ApoE^{-/-} and C57 mice. Therefore, the abnormal lipid metabolism caused by ApoE gene knockout may account for the lower plasma adiponectin levels observed in the ApoE^{-/-} mice. As shown in tables 1 and 2, the effects of the aerobic exercise on the body weights and the majority of blood lipid indices were negligible, indicating that the aerobic exercise utilized in this study was not sufficient to significantly impact the body fat compositions of animals in the absence of diet control. The adiponectin secreted by adipose tissue is a key factor in the regulation of plasma adiponectin levels. To significantly change the plasma level of adiponectin via exercise, it may be necessary to increase either the intensity of the exercise or the length of the exercise.

The effects of ApoE knockout and aerobic exercise on adiponectin-related protein expression in myocardial tissue

The effects of aerobic exercise on the expression of the adiponectin-related proteins AdipoR1, PPAR α , AMPK and P-AMPK in myocardial tissue have seldom been reported, although the effects of exercise on the expression of these proteins have been described in previous studies involving skeletal muscle. Vu et al. (2007) reported that low intensity training may have resulted in in-

creased AdipoR1 expression in skeletal muscle of Wistar rats; however, short-term exercise did not significantly increase AdipoR1 expression in skeletal muscle and decrease AdipoR2 expression. Therefore, it has been hypothesized that only long-term aerobic exercise increases the expression of both AdipoR1 and AdipoR2 in skeletal muscle. It has also been reported that eight weeks of aerobic exercise increases PPAR expression in skeletal muscle, effectively inhibiting cholesterol transport (Thomas et al., 2012). Coven et al. (2003) and Musi et al. (2005) determined that acute exercise increases both the activity and the expression of AMPK in myocardial tissue; however, David et al. (2009) reported that exercise has no effects on either the phosphorylation or the expression of AMPK in myocardial tissue. Based on the findings of these studies, the effects of exercise on AdipoR1, PPAR α , AMPK and P-AMPK expression in myocardial tissue have not yet been conclusively determined, due to the use of different exercise intensities and exercise durations in individual experiments.

Myocardial adiponectin functions primarily by combining with AdipoR1. In this study, the effects of the ApoE knockout and aerobic exercise were both found to be significant on myocardial AdipoR1 levels, which might effectively enhance the sensitivity of myocardial tissue to adiponectin.

PPAR α is a key protein (Madrazo and Kelly, 2008) in the regulation of the fatty acid oxidation in the heart. In this study, the overall effects of both the ApoE gene knockout and aerobic exercise were significant on PPAR α expression in myocardial tissue. Notably, however, after 12 weeks' aerobic exercise, the change in PPAR α expression was not significant in normal mice, but was significant in ApoE $^{-/-}$ mice. These findings indicate that the exercise utilized in this study may not have been sufficient to alter myocardial lipid metabolism in the normal mice, and to increase PPAR α expression, but was sufficient to increase myocardial PPAR α expression in the ApoE $^{-/-}$ mice ($p < 0.05$).

AMPK is an enzyme that regulates intracellular energy metabolism, which it accomplishes by promoting energy production and maintaining energy balance via the activation of downstream signal transduction pathways. Experimental evidence indicates that AMPK plays an important role in cardiac development, myocardial energy metabolism, and protection against myocardial ischemia-reperfusion injury (Kim et al., 2009). In this study, 12 weeks of aerobic exercise effectively promoted the AMPK expression and phosphorylation in myocardial tissue in both types of mice, but appeared to have more significant effects in the ApoE $^{-/-}$ mice than in the normal mice. As shown in table 4, P-AMPK expression was increased by more than 100% in the AE group mice, which was a much greater increase than that in the CE group mice.

The aerobic exercise utilized in this study was effective in stimulating the adiponectin-related protein expression in myocardial tissue of the both types of mice, but the effectiveness of this stimulation was more significant in the ApoE $^{-/-}$ mice. After the 12 weeks of aerobic exercise, the increases in expression of the four adiponec-

tin-related proteins in the myocardial tissue of ApoE $^{-/-}$ mice were more significant than those of normal mice. ApoE $^{-/-}$ mice are born with disorders of cardiovascular system and impaired energy metabolism, causing these mice having a lower exercise capacity than normal mice. In this study, although the exercise models used for both types of mice were the same, the stimulation might have differed due to the different exercise capacities, thus the same exercise might have appeared more effective in promoting adiponectin-related protein expression in myocardial tissue in the ApoE $^{-/-}$ mice than in the normal mice.

Conclusion

The effects of the ApoE gene knockout were significant on the body weight, blood lipids, plasma adiponectin and adiponectin-related protein expression in the myocardial tissue, and the effects of aerobic exercise were also significant on adiponectin-related protein expression and some indices of blood lipid indices. In summary, adiponectin signaling may play an important role in the progression of cardiovascular diseases induced by ApoE knockout, and the healthy effect of aerobic exercise on ApoE $^{-/-}$ mice may be mainly from the increased adiponectin-related protein expression in myocardial tissue

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AUTHOR BIOGRAPHY

Xiao-Juan ZHU

Employment

Key Laboratory of Training Monitoring and Intervention on the Sports in Water, State Sports General Administration of China; Institute of Physical Education, Jiangxi Normal University, China.

Degree

MD

Research interests

Health and environment, physical fitness and sports physiology.

E-mail: zhxjhh@163.com

Li-Hui CHEN

Employment

Key Laboratory of Training Monitoring and Intervention on the Sports in Water, State Sports General Administration of China; Institute of Physical Education, Jiangxi Normal University, China.

Degree

MD

Research interests

Sports medicine and sports psychology.

E-mail: 371051670@qq.com

Jiang-Hua LI

Employment

Key Laboratory of Training Monitoring and Intervention on the Sports in Water, State Sports General Administration of China; Institute of Physical Education, Jiangxi Normal University, China.

Degree

PhD

Research interests

Sports medicine, sports physiology, physical fitness and metabolomics.

E-mail: lijianghua8@sina.com

Key points

- A twelve-week aerobic exercise program exerted only limited effects on the body weights and the plasma adiponectin levels of both the normal mice and the ApoE^{-/-} mice but did effectively regulate the blood lipid levels of the normal mice (but not the ApoE^{-/-} mice).
- After 12 weeks of aerobic exercise, expression of the adiponectin-related proteins in the myocardial tissue of the ApoE^{-/-} and normal mice was increased, but the increased amplitudes of these proteins in the ApoE^{-/-} mice were much larger in the ApoE^{-/-} mice than in the normal mice.
- Aerobic exercise might not alter the plasma adiponectin levels and blood lipid levels of ApoE^{-/-} mice, but improve myocardial energy metabolism and relieve cardiovascular disease symptoms by increasing adiponectin-related protein expression in myocardial tissue.

✉ Jiang-Hua Li

Key Laboratory of Training Monitoring and Intervention on the Sports in Water, State Sports General Administration, Institute of Physical Education, Jiangxi Normal University, Nanchang, China