Journal of Sports Physiology and Athletic Conditioning (J.S.P.A.C) 2025; 5 (16): 1-10

Research Article

The effect of resistance exercise on the expression of the acute steroidogenic regulatory protein gene in rats fed a high-fat diet

Faezeh Saebi[1], Shahram gholamrezaei darsara [2]*, Mohammad Ali Azarbayjani[3], Mohammad Reza Fadaei Chafi[4]

1. Department of Physical Education and Sports Sciences, Ra.C., Islamic Azad University, Rasht, Iran.
2. Department of Physical Education and Sports Sciences, Ra.C., Islamic Azad University, Rasht, Iran.
3. Department of Physical Education and Sport Sciences, CT.C., Islamic Azad University, Tehran, Iran.
4. Department of Physical Education and Sports Sciences, Ra.C., Islamic Azad University, Rasht, Iran.

Received: 12 July2025
Accepted: 23 August 2025

Abstract

The present study aimed to investigate the effect of resistance training on the expression of the Steroidogenic acute regulatory protein (StAR) gene in the testicular tissue of rats consuming a high-fat diet (HFD). Eighteen male Wistar rats were selected from the animal house of Islamic Azad University, Central Tehran Branch. They were randomly divided into three groups: 1- control group fed with a normal diet, 2- control group fed with HFD and 3- resistance training group with HFD. HFD was administered for eight weeks. Resistance training was also performed during the first four weeks of HFD. Forty-eight hours after the last training session, the animals were anestheticated with ketamine and xylazine. Testicular tissue was removed to determine StAR gene expression using Real Time PCR. Results showed that StAR gene expression in the high-fat diet-fed control group was significantly lower than that in the normal diet-fed control group (P=0.001). StAR gene expression in the resistance-exercise-HFD group was significantly higher than in the HFD control group (P=0.008). Based on this, it is concluded that resistance training is a suitable strategy for protecting testicular tissue from damage caused by HFD. It can prevent a decrease in testosterone production and spermatogenesis due to increased STAR gene expression.

Keywords:
Aerobic exercise, gallic acid, Beclin-1, ATG5, and LC3

Introduction

***Corresponding author:** Shahram gholamrezaei darsara
Address: Department of Physical Education and Sports Sciences, Ra.C., Islamic Azad University, Rasht, Iran.
Email: shahram1347@iau.ac.ir **Tell**:
SH GH: 0000-0001-7271-4472

1. Introduction

In the current century, obesity has developed greatly due to lifestyle changes and its prevalence is increasing (1). Available data show that overweight and obesity rates have increased globally and regionally and in all countries between 1990 and 2021 (2). In line with the increase in obesity, a significant increase in infertility has been reported in recent years, especially during the last two decades (3). Findings from a comprehensive, evidence-based meta-analysis indicate a decline in sperm concentration among European men over the past 50 years, which is one of the reasons for male infertility (4). Various factors contribute to male infertility, including obesity (5). Studies have reported an association between obesity and disturbances in reproductive hormones or sperm parameters in adult men (6). Evidence suggests that obesity is associated with disturbances in reproductive hormones, which can cause infertility (7,8). Although the exact mechanism by which obesity reduces sexual hormones in men is not fully understood, it seems that obesity can cause infertility through negative effects on the hypothalamic-pituitary-gonadal (HPG) axis (9), SHBG concentration (10), and factors responsible for testosterone synthesis in Leydig cells (11). Obesity inhibits Steroidogenesis Pathway Enzymes and decrease testosterone production (12). Obesity can also negatively affect other factors involved in steroidogenesis. One of these factors is the steroidogenic acute regulatory protein. StAR is a transport protein that plays a crucial role in steroid hormone biosynthesis by regulating cholesterol transfer from the outer to the inner mitochondrial membrane.

This step is essential because cholesterol must reach the inner membrane to be converted to pregnenolone by the enzyme cytochrome P450scc (CYP11A1), which is the first enzymatic step in steroidogenesis (13,14). Previous studies have shown that HFD can decrease StAR expression and reduce testosterone production (15,16,17). Since regular physical activity can reduce many obesity negative effects, Yi et al. (2017) show that moderate-intensity aerobic exercise in obese mice significantly increased StAR mRNA and protein expression and reversed HFD inhibition (18). Xu et al. (2022) reported increased StAR expression after treadmill running in male mice fed a high-fat diet (19). However, studies in this area are very limited. A review of studies shows that the effect of resistance training on StAR expression has not been investigated. Accordingly, the present study aimed to determine the effect of resistance training on the expression of the steroidogenic acute regulatory protein gene in male rats fed a high-fat diet.

2. Materials and Methods

Animals

In a preclinical trial, 15 adult male rats, 12 weeks old and weighing 180 to 200 grams, were selected from the animal house of Islamic Azad University, Central Tehran Branch, as subjects. All animals were kept under standard laboratory conditions in autoclavable transparent polycarbonate cages at a temperature of (20-22°C), relative humidity (55%), and free access to water and sufficient food (a product of Behparvar Company, Iran) with a 12-hour dark/light cycle. After a week of acclimatization, the subjects were randomly divided into three groups (six rats in each group).

These groups included 1- the control group fed with normal food, 2- the control group fed with high-fat food, and 3- the group fed with high-fat food and resistance training. The subjects were maintained and studied according to guidelines for working with laboratory animals approved by the Ministry of Health and Medical Education of the Islamic Republic of Iran.

High-fat diet

Rats in the control group – normal diet – were fed pelleted rat food produced by Behparvar Company. The normal diet used in the present study contained 3.5-4.5% fat, 4.5-4.5% fiber, 19.5-50.20% crude protein, 1.0-95% calcium, 0.5-55% salt, 1.15% lysine, 0.7-65% phosphorus, 0.33% methionine, 0.25% tryptophan, and 0.72% threonine. The high-fat diet was prepared by adding 20% palm oil, 1.5% cholesterol, and 0.25% cholic acid to a normal diet (20). Subjects were fed the HFD for 8 weeks (four weeks before and four weeks during the resistance training period).

Resistance training protocol

The resistance exercise program used in the present study was ladder climbing, a rodent-specific exercise program. First, rats were trained to climb ladders for one week. Then their weights were calculated and the training program was adjusted based on their initial weights. After that, the rats underwent resistance training for four weeks, five days a week. This included climbing a vertical ladder (height: 110 cm, slope: 80 degrees, and distance between steps: 2 cm) according to Hornberger et al's instructions. (2004) (21).

Testicular biopsy method

Twenty-four hours after the last training session and cannabis extract administration and after a 12-hour fast, the rats were anesthetized using a mixture of 2.7 ml xylazine and 10 ml ketamine at a dose of 100 µL/100 g. body weight. After complete anesthesia, the chest was opened and blood was collected from the left ventricle. After the animal was killed, the testicles were removed from both sides. The testicular fat and epididymis were removed and stored at -80°C for RT-PCR.

Gene expression assay

Real-time-PCR technique was used to assay StAR gene expression in testicular tissue. First, primers were designed. Total RNA was extracted from testicular tissue and converted to cDNA. Then, cDNA was amplified by PCR and measured for expression of the studied genes. Table 1 presents the primer sequences.

Statistical Model

All data are presented based on the mean and standard deviation. First, the assumptions of parametric tests, including normality of data distribution and homogeneity of variances, were tested by Shapiro-Wilk and Levene tests. To compare StAR gene expression between the control group fed with normal food, the control group fed with high-fat food, and the group fed with high-fat food and resistance training, one-way analysis of variance was used for independent groups. If a significant difference was observed, the Bonferroni test was used to determine the location of the difference. The significance level for all calculations was considered P=0.05.

Gene	Forward	Reverse
StAR	AGGAAGTCTGCAAGTCTGTTC	AGCCTCAGTTCTGTTTTCCTG
GAPDH	AACCCATCACCATCTTCCAG	CCAGTAGACTCCACGACATAC

3. Results

Comparison of StAR gene expression between the study groups showed that the expression of this gene in the control group-fed with high-fat food was significantly lower than in the control group-fed with normal food (P=0.001). The expression of the StAR gene in the resistance exercise-HFD group was significantly higher than in the control-HFD group (P=0.008). Figure 1

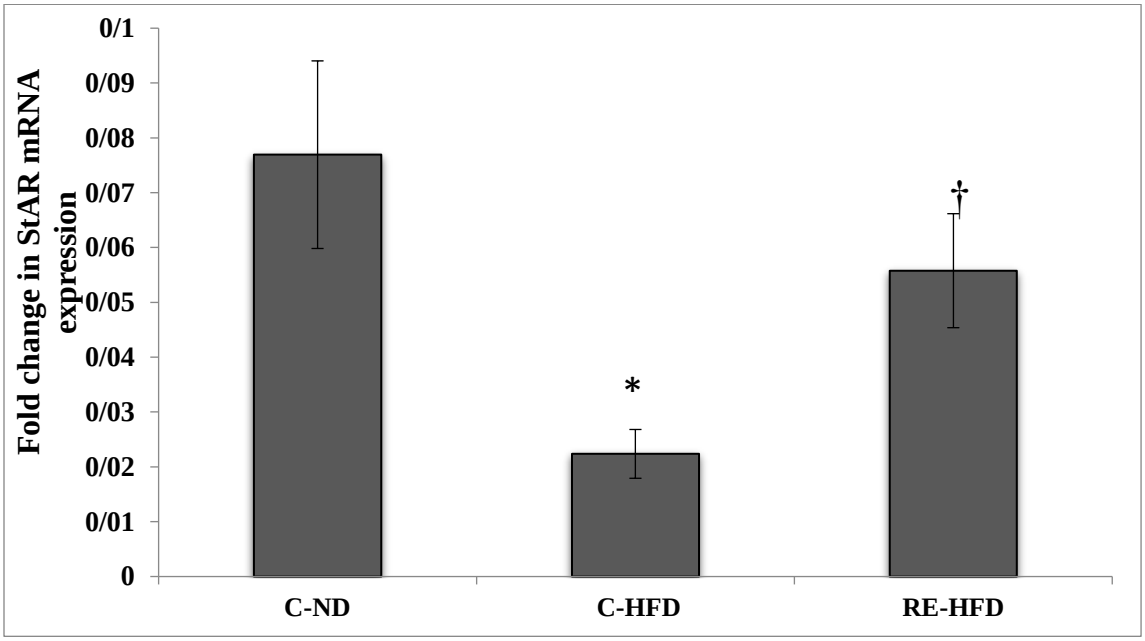

Figure 1- StAR gene expression in testicular tissue in the studied groups.C-ND: control normal diet.C-HFD:control high fat diet. RE-HFD: resistance exercise-high fat diet * Indicates significant difference compared to the C-ND group. † Significant difference compared to the C-HFD group. Data are reported based on mean and standard deviation.

4. Discussion

The present study found that high-fat feeding reduced StAR gene expression. Testosterone, a steroid hormone, is synthesized and secreted mainly (95%) by Leydig cells in the testis (22). In response to luteinizing hormone (LH), testicular testosterone biosynthesis is a multistep process involving a complex of steroidogenic proteins and enzymes, such as the steroidogenic protein (StAR), cholesterol side-chain cleavage enzyme (P450scc or CYP11A), and 3β-hydroxysteroid dehydrogenase (3β-HSD) (23). StAR facilitates the first and rate-limiting step of testosterone biosynthesis by accelerating the transfer of cholesterol from the outer mitochondrial membrane to the inner mitochondrial membrane (24). In support of the role of StAR in testosterone production, it has been reported that individuals with congenital adrenal hyperplasia caused by a mutation in the StAR gene and mice lacking StAR are almost completely unable to synthesize steroids, indicating that the StAR protein is an essential element in steroids synthesis (25). It has been reported that both pharmacological inhibition of ATP synthesis and loss of mitochondrial membrane potential (Δψm) can inhibit steroidogenesis by reducing StAR expression, demonstrating that StAR activity requires mitochondrial homeostasis (26). Evidence suggests that high-fat feeding can impair StAR function by negatively affecting mitochondria, thereby reducing testosterone production and subsequent spermatogenesis in testicular tissue. As a result, HFD can negatively affect testicular function by increasing oxidative stress and inflammation. Transient opening of the mitochondrial permeability transition pore (mPTP), a multi-protein tunnel-like complex located across the inner and outer mitochondrial membranes, is of critical importance in maintaining mitochondrial homeostasis in response to natural stimuli (27). However, under severe stress conditions, the irreversible opening of the mPTP leads to mitochondrial dysfunction, characterized by loss of Δψm, uncoupling of oxidative phosphorylation, and subsequent inhibition of ATP production, increased production of reactive oxygen species, mitochondrial swelling, rupture, and death (28).

Although the molecular components of the mitochondrial permeability transition pore remain unclear to date, cyclophilin D (CypD), a peptidyl prolyl cis-trans isomerase F (PPIF) in the mitochondrial matrix, is an essential regulator of mitochondrial permeability transition pore opening (29). Cyclophile D has been shown to translocate from the matrix to the inner mitochondrial membrane and bind to major candidate components of the mitochondrial permeability transition pore, including adenine nucleotide translocatable (ANT), phosphate carrier (PiC), and oligomycin-sensitive conferring protein FoF1 ATP synthase (OSCP) across the inner mitochondrial membrane, which is a key step in the process of facilitating the opening of the permeability transition pore in response to various stress stimuli (30). High-fat diet feeding can increase expression levels of cyclophilin D protein and can cause mitochondrial dysfunction in the mouse liver Evidence suggests that cyclophilin D-dependent mitochondrial dysfunction in testicular Leydig cells plays a role in the downregulation of StAR expression induced by HFD (14). A significant decrease in StAR mRNA was reported under HFD conditions, suggesting that high-fat diet feeding may decrease testosterone levels through transcriptional repression of StAR. Mitochondrial homeostasis is essential for StAR expression and activity. Leydig cells are particularly sensitive to reactive oxygen species produced by oxidative stress. Previous studies have shown that loss of mitochondrial membrane potential (ΔΨm) can induce StAR degradation. Reduced mitochondrial ATP levels reduce StAR translation. Furthermore, ATP is critical for StAR phosphorylation, which increases its activity and stability. Long-term HFD can cause abnormal mitochondrial membrane structure or even rupture, due to increased permeability to the inner mitochondrial membrane. This allows some small molecules to gently penetrate into the intermembrane space, disrupting mitochondrial homeostasis. In summary, these results both in vivo and in vitro demonstrate that long-term high-fat or palmitic acid feeding leads to mitochondrial dysfunction including decreased mitochondrial ATP production, increased ROS, and loss of ΔΨm, which can reduce StAR expression.

Accordingly, it is clear that the present study showed that feeding on a high-fat diet containing palmitic acid reduces the expression of the StAR gene in testicular tissue, both by increasing reactive oxygen species and by affecting the aging of the mitochondrial membrane. Another finding of the present study showed that StAR gene expression was significantly higher in the resistance training-high-fat diet group than in the control-HFD group. Although the effect of resistance exercise on StAR gene expression has not been studied, few studies have studied the effect of aerobic training on this gene's expression. However, it seems that since the resistance exercise used was not of high intensity, the increase in StAR gene expression can be explained based on the proposed molecular mechanisms resulting from aerobic exercise. In all three studies, increased StAR gene expression has been reported in rodents fed an HFD after moderate-intensity aerobic swimming and interval training on a treadmill (18,19,31). Although the exact molecular mechanism of increased StAR expression following regular physical activity, especially resistance training, under HFD conditions is not well understood, some mechanisms have been proposed for stimulating its expression. Evidence suggests that regular physical activity counteracts HFD inhibitory effects on testicular steroidogenesis by modulating key molecular pathways. One proposed mechanism is the restoration of leptin signaling and activation of the JAK-STAT pathway. HFD-induced leptin resistance disrupts the testicular leptin-JAK-STAT pathway and suppresses steroidogenic enzymes such as SF-1 and P450scc and StAR expression. Regular physical activity (e.g., aerobic exercise) restores leptin sensitivity and reactivates the JAK-STAT pathway. This upregulates StAR mRNA and protein expression and enhances cholesterol transport to mitochondria for testosterone synthesis (18). Increased antioxidant defense capacity resulting from regular physical activity is another proposed mechanism for increasing STAR expression. HFD creates reactive oxygen species (ROS) that damage testicular cells and suppress StAR expression.

Regular physical activity promotes the expression of Nrf2, a master regulator of antioxidant defense, reduces oxidative stress and protects steroidogenesis processes. This increases StAR gene expression in obesity conditions induced by HFD (31-35). On the other hand, regular physical activity activates AMPK as an energy sensor that stimulates mitochondrial biogenesis via PGC-1α. Improved mitochondrial efficiency supports cholesterol transport and StAR function. Increased systemic and tissue inflammation inhibits steroidogenesis in obesity. HFD increases proinflammatory cytokines (TNF-α, IL-1β) which decrease StAR and steroidogenic enzymes. Regular physical activity reduces systemic inflammation by reducing proinflammatory cytokines. It prevents the activation of the NFkB signaling pathway in testicular tissue, thereby minimizing steroidogenesis suppression caused by inflammation.

5. Conclusion

The results of the present study show that a high-fat diet reduces the expression of the StAR gene, one of the most critical regulators of testosterone production in testicular tissue. These findings indicate that a high-fat diet causes testicular dysfunction. This finding confirms the negative effects of obesity on testicular tissue and justifies one of the molecular mechanisms of testicular damage in obese individuals. In contrast, resistance training was able to increase the StAR gene expression. This indicates that resistance training can reduce the likelihood of decreased testosterone production in testicular tissue under HFD. Based on this, it is concluded that resistance training is a suitable strategy to protect testicular tissue from the damage caused by HFD. It can prevent testosterone production decrease and subsequent spermatogenesis due to increased STAR gene expression.

Acknowledgements

This article is based on a doctoral thesis in exercise physiology. It was conducted at the Department of Exercise Physiology, Faculty of Physical Education and Sport Sciences, Islamic Azad University, Central Tehran Branch. The authors of this article would like to thank the officials of the Faculty of Physical Education for their invaluable assistance in conducting this study.

Funding

This study did not have any funds.

Compliance with ethical standards

Conflict of interest None declared.

Ethical approval the research was conducted with regard to the ethical principles.

Informed consent Informed consent was obtained from all participants.

Author contributions

Conceptualization: F.S, SH.GH.D, M.A.A, M.R.F.GH ; Methodology: F.S, SH.GH.D.; Software: F.S, SH.GH.D.; Validation: F.S, SH.GH.D.; Formal analysis: F.S, SH.GH.D.; Investigation: F.S, SH.GH.D.; Resources: F.S, SH.GH.D.; Data curation: F.S, SH.GH.D.; Writing - original draft: F.S, SH.GH.D.; Writing - review & editing: F.S, SH.GH.D.; Visualization: F.S, SH.GH.D.; Supervision: F.S, SH.GH.D.; Project administration: M F.S, SH.GH.D.; Funding acquisition: F.S, SH.GH.D.;

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