



# The Effect of Resistance Training on Heart Damage Risk Factors in Adult Male Rats Exposed to Nandrolone

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ARTICLE INFO	ABSTRACT
<p><i>Article type:</i> Research Paper</p>	<p><b>Introduction:</b> Athletes and non-athletes use nandrolone (Na) for different purposes as a derivative of testosterone, and various results have been reported regarding its use. This study aimed to evaluate the effect of a period of resistance training (RT) on SOD, MDA, CRP, and cTn-C in the cardiac tissue of Na-exposed rats.</p> <p><b>Methods:</b> In the present experimental research, 20 male Wistar rats aged 8-10 weeks were randomly divided into four groups of five animals: (1) control, (2) sham, (3) Na (10mg/kg), (4) Na+RT. The RT group performed 1m ladder climbing in three sessions weekly for eight weeks. One-way ANOVA and Tukey's post hoc tests were implemented to analyze the data (<math>P \leq 0.05</math>).</p> <p><b>Results:</b> Levels of SOD were significantly lower in the Na+RT group compared to the control group (<math>P=0.006</math>). However, no significant difference was observed in levels of SOD in the Na group compared to the Na+RT group (<math>P=0.99</math>). In addition, the levels of MDA and CRP in the Na+RT group were significantly lower than in the Na group (<math>P \leq 0.05</math>). Histological studies showed that Na intoxication, RT, and RT with Na use did not affect tissue changes in the study groups.</p> <p><b>Conclusion:</b> Based on the results, nandrolone with and without resistance training increases heart disease risk indicators. However, the risk of heart disease in training and abusing nandrolone is less than the normal condition of this steroid without training.</p>
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## Introduction

Anabolic-androgenic steroids (AAS) include testosterone and a number of its derivatives that athletes and non-athletes use regarding their anabolic effects (1-3). Increased use of AASs in non-athletes for weight loss, body mass index (BMI), and improved body image in developed countries, followed by heart disease, anxiety, depression, and hippocampal dysfunction, have concerned the world (4). Abuse of these drugs that appear to cause hormonal disorders, precocious puberty, alterations in physical fitness, pre-adolescent sexual behaviors, altered glucose tolerance, increased insulin resistance, and reduced thyroid hormones might lead to diseases such as osteoporosis, aplastic anemia, kidney and liver failure, cancer and heart diseases (2,3).

The precise mechanism of the effect of nandrolone as a testosterone derivative has not been fully identified. However, researchers

believe that myocardial tissue is sensitive to anabolic steroids, and their abuse might increase the expression of its receptors in cardiac tissue and lead to enhanced production of reactive oxygen species (ROS) and disruption of oxidative metabolism (5). A six-week administration of nandrolone and nandrolone combined with swimming training increased heart and coronary artery hypertrophy and DNA damage markers. Additionally, results showed increased levels of low-density lipoprotein and cholesterol, and severe cardiac and coronary artery fibrosis was observed in pathology images (3). The lipid peroxidation levels in the nandrolone + training and nandrolone groups were accompanied by increased thickness of the left ventricular wall and left ventricular diameter of rats compared with the training group without nandrolone and controls. On the other hand, the findings of another research have demonstrated no considerable changes in total antioxidant capacity in the heart tissue of animals in the

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nandrolone, nandrolone + training, control, and sham groups (6). Studies have shown that the abuse of AAS by disrupting the metabolism of cardiac cells increases C-reactive protein (CRP) as a strong indicator of cardiovascular risks (7). In addition, researchers have indicated that nandrolone decanoate abuse increases cardiac troponin I in rats via increasing lipid peroxidation and decreasing antioxidants (8). Furthermore, in another study, researchers assessed the acute effect of nandrolone on the heart tissue of rats, and the results revealed that the use of nandrolone at dosages of 1, 10, and 100mg had no considerable effect on oxidative markers in the heart tissues of animals (9). In another study, 15 mg/kg nandrolone for two weeks revealed no significant effect on cardiac tissue hypertrophy; nevertheless, longer-term 10-week use of 5mg/kg nandrolone two times weekly increased the extent of ischemia in the heart tissue and decreased cardiac function. Accordingly, nandrolone might have different effects on cardiac tissue, as in some cases, which improved metabolism and increased the expression of antioxidant proteins in ischemic heart tissue by increasing the synthesis of proteins involved in energy consumption (10). There are inconsistent findings regarding the efficacy of nandrolone on the heart tissue, and these effects are dose- and duration-dependent. Despite the prohibition of anabolic steroids during exercise by the World Doping Organization, there is a growing trend in non-therapeutic drugs among athletes and non-athlete individuals. As a result, more accurate information could provide medical-athletic communities with the knowledge to determine these drugs' amount, timing, and effects. Consequently, the present research aimed to evaluate the efficacy of resistance training on the concentration of superoxide dismutase enzyme in the cardiac tissue of nandrolone-exposed rats. The study aimed to investigate the effect of a period of resistance training on the concentration of heart damage risk factors in the cardiac tissue of nandrolone-exposed rats.

## Materials & Method

### *Animal Care and Experimental Design*

This article is extracted from a Ph.D dissertation of Sport Physiology and has been registered with the code IR.SSRC.REC.1398.010 at the Ethics Committee of the Institute of Physical Education.

In this experimental study, 20 male Wistar rats aged 8-10 weeks with a mean weight of  $190 \pm 9g$  were purchased, transferred to a laboratory, and maintained there for two weeks to get accustomed to training protocols and adapt to the laboratory environment.

The rats were kept in rodent cages made of PVC with metal mesh lids, the floor of which was covered with clean wood chips, at an ambient temperature of  $22 \pm 2^{\circ}C$ , 50-60% humidity, and 12-hour dark-light cycle and had *ad libitum* access to special water and food. The rats were then randomly divided into four groups of 5 animals: (1) control, (2) sham (receiving nandrolone solvent or normal saline peritoneally), (3) nandrolone, and (4) nandrolone + resistance training.

### *Consumption of Nandrolone*

A calibrated insulin syringe was used to administer the drug at a precise dose and specific time to the animals. Given the significance of evening training, nandrolone (manufactured by Iran Hormone Company) (11) was injected deeply at the dose of 10mg/kg in the quadriceps muscles and back of all groups (except the control group) once a week at 9:00 am.

### *Resistance Training Protocol*

In this study, the rats in the training groups climbed a ladder with a one-meter height, 26 steps, and a slope of 85 degrees for eight hours. The training sessions included five repetitions of three sets, with 60 seconds of rest among repetitions and 120 seconds of rest between sets. In this vein, the training protocol started after tying a weight to the animals' tails. In the first week, the weight attached to the rats' tails was 50% of their weight, which was added to 10% per week, which reached 120% of the animal's body weight in the final week. Rats were trained to climb the ladder a few weeks before the training protocol and were forced to climb it by manual stimulus if they refused. The control group was also present to go through all the conditions at the training site (12).

### *Sampling*

The rats were anesthetized with diethyl ether 48 hours after the final training session (made by Merck Co., Germany), and then their chest was dissected, and their hearts were extracted and preserved at  $-20^{\circ}C$  to examine the desired variables (13). After dissection, the rats' bodies

were burned at 3000 degrees Celsius, and their ashes were delivered to the studied hospital.

### Measuring Variables

This research used the ELISA method to measure superoxide dismutase at the U/ml scale using the Navand Lab kit (Version 0.6 Last updated 15). MDA levels were measured at  $\mu\text{M}$  scale using the ELISA method and ZellBio GmbH kit (Made in Germany) with economic code ZB-MDA-96A. In addition, CRP levels were measured at pg/mL scale using the MYBIOSOURCE kit (made in China) with economic code Catalog No: MBS2508830. Troponin levels were also measured at ng/L scale using the Pars test kit (made in Iran). Further, the hematoxylin-eosin (H&E) staining method was used to assess the oxidative levels of heart tissues.

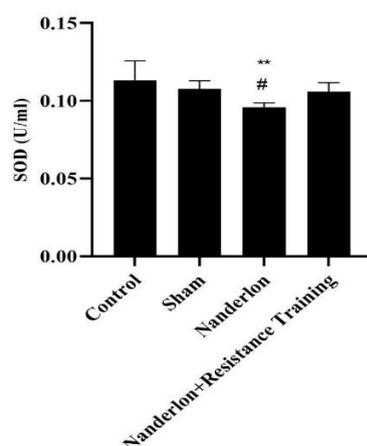
### Hematoxylin-eosin Method

Biopsy samples of cardiac tissues were fixed in formalin 10%. After processing, a thin slice of cardiac tissue was prepared, and laboratory slides were provided for all tissue slices. Then,

the H&E technique was used. The stages of staining were floating in two containers of xylol (ten minutes each), 96% alcohol (five minutes), 100% alcohol (five minutes), and hematoxylin (ten minutes), immersion in an acid container one time and later Lithium carbonate, three steps of water wash, eosin (3min), incubation in 96% and 100% alcohol container and finally xylol clearance, respectively. As a result, a pathological examination of the samples was conducted to determine how much oxidative stress (OS) was present in the heart tissues in the study.

### Data Analysis Procedure

In this study, the Shapiro-Wilk test was implemented to show the normality of data. One-way analysis of variance and Tukey's *post hoc* tests were also performed to compare the study groups. Statistical statistics were analyzed using SPSS software version 22, and  $P \leq 0.05$  was assumed as the significance level in the study. In other words, the confidence interval for the analysis was considered 95%.



**Figure 1.** Mean and standard deviation of SOD levels in the heart tissue of rats in the four study groups.

\*\*( $P = 0.01$ ) Significant decrease in nandrolone group compared to the control group.

# ( $P=0.05$ ) Significant decrease in nandrolone group compared to the sham group.

## Results

The results of the one-way analysis of variance test showed a significant difference in the levels of SOD ( $P=0.002$ ,  $F=5.73$ ), MDA ( $P=0.001$ ,  $F=21.40$ ), CRP ( $P=0.001$ ,  $F=13.42$ ) and troponin ( $P=0.001$ ,  $F=10.19$ ) in the research groups.

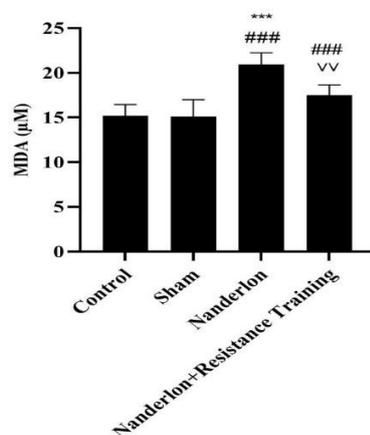
The results of Tukey's *post hoc* test showed no significant difference in the control and sham groups ( $P=0.52$ ), but the levels were significantly lower in the nandrolone group than in the control ( $P=0.0015$ ) and sham ( $P=0.04$ ) groups. In

addition, there was no significant difference in the nandrolone + resistance training group compared to the control ( $P=0.29$ ) and sham ( $P=0.97$ ) groups. Further, no significant difference was observed in the nandrolone groups compared to the nandrolone + resistance training group ( $P=0.09$ ) (Figure 1).

There was no significant difference in MDA levels in the control and sham groups ( $P=0.99$ ). However, the levels were significantly higher than in the control in the nandrolone group ( $P=0.001$ ) and sham ( $P=0.001$ ) groups. In

addition, the levels in the nandrolone + resistance training group were significantly higher than in the sham group ( $P=0.04$ ). The levels in the nandrolone + resistance training group were significantly lower than in the

nandrolone group ( $P=0.002$ ). However, there was no significant difference between the nandrolone + resistance training group and the control group ( $P=0.57$ ) (Figure 2).



**Figure 2.** Mean and standard deviation of MDA levels in the heart tissue of rats in the four study groups.

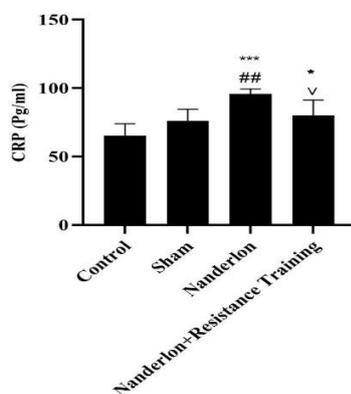
\*\*\*( $P = 0.001$ ) Significant increase in nandrolone group compared to the control group.

### ( $P=0.001$ ) Significant increase in nandrolone group compared to the sham group.

√ ( $P=0.01$ ) Significant decrease in nandrolone+resistance training group compared to the sham & nandrolone groups.

CRP levels in the sham and control groups were not significantly different ( $P=0.15$ ), but the levels were significantly higher than in the control group in the nandrolone ( $P=0.001$ ) and nandrolone + resistance training ( $P=0.02$ ) groups. The levels were also significantly higher

in the nandrolone group than in the sham group ( $P=0.003$ ). However, the levels in the nandrolone + resistance training group were significantly lower than in the nandrolone group ( $P=0.02$ ) (Figure 3).



**Figure 3.** Mean and standard deviation of CRP levels in the heart tissue of rats in the four study groups.

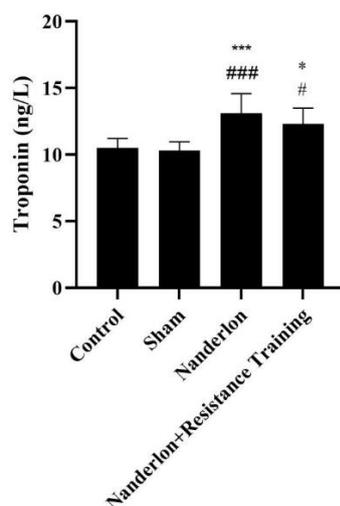
\*( $P=0.05$ ), \*\*\*( $P = 0.001$ ) Significant increase in nandrolone group compared to the control group.

## ( $P=0.01$ ) Significant increase in nandrolone group compared to the sham group.

√ ( $P=0.05$ ) Significant decrease in nandrolone+resistance training group compared to the sham & nandrolone groups.

There was no significant difference in troponin levels in the control and sham groups ( $P=0.98$ ). However, the levels were significantly higher in the nandrolone ( $P=0.001$ ) and nandrolone + resistance training ( $P=0.03$ ) groups than in the control group. The levels were also significantly

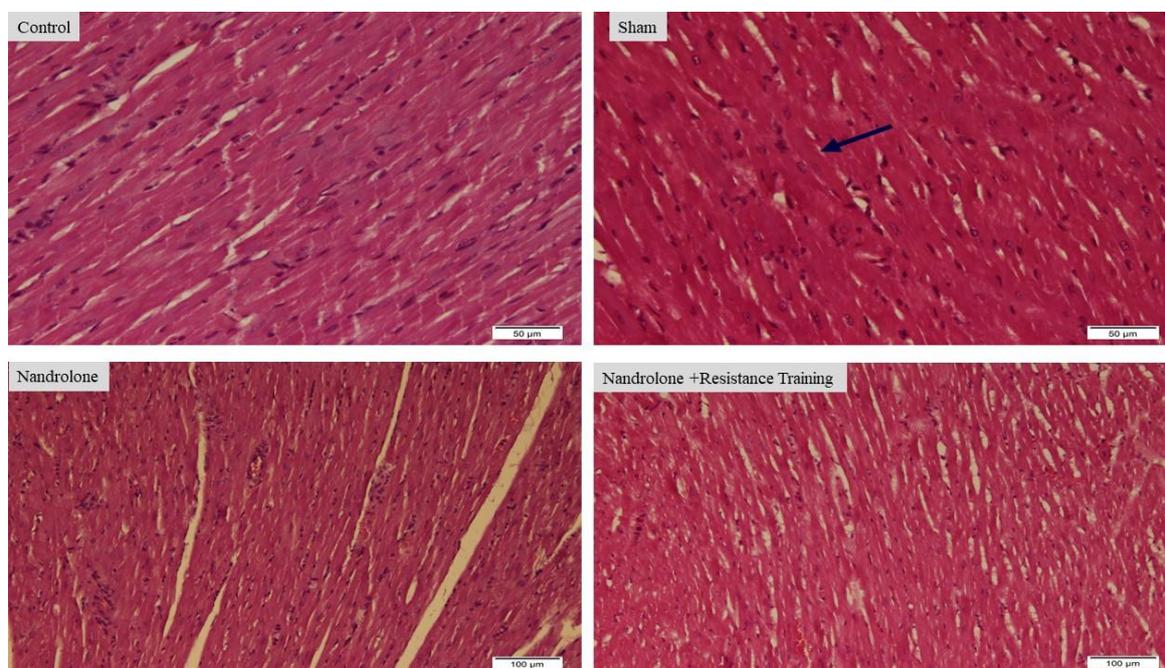
higher in the nandrolone ( $P=0.001$ ) and nandrolone + resistance training ( $P=0.017$ ) groups than in the sham group. In addition, there was no significant difference in the nandrolone and nandrolone + resistance training groups ( $P=0.55$ ) (Figure 4).



**Figure 4.** Mean and standard deviation of troponin levels in the heart tissue of rats in the four study groups. \*(P=0.05), \*\*\*(P=0.001) Significant increase in nandrolone training group compared to the control group. # (P=0.05), ### (P=0.001) Significant increase in nandrolone group compared to the sham group.

Parallel orientation and normal architecture of myocardial cells in the control group were H&E stain (x200). Cardiac myocytes in a sham group showed normal diameter and structure, H&E stain (x200). Cardiac myocytes in the nandrolone group revealed no evidence of inflammation or fibrosis, H&E stain(x100). In the nandrolone + resistance training group, cardiac myocytes illustrated no pathologic change, H&E

stain(x100). According to Figures 2-5, the results obtained by light microscopy from the heart tissue of rats in the study groups indicate that the heart tissue has normal tissue characteristics, and cell cytoplasm and basophilic nuclei are observed with normal margins. In addition, intercellular connections are evident, and no bleeding, inflammation, or abnormal features are observed.



**Figure 5.** Oxidative stress profile in the research groups

## Discussion

The present research demonstrated that nandrolone administration had no considerable effect on levels of superoxide dismutase, but MDA, CRP, and troponin levels in the Nandrolone group increased compared to the control group. Contradictory findings have been reported regarding the efficacy of nandrolone on the heart tissue. For instance, similar to our study, researchers have shown that using 10mg/kg nandrolone for 14 weeks had no significant effect on changes in GPX levels in rats (14). Results of a study showed that two weeks of 15mg/kg nandrolone administration had no significant effect on cardiac tissue hypertrophy in rats and could increase the expression of antioxidant enzymes under stressful conditions (15). Nandrolone and anabolic steroids could increase metabolic proteins by contributing to increased protein expression. As reported in one study, anabolic steroids improved lipid metabolism (16), but inconsistent with the present study, nandrolone alone could increase the ratio of MDA to GPX in the heart tissue of rats (17). The contradictory effects of nandrolone could be attributed to the effects of corticosteroids, which can increase glucocorticoids. Glucocorticoids increase the production of cellular peroxides, which induce antioxidants (15), or the hypothalamic-pituitary-gonadal axis disruption disturbs glucocorticoid function and carbohydrate and lipid metabolism, leading to increased metabolic stress (2,3). On the other hand, different outcomes of nandrolone consumption could be related to the purpose (therapeutic or doping) and duration of administration. Therefore, no undesired effect was observed after drug administration for two weeks, and drug consumption could cause tissue ischemia for ten weeks.

The results showed that the levels of SOD were significantly lower in the nandrolone + resistance training group than in the controls, but there were no significant differences in SOD levels of the nandrolone group compared with the nandrolone + resistance training group. In addition, the results indicated that the levels of MDA and CRP in the nandrolone + resistance training group were significantly lower than in the nandrolone group. In addition, pathologic studies showed that the tissues were normal in different groups, and there were no considerable differences in oxidative stress in the four study

groups. Consistent with the present study, 20mg/kg nandrolone decanoate intake and swimming exercise (one hour daily) for four weeks induced left ventricular wall thickness compared to the nandrolone and control groups. In addition, the H&E method showed increased collagen in the exercise + nandrolone group. The findings demonstrated that swimming exercise and nandrolone administration caused pathological changes in the left ventricular tissue of rats (18).

Using nandrolone with and without resistance training dependent on increased collagen in left ventricular tissue impaired systolic and diastolic pressure and led to heart disorders (19). However, inconsistent with our research findings, ten weeks of 10mg/kg nandrolone administration had no significant effect on the antioxidant changes in the heart tissue. In addition, nandrolone combined with exercise had no significant impact on myocardial GPX concentration in aged rats (20).

Nandrolone intake, moderate-intensity resistance training, and a combination of nandrolone and RT led to hypertrophy of the heart tissue. However, the training modified the ratio of MDA to GPX. Moderate-intensity endurance training and nandrolone intake had no significant effect on hypertension, heart rate, baseline levels of electrocardiographic parameters, and composition of exercise; however, nandrolone increased left ventricular fibrillation in the heart tissue of rats (17). Similarly, another study showed that high doses of testosterone had no significant effect on changes in antioxidant levels in the myocardium of elderly rats (20). In a study, the researchers concluded that apoptosis markers and oxidative stress were lower than in the stanazol consumption group in resistance training groups with stanazol consumption (21). In addition, a study showed that SOD and GPX levels in the nervous system tissue of the resistance training + stanazol consumption group were higher than the stanazol consumption group. In contrast, the MDA values in the resistance training + stanazol consumption group were lower than the stanazol consumption group (22). In addition, Grace and Davies showed that CRP levels in bodybuilders who used anabolic steroids were much higher than in bodybuilders who did not use any AAS (7). The inconsistencies between these studies and the present study could be due to the

differences in the dosage and duration of the drug intake. Researchers have believed that long-term administration of anabolic steroids by athletes decreases gonadotropin-releasing hormone secretion as well as levels of glucocorticoid and mineralocorticoid and therefore causes disorders to improve muscle strength and volume with negative feedback to the hypothalamic-pituitary-gonadal axis. This encounters individuals with increased reactive oxygen species and decreased antioxidant enzymes such as GPX and SOD, improving cardiovascular risk factors (1–4,23). Resistance training with transcription of the nuclear factor erythroid 2-related factor 2 (NRF2) gene seems to increase the expression of antioxidant enzymes such as SOD and catalase. In addition, exercise leads to improved angiogenesis by increasing the energy requirement, reducing oxidative stress, and thus improving cardiovascular function (22). Therefore, the reduction of CRP and troponin T following exercise in rats exposed to nandrolone can be related to activating antioxidant pathways and improving cell metabolism. Regarding the differences in the measurement of the levels of variables, one of the limitations of the present study is the lack of measurement of oxidative stress variables by ELISA and western blot. Thus, it is recommended that antioxidant markers be evaluated in different ways in future studies. As nandrolone use and resistance training result in pathological, physiologic, and systolic and diastolic blood pressure changes, this research appears to lack investigation of these factors. Consequently, evaluating these factors is recommended through further studies.

## Conclusion

Based on the results, taking nandrolone with and without resistance training increased heart disease risk indicators. However, the risk of heart disease in training and abusing nandrolone was less than the normal condition of this steroid without training.

## Conflict of Interests

The authors declare no conflict of interests.

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