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RESEARCH ARTICLE

Preventive Role of Omega-3 Fatty Acid on the Some Biomarkers in Heart Aging Induced by Fructose on the Male Rate

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Abstract: Investigating the potential protective effects of food supplements including powdered omega-3 fatty acids against heart degeneration is the goal of the present research. 20 rabbit male had been similarly and arbitrarily assigned to four categories: regular as the control category, fifteen hundred milligram BW S/C every day about fructose actos just like the GII, 500 milligrams per kilogram body weight by swallowing of omega-3 fatty acid supplements as the GIII, and 150 mg per kilogram of BW every day S/C consumption about the fructose actos with five hundred milligrams per kilogram BW by mouth of omega-3 fatty acid as the GIV category for the duration of four weeks. According according to the investigation's findings, the group of individuals that got omega-3 fatty acids had much higher levels of glutathione as well as nitric oxide, and significantly lower levels of cardiac troponin I and per oxynitrate than the aging category GII. In conclusion, our findings show that the feed addition of powdered omega-3 fatty acids had a protective effect towards cardiac ageing.

Keywords: omega-3 fatty acid, fructose actos, heart aging, male rabbits

INTRODUCTION

Dietary abnormalities in metabolism are thought to be a significant risk factor for developing any number serious cardiovascular illnesses, which continue to be the worldwide leading contributing factor to death and disability [1]. Because fructose contributes to the development of metabolic syndrome, diabetes, insulin resistance, and cardiac disorders, it has drawn a lot of interest within nutritional sugars [2]. In research on animals, excessive consumption of fructose was demonstrated to hasten myocardial degeneration by causing damage to mitochondria, inflammation, and changes in heart functioning and structure [3,4].

Increasing alterations in cardiovascular structure and functioning, which involve enlargement, inflammation, and decreased contractility are hallmarks of maturing for the circulatory system and are made worse by biochemical assaults especially consuming too much fructose [5]. Furthermore, elevated indicators like troponin I and peroxynitrite, decreased nitric oxide bioavailability, and enhanced damage from oxidation are linked to fructose-associated heart injury [6].

It was recently suggested that nutrition therapies including bioactive substances could be used to combat dietary-induced heart degeneration. Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), two of those omega-3 polymorphic fatty acids (PUFAs), are especially important for protecting the cardiovascular system [7]. Omega-3 omega-6 fatty acids have anti-inflammatory, antioxidant, anti-rhythmic, and prevent hypertrophic qualities, among additional beneficial attributes [8,9].It is currently demonstrated that taking omega-3 fatty acid supplements enhances the activity of antioxidants, decreases inflammation, and improves cardiac performance in laboratory animals [10].

MATERIAL AND METHOD

The experiment protocol

Rats were split into 4 identical and randomized categories: regular as the untreated category, GroupI, ani 20 male rats in good condition were put within the veterinary medical college's animals facility at Kerbala University. Males were administered twenty g/kg BW of glucose in water to consume every day as Group II, whereas the rats had been given four hundred milligrams per kilogram BW of omega-3 fatty acid orally as GIII during the same period. The previously Group IV category was given twenty g/kg BW in gastrointestinal water daily of fructose along with four hundred mg/kg body weight through oral ingestion of omega-3 fatty acid to stay a period of 4 weeks. A blood sample had been collected at the conclusion of the research using the technique described by ().

Assessment of serum cardiac troponin I (ng/ml) An instrument made in Guangzhou, the ruling People's Republic of China, was used for determining cardiovascular troponin I (cTnI). [11]
A serum estimate of nitric oxide (NO) M/L The nitrogen oxide (NO) per oxynitrate (ONOO) m/L estimate has been calculated using the [12]methodology. Peroxynitrate was measured employing [13]technique. estimate of blood lower levels of glutathione (mg/dl) The Ellmans reagent method, which was first employed by the researchers [13]was used to measure the amount about glutathione in blood.

RESULTS

The mean values of serum Cardiac troponin 1 at the end of the experiment were (0.00 ± 0.00C, 1.03±0.02A, 0.00 ±0.00C, 0.33±0.03B) for groups Control, fructose , *omega-3 fatty acid* and *omega-3 fatty acid* +fructose respectively(LSD =0.034) as showen in Figure (1), show a significant increases (p≤0.05) in GII group when compared with GI, GIII and GIV groups. On the other hand the main value of serum Cardiac troponin 1 show a significant decrease (p≤0.05) in GIV group when compared with GI, GIII and GIV groups. While there is no significant (p≥0.05) differences between GI and GIII groups.

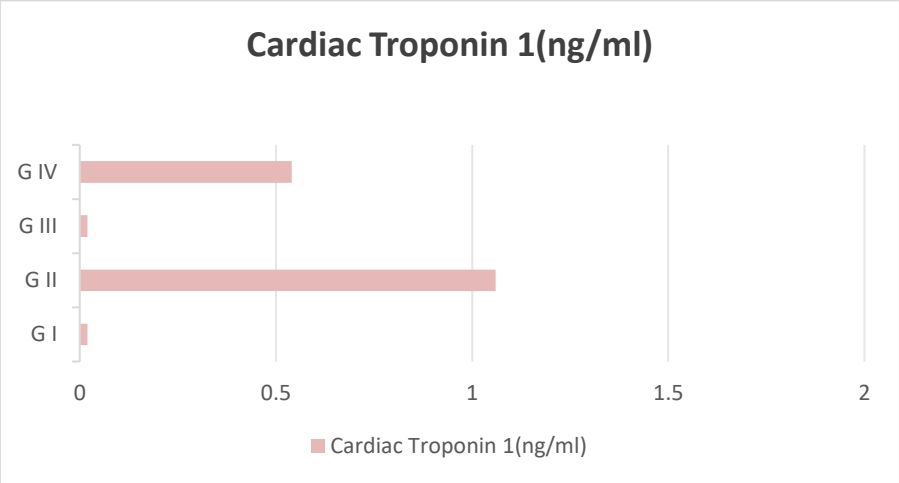

Figure (1) Effect of daily oral intubation of *omega-3 fatty acid* for 4 weeks on serum Cardiac Troponin 1(ng/ml) concentration of fructose actos treated male rabbits

The result in figure (1) showed a significant increase in Cardiac troponin I(cTnI) in GII received fructose as comparing with other groups

According illustrated in Figure (2), the average serum nitric oxide levels at the conclusion about the trial significantly 30.36 ±0.06B, 24.80±0.18C, 43.82 ±1.00A, and 29.79±0.12B for the Control, fructose, omega-3 fatty acid, and omega-3 fatty acid + fructose groups, respectively (LSD= 2.316). Comparing the GII category to the GI, GIII, and GIV groups, the primary value revealed a substantial reduction. In addition, the GIII category's serum nitric oxide primary value was noticeably higher than the GII comparison group's. Although the GI and GIV groups are not separated significantly.

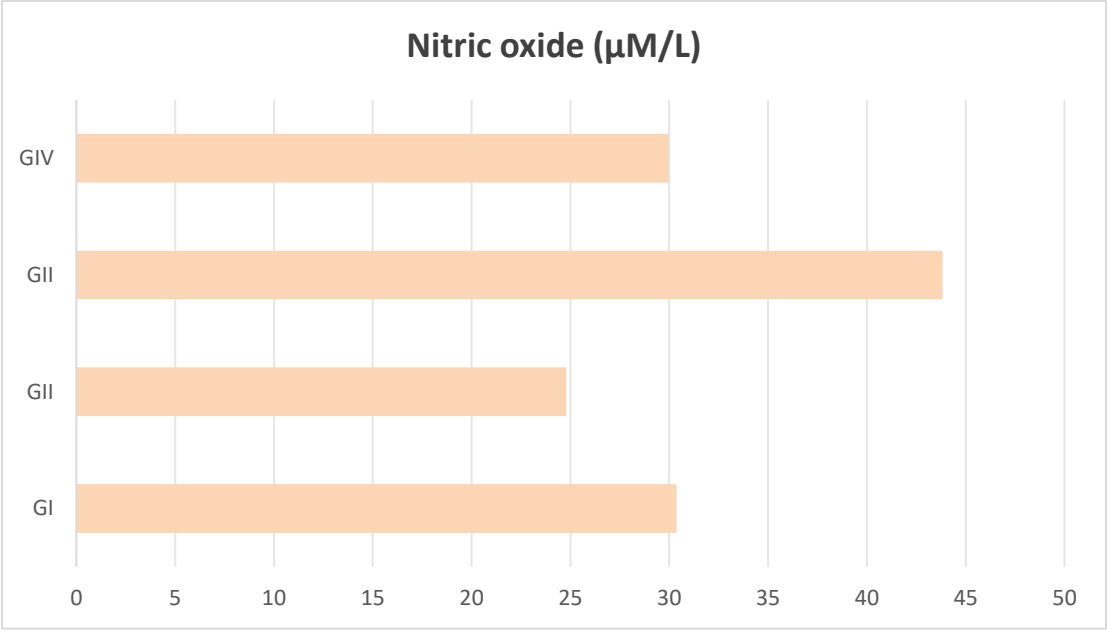

Figure (2) Effect of daily oral intubation of *omega-3 fatty acid* for 4 weeks on serum Nitric oxide(µM/l) concentration of fructose actos treated male rabbits Within the completion of the investigation, the average serum ONOO measurements for the Control, fructose, omega-3 fatty acid, and omega-3 fatty acid + fructose categories were 4.05 ± 0.01B, 7.31± 0.14A, 3.51 ± 0.15C, and 3.85± 0.15B, consequently (LSD = 0.583). Figure (3) shows that the GII category had a significantly

higher serum ONOO than the GI, GIII, along with GIV categories. In addition, the GIII sample's serum per oxynitrate of significant amount was much lower than that of the GII category. Although the GI and GIV groups do not differ significantly.

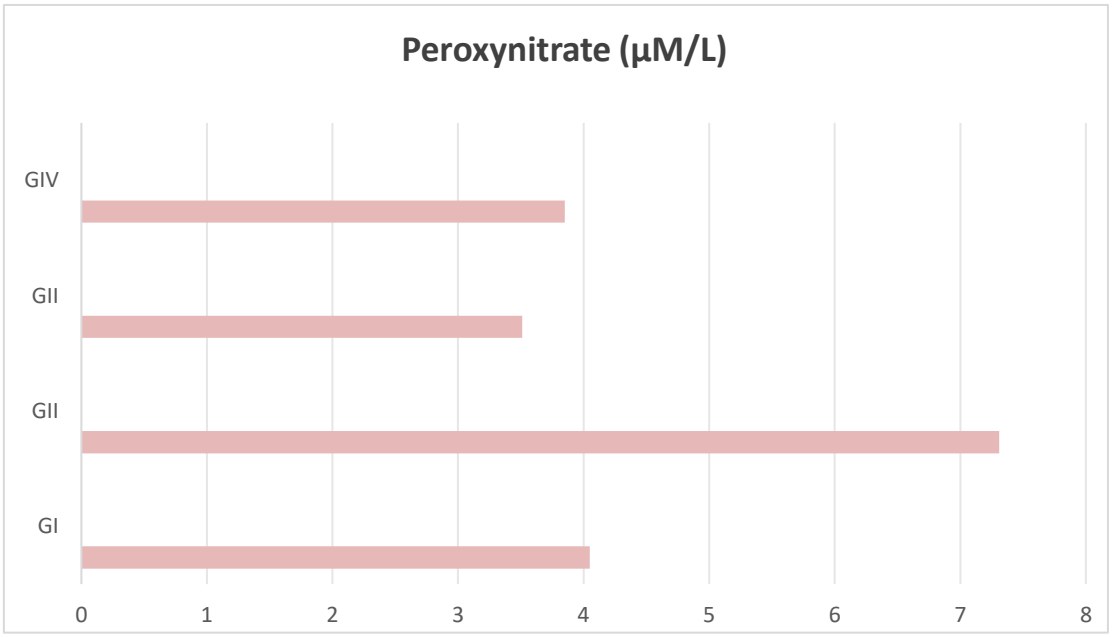

Figure (3) Effect of daily oral intubation of *omega-3 fatty acid* for 4 weeks on serum Peroxynitrate (µM/l) concentration of fructose actos treated male rabbits

The investigation's average serum GSH amounts were 9.85 ± 0.055BC, 5.81±0.14C, 17.44 ±0.13A, as well as 13.64±0.13AB within the Control, fructose, omega-3 fatty acid, and omega-3 fatty acid + fructose groups, accordingly (LSD=0.551), as shown in Figure (4). The GII group's average serum GSH levels had been significantly lower than those of the GI, GIII, and GIV categories. In addition, the GIII group's primary serum GSH value was substantially higher than the GII group's.

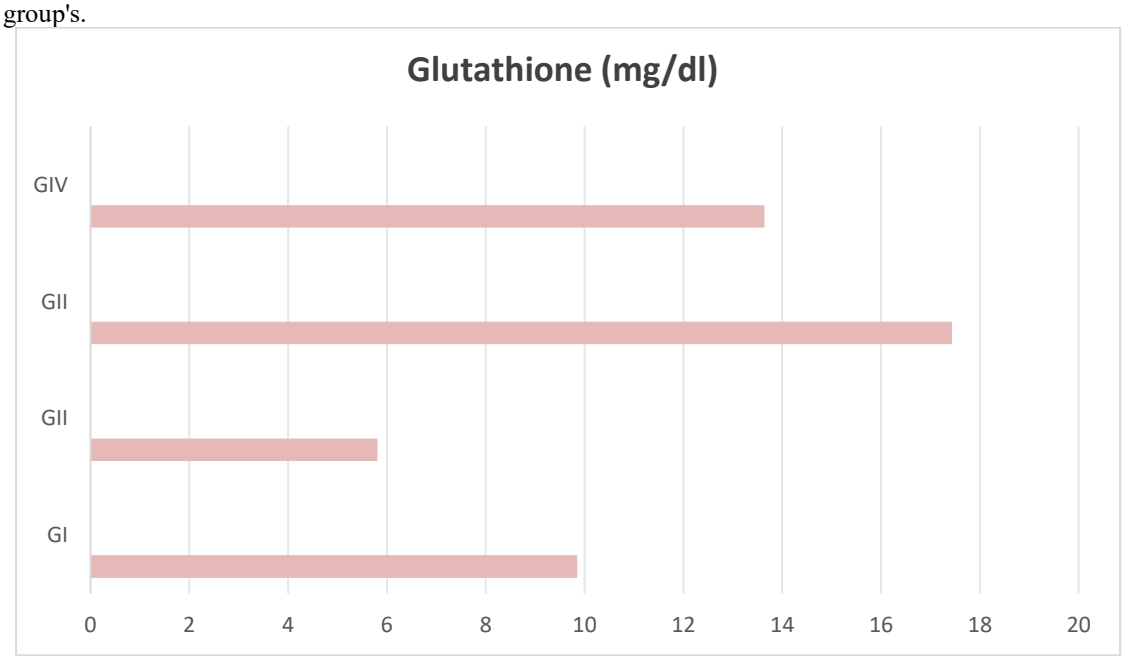

Figure (4) Effect of daily oral intubation of *omega-3 fatty acid* for 4 weeks on serum Glutathione (mg/dl) concentration of fructose actos treated male rabbits

The investigation's mean total body weight (gm/kg) was 1285.83±15.62 C, 1715.00±42.32 A, 1095.33±31.14 D, and 1415.00±12.31 B) for the controls, fructose, omega-3 fatty acid, and omega-3 fatty acid + fructose categories, accordingly

(LSD=0.551), as shown in Figure (5). The GII grouping had a significantly higher body weights than the GI, GIII, and GIV categories. Along with a notable drop in the GIII group's primary body weight value relative to the GII group

Figure (5) Effect of daily oral intubation of *omega-3 fatty acid* for 4 weeks on Body weight(gm/kg) concentration of fructose actos treated male rabbits

The mean values of heart weight(gm/kg), at the end of the experiment were(8.63±0.06 B ,12.43±0.32 A ,7.50±0.13 C ,8.69±0.10 B)for groups Control, fructose , *omega-3 fatty acid* and *omega-3 fatty acid* +fructose respectively(LSD=0.551) as showen in Figure (6), show a significant increase in GII group when compared with GI, GIII and GIV groups. Beside a significantly decreases in the main value of heart weight in GIII group when compared with GII group.

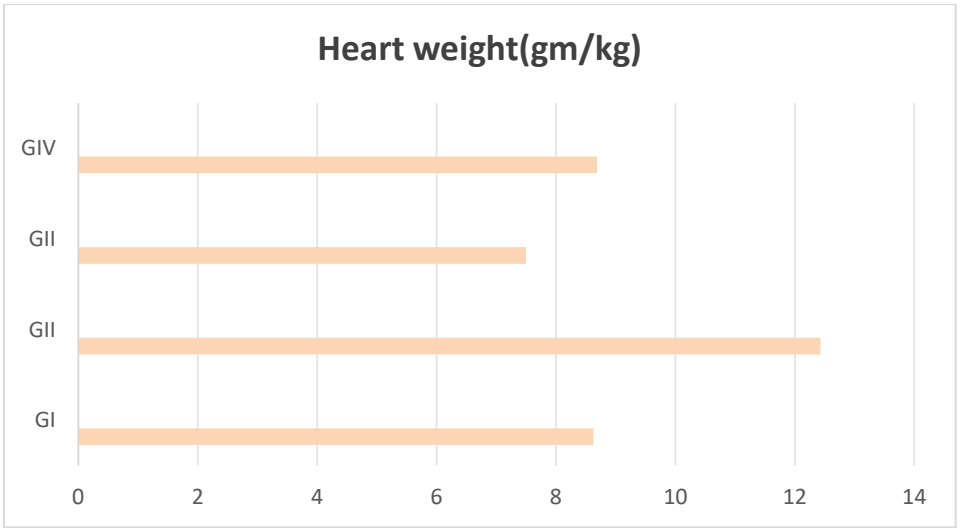

Figure (6) Effect of daily oral intubation of *omega-3 fatty acid* for 4 weeks on heart weight(gm/kg) concentration of fructose actos treated male rabbits

DISCUSSION

Currently, all kinds of proteins usually automatically employed as guidelines-advocated indicators for resource utilization with within the likelihood of an acute heart attack, and heart troponin was suggested as a sign of cardio mobileular death. The identification of cTnI was the bloodstream of rats ingesting fructose acting is something that was not previously investigated [14].The increased cTnI could be explained by the properties of cTnI, which is a small-molecule peptide component of the cardiac muscular myocardial mechanical equipment [15]. Rising plasma cTnI indices in fructose-injected rats

have been linked to significant increases in plasma CK-mitochondrial function, which could have been associated with the the amount of biological indicators of heart disease which circulate through the bloodstream after cells in the myocardium have been injured [16,17]. Elevated cTnI amounts may indicate the possibility of ischemia of the myocardium in stress-stressed individuals cardiovascular loss of cells and following coronary artery disease. The cardiac heart's membranes becomes transparent or bursts whenever exposed to insufficient oxygen or nutrition, allowing cytosolic digestive enzymes to enter the bloodstream and raise serum concentrations [18].

Organisation GIII saw a significant decrease in cardiac troponin I as a result of the fatty acid omega-3 treatment. Results of this investigation correspond by those of additional investigations [19]. In mice treated with omega-3 fatty acids, the cTnI phases returned to normal after blocking this reaction while significantly reducing the various phases of cTnI produced by fructose action. The presence of antioxidants like beta-carotene as well as cytocyanin in omega-3 fats may be the cause of such. The results of the present study support the idea that omega-3 fatty acids may safeguard the myocytes in the heart against Dgalactose-induced damage from oxidation [20,21,22]. Within along with increasing animal health, the elimination about unsecured oxygen radicals by the omega-three fat acid's organic antioxidants and antimicrobial enzymes is responsible for successfully removing unsecured radical-induced cellular damage, enhancing immune system function, along with preventing diseases. Omega-3 fatty acids have consequently being heralded as a superfoods that may avoid malignancies, delay age-related changes, fight viruses, and solve a host of health problems [23].

Those who consumed fructose experienced a significant decrease in NO compared to the manipulation groups. The results from the investigation are consistent those of [24]. Endothelial cells in the artery walls generated naturally occurring nitrogen oxide (NO) through the stimulation of epithelial NO synthesized (eNOs) as well as the expulsion of NO compared to NO donating compounds through the manipulation through the sGC-cGMP-PKG process (soluble guanylyl cycle, cyclic GMP, as well as the protein kinase G), which is followed by a stimulation of blood vessel dilation. Its cellular calcium level is higher in vascular cells which connect to calmodulin to initiate eNOS. Through a decrease in [Ca] in vascular clean muscle cells, it can induce an increase in [Ca] in endothelial cells. [25]. Decreased NO levels could possibly contribute to damage to tissues caused by fructose considering NO was a necessary endothelial-produced vasodilation as well as . Significantly lower nitrogen oxide (NO) production is typically indicative of increased antioxidant pressure, and this may also reduce NO's cardiovascular protective properties. When compared to the control category, GIII that received omega-3 fatty acid showed a significant increase in NO. The results of this investigation correspond that the results from other research [26].The endothelium's basal production and release of nitric oxide and the cyclooxygenase-structured vascular narrowing substance prostanoid, were both increased under laboratory conditions by omega-3 fatty acid extracts.progressively, S. platensis's compounds, such as the formation of can increase the production of nitric oxide from endothelial cells synthesis, which will boost the absorption and utilization of nitric oxide [27].

A recent investigation revealed daily omega-3 fatty acid supplementation substantially altered blood cardiac troponin I (cTnI) levels in male rats exposed to stress to metabolism caused by fructose. One well-known indicator of stress on the heart and harm to the myocardium is increased cTnI. Blood cTnI increased in the present investigation after fructose was administered, suggesting preclinical heart injury could have been brought on by dysfunction of the endothelial system, high cholesterol levels, and a state of oxidative stress. It is well recognized that omega-3 fatty acids, especially DHA (docosahexaenoic acid along with the acid eicosapentaenoic (EPA), have cardiovascular protection, anti-inflammatory, and antioxidant qualities. Antioxidant addition reduced the rise in cTnI, indicating that cardiac tissue was protected. The result is consistent with earlier research in animal models involving heart illness, which showed that taking omega-3 fatty acids increased the metabolism of lipids, strengthened the defenses against antioxidants, and decreased necrosis of the myocardium. By stabilizing cardiac structures, modifying ions, which are and inhibiting inflammatory substances, the fatty acids omega-3 may be included in the walls of cells as part of the cardioprotective process. Furthermore, omega-3 fatty acids are believed to enhance the health of endothelial cells and lessen damage caused by oxidation, which may account for the investigation's finding of a decrease in heart disease indicators.

Our findings correspond to conformity to past research showing the cardiovascular benefits of omega-3 fatty acids in rats that were fed a fructose-rich dietary or had coronary artery disease caused by isoproterenol. nevertheless, the level of cardio protection may vary depending from the amount administered, period, particular animal breed. To validate these results, more research with bigger sample sizes and more biomarkers relevant to (CK-MB, LDH, etc.) is advised. For contradiction to the untreated category, this research showed that giving male rats fructose resulted in a considerable decrease in weight for the animals. This result coincides with line with other research showing which a diet packed with fructose interferes with energy metabolism, causing changes in the oxidation of fats and carbohydrates which may hinder typical development and obesity[28,29].

Conversely, supplementation with omega-3 fatty acids significantly improved body weight compared with the fructose-only group. Omega-3 fatty acids are known to exert beneficial metabolic effects by enhancing insulin sensitivity, modulating lipid metabolism, and reducing oxidative stress[30]. The improvement in body weight observed in omega-3 treated groups may be attributed to the ability of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) to counteract the metabolic disturbances induced by fructose Interestingly, the combination of fructose and omega-3 fatty acids showed a partial improvement in body weight compared with the fructose-only group, but values remained lower than the control. This suggests that while omega-3 fatty acids mitigate some of the deleterious effects of fructose, they may not fully reverse fructose-induced metabolic imbalance

Our findings agree with previous studies in male rats showing that dietary omega-3 fatty acids improve metabolic health and body weight regulation under conditions of high-sugar or high-fat diets[31,32]. The present study revealed significant alterations in relative heart weight in male rats following different dietary treatments. Rats administered fructose alone exhibited an increase in heart weight compared with the control group. This observation aligns with previous studies indicating that high fructose intake induces cardiac hypertrophy and remodeling due to metabolic disturbances, increased lipogenesis, and oxidative stress [33,34]. Fructose feeding is also associated with hyperinsulinemia and hypertriglyceridemia, which promote structural and functional changes in cardiac tissue [35]

On the other hand, omega-3 fatty acid supplementation significantly reduced the adverse impact of fructose on heart weight. Omega-3 fatty acids, particularly EPA and DHA, are known to exert cardioprotective effects by modulating inflammatory pathways, improving lipid metabolism, and reducing cardiac hypertrophy[36,37]. Their role in enhancing mitochondrial function and attenuating oxidative stress may explain the normalization of heart weight in omega-3 treated groups.

These results are consistent with earlier findings in male rats, where omega-3 fatty acids were shown to suppress myocardial hypertrophy, improve cardiac contractility, and protect against diet-induced cardiometabolic syndrome [38,39].

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