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Original Research Article

Restoration of Atherosclerosis Plaque Formation in Wistar Rat through the Combination of Olive Oil and Gallic Acid

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Abstract: The aim of the current study was investigating the antiatherogenic effects of olive oil and gallic acid as a combination. Forty male atherogenic rats were arbitrary divided into five groups (n=8), 1 was given 1ml distilled water as control group (G1), while the groups G2, G3, were drenched 1ml of olive oil and 100mg/kg.bw of Gallic acid respectively. As well as G4 was given 40mg/kg.bw of Atorvastatin and G5 used as a combination group drenched with the same dose of G2 and G3 together for 30 consecutive days. The higher body weight of experimental study rats at zero time was (162±4.05g) on average in gallic acid group. All the atherosclerosis induced rats demonstrated suppression of increase in body weight as compared to the control positive group which reported the high body weight (329±5.09g). The significant ($P < 0.05$) suppressions of body weight gain in atherogenic rats treated with Atorvastatin was observed at the end of the study as compared to the body weight of those in G1, G2, G3 and G5 respectively. Moreover, the results of the Nitric oxide (NO) revealed there was a significant difference ($P<0.05$) in all experimental groups after treatment. The highest means were shown in G4 (41.61 µmol/L) and G5(33.50 µmol/L) with non-significant difference between them, followed by G3 (23.92 µmol/L) which was significantly ($P<0.05$) lower than G4 and G5, but significantly ($P<0.05$) respectively. In the same line the histopathological section Aorta of control positive rats showed moderate to severe atherosclerosis was seen in the affected aorta wall, with three plaques with thickening of aorta wall. Alos, the aggregation of Atheroemboli that occupied 35% of lumen area of the affected aorta. While there was mild atherosclerosis was seen in the affected aorta wall. Note two mild atherosclerosis plaques, were observed as abnormal thickening in the affected aorta wall of Atorvastatin and combination group respectively. In conclusion, the results indicate that Olive oil and Gallic acid showed potential antiatherogenic effects but may require additional exploration to maximize their therapeutic benefits.

Keywords: Gallic Acid, Atherosclerosis, Atorvastatin, Nitric Oxide.

INTRODUCTION

The leading cause of death and disability in modern medicine and healthcare is atherosclerosis and atherosclerotic disorders, which in turn cause myocardial infarction, stroke, and sudden death. A long asymptomatic phase occurs during the development of atherosclerotic lesions (Jebari-Benslaiman *et al.*, 2022). In most cases, the arterial lumen is significantly narrowed by the time the first clinical signs of atherosclerosis emerge, which means that atherosclerosis is the main degenerative mechanism at work (Fan & Watanabe, 2022). Pathological changes to the artery wall layers, known as atherosclerosis, start developing in childhood and go unnoticed for many years until they reach a more advanced level (Bonafiglia *et al.*, 2022). Inflammation and early lipid buildup in the arterial wall are hallmarks of several animal models of atherosclerosis. In both animals and humans, the first lesions of atherosclerosis are populated by blood leukocytes, which play a role in host defenses and inflammation (Soehnlein & Libby, 2021). The processes behind this recruitment of leukocytes have been greatly illuminated by the fundamental science of inflammatory biology as it pertains to atherosclerosis (Ajoolabady *et*

al., 2024). In addition to improving patients' inflammatory and lipidemic profiles, the polyphenols in Olive Oil (OO) offer antioxidant capabilities via lowering free radicals. The polyphenols in olive oil have an antioxidant effect and can effectively neutralize free radicals, which are responsible for the oxidation of LDL cholesterol. Additionally, the nutritional and sensory qualities of OO are excellent. Consumption of extra virgin olive oil has been linked to the primary or secondary prevention of cardiovascular illnesses, according to recent studies. Still up in the air, though, is whether a lower dosage of OO with a greater concentration of polyphenols or a larger dosage of OO with a lower concentration is better (Kourek *et al*., 2024). Gallic acid, a trihydroxybenzoic acid, is found in certain plants, such as tea leaves. Gallic acid lessens the impact of transverse aortic constriction (TAC) stimuli on cardiac hypertrophy, dysfunction, and fibrosis in living organisms and on transforming growth factor β1 (TGF-β1) in laboratory settings. The left ventricle's end-diastolic and end-systolic dimensions are reduced, and the reduced fractional shortening in TAC is regained (Jin *et al*., 2018). It also lowers the amounts of skeletal α-actin, β-myosin heavy chain, brain natriuretic peptide, atrial natriuretic peptide, and skeletal α-actin. The results of Trichrome II Blue staining reveal that the administration of gallic acid decreases perivascular fibrosis, as well as the expression of connective tissue growth factor and collagen type I (Okafor *et al*., 2024). In this study Here, we report the effect of gallic acid on cardiac dysfunction and fibrosis in a rat model of atherosclerosis-induced heart failure and in primary rat cardiac fibroblasts and compare the effects of gallic acid with those of olive oils and Atorvastatin.

MATERIALS AND METHODS

Chemicals:

Olive oil purchased from the local market at Babylon city, as well as Gallic acid obtained from sigma Aldrich (USA), cholesterol, Hydrogen peroxide and Triton.

Experimental Animals and Management

The current investigation made use of forty male albino rats, all of which were at least two months old and weighed between 150 and 180 grams, procured from the animal house of the College of Veterinary Medicine/University of Al-Qasim. The rats were given a regular pellet meal and were given water from the faucet. The animals were housed in the animal home of the College of Veterinary Medicine, University of Al-Qasim, and kept under regular settings for two weeks to help them adjust. These conditions included a 12 / 12-hour light-dark cycle, temperatures between 20-25 °C, and air conditioning. The mulch in the bed was changed twice a week.

Induction of Atherosclerosis

40 rats were used to induction of Atherosclerosis by oral administration of hydrogen peroxide 0.5% in drinking water and high cholesterol fed diet (1.50% cholesterol) daily for three weeks (Pashaie *et al*., 2017; Jasim and Hassan, 2019). In addition, a single dose of triton 10mg/kg was administrated intra-peritoneal at the end of the last three weeks of the experiment. After the end of the periods the levels of cholesterol were estimated to ensure the disease was induced.

Experimental Design

After the induction of atherosclerosis, the animals were divided into five groups (n=8), rats divided equally into five treatment groups according to the following:

Group1: 8 rats drenched of (1ml/rat/day) of distilled water as control positive.

Group2: 8 rats drenched of (1ml/rat/day) of Olive Oil daily (Vazquez *et al.*, 2019).

Group3: 8 rats drenched of (1ml/rat/day) of distilled water contain (100mg/kg.bw) of Gallic Acid (Jin *et al.*, 2018).

Group4: 8 rats drenched of (1ml/rat/day) of distilled water contain (40mg/kg.bw) of Atorvastatin (Oh *et al.*, 2021).

Group 5: 8 rats drenched of (1ml/rat/day) of Virgin Olive Oil & (100mg/kg.bw) of Gallic Acid, for 30 days.

Body weight:

The weight of the experimental animals was measured throughout the period of the experiment using an electric scale, the weights were taken every 7 days to determine the weight of each group (Al-Fayadh and Naji, 1989).

Heart Biomarker (Nitric Oxide):

We used an ELISA kit to measure Nitric oxide levels, following the manufacturer's instructions. It was tested the absorbance at 450 nm using a microplate ELISA (Aboktifa *et al*., 2025).

Histopathology:

A diethyl ether euthanasia chamber was used to put the animals to sleep once their treatments were complete. The College of Veterinary Medicine's Ethics Committee at Al-Qasim Green University approved the present protocol. The sternum bone was cut longitudinally. Then, the histology lab would create slides following the usual protocols outlined by Luna (1968) after isolating the aorta and coronary arteries and fixing them in 10% neutral formalin (Ayad *et al*., 2021).

Statistical Analysis:

In the present study, we used one- and two-way ANOVA, and to find out if there were any significant differences between the group means, we used the least significant differences (LSD) test, which is a statistical method available in SAS (Statistical Analysis System, version 9.1). According to SAS (2010), a p-value less than 0.05 was deemed statistically significant, and the

findings were presented as the mean plus or minus the standard error.

RESULTS AND DISCUSSION

Body Weight

In the present study, experimental rats were fed with high cholesterol and fat diets for atherosclerosis induction, their body weights increased. The higher body weight of experimental study rats at zero time was (162±4.05g) on average in gallic acid group. After the atherosclerosis modeling, the rats were treated with Gallic acid, olive oil and Atorvastatin, and their body weights were monitored. All the atherosclerosis induced rats demonstrated suppression of increase in body weight as compared to the control positive group which reported the high body weight (329±5.09 g) table 1 and (Fig. 1). The significant ($P < 0.05$) suppressions of body weight gain in atherogenic rats treated with Atorvastatin was observed at the end of the study as compared to the body weight of those in G1, G2, G3 and G5 respectively as illustrated in table 1 and (Fig. 1).

Table 1: Body weight gains along of study period

Groups	Week 0	Week4	Week8
G1(control positive)	128±3.44 Bc	267±1.45 Bb	329±5.09 Aa
G2 (olive oil)	162±4.05 Ac	246±3.97 Cb	315±1.58 Ba
G3 (Gallic acid)	130±3.99 Bc	286±5.60 Ab	236±4.74 Ca
G4 (Atorvastatin)	125±2.95 Bc	278±1.69 Ab	224±2.66 Ea
G5 (olive+ Gallic acid)	160±3.65 Ac	238±5.43 Db	231±1.94 Da
LSD	9.22		

Means with a different small letter in the same column are significantly different (P<0.05).
Means with a different capital letter in the same row are significantly different (P<0.05).

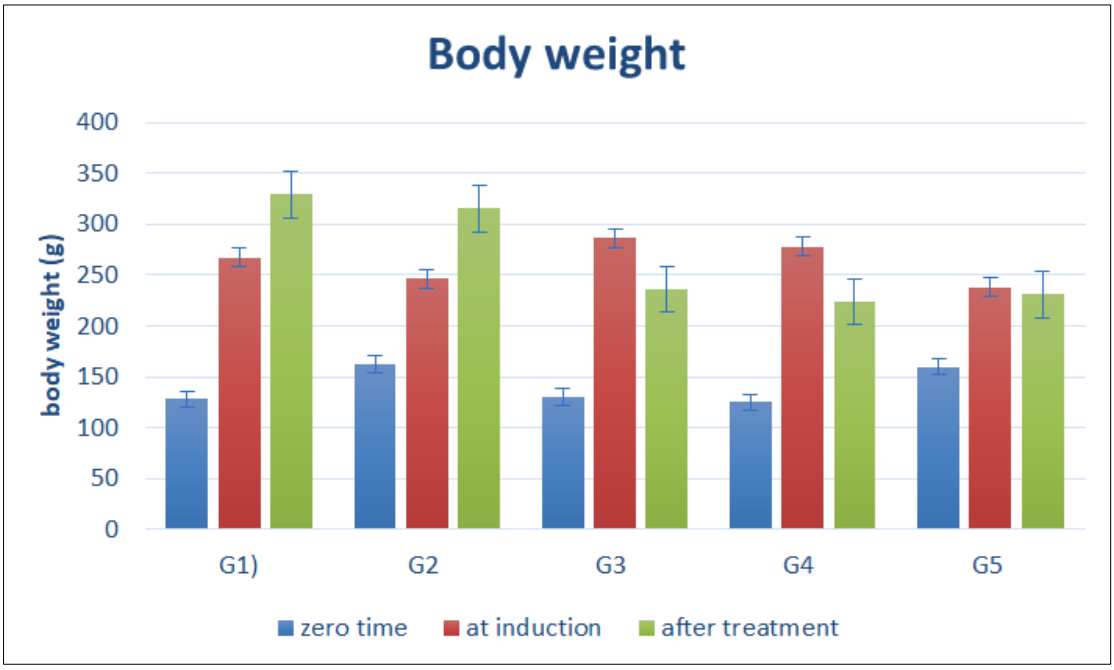

Figure 1: body weight of different treated groups

Nitric Oxide

The results of the Nitric oxide (NO) illustrated in table (2) and figure (2) showed that the levels of nitric oxide at the induction were not significant differences among groups. In contrast, there was a significant difference (P<0.05) in all experimental groups after treatment. The highest means were shown in G4 (41.61 µmol/L) and G5(33.50 µmol/L) with non-significant difference between them, followed by G3 (23.92 µmol/L) which was significantly (P<0.05) lower than G4 and G5, but significantly (P<0.05) respectively, Concerning the differences between NO after 1 and 2 months, results obtained that the means of NO in groups G3, G4 and G5 increased significantly (P<0.05) after treatment with (gallic acid, olive oil, and Atorvastatin) as compared with the corresponding means at atherosclerosis induction (control positive).

Table 2: The mean±SE of Serum nitric oxide levels (µmol/L)

Groups	Ni. after induction (1month)	Ni. after treatment (2 months)
G1(control positive)	11.02±1.22 Aa	13.60±0.87Da
G2 (olive oil)	13.96±2.07 Aa	21.57±4.60CDa
G3 (Gallic acid)	10.43±0.98 Ab	23.92±3.89Ca
G4 (Atorvastatin)	12.06±2.13 Ab	41.61±6.80Aa
G5 (olive+ Gallic acid)	13.32±1.99 Ab	33.50±5.13Aa
LSD	8.41	

Means with a different small letter in the same column are significantly different (P<0.05).
Means with a different capital letter in the same row are significantly different (P<0.05).

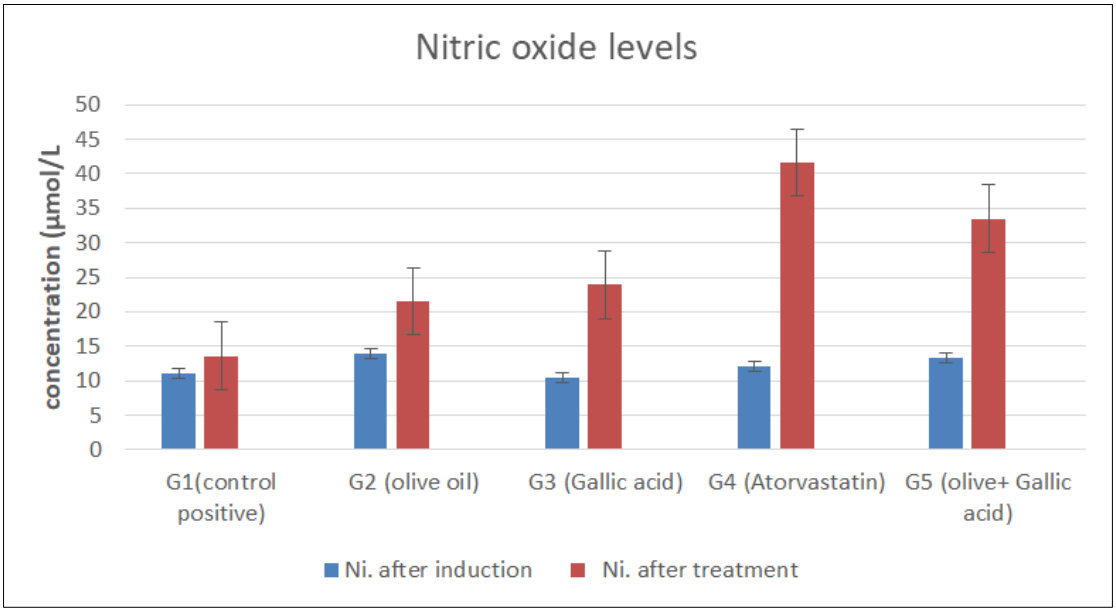

Figure 2: Serum levels of nitric oxide in different treated groups

Histopathological Study of Aorta:

Negative Control:

Normal histological architecture of aorta wall. Note tunica intima (black arrow), tunica media (yellow arrow) and tunica adventitia (blue arrow) (fig3A).

Positive Control:

Moderate to severe atherosclerosis was seen in the affected aorta wall, with three plaques (black arrow) noted. Note the thickening of aorta wall with presence of collagen fiber elastic fibers in the affected area. Alos, aggregation of Atheroemboli (involved cholesterol crystal or necrotic cells debris) or fibrin particles (yellow arrow) that occupied 35% of lumen area of the affected aorta (fig3B).

Atorvastatin:

Mild atherosclerosis was seen in the affected aorta wall. Note one atherosclerosis plaque (black arrow), was observed in the affected area (fig3C).

Gallic Acid:

Mild to moderate atherosclerosis was seen in the affected aorta wall, where one markable atherosclerosis plaque (black arrow), and two thickening areas (yellow arrow) were observed in the affected aorta wall. Also, aggregations of atheroemboli or fibrin particles (red arrow) were observed in lumen of affected aorta. However, these aggregations of atheroemboli or fibrin occupied about 10% of aorta lumen (fig3 D).

Olive Oil:

Moderate atherosclerosis was seen in the affected aorta wall, with three plaques (black arrow) noted. Note the thickening of aorta wall with presence of collagen fiber elastic fibers in the affected area. However, a few aggregations of atheroemboli or fibrin particles (yellow arrow) compared with control positive group were noted in the lumen of the affected aorta (fig3E).

Gallic+Olive (Combination):

Mild atherosclerosis was seen in the affected aorta wall. Note two mild atherosclerosis plaques (black arrow), were observed as abnormal thickening in the affected aorta wall (fig3F). All slides were stained with H&E 40x.

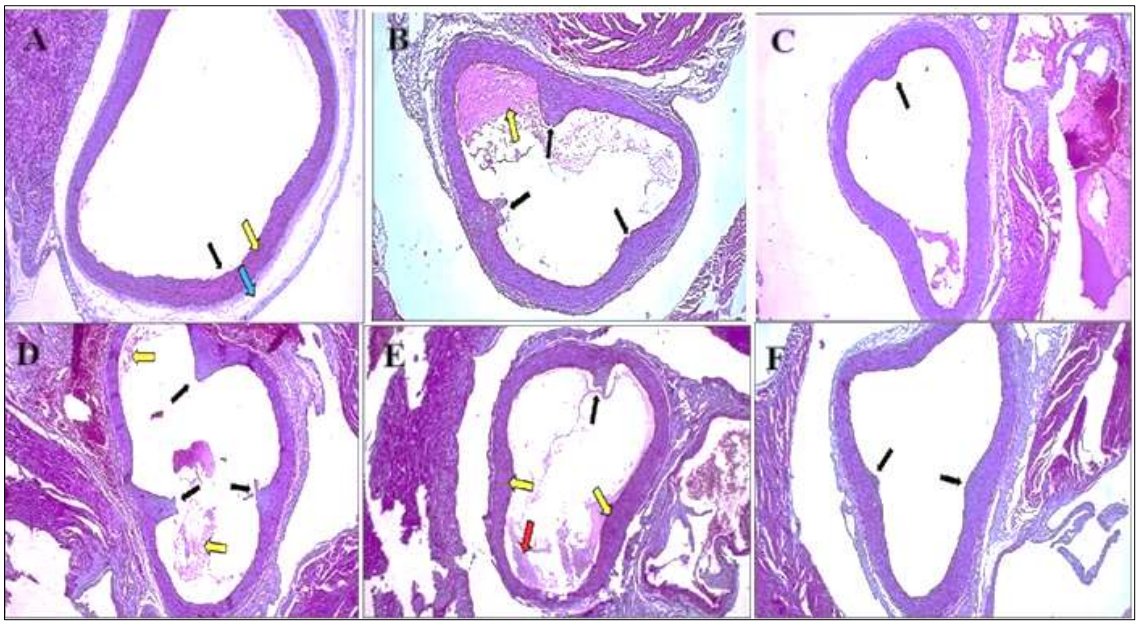

Figure 3: Photomicrograph of Aorta of rat in different groups, A: control negative, B: control positive, C: Atorvastatin group, D: Gallic acid group, E: olive oil group, F: combination group. All slides were stained with H&E 40x

DISCUSSION

The current findings show that atherosclerosis was produced in rats by feeding them a high-fat diet and cholesterol while simultaneously administering oral hydrogen peroxide every day for a month. In addition to developing atheromatous lesions in their arteries, the experimental animals showed high plasma levels, a low serum nitric oxide content, and a high body mass index. Supporting the current findings, previous research demonstrated that subjecting albino rats to a high-fat diet elevated atherogenic indices and caused arterial endothelial dysfunction in the isolated aorta of rats fed an atherogenic diet (Nakagawa *et al*., 2009; Kubra *et al*., 2025). According to Bello *et al*., (2016), there is substantial evidence linking obesity to an increased risk of atherosclerosis and cardiovascular illnesses. Accordingly, atherosclerosis may be affected by measures taken to reduce weight gain (Ayad *et al*., 2021). We found a positive association between increasing body weight and the production of atherosclerosis, which was statistically significant. Evidence from clinical trials shows that metabolic diseases, an aberrant distribution of adipose tissue, and an elevated risk of morbidity and mortality are associated (Osayande *et al*., 2018; Malik *et al*., 2021). In order to determine the amount of body fat, researchers have looked at anthropometric measures like waist circumference (WC), body mass index (BMI), waist-hip ratio (WHR), and waist-to-height ratio (WHtR). These measurements have been linked to an increased risk of death from cardiovascular diseases (Song *et al*., 2015; Salari *et al*., 2016). An increase in NO, a key component in the endothelium's anti-atherogenesis capabilities, demonstrates the beneficial impact of the (Olive+Gallic acid) combination and gallic acid (Ross, 1993). This study's findings corroborate those of a previous one by Gauthier and colleagues, who found that NO inhibits leukocyte adherence to the endothelium and other critical processes in atherosclerosis progression in vitro (Gauthier *et al.*, 1995). Moreover, NO acts to decrease endothelial permeability, reduces vessel tone, and decreases lipoproteins flux into the vessel wall (Cardona-Sanclemente and Born, 1995). Several studies in vivo and in vitro confirmed the role of NO to inhibit the proliferation of the vascular smooth muscle cell and migration (Garg and Hassid, 1989). In this study, the histopathological examination of the Aorta of atherosclerosis treated rats in control positive group showed moderate atherosclerosis was seen in the affected aorta wall, with three plaques as well as thickening of aorta wall with presence of collagen fiber elastic fibers in the affected area. Alos, aggregation of Atheroemboli (involved cholesterol crystal or necrotic debris) or fibrin particles that occupied 25% of lumen area of the affected aorta (fig1B). However, the animal's treatment with olive oil, gallic acid and combination (olive oil+ gallic) as shown in (fig3, D, E, and F) resulted mild to moderate atherosclerosis with mild thickening of aorta wall and a restoration of the parameters mentioned earlier, which was like that of the standard (Atorvastatin) group (fig3 C). These findings strongly supported by an earlier study reported on medicinal plant “Rheum turkestanicum” belongingto family polygonaceae decreased inflammatory cells and myocardial degeneration (Hosseini *et al*., 2022; Ibrar *et al*., 2025). This study shows that atherosclerotic rats can benefit from a mixture of olive oil and gallic acid in many ways. These include lowering lipid levels, improving reperfusion injury, stabilizing membrane potential, antioxidant activity, and restoring enzyme levels. Olive oil and gallic acid may help manage atheromatous plaque and the cardiovascular problems it causes, according to this study's ameliorative results. Related plants, such as Solanum melongena, have been found to exhibit similar

behaviors (Guimarães *et al*., 2000; Ossamulu, *et al*., 214). Avastin, fbrates, atorvastatin, fuvastatin, and lovastatin are among of the antihyperlipidemia medications that are commonly prescribed for these conditions (Huang *et al*., 2018; Osebhahiemen, and Ehimwenma, 2022). The anti-atherosclerosis benefits of gallic acid and olive oil are due to their bioactive components, which include terpenoids, unsaturated fatty acids, saponins, and flavonoids. These chemicals exert their effects through various molecular pathways (Widmer *et al*., 2013). Flavonoids inhibit HMG-CoA reductase, reduce cholesterol synthesis, and upregulate ABCG5/ABCG8 transporters to enhance cholesterol excretion (Kumar and Pandey, 2019; Gupta *et al*., 2020). Saponins promote bile acid synthesis and reduce lipid levels through binding to intestinal cholesterol (Pai *et al*., 2009). Furthermore, the PPAR- α/γ pathways are activated by the abundant flavonoids and terpenoids in olive oil and gallic acid, leading to an increase in fatty acid oxidation and a decrease in foam cell production, which in turn prevents atherosclerosis (Syamsu, 2024). The promise of olive oil and gallic acid as a cardiovascular therapeutic agent is supported by these mechanisms, which work together to lower lipids, reduce inflammation, and protect endothelial cells (Ayad *et al*., 2021; Antonopoulou and Demopoulos, 2023).

CONCLUSIONS

The results indicate that Olive oil and Gallic acid showed potential antiatherogenic effects but may require additional exploration to maximize their therapeutic benefits. Further studies could explore the mechanisms behind these observations and assess long-term effects on cardiovascular health.

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