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EFFECTS OF ETHANOLIC GARLIC (*Allium sativum*) AND BITTER MELON (*Momordica charantia*) EXTRACTS ON SERUM CHOLESTEROL AND MALONDIALDEHYDE IN HIGH-FAT-DIET RATS

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ABSTRACT

This study compared the effects of ethanolic garlic (Allium sativum) and bitter melon (Momordica charantia) on total cholesterol and serum MDA in high-fat-diet rats. Thirty-five male Rattus norvegicus were randomized into seven groups (n=5): standard-diet control, untreated high-fat-diet control, simvastatin comparator, garlic extract at 300 or 600 mg/kg body weight, and bitter melon extract at 300 or 600 mg/kg body weight. Following 14 days of high-fat-diet induction, treatments were administered orally for 14 days. Total cholesterol levels were measured before and after the intervention (pre-test and post-test), while serum MDA was measured by the thiobarbituric acid-reactive substances method. Nonparametric analyses used Kruskal–Wallis tests followed by pairwise comparisons with Bonferroni adjustment for multiple comparisons. Post-test cholesterol differed among groups (H=15.396; p=0.017), as did cholesterol change (H=15.208; p=0.019); however, the untreated high-fat-diet group had lower post-test cholesterol than the standard-diet and simvastatin groups 40 mg/kg body weight, and none of the extract groups differed significantly from the untreated high-fat-diet group. By contrast, MDA differed markedly (H=21.692; p=0.001). Garlic at 300 and 600 mg/kg and bitter melon at 300 and 600 mg/kg significantly lowered MDA relative to the untreated high-fat-diet group, with the lowest mean in the bitter melon 600 mg/kg group (2.72±1.52 nmol/mL). Both extracts demonstrated antioxidant activity, but the cholesterol findings require verification against the raw laboratory records before firm hypolipidemic conclusions are drawn.

INTRODUCTION

Dyslipidemia and metabolic syndrome are increasingly associated with dietary patterns rich in saturated fat, repeatedly heated oils, refined carbohydrates, and highly processed foods. Sustained exposure to these diets increases hepatic fatty-acid delivery, disrupts lipoprotein metabolism, and promotes metabolic inflammation. Total cholesterol is commonly used as an accessible indicator of altered lipid metabolism, although it does not distinguish among low-density lipoprotein, high-density lipoprotein, and other lipoprotein fractions. In experimental animals, high-fat feeding is therefore often evaluated using both circulating lipid measures and downstream indicators of cellular injury (Widjaja et al., 2020; Jha et al., 2023; St-Amand et al., 2020).

Abnormal cholesterol metabolism is biologically linked to oxidative stress. Excess free fatty acids and cholesterol can increase mitochondrial electron leakage, activate oxidase systems, and facilitate low-density lipoprotein oxidation. The resulting reactive oxygen species (ROS) attack membrane polyunsaturated fatty acids and generate lipid hydroperoxides. If these reactions persist, they amplify endothelial dysfunction, inflammation, and atherogenic processes. Nevertheless, cholesterol concentration and oxidative damage do not always change in parallel because ROS formation is also influenced by antioxidant capacity, inflammatory activity, duration of dietary exposure, and tissue-specific metabolism (Masenga et al., 2023; Rives et al., 2020).

Malondialdehyde (MDA) is a secondary aldehydic product formed during the decomposition of oxidized polyunsaturated fatty acids. It is widely used as an indirect marker of lipid peroxidation because its concentration reflects cumulative oxidative injury to membrane lipids. Measuring total cholesterol together with MDA can provide complementary information: cholesterol describes a component of lipid status, while MDA indicates the oxidative consequences of disturbed lipid metabolism. However, thiobarbituric acid-reactive substances assays require cautious interpretation because other carbonyl compounds may contribute to absorbance (Ito et al., 2019; Tsikas et al., 2023; Valgimigli, 2023).

The body limits oxidative injury through enzymatic antioxidants, including superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx), as well as nonenzymatic molecules. During prolonged metabolic stress, endogenous defenses

may be insufficient, creating interest in plant-derived compounds that can donate electrons or hydrogen atoms, chelate pro-oxidant metals, inhibit lipid oxidation, or activate endogenous antioxidant pathways (Di Meo & Venditti, 2020; Kozlov et al., 2024).

Garlic (*Allium sativum*) contains allicin, diallyl disulfide, diallyl trisulfide, S-allyl cysteine, and other organosulfur compounds. These constituents have been associated with free-radical scavenging, inhibition of lipid oxidation, modulation of antioxidant enzymes, and possible suppression of cholesterol synthesis. Previous studies have shown that ethanolic garlic extract contains phytochemicals with potent free-radical inhibitory activity, supporting its potential as a natural antioxidant. Reviews and clinical syntheses suggest that garlic preparations can improve selected lipid outcomes, but the magnitude of effect varies with preparation, sulfur-compound stability, dose, and duration (Batiha et al., 2020; Borlinghaus et al., 2021; Du et al., 2024; Li et al., 2022; Priska et al., 2020).

Bitter melon (*Momordica charantia*) is rich in charantin, phenolic acids, flavonoids, saponins, triterpenoids, carotenoids, and vitamin C. These compounds may interrupt radical-chain reactions, enhance endogenous antioxidant activity, and influence glucose and lipid metabolism. Experimental evidence supports its potential as a functional food ingredient, but responses differ across cultivars, extraction procedures, doses, and disease models (Gayathry & John, 2022; Zhang et al., 2023).

Previous investigations have usually examined garlic and bitter melon separately and have often focused on either lipid concentrations or general antioxidant capacity. Direct comparison of both extracts at two doses within a single high-fat-diet model, while evaluating total cholesterol and a specific lipid-peroxidation biomarker, remains limited. Therefore, this study compared ethanolic garlic and bitter melon extracts at 300 and 600 mg/kg body weight with standard-diet, untreated high-fat-diet, and simvastatin groups. The primary objective was to evaluate the effects of garlic and bitter melon extracts on lipid peroxidation, as reflected by serum MDA concentrations. At the same time, total cholesterol was assessed as a complementary metabolic outcome.

MATERIALS AND METHODS

Study design, setting, and ethics

A randomized controlled animal experiment was conducted from January to March 2026 at the Experimental Animal Laboratory and Biochemistry Laboratory, Faculty of Medicine, Universitas Wijaya Kusuma Surabaya, Indonesia. The protocol used a post-intervention comparison across seven experimental groups. Ethical approval was granted by the Ethics Committee of the Faculty of Medicine, Universitas Wijaya Kusuma Surabaya (No. 26/SLE/FK/UWKS/2026).

Animals, sample size, and allocation

Thirty-five healthy male white rats (*Rattus norvegicus*) aged 8–12 weeks and weighing approximately 200 g were obtained from the Satwa Sehat Animal Clinical Laboratory, Malang. Animals were eligible when active, free of visible injury or disease, and feeding normally. White rats that refused food, sustained major injury, or died during the experiment were excluded. The sample consisted of five animals per group, based on the Federer experimental formula and an allowance for dropout; small-group animal studies nevertheless require cautious interpretation because biological variability can materially affect statistical power (Zhang & Hartmann, 2023). Animals were assigned by simple random sampling to the seven groups described in Table 1.

Table 1. Experimental groups and interventions

Group	Diet and treatment	n
K−	Standard feed and water; no high-fat-diet or active treatment	5
K+	High-fat-diet; no active treatment	5
P0	High-fat-diet plus simvastatin comparator 40mg/kg body weight/day	5
P1	High-fat-diet plus garlic extract, 300 mg/kg body weight/day	5
P2	High-fat-diet plus garlic extract, 600 mg/kg body weight/day	5
P3	High-fat-diet plus bitter melon extract, 300 mg/kg body weight/day	5
P4	High-fat-diet plus bitter melon extract, 600 mg/kg body weight/day	5

K−, negative control; K+, untreated high-fat-diet control; P0, simvastatin comparator; P1–P4, plant-extract groups. The thesis contains two inconsistent simvastatin doses (40 mg/kg body weight); the dose should be verified before submission.

Preparation of plant extracts

Fresh garlic bulbs (*Allium sativum*) and bitter melon fruits (*Momordica charantia*) were processed separately. The plant materials used in this study were 5 kg of garlic

bulb powder (*Allium sativum*) and 3 kg of bitter melon fruit powder (*Momordica charantia*). The powders were separately macerated using 96% ethanol as the solvent. The resulting filtrates were separated and concentrated using a rotary evaporator (IKA HB 10). The extraction approach was selected to recover polar and moderately non-polar antioxidant constituents, including phenolics, flavonoids, sulfur-containing compounds from garlic, and saponins and triterpenoids from bitter melon.

Housing, high-fat-diet induction, and treatment

Rats were maintained at 22–25 °C under a 12-hour light–dark cycle in ventilated cages with regularly replaced wood-shaving bedding. During a seven-day acclimatization period, animals received standard feed and water ad libitum. Except for K−, all groups were then exposed to a high-fat diet for 14 days, consisting of standard feed supplemented with repeatedly heated cooking oil, providing approximately 40–50% of daily energy from fat. After induction, Oral treatments were administered once daily by gastric gavage for 14 days, with the administration volume adjusted according to the body weight of each rat, ranging from 0.5 to 1.6 mL per rat. Body weight, feeding behavior, activity, and general signs of distress were monitored throughout the intervention.

Blood collection and biochemical measurements

Total cholesterol was recorded before and after the intervention as reported in the study laboratory dataset. The source thesis does not state the cholesterol assay principle, reagent manufacturer, catalog number, or analytical wavelength; these details must be completed from the original laboratory worksheet before submission. At termination, rats were fasted for 12 hours and anesthetized with ketamine (80 mg/kg body weight) and xylazine (10 mg/kg body weight). Blood without a coagulant was collected from the heart. Serum was separated by centrifugation at 3,000 rpm for 10 minutes at 4 °C and stored at −40 °C. Until analysis, Serum MDA was analyzed in duplicate using the thiobarbituric acid-reactive substances (TBARS) method after incubation with thiobarbituric acid at 95 °C for 30 minutes; absorbance was read at 532 nm using a spectrophotometer, and the results were expressed as nmol/mL.

Statistical analysis

Data were processed using the Statistical Package for the Social Sciences (SPSS). Descriptive values are presented as mean±standard deviation, median, minimum, and maximum. Normality was assessed with the Shapiro–Wilk test and variance homogeneity with Levene's test. Because at least one group was non-normally distributed for post-test cholesterol, cholesterol change, and MDA, overall group differences were analyzed with the Kruskal–Wallis test followed by adjusted pairwise comparisons. Pre-test and post-test cholesterol within each group were compared using the Wilcoxon signed-rank test. Statistical significance was set at $p<0.05$.

RESULTS AND DISCUSSION

Total cholesterol outcomes

All 35 animals were included in the analysis. Post-test total cholesterol differed significantly among the seven groups (Kruskal–Wallis H=15.396; df=6; p=0.017). The highest mean was observed in P0 (84.27±19.44), followed by K− (79.36±10.14), whereas K+ had the lowest mean (48.32±8.79). The garlic groups had means of 51.57±29.50 and 53.09±31.21, and the bitter melon groups had means of 54.36±31.83 and 55.45±31.54. Thus, although an overall difference existed, the descriptive direction did not demonstrate the expected elevation in the untreated high-fat-diet group or a consistent cholesterol-lowering pattern in the extract groups.

Table 2. Post-test total cholesterol in the experimental groups

Group	n	Mean±SD	Median	Minimum–maximum
K−	5	79.36±10.14	81.64	62.72–90.00
K+	5	48.32±8.79	48.18	36.36–57.98
P0	5	84.27±19.44	83.08	56.36–109.09
P1	5	51.57±29.50	67.27	0.00–69.09
P2	5	53.09±31.21	59.09	0.00–76.36
P3	5	54.36±31.83	65.45	0.00–78.18
P4	5	55.45±31.54	67.27	0.00–76.36

K−, negative control; K+, untreated high-fat-diet control; P0 = simvastatin comparator 40 mg/kg body weight; P0 = simvastatin group; P1 = garlic extract group at 300 mg/kg body weight; P2 = garlic extract group at 600 mg/kg body weight; P3 = bitter melon extract group at 300 mg/kg body weight; P4 = bitter melon extract group at 600 mg/kg body weight.

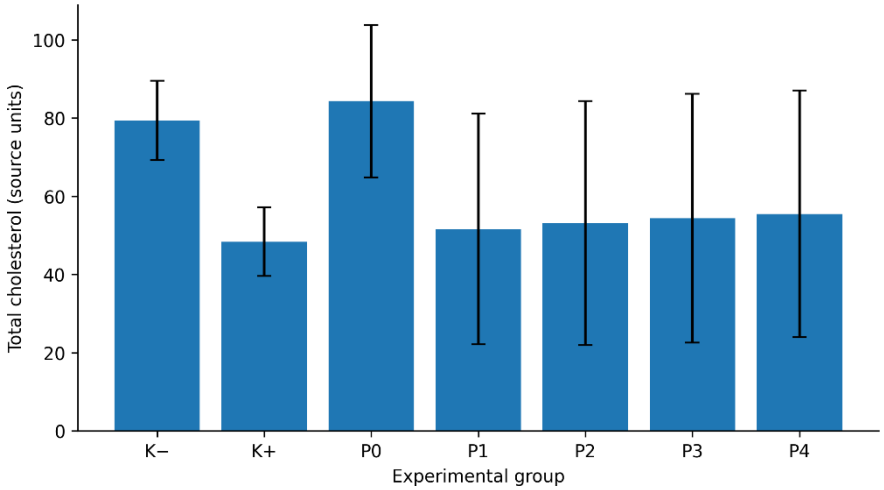

Figure 1. Mean post-test total cholesterol in each experimental group. Error bars represent standard deviations

Pairwise comparisons showed that K+ differed from K− (p=0.003) and P0 (p=0.002), but not from P1 (p=0.379), P2 (p=0.294), P3 (p=0.266), or P4 (p=0.200). P1 differed from K− (p=0.033) and P0 (p=0.031), while P2 differed from K− (p=0.050) and P0 (p=0.046). The overall change in cholesterol also differed among groups (H=15.208; p=0.019). Wilcoxon analysis identified significant pre–post differences in K− and P0 (both p=0.043), but not in K+, P1, P2, P3, or P4.

Table 3. Key statistical results for total cholesterol

Outcome/comparison	Test	Statistic or p-value	Interpretation
Post-test cholesterol: overall	Kruskal–Wallis	H=15.396; p=0.017	Significant
Cholesterol change: overall	Kruskal–Wallis	H=15.208; p=0.019	Significant
K+ vs K− post-test	Pairwise comparison	p=0.003	Significant
K+ vs P0 post-test	Pairwise comparison	p=0.002	Significant
K+ vs P1/P2/P3/P4	Pairwise comparison	p=0.379/0.294/0.266/0.200	Not significant
K− pre–post	Wilcoxon signed-rank	p=0.043	Significant
P0 pre–post	Wilcoxon signed-rank	p=0.043	Significant
K+, P1, P2, P3, P4 pre–post	Wilcoxon signed-rank	p>0.05	Not significant

AUTHOR QUERY: The source thesis contains internal inconsistencies in the cholesterol tables: the K− pre-test mean is incompatible with its reported median and range, and the K−/K+ change values do not arithmetically correspond to the reported pre-test and post-test means. These values must be checked against the raw dataset before journal submission.

Serum malondialdehyde outcomes

Serum MDA showed a clearer treatment pattern. K+ had the highest mean MDA concentration (6.06±1.16 nmol/mL), whereas P4 had the lowest value (2.72±1.52 nmol/mL). Relative to K+, mean MDA was lower by 45.04% in P1, 46.53% in P2,

52.64% in P3, and 55.11% in P4. The simvastatin group showed only an 11.71% descriptive reduction.

Table 4. Serum malondialdehyde (MDA) concentrations and reductions relative to K+

Group	n	Mean±SD (nmol/mL)	Median (minimum– maximum)	Reduction vs K+ (%)
K−	5	4.11±0.80	3.92 (3.44–5.38)	32.17
K+	5	6.06±1.16	6.35 (4.41–7.32)	0.00
P0	5	5.35±0.68	5.38 (4.41–6.35)	11.71
P1	5	3.33±1.91	4.41 (0.00–4.41)	45.04
P2	5	3.24±1.85	3.92 (0.00–4.41)	46.53
P3	5	2.87±1.61	3.44 (0.00–3.93)	52.64
P4	5	2.72±1.52	3.44 (0.00–3.44)	55.11

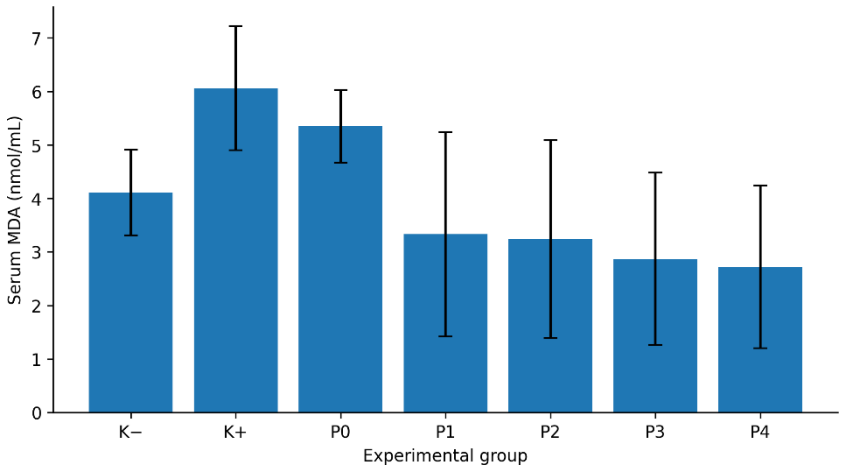

Figure 2. Mean serum malondialdehyde (MDA) concentration in each experimental group. Error bars represent standard deviations.

The Kruskal–Wallis test showed a significant overall difference in MDA ($H=21.692$; $df=6$; $p=0.001$). K+ differed significantly from K− ($p=0.032$), P1 ($p=0.023$), P2 ($p=0.014$), P3 ($p=0.002$), and P4 ($p<0.001$). P2, P3, and P4 were also significantly lower than P0 ($p=0.039$, $p=0.006$, and $p=0.001$, respectively). Differences between the two garlic doses and between the two bitter melon doses were not significant.

Interpretation of the cholesterol response

The cholesterol findings require a more cautious interpretation than the MDA findings. High-fat feeding is expected to increase circulating cholesterol or alter lipoprotein distribution. Still, the untreated high-fat-diet group in this dataset had a lower post-test mean than both the standard-diet and simvastatin groups. Consequently, the significant omnibus and pairwise tests indicate that the groups differed. Still, they do not by themselves establish successful hypercholesterolemia induction or a cholesterol-

lowering effect of the plant extracts. Statistical significance must be interpreted together with the direction, magnitude, baseline balance, and biological plausibility of the observed changes.

Several factors may explain an atypical cholesterol pattern. The induction period was relatively short, and two weeks of a Western or high-fat diet can alter hepatic cholesterol-regulatory pathways before producing a stable increase in circulating total cholesterol (St-Amand et al., 2020). Total cholesterol also aggregates several lipoprotein fractions and may conceal opposing changes in LDL and HDL. In addition, the small group size and large standard deviations in the extract groups indicate substantial biological or analytical variability. The apparent zero values in several groups further warrant verification of assay detection limits, sample labeling, and data entry. Until the raw records are checked, cholesterol should be treated as an exploratory secondary outcome rather than definitive evidence of hypolipidemic efficacy.

High-fat diet, oxidative stress, and MDA

In contrast to total cholesterol, MDA provided a coherent biological pattern. The untreated high-fat-diet group had significantly higher MDA than the standard-diet group, supporting the presence of enhanced lipid peroxidation. High-fat feeding increases substrate delivery to mitochondria and can intensify electron leakage, ROS production, and oxidation of membrane polyunsaturated fatty acids. Decomposition of lipid hydroperoxides generates MDA, which explains why its concentration can rise even when total cholesterol does not show a simple parallel response (Ito et al., 2019; Masenga et al., 2023; Rives et al., 2020; Valgimigli, 2023).

The present study did not perform a direct correlation analysis between cholesterol and MDA. Therefore, it would be inappropriate to infer that a reduction in cholesterol mediated every reduction in MDA. MDA is influenced not only by lipid abundance but also by the degree of fatty-acid unsaturation, inflammatory signaling, endogenous antioxidant activity, and the direct radical-scavenging properties of the interventions. The two outcomes should therefore be interpreted as complementary but mechanistically distinct measures.

Effects of garlic extract

Garlic extract at 300 and 600 mg/kg significantly reduced MDA relative to K+, demonstrating antioxidant activity at both doses. Allicin and related organosulfur compounds can react with oxidants, interrupt radical propagation, reduce LDL oxidation, and modulate cellular antioxidant defenses. These mechanisms are consistent with the observed decrease in lipid-peroxidation products (Batiha et al., 2020; Borlinghaus et al., 2021; Li et al., 2022; Shang et al., 2019). The small numerical difference between the two garlic doses was not statistically significant, suggesting either an early plateau of antioxidant activity or insufficient power to detect a modest dose response.

Although garlic has been associated with improved lipid profiles in meta-analyses and mechanistic reviews, the cholesterol results in this experiment do not independently confirm a hypolipidemic effect (Du et al., 2024; Li et al., 2022). Variability in allicin yield, extraction conditions, storage, and treatment duration may contribute to inconsistent cholesterol responses. Standardization of the extract is therefore essential before its lipid-modifying effects can be compared across studies.

Effects of bitter melon extract

Bitter melon produced the lowest MDA values, with the largest descriptive reduction at 600 mg/kg. Its phenolic compounds, flavonoids, charantin, triterpenoids, carotenoids, and vitamin C may donate electrons or hydrogen atoms, neutralize peroxyl radicals, and support endogenous antioxidant enzymes. These mechanisms provide a plausible explanation for the substantial reduction in MDA (Gayathry & John, 2022; Zhang et al., 2023). Nevertheless, P3 and P4 did not differ significantly, so the data do not establish superiority of 600 mg/kg over 300 mg/kg.

Comparison with simvastatin and clinical implications

The simvastatin comparator did not significantly reduce MDA relative to K+ in this experiment, whereas the higher garlic dose and both bitter melon doses were significantly lower than P0. However, this result should not be interpreted as evidence that the extracts are clinically superior to statins. Simvastatin is primarily prescribed for the inhibition of hepatic cholesterol synthesis. At the same time, its antioxidant effects are indirect. They may depend on dose, exposure duration, and the validity of the induced hyperlipidemic

model (Zhang et al., 2020)—the unresolved simvastatin dose and the unexpected cholesterol pattern further limit pharmacological comparison.

The findings support further investigation of garlic and bitter melon as adjunctive antioxidant candidates rather than replacements for established lipid-lowering therapy. Future studies should use a verified cholesterol assay, longer induction and treatment periods, larger sample sizes, standardized phytochemical profiles, and complete lipid panels including LDL, HDL, and triglycerides. Additional measurements of superoxide dismutase, catalase, glutathione peroxidase, inflammatory markers, and tissue histopathology would clarify whether the reduction in MDA corresponds to broader protection against metabolic and vascular injury (Mohd Rosmi et al., 2021).

Limitations

The principal limitations were the small sample size, substantial within-group variability, the use of total cholesterol without lipoprotein fractions, and the absence of phytochemical standardization. The source thesis also did not specify the cholesterol assay method or unit and contains internal inconsistencies in selected pre-test and change values. These issues do not invalidate the MDA findings, which were internally coherent, but they prevent firm conclusions about cholesterol lowering and must be resolved before the manuscript is submitted.

CONCLUSION

Ethanolic garlic and bitter melon extracts reduced serum MDA in high-fat-diet rats, indicating meaningful radical-scavenging and lipid-peroxidation-inhibiting activity. Bitter melon at 600 mg/kg produced the lowest descriptive MDA value, although no significant dose difference was demonstrated within either plant. Total cholesterol differed among groups, but the unexpected direction of the control and comparator results, together with inconsistencies in the source cholesterol tables, prevented a definitive conclusion that either extract lowered cholesterol. The raw cholesterol dataset, assay method, measurement unit, and simvastatin dose should be verified before submission. After these issues are resolved, the study can provide a useful preclinical basis for evaluating standardized garlic and bitter melon preparations as adjunctive antioxidant interventions.

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