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Protective Effects of *Coriandrum sativum* L. Aerial Parts and Seed Extracts against Aluminum-Induced Hepatic and Biochemical Alterations in Rats: GC-MS Profiling and Antioxidant Evaluation

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ABSTRACT

Aluminum exposure is linked to oxidative stress and liver damage, primarily due to the excessive formation of reactive oxygen species. Accordingly, this study investigated the hepatoprotective and antioxidant effects of methanolic and ethanolic extracts, which were prepared from both the aerial parts and seeds of *Coriandrum sativum* L. in Wistar rats with aluminum-induced hepatotoxicity. The animals were randomly assigned to different experimental groups and received the treatments for 30 days. We evaluated the liver function indicators, lipid profile changes, antioxidant enzyme activities, DPPH radical scavenging ability, and also the expression levels of antioxidant-related genes like *CAT*, *GPX1*, and *SOD2*, within the same experimental protocol. Then, GC-MS analysis revealed several bioactive phytochemicals with reported antioxidant potential. After aluminum administration, the liver enzyme levels increased significantly, and the oxidative stress markers were significantly elevated, while antioxidant gene expression decreased significantly. Treatment with *Coriandrum sativum* L. extracts improved the biochemical and antioxidant parameters significantly compared to the aluminum-treated group ($P<0.05$), particularly in the methanolic seed extracts. These results suggest that *Coriandrum sativum* L. has antioxidant and hepatoprotective properties, which may help attenuate aluminum induced liver injury.

Keywords: *Coriandrum sativum*, Hepatoprotection, Aluminum toxicity, Bioactive Compounds, Medicinal Plants

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INTRODUCTION

Aluminum (Al) is a naturally occurring metal that is widely distributed in the environment and extensively used in industrial processes, making it a common environmental contaminant of concern [1]. It poses significant toxicological risks to human health because of its persistence in the human body and accumulation over time [2]. Long-term exposure to aluminum is known to significantly alter liver enzymes, such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST), along with alkaline phosphatase (ALP). It also disturbs lipid metabolism and depletes glutathione, thereby leading to damage to cellular membranes [3]. Given its role as a primarily detoxifying organ, the liver is especially prone to oxidative stress, secondary to metal toxicity, calling for preventive measures against aluminum-induced hepatotoxicity [4]. These findings emphasize that additional potential hepatoprotective drugs are still required to counter the toxic effects of aluminum and to maintain liver function [5]. Given the increasing burden of various hepatic disorders caused by environmental pollutants, especially in industrial zones and developing countries, the search for cheap natural hepatoprotectors has been a vital research pursuit [6]. Recent epidemiological studies may have seemed to strengthen the case of heavy metals contributing to liver dysfunction cases, emphasizing pharmacological interventions for prevention [7]. Due to their ability to scavenge free radicals, enhance the activities of endogenous antioxidant enzymes, and protect cellular macromolecules from oxidative injury, herbal antioxidants are considered promising natural agents to counteract metal toxicity [8]. The heightened interest in phytochemical therapeutic agents has been stimulated by the availability and low toxicity of dietary

phytochemicals and their many biological activities, including their capability to counter oxidative damage from environmental pollutants such as aluminum [9]. Among these, traditional medicinal plants have received considerable attention due to their multifunctional bioactivity and relatively fewer adverse effects and hence have garnered special attention [10]. Researchers have observed that natural antioxidants of plant origin have a two-fold function of firstly assisting in the alleviation of oxidative stress and secondly in the regulation of cell death pathways and mitochondrial functions of hepatocytes [11]. *Coriandrum sativum* L., better known simply as "coriander", is an annual herbaceous plant originating in Mediterranean countries [12, 13]. The plant is characterized by intricately branched foliage, with its leaves finely divided. The inflorescences are small and white, while the fruit is nearly spherical to ovoid, distinguished on all sides by prominent longitudinal ridges [14]. The leaves are edible and are used in salads, while the seeds can be roasted and then ground, and along with the fruit are believed to be a good treatment for cold, served when sniffed [15]. It has long been used by many other traditional medical systems, such as Ayurveda and Traditional Iranian Medicine, to treat a variety of gastrointestinal, metabolic, and hepatic ailments [12]. It is precise to consider that fresh coriander contains approximately 84% water, low levels of saturated fat and cholesterol, and high levels of thiamine, zinc, and dietary fibers [16]. Coriander seeds are indeed a source of vitamins, lipids, and minerals like potassium, calcium, phosphorus, magnesium, sodium, and zinc [17]. Pepper, according to the study, is a source owing to the high fatty alcohols, particularly chenopodiaceous carotenoids [18]. *Coriandrum sativum* L. contains a wide range of secondary metabolites such as linalool, vanillic acid, 27 alkaloids, quercetin, tannins, limonene, saponins, dodecanal, zeaxanthin, phyllodulcin, glycosylated apigenin, oxygenated monoterpenes, γ-terpinene, and anethole [19]. The hepatoprotective properties may be attributed to the antioxidant potential of phenolic compounds present in *Coriandrum sativum* L., which simultaneously exert a cardiovascular protective effect [18, 20]. The plant's healing capability can be attributed to an assortment of natural chemicals with active properties, including limonoids, flavonoids, terpenes, and alkaloids, which have been implicated in several biological activities [21-23]. Different plant parts have been reported to possess various pharmacological attributes, including antibacterial, antiviral, antifungal, antifeedant, antioxidant, anti-inflammatory, antiallergic, antidiabetic, analgesic, and nematicidal [19, 24, 25]. While several studies have reported the antioxidant activity of *Coriandrum sativum* L., little is known on a molecular basis with respect to its effects on endogenous antioxidant gene expression [26]. Such genes, including catalase (CAT), glutathione peroxidase (GPX1), and superoxide dismutase 2 (SOD2), are the main regulators of hepatic oxidative balance and, subsequently, cellular defense associated with metal-induced ROS accumulation [27, 28]. The increased expression of these genes is considered an important biomarker of antioxidant therapeutic efficacy, marking an enhanced ability to enzymatically detoxify and maintain mitochondrial stability when oxidative stress arises [29, 30]. Recent reports have indicated that chitosan has an ability to control the expression of different genes responsible for antioxidant defense and stress response, including APX, SOD, PAL, P5CS, and PIP, under abiotic stresses [31- 33].

In addition to the aluminum-induced hepatotoxicity model, a cholesterol induced hyperlipidemia model was included in this study to provide additional insight into the biological activity of *Coriandrum sativum* L.. Given that oxidative stress and lipid metabolism have a close connection with hepatic dysfunction, the use of both experimental models allowed the evaluation of the antioxidant, hepatoprotective, and lipid-modulating properties of the extracts. Accordingly, the cholesterol model was employed as a complementary experimental approach to broaden the biological evaluation of the extracts rather than as an independent study focus.

Aiming to strengthen the scientific basis, this study explores the biochemical and molecular parameters of hepatic injury. Furthermore, taking into account the antioxidant mechanism, the expression levels of genes encoding critical antioxidant enzymes, CAT, GPX1, and SOD2, have been analyzed in the livers of aluminum-intoxicated rats. This integrated gene expression analysis enables us to make mechanistic claims about the molecular aspects of how *Coriandrum sativum* L. extracts exert hepatoprotection, and it provides the necessary translational evidence for the use of *Coriandrum sativum* L. as a functional nutraceutical or an adjunct to heavy metal toxicity. This study provides new insights into the mechanisms by which *Coriandrum sativum* L. exerts hepatoprotective effects, through gene expression studies and GC-MS metabolite profiling, supporting its potential development as a phytomedicine against heavy metal-induced oxidative damage to the liver. Altogether, the current work integrates molecular, biochemical, and metabolomic data to provide a holistic understanding of how coriander extracts might mediate hepatic protection, thus corroborating *Coriandrum sativum* L. as a potential nutraceutical candidate against aluminum-induced oxidative hepatotoxicity. Consequently, to the best of our knowledge, this study is among the first to employ a combined biochemical and gene expression approach to investigate the antioxidant effects and regulation of antioxidant-related genes of *Coriandrum sativum L.* in liver tissues affected by aluminum, adding a different approach to the growing field of phytogenomics in hepatoprotection.

MATERIALS AND METHODS

Plant Material

Collection of Plant Material

Fresh aerial parts and seeds of *Coriandrum sativum L.* were collected during the flowering stage from an agricultural field in Isfahan, Iran, and taxonomically identified by a botanist; representative samples were preserved for reference (Fig. 1).

Preparation of Plant Material and Extraction Procedure

The aerial portions (stems, leaves, and fruits) of *Coriandrum sativum* L. were gathered from a nearby farming plot in Isfahan Province, Iran, during the period of active growth. The plant specimens were taxonomically recognized, by looking at usual botanical traits, by a qualified botanist in the Department of Biology, Islamic Azad University, Falavarjan Branch, Isfahan, Iran, before they were taken into the experimental procedures.

The gathered plant materials were washed thoroughly with distilled water to get rid of surface contaminants, and then they were shade-dried at room temperature (25 ± 2 °C) for 7–10 days, until a constant weight was reached. After that, the dried plant parts were ground to a fine powder using a lab mill, and they were kept in airtight containers at 4 °C until the next step of extraction. The extraction method was picked based on earlier phytochemical reports, and also on the polarity of the target bioactive compounds. A hydroalcoholic solvent system, made of methanol and n-butanol, was used to extract a broad range of polar plus semi-polar phytochemicals. This included phenolic compounds, flavonoid

types, and other antioxidant related constituents. The powdered plant material was macerated with the solvent at a ratio of 1:10 (w/v) for 72 h at room temperature, with intermittent shaking to improve extraction efficiency. After extraction, the mixtures were passed through Whatman No. 1 filter paper. The filtrates were then concentrated under reduced pressure with a rotary evaporator (Heidolph, Germany) at 40–45 °C until complete removal of the organic solvents. These concentrated fractions were afterward dried to get the crude extract. The extraction yield was calculated as a representative example the percentage of the dried crude extract compared to the initial dry weight of the plant material using this equation.

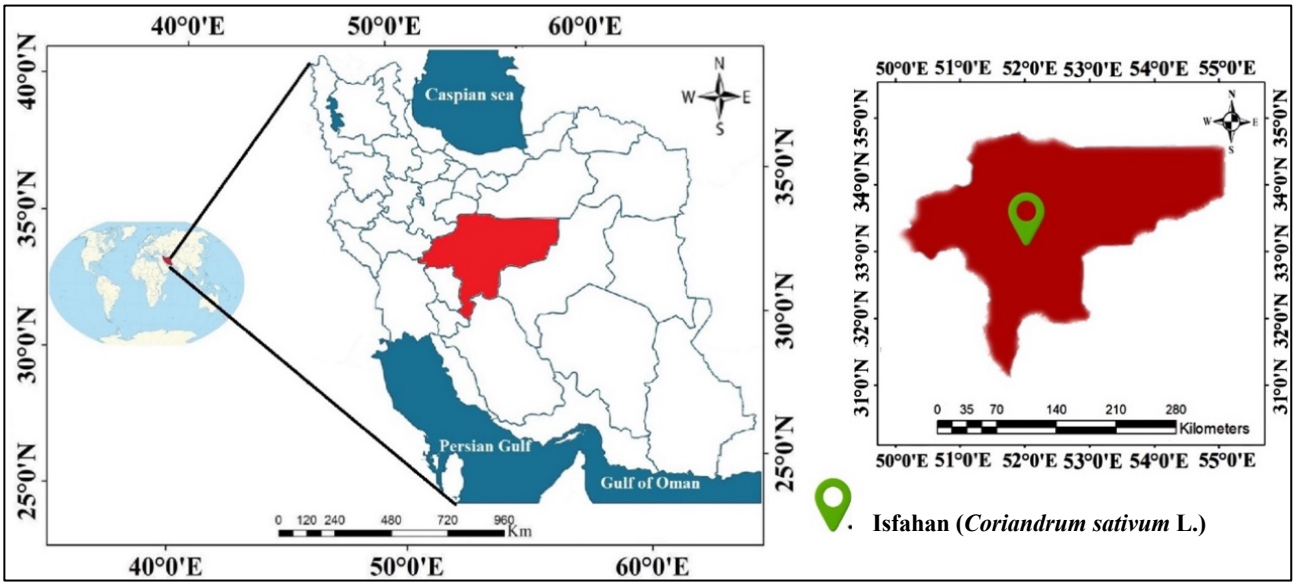


Fig. 1 Geographical coordinates (latitude and longitude) of the sampling sites.

Extraction yield (%) = (Weight of dried extract / Weight of dried plant material) × 100

The dried crude extracts were moved into airtight amber glass containers and then stored at −20 °C until the phytochemical characterization and the biological analyses were done. All extractions were carried out under the same experimental conditions to ensure methodological consistency throughout the study [34].

Gas Chromatography–Mass Spectrometry (GC-MS) Analysis

The chemical composition of the volatile constituents in the *Coriandrum sativum* L. extracts was determined using a gas chromatography–mass spectrometry (GC-MS) system consisting of an Agilent 7890A gas chromatograph coupled to an Agilent 5975 mass selective detector operating in electron ionization (EI) mode. Chromatographic separation was achieved on an HP-5MS capillary column (30 m × 0.25 mm i.d., 0.25 µm film thickness). The injector temperature was set at 280 degrees Celsius using helium as the carrier gas at a constant flow rate of 1.2 mL/min. The tune mode of the MS was used with an ionization energy of 70 eV. The ion source was set at 150 °C whilst the quadrupole had a temperature of 230 °C and the GC-MS interface was kept at 280 °C. The temperature program was initiated, leaving the oven at a temperature of 50 °C for 3 min, then ramped to 200 °C at 8 °C/min, then increased to 290 °C at 12 °C/min and kept isothermal for 3 min at 290 °C. For the volatile compound analysis, the headspace method was used, whereby the samples were incubated at 85 °C for 3 min prior to injection. Compounds were identified by comparing the mass spectra to NIST and Wiley libraries, and the retention index (RI) was calculated compared to a set of n-alkanes (which were run with the sample under the same conditions) [35]. GC-MS analysis was done in triplicate, with independently prepared extract samples so that the reproducibility of the chromatographic profiles is ensured. In the Results section, you can find the representative chromatograms.

Animal Model and Experimental Design

In all, 120 adult male Wistar rats (200–250 g) were procured from Royan Institute (Isfahan, Iran). Animals were housed in the animal facility of the Research Laboratory at Islamic Azad University, Falavarjan Branch, under standardized environmental conditions (temperature: 22 ± 2 °C, humidity: 55 ± 5%, 12:12-h light/dark cycle). Standard pelleted diet and tap water were provided ad libitum. Animals were acclimatized for one week prior to the commencement of experimental procedures.

The study design involved 20 experimental groups (n=6 per group), designed to assess the individual and combined effects of aluminum chloride, cholesterol, and hydroalcoholic (based on ethanol and methanol) extracts of coriander (*Coriandrum sativum* L.). The grouping was as follows: A negative control was also included when phosphate buffered saline (PBS; 0.9%; intraperitoneally) was administered. Two groups each received aluminum chloride at doses of 100 and 200 mg/kg (i.p.) to induce hepatotoxicity. Four groups received coriander extract alone, with extract being made from ethanol or methanol or being derived from seed or aerial parts (100 mg/kg, p.o.). Eight groups received aluminum chloride at 100 mg/kg or 200 mg/kg (i.p.), with the same coriander extracts based on ethanol or methanol, seed or aerial parts at 100 mg/kg (p.o.), to evaluate their protective effects. One group received cholesterol alone (50 mg/kg, p.o.) to generate hyperlipidemia, while the other five groups were given cholesterol (50 mg/kg, p.o.) and the plant extracts from ethanol or methanol of both the seed and aerial parts of coriander (100 mg/kg, p.o.) to find their antihyperlipidemic potential [36, 37]. The selected dose of coriander extract (100 mg/kg body weight) was chosen based on earlier in vivo work that reported strong antioxidant and hepatoprotective activities, and also no clear signs of treatment

related toxicity. This amount had already been used successfully in animal experiments to measure the biological efficacy of *Coriandrum sativum* extracts, so it was considered appropriate for the current study [38, 39].

All experimental procedures received prior approval from the Institutional Animal Ethics Committee of Islamic Azad University, Najafabad Branch (Approval Code: IR.IAU.NAJAFABAD.REC.1397.022) and were carried out accordingly, in accordance with the respective national and international guidelines on care and use of laboratory animals.

Preparation and Administration of Test Compounds

Phosphate-buffered saline (PBS, 0.9%) was mixed on a daily basis by diluting the concentrated PBS solution with distilled water. The negative control group was treated via intraperitoneal injections of 2 mL PBS as the vehicle blank. Stock solutions of aluminum chloride ($AlCl_3$) were freshly prepared before each injection by dissolving the appropriate amount of $AlCl_3$ in PBS to provide doses of 100 or 200 mg/kg body weight via intraperitoneal injection as a positive control in aluminum-induced toxicity. The selected $AlCl_3$ regimen (100 mg/kg body weight oral gavage for 30 days) was chosen based on earlier experimental models that show this type of exposure reliably induces hepatic oxidative stress, biochemical malfunction, and also provides a reproducible framework for testing hepatoprotective interventions [40, 41]. The coriander seed and aerial part extracts were prepared using methanol and ethanol as extraction solvents. After filtration, these were concentrated under reduced pressure to about 25% solids; dried extracts weighing approximately 7 g were obtained. Extract suspensions were freshly prepared in distilled water and were administered daily by oral gavage at 100 mg/kg body weight for groups receiving coriander alone or in combination with aluminum or cholesterol substances. The administration of cholesterol was done via oral gavage at the specified dose after being dissolved in a suitable vehicle (olive oil or Tween-80 solution). The cholesterol-alone group was taken as a positive control for hyperlipidemia, while the combined treatment groups were used to evaluate the protective effects of coriander extracts.

Serum Biochemistry and Liver Sampling

After completing the 28-day treatment, animals were fasted overnight and anesthetized with ketamine (90 mg/kg) and xylazine (10 mg/kg) intraperitoneally. Once deep anesthesia was confirmed, blood samples were taken by cardiac puncture using sterile syringes. The collected blood was put inside serum-separating tubes with clot activator gel and centrifuged at 3000 rpm for 10 minutes at 4 °C. The obtained serum was then distributed into sterile microtubes that were stored at −20 °C until further biochemical analyses were performed. Liver function markers were measured: alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total bilirubin (TBIL), and direct bilirubin (DBIL). The methods used for determination were performed on an automated biochemistry analyzer (e.g., BT3000 Plus, Biotecnica Instruments, Italy), according to the instructions supplied by the manufacturer. Liver tissues were removed surgically, washed with ice-cold saline, and portioned for biochemical assays [42-44].

Determination of Antioxidant Enzymes (SOD and CAT)

The SOD and CAT activities were measured in liver tissue homogenates using the ELISA kit for rats by Novand Salamat Co. (Iran) following the manufacturer's instructions. The homogenization took place in cold phosphate-buffered saline (PBS), with a pH of 7.4; A centrifugation step was performed at 10,000 × g for 15 minutes at 4 °C. After centrifugation, the supernatant was taken for biochemical analysis. The activity of SOD was quantified at 405 nm using a sandwich ELISA method. The kit determines SOD concentration based on the interaction of the antigen and antibody through color development by enzyme activity. The SOD activity was expressed as U/mg protein. The CAT activity was obtained from an ELISA kit that measured the absorbance at 540 to 550 nm following a colorimetric detection of the catalase enzyme concentration. The assay utilized the conversion of a substrate followed by a colorimetric reaction. The data were expressed as U/mg protein. The assay was measured in duplicate, and the optical density was determined using a microplate reader (BioTek ELX800) that had been zeroed. The manufacturer supplied the sensitivity and repeatability values for the assay, which were determined to be acceptable.

Each biological specimen stood for one individual animal, with n = 6 per group and all ELISA readings were done in duplicate technical replicates, namely two measurements per sample so it was to ensure analytical consistency.

Measurement of Free Radical Scavenging Activity (DPPH Assay)

DPPH assay measures the free radical scavenging activity of the extracts of *Coriandrum sativum* L., described as the most common method for assessing antioxidant activity. In each experimental group (Control; Lead Acetate; Soxhlet Extract; Soaking Extract), 1 mL of liver tissue homogenate was prepared and mixed with 2 mL of 0.1 mM DPPH solution in methanol. The mixtures were allowed to incubate for 30 mins at room temperature in the dark prior to measurements of absorbance at 517 nm using a UV-Vis spectrophotometer. The percentage of scavenging activity towards DPPH radicals was measured using the following formula: $[(A_0 - A_1)/A_0] \times 100$, where A_0 is the absorbance of DPPH without a sample (control) and A_1 is the absorbance with a sample (under investigation). All experiments were done in triplicate, and results are reported as mean ± SD. The data obtained were statistically analyzed using one-way ANOVA, followed by Tukey's post hoc test, and a p-value less than 0.05 was considered significant.

RNA Extraction and cDNA Synthesis

Total RNA was extracted from liver tissue via the lithium chloride method as detailed in [45] and [46]. RNA was extracted from liver tissue containing high levels of polysaccharides and secondary metabolites. The initial spectrophotometric analysis was completed to evaluate RNA purity and integrity. cDNA was synthesized using the GeneALL cDNA synthesis kit (catalog no. 601-602, GeneALL Biotechnology, South Korea), and the protocol was performed according to the manufacturer's protocol. In brief, 2.5 µL of total RNA was combined with 0.5 µL of dNTP mix, 0.5 µl of oligo (dT), and 0.5 µL of random hexamer primers into a sterile 0.2 mL PCR tube. The mixture was incubated at 70 °C for 5 minutes to denature any secondary structures present in the RNA, and then placed on ice for 2 minutes. Next, 0.5 µL of reverse transcriptase enzyme, 0.5 µL of 2X reaction buffer, and 0.5 µL of an RNase inhibitor (RNasin) were added and the procedure continued with primer annealing (25 °C for 10 min), and next, cDNA synthesis (42 °C for 50 min) and finally enzyme inactivation (80 °C for 10 min). In the next step, cDNA was put at −20 °C until the time the qPCR analysis for its expression could be performed.

Real-Time PCR Analysis

Quantitative real-time PCR was performed using the ABI StepOne™ Real-Time PCR System (Applied Biosystems, USA) according to the manufacturer's guidelines, utilizing the BioFACT™ Real-Time PCR MasterMix. The real-time system recorded the Ct values automatically after amplification; data were subsequently exported to a worksheet in Microsoft Excel for further examination. The gene expression data were analyzed by the ΔΔCt method relative to the housekeeping gene used. Relative expression differences were calculated using GenEx (MultiD, Sweden); statistical analysis/graphing were performed using SPSS 20.0. Primers targeting the genes CAT, GPX1, SOD2, and the reference gene 18S rRNA, were designed by Gene Runner v6.5.5 x64 software. Primers were designed to ensure sequence specific optimum target specificity with high-efficiency amplification based on the referenced protocol described [36]. Specificity primers were checked by observing one amplification product and only one peak in the melting curve; analysis was done for each target gene. The amplification efficiencies were within the acceptable range, about 90–110% and that backs the use of the 2^−ΔΔCt method for relative gene expression. The reference gene, 18S rRNA, was selected since its transcript levels remain stable in all experimental conditions, even across the different groups. Also, no template controls (NTCs) were run in every batch to rule out carryover contamination or non-specific amplification. Oligonucleotides were synthesized on an ABI 394 DNA/RNA synthesizer (Applied Biosystems, USA) by standard phosphoramidite chemistry to provide assurance of sequence fidelity and purity. Table 1 lists Primer sequences, Amplicon sizes, Annealing Temperature, and NCBI accession numbers.

formula1: ΔCT = CT (a target gene*) – CT (a reference gene**)

*= CAT ،GPX1 و SOD2

**= mCtrRNA

Table 1 Primers for RT-qPCR analysis of antioxidant and inflammatory gene expression.

Gene Symbol	Forward Primer (5′–3′)	Reverse Primer (5′–3′)	Product Size (bp)	Tm (°C) OLIGO
CAT	GCTCAGCTGACACAGTTCGT	TTGCCATCGCCAATGGCAAT	172	52-54
GPX1	TTGAGTCCCAACATCTCCAGTA	TACTGGAGATGTTGGGACTCAA	198	53
SOD2	TGGTGAACCAGTTGTGTTGTC	CCTGGTACTTCTCCTCGGTG	~185	52-56

The gene expression analysis was conducted with six biological replicates per experimental group. Each sample was analyzed in triplicate technical replicates according to standardized conditions.

Statistical Analysis

When the observational data were collected, the initial statistics provided an indication of the potential to study them as potential candidates for Analysis of Variance (ANOVA). Both Pearson correlation matrices and hierarchical heatmap clustering were used to evaluate linear correlative relationships among all parameters studied. To lessen the effects of discrepancies in magnitude on the measurement scales, the data were Z-score standardized (mean = 0; standard deviation = 1) before running any analyses. Data visualization was performed using XLSTAT (version 2019) for graphical presentation, ClustVis (https://biit.cs.ut.ee/clustvis/) for hierarchical heatmap clustering, and SRplot (https://www.bioinformatics.com.cn/en) for Pearson correlation matrix visualization. Next, the results were interpreted and presented graphically and in a Tabular format. All quantified results are shown as mean ± SD, as in the average value plus standard deviation. Then statistical analyses were carried out with SPSS software (version 20.0; IBM Corp., Armonk, NY, USA), including one-way analysis of variance (ANOVA), Tukey's multiple comparison post hoc test, and the Shapiro–Wilk normality test. When comparing differences between groups, we used one-way analysis of variance (ANOVA) and, when appropriate, followed by Tukey's multiple comparison post hoc test. Before running the ANOVA, we checked normality of the data and whether the variances were similar, using the Shapiro–Wilk test and Levene's tests, respectively. A P value that was <0.05 was taken as statistically significant.

RESULTS

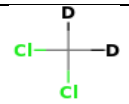
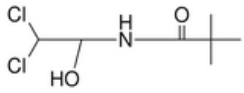
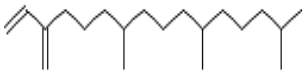
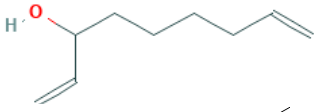
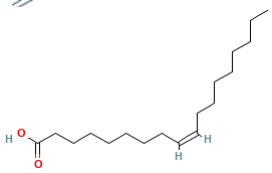
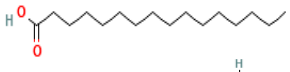
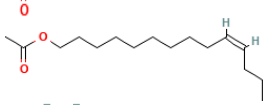
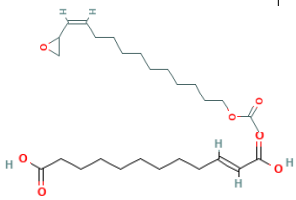
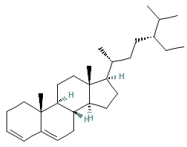
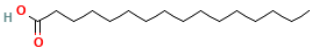
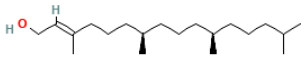
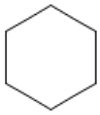
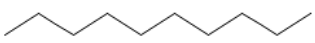
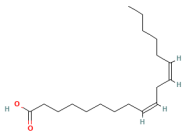
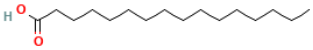
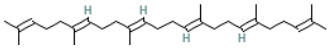
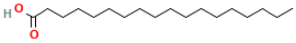
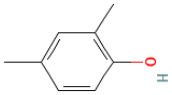
Gas Chromatography-Mass Spectrometry (GC-MS) composition of *Coriandrum sativum* L. aerial part extracts obtained using methanol and ethanol.

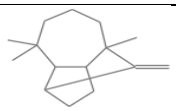
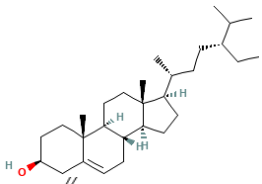
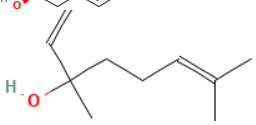
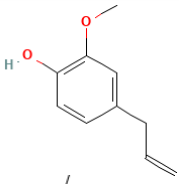
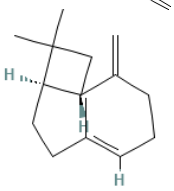
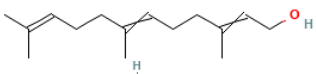
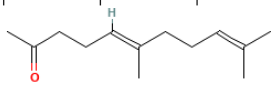
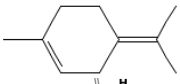
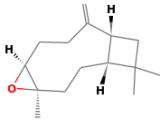
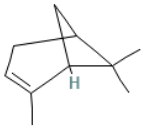
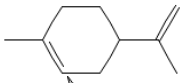
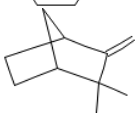
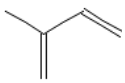
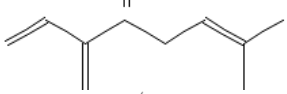
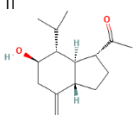
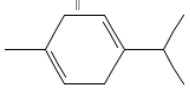
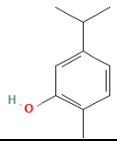
Following extraction of the aerial parts of *Coriandrum sativum* L. with methanol and ethanol and their GC-MS analysis, GC-MS analysis revealed a diverse range of phytochemical constituents in both extracts (Table 2). The methanolic extract was characterized by the presence of several major compounds, including: Methane, dichloro (10.49%), Neophytadiene (7.34%), Stigmastan-3,5-diene (8.94%), and Oleic acid (2.16%), as well as n-Hexadecanoic acid (0.82%) and trans-Traumatic acid (1.11%). Z-(13,14-Epoxy) tetradec-11-en-1-ol acetate (0.78%), Methylene chloride (1.39%). These results indicate the chemical complexity of the methanolic extract. In contrast, the ethanol extract showed a different chemical profile, with the predominant compounds being Linoleic acid at 40.814%, Decane at 12.761%, and Cyclohexane at 20.455%. Other abundant compounds detected in the ethanol extract included n-Hexadecanoic acid at 9.673%, Palmitic acid at 2.221%, Pulegone at 8.149%, and Methyl chavicol at 3.095%. Major terpenoid constituents included Beta-pinene – 5.321%, Farnesene – 3.776%, Nerolidol – 3.912%, and Carvacrol – 3.086%. The ethanol extract contained a broad range of fatty acids, terpenes, and aromatic compounds, which have been reported to exhibit biological activities. The methanol extract, having considerable fatty acids, hydrocarbons, and sterols, contained a relatively high proportion of Stigmastan-3,5-diene. In addition, both extracts revealed diverse phytochemical profiles of *Coriandrum sativum* L., with terpenes and fatty acids representing major constituents in the ethanol extract (Table 2).

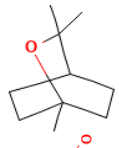
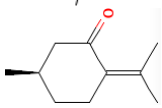
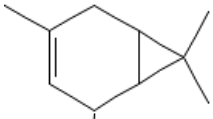
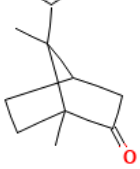
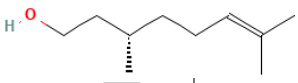
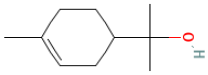
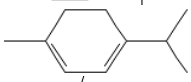
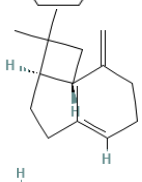
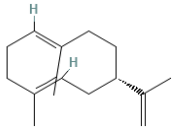
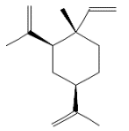
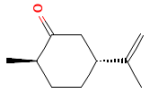
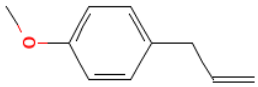
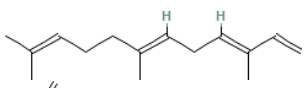
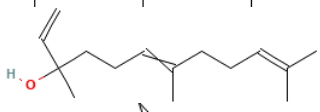
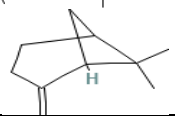
Comparative Clustering of Biochemical and Antioxidant Profiles via Heatmap Analysis

The heatmap displays the hierarchical clustering and relative expression profiles of many biochemical and molecular indicators for twenty experimental groups. These indicators consisted of liver function enzymes such as ALT, AST, and ALP; lipid profile indicators such as LDL, HDL, total cholesterol, and triglycerides; indicators of oxidative stress; and gene expression of common antioxidant genes *SOD2*, *CAT*, *GPX1,* and DPPH radical scavenging capacity.

Table 2 GC-MS Composition of *Coriandrum sativum* L. Aerial Part Extract Using Methanol and Ethanol

No	Phytochemical compound	Retention time (RT, min)	Molecular formula (M.F.)	Molecular weight (M.Wt)	Peak area %	Chemical structure	Extraction solvent
1	Methane, dichloro-	4.007	CD_2Cl_2	86.945	10.49%		Methanol
2	N-(2,2-Dichloro-1-hydroxy-ethyl)-2,2-dimethyl	20.517	$C_7H_{13}Cl_2NO_2$	214.093	1.59%		Methanol
3	Neophytadiene	31.71	$C_{20}H_{38}$	278.52	7.34%		Methanol
4	Methylene chloride	31.797	CH_2Cl_2	84.93	1.39%		Methanol
5	1,8-Nonadien-3-ol	34.816	$C_9H_{16}O$	140.22	1.2%		Methanol
6	Oleic Acid	36.719	$C_{18}H_{34}O_2$	282.46	2.16%		Methanol
7	*n*-Hexadecanoic acid	36.091	$C_{16}H_{32}O_2$	256.43	0.82%		Methanol
8	Z-10-Tetradecen-1-ol acetate	38.419	$C_{16}H_{30}O_2$	254.41	0.9%		Methanol
9	Z-(13,14-Epoxy) tetradec-11-en-1-ol acetate	38.633	$C_{16}H_{28}O_3$	268.39	0.78%		Methanol
10	*trans*-Traumatic acid	41.758	$C_{12}H_{20}O_4$	228.28	1.11%		Methanol
11	Stigmastan-3,5-diene	46.238	$C_{29}H_{48}$	396.68	8.94%		Methanol
12	*n*-Hexadecanoic acid	3.715	$C_{16}H_{32}O_2$	256.42	9.673%		Ethanol
13	Phytol	3.89	$C_{20}H_{40}O$	296.55	2.037%		Ethanol
14	Cyclohexane	4.148	C_6H_{12}	84.16	20.455 %		Ethanol
15	Tetradecane	4.187	$C_{14}H_{30}$	198.39	1.269%		Ethanol
16	Decane	4.431	$C_{10}H_{22}$	142.29	12.761 %		Ethanol
17	Linoleic acid	4.012	$C_{18}H_{32}O_2$	280.44	40.814 %		Ethanol
18	Palmitic acid	4.475	$C_{16}H_{32}O_2$	256.42	2.221%		Ethanol
19	Squalene	5.385	$C_{30}H_{50}$	410.72	1.222%		Ethanol
20	Octadecanoic acid	9.402	$C_{18}H_{36}O_2$	284.48	2.802%		Ethanol
21	2,4-Dimethylphenol	9.28	$C_8H_{10}O$	122.17	1.881%		Ethanol

22	Longifolene	16.807	$C_{15}H_{24}$	204.3511	1.290%		Ethanol
23	β-Sitosterol	17.138	$C_{29}H_{50}O$	414.72	0.921%		Ethanol
24	Linalool	19.665	$C_{10}H_{18}O$	154.25	1.083%		Ethanol
25	Eugenol	δ22.8	$C_{10}H_{12}O_2$	164.21	1.637%		Ethanol
26	β -Caryophyllene	24.285	C15H24	204.36	1.234%		Ethanol
27	Farnesol	25.395	$C_{15}H_{26}O$	222.37	0.706%		Ethanol
28	Geranyl acetone	25.41	$C_{13}H_{22}O$	194.31	0.719%		Ethanol
29	Terpinolene	27.333	$C_{10}H_{16}O$	152.23	1.268%		Ethanol
30	Caryophyllene oxide	31.69	$C_{15}H_{24}O$	220.35	6.469%		Ethanol
31	α-Pinene	31.71	$C_{10}H_{16}$	136.23	1.516%		Ethanol
32	Limonene	32.05	$C_{10}H_{16}$	136.23	1.067%		Ethanol
33	Camphene	32.318	$C_{10}H_{16}$	136.23	1.896%		Ethanol
34	Isoprene	32.333	C_5H_8	68.12	0.749%		Ethanol
35	Myrcene	33.652	$C_{10}H_{16}$	136.23	1.295%		Ethanol
36	Sesquiterpenes	33.793	$C_{15}H_{24}O_2$	204.36	1.070%		Ethanol
37	δ-Terpinene	33.813	$C_{10}H_{16}$	136.23	0.736%		Ethanol
38	Carvacrol	35.765	$C_{10}H_{14}O$	150.22	3.086%		Ethanol

39	1,8-Cineole (Eucalyptol)	35.824	$C_{10}H_{18}O$	154.25	1.261%		Ethanol
40	Pulegone	35.838	$C_{10}H_{16}O$	152.23	8.149%		Ethanol
41	3-Carene	35.887	$C_{10}H_{16}$	136.23	0.751%		Ethanol
42	Camphor	36.082	$C_{10}H_{16}O$	152.23	1.345%		Ethanol
43	Citronellol	36.101	$C_{10}H_{20}O$	156.26	1.133%		Ethanol
44	Terpineol	36.72	$C_{10}H_{18}O$	154.25	1.871%		Ethanol
45	α-Terpinene	36.953	$C_{10}H_{16}$	136.23	0.801%		Ethanol
46	Caryophyllene	41.208	$C_{15}H_{24}$	204.36	1.085%		Ethanol
47	Germacrene	41.759	$C_{15}H_{24}$	204.36	0.719%		Ethanol
48	β-Elemene	42.888	$C_{15}H_{24}$	204.36	0.703%		Ethanol
49	Dihydrocarvone	43.278	C10H16O	152.23	1.804%		Ethanol
50	Methyl chavicol	43.307	$C_{10}H_{12}O$	148.20	3.095%		Ethanol
51	Farnesene	45.269	$C_{15}H_{24}$	204.36	3.776%		Ethanol
52	Nerolidol	46.184	$C_{15}H_{26}O$	222.36	3.912%		Ethanol
53	β-Pinene	46.233	$C_{10}H_{16}$	136.23	5.321%		Ethanol

The color scheme ranged from blue (low expression or activity) to red (high expression and activity), with standardized Z-scores presented for comparison purposes across all groups (Fig. 2). The Aluminum 200 mg/kg group significantly increased the liver damage markers, particularly ALP and AST (deep red), as well as LDL and total cholesterol levels, and decreased the expression of the antioxidant genes *SOD2*, *CAT*, and *GPX1,* indicating the hepatotoxic and pro-oxidative effect of high-dose aluminum. In contrast, The Aluminum 200 + coriander extract treated groups (both ethanol and methanol extracts, seed and aerial parts) partially reversed these pathological changes through decreased liver enzyme levels (shift towards green/yellow) and increased antioxidant gene expression (shift away from blue towards green). Among them, the combination with higher polyphenol content and antioxidant DPPH activity profile showed a more pronounced protective effect in the methanol seed extract. The Control (PBS) group is used as a comparison because it shows normal physiological values by having stable baseline values for all parameters. In the non-Coriander extract group, all parameters showed expression of antioxidant genes and small decreases in hepatic enzymes compared to the Control group, which indicates a possible hepatoprotective and antioxidant effect conferred by the

extract without induced toxicity (Fig. 2). The methanol extracts exhibited stronger effects than the ethanol extracts, although all extracts are effective in inducing expression of *SOD2*, *CAT*, and *GPX1*. The Cholesterol-only group showed a significant rise in LDL, total cholesterol, and triglycerides, thus confirming hyperlipidemia induction. However, the Coriander extract treatments, especially the seed methanol and aerial parts methanol extracts, significantly reduced these elevations and positively affected the HDL level, suggesting potential antihyperlipidemic effects. In addition, increases in DPPH scavenging activity, which were also more pronounced than in the Cholesterol-only group, were observed along the same trend. This suggests a potential association between the lipid-lowering effect and the activity of the extracts as antioxidants. Moreover, hierarchical clustering shows that the treatment groups separate according to similarity in biochemical profiles. The Aluminum 200 mg/kg group clustered separately from most of the treatment groups, indicating its unique pathological signature. The coriander-treated groups formed sub-clusters closer to the control group, thus showing their ameliorative effect against aluminum-and cholesterol-induced damage (Fig. 2).

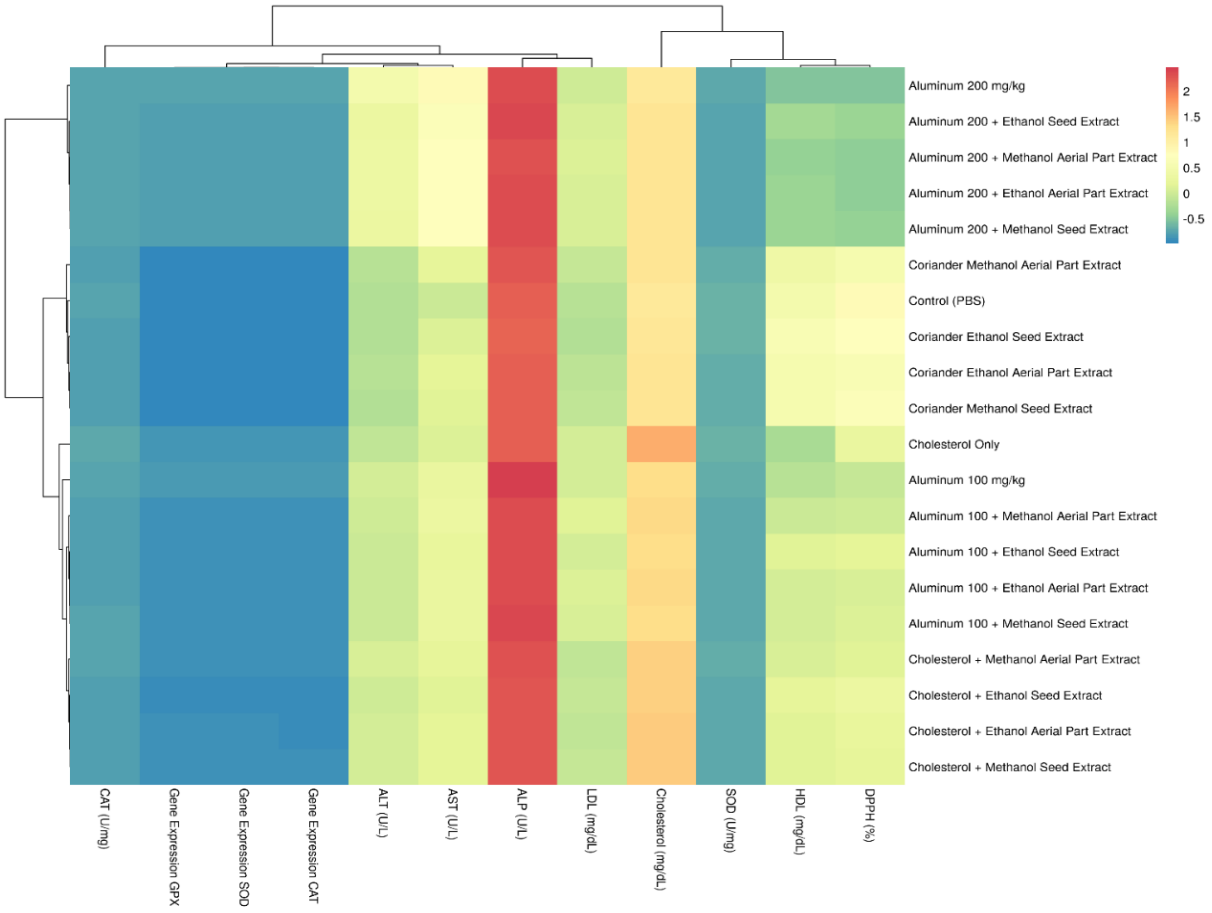


Fig. 2 Clustered heatmap for combined experimental groups showing biochemical, antioxidant, and gene expression outcomes. The heatmap shows group-wise standardized z-scores for serum indicators of liver function (ALT, AST, ALP), lipid profile indicators (LDL, HDL, total cholesterol, triglycerides), activities of antioxidant enzymes (SOD, CAT), gene expression (*SOD2*, *CAT*, *GPX1*), and elimination potential of DPPH radicals. Dendrograms and heatmaps were created with two-way clustering based on Pearson Correlation using Euclidean distance, while treatments were clustered with a full linkage method. Red in the heatmap indicates upregulation/increased value and blue indicate downregulation/decreased value compared to the mean of the respective features.

Integrative Visualization of Parameter Deviations Using Flower Plot

The flower plot is a graphical visualization that represents relationships among various parameters and their respective deviation from a defined reference value (value: 500) or standardized reference state. Each individual petal radiating from the central core represents a specific biochemical or molecular parameter; the petals represent the measured values during this specific research project, reflecting the measured concentration of that particular parameter in the specific sample under consideration. This study included 12 parameters. These include markers of hepatic function (ALT, AST, ALP), lipid profile (LDL, HDL, total cholesterol), oxidative stress enzymes (SOD, CAT), gene expressions of antioxidant markers (*SOD2*, *CAT*, *GPX1*), and total antioxidant capacity (DPPH %) (Fig. 3). The range of colors on the petals indicates departure from normal: green values are close to or slightly above the reference, indicating a favorable biological status; red indicates markedly increased values or imbalances, largely indicative of pathological changes. Red petals were observed for the ALP (121 U/L) and cholesterol (81 mg/dL) results, showing significantly elevated values relative to the reference range, illustrating hepatic stress or cholestatic injury and hypercholesterolemia respectively- typically caused by aluminum or dietary cholesterol exposure (Fig. 3). Both ASTM (36 U/L) and DPPH scavenging activity (69%) are moderately high, in the intermediate range, suggesting a moderate oxidative/hepatic imbalance, or evidence of a partial response. Both LDL, at 31 mg/dL, and ALT, at 29 U/L, within the normal range, represented gray-green petals, meaning that while some indicators were elevated (e.g. ALP, total cholesterol) and some remained relatively stable, indicating either: either selected organ involvement or, possibly, partial relief from organ malfunction due to intervention.

Therefore, the flower plot seeks to represent a comprehensive integrated snapshot of the biological response to experimental interventions, based on the evaluated parameters. A selective recovery of antioxidant gene expression (*SOD2*, *CAT*, *GPX1*) and enzymatic activity (SOD, CAT) is found, likely mediated by the protective effects of coriander extracts; meanwhile, parameters like ALP and cholesterol remain elevated, suggesting that while oxidative stress may not be as severe, hepatic and lipid dysfunctions are not completely restored (Fig. 3).

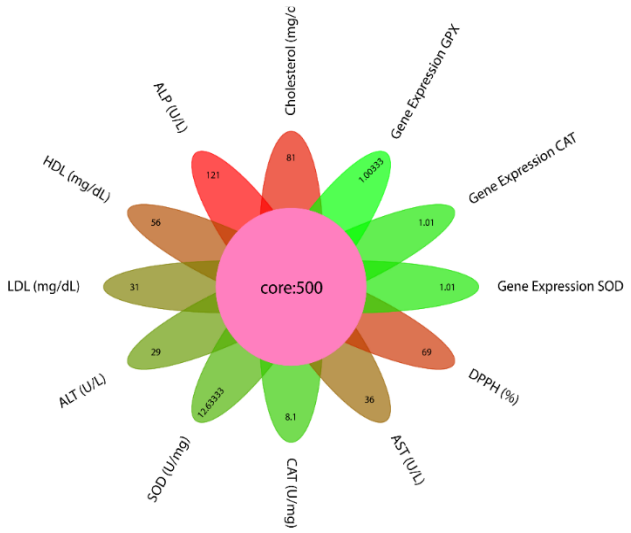


Fig. 3 A flower plot depicting deviations from each biochemical and molecular parameter as compared to the reference core value. In the flower plot, each petal corresponds to a single measured parameter: the liver enzymes actively released into plasma (ALT, AST, ALP), lipid components (LDL, HDL, cholesterol), antioxidant enzyme activities (SOD, CAT), the expression levels of antioxidant genes (*SOD2*, *CAT*, *GPX1*), and DPPH activity. The center of the plot (value =500) corresponds to the standardized baseline. The color of the petal refers to the magnitude of deviation from green, referring to those parameters that are close to the core (normal/restored), to red, which describes a significant elevation or pathological change from baseline.

Correlation Patterns Among Biochemical, Antioxidant, and Gene Expression Parameters

The Pearson correlation matrix of the major biochemical parameters (cholesterol, HDL, LDL, ALT, AST, ALP), the antioxidant enzyme activities (SOD, CAT), the non-enzymatic antioxidant capacity (DPPH), and the expression of the major antioxidant enzymes (*SOD2*, *CAT*, *GPX1*) is illustrated in figure 4.

The findings showed a moderate negative relationship between serum lipid markers (cholesterol and LDL) with the liver enzymes (ALT, AST, and ALP) both in relation to enzymatic antioxidant activity and the gene expression of the enzymes that were related to the antioxidants, with a high level of confidence statistically. This indicates that while increased oxidative stress would negatively impact hepatic functioning and lipid metabolism, increases in antioxidant defense may bolster biochemical status. The antioxidant gene expression levels exhibited a markedly different and more robust positive correlation statistically with the activities of their respective enzymes, providing evidence of the congruency between transcriptional regulation and functional antioxidant response. Expression of *GPX1*genes exhibited a positive correlation with DPPH radical scavenging activity, suggesting that the *GPX1*genes are valuable for the removal of ROS. Interestingly, when assessing the entire lipid profile, HDL was the only measured variable that was positively correlated with antioxidant parameters, which aligns with the presumed protective effects of HDL against oxidative damage. Overall, our findings underscore the central importance of modulating oxidative stress in maintaining both metabolic and hepatic health, thereby suggesting the expression of certain antioxidant-related genes as potential biomarkers for monitoring oxidative and physiological status (Fig. 4).

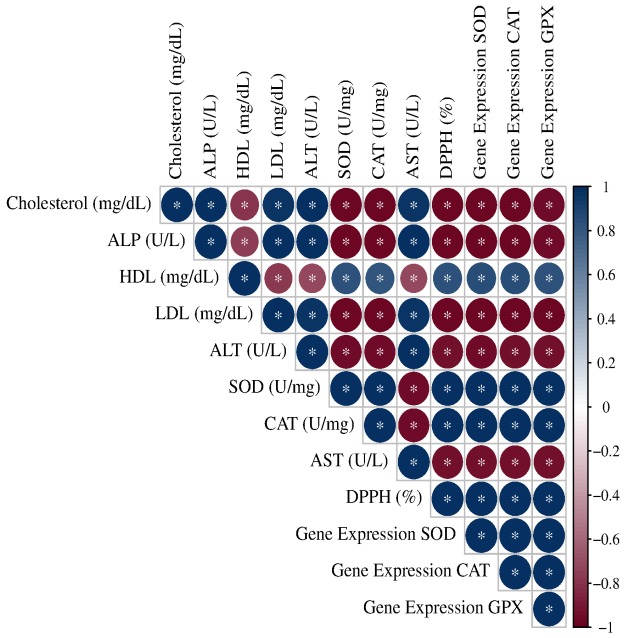


Fig. 4 Correlation matrix based on the Pearson correlation coefficient among biochemical markers (cholesterol, HDL, LDL, ALT, AST, ALP), enzymatic antioxidants (SOD, CAT), non-enzymatic antioxidant capacity (DPPH), and antioxidant enzyme gene expression (*SOD2*, *CAT*, *GPX1*). The intensity of color indicates correlation strength and direction (red = positive correlation; blue = negative correlation). Asterisks (*) denote statistically significant correlations ($P < 0.05$).

DISCUSSION

In the present study, the modulatory and protective activity of aerial part extracts (methanolic and ethanolic) of *Coriandrum sativum* L. (coriander) was tested for aluminum-induced liver damage and disturbance of lipid metabolism. The findings show that the administration of coriander extracts, especially the methanolic fraction, has opposed aluminum generated pathological biochemical changes. These abilities to potentially have therapeutic actions are associated with the phytochemical constituents of the extracts and their ability to modify important antioxidant gene expression, including *SOD2*, *CAT*, and *GPX1*; restore enzymatic antioxidant activity and lipid profile homeostasis. Physiological mechanisms can be interpreted by considering biochemical, genomic, and antioxidant activity data through coriander's phytochemicals acting upon cellular redox signaling and liver metabolic pathways. The assessment of the extracts using GC-MS, phytoconstituents were identified that had a wide range of variation. The methanolic and ethanolic extracts had different chemical profiles. The methanol extract of the aerial parts of *Coriandrum sativum* L. contained many bioactive components such as Neophytadiene, Stigmastan-3,5-diene, and oleic acid. Of particular interest, Stigmastan-3,5-diene is a plant sterol that is widely recognized for its cholesterol reducing and anti-inflammatory activity by inhibiting cholesterol absorption in the gut and altering membrane fluidity [47-49]. Neophytadiene is

a diterpene hydrocarbon that has been reported to show antioxidant and hepatoprotective properties, primarily by reducing oxidative stress and also by blocking lipid peroxidation [43, 44]. Its presence in the methanolic extract may have contributed to the biological effects we saw in the present study [50, 51]. Additionally, oleic acid plus trans-tubenoic acid have been reported as stimulators of cellular antioxidant defenses, and they also seem to reduce the inflammatory load via activation of PPAR-α and inhibition of the NF-κB signaling route, suggesting that these pathways may be regulated in opposite directions [52, 53]. The ethanolic extract contains the highest amounts of linoleic acid, cyclohexane, decanβ-pinene, nerolidol, and carvacrol. Linoleic acid, a polyunsaturated fatty acid, operates in modulating lipid metabolism, the oxidation of LDL, and the suppression of pro-inflammatory eicosanoid production [54, 55]. Terpenes such as β-pinene and nerolidol have been shown to scavenge ROS, stabilize membranes, and modulate genes, specifically highlighting the activation of the *Nrf2*/*ARE* pathway, which is important for regulating antioxidant gene expression [56, 57]. Carvacrol is an aromatic monoterpenoid that can counter oxidative stress and enhance lipid parameters in HFD models through a mechanism of direct downregulation of hepatic lipogenic enzymes and upregulation of endogenous antioxidant enzymes [58, 59]. The differences in the biological activities of the methanolic and ethanolic extracts might be linked with the differences in their phytochemical composition and also with possible synergistic interaction among their bioactive components. The methanolic extract is enriched in sterols and some unsaturated fatty acids and could have improved hepatoprotective and antioxidant capacities simply through a synergism of a polar and non-polar component [60, 61]. On the other hand, due to its strong antioxidant action, methanol extraction was found to be more powerful in extracting polyphenols, flavonoids, and other polar compounds from the extract. Therefore, biochemical analysis results for the current study show that Al intoxication significantly increased serum enzyme activities of AST, ALT, and ALP, suggesting possible liver damage, in addition to increased LDL and total cholesterol, consistent with other publications identifying aluminum as a hepatotoxic and hyperlipidemic agent [62, 63]. Moreover, Al affects normal iron storage, thus inducing the breakdown of allied defensive antioxidant enzymes such as catalase and superoxide dismutase, developing an oxidative stress factor [64]. The administration of extracts of *Coriandrum sativum* L. promoted recovery from these biochemical changes. The methanolic extracts from the seeds, and also the aerial parts, showed comparatively stronger effects in reducing serum liver enzyme levels, along with lipid markers too, and even steering the expression of genes that are associated with antioxidant defenses. The liver protective effects in this case may be due to increases in enzymatic antioxidant defenses (*SOD*, *CAT*), changes in lipid metabolism regarding cholesterol biosynthesis and absorption, and/or inhibition of pro-inflammatory cytokines [65, 66]. Since *SOD2, CAT,* and *GPX1* expression were up in the treated groups, coriander extracts could be affecting antioxidant related transcription regulation indirectly. *Nrf2* signaling involvement remains a plausible mechanism, but it really needs more investigation [67]. Also referred to as nuclear factor erythroid 2-related factor 2 (*Nrf2*) it is important for maintaining redox balance. *Nrf2*, upon activation, will interact with *many specific* sequences of the antioxidant response element (*ARE*). These sequences are located in the promoter region of certain genes. This binding leads to increased transcription of genes that code for antioxidant enzymes [68]. Terpenoids and polyphenols identified in coriander have been previously reported to activate the Nrf2 signaling pathway. So, this suggests that signaling activation might at least partly account for the higher expression of SOD2, CAT, and GPX1 that we saw in the current study, even though that specific mechanism was not directly examined [69]. Although the heightened expression of genes tied to antioxidant defense observed in the present study is consistent with a possible role for *Nrf2/ARE* signaling, we did not directly test activation of this pathway, so the contribution of *Nrf2* signaling remains a plausible mechanism requiring further investigation. Flower plot and heatmap analyses support this case and present visually how the normalization of hepatic enzymes and antioxidant gene expression occurs in coriander treated groups. The decreases in ALP and cholesterol did not fully revert to control levels but did remain a plausible mechanism requiring further investigation into hepatocellular damage and lipid dysregulation. The research conducted by Mishra *et al.* [67], "Lowers Blood Sugar and Antioxidative Potential of *Coriandrum sativum* L. Seed Extract in Alloxan-Induced Diabetic Rats," indicated that a treatment with crude methanolic coriander seed extract significantly increased the activity of antioxidant enzymes in the kidney tissue, including catalase (*CAT*) and glutathione peroxidase (*GPx*), compared to the untreated diabetic controls [70]. Collectively, all these observations support the hypothesis that the therapeutic response attenuation of coriander is a form of response mediated by gene-regulatory mechanisms in addition to free-radical scavenging. The Pearson correlation matrix indicated that cholesterol, LDL, and liver enzymes had a strong negative correlation with antioxidant enzyme activities and gene expression. HDL had a positive correlation with the antioxidant markers, which is expected with HDL's well-studied role in reverse cholesterol transport, and HDL has antioxidant properties of its own, transduced via paraoxonase-1. The substantial and strong association between *GPX1* and DPPH is especially noteworthy, further demonstrating that the increased antioxidant status in the coriander-treated groups is functionally related to increased expression of endogenous antioxidant genes. Coriander's biochemical effects have broader consequences. Beyond activating *Nrf2*, its active terpenoids and fatty acids might have an impact on pathways that control inflammation, lipid metabolism, and the creation of new mitochondria [54, 71]. Other polyunsaturates, such as linoleic acid, may also affect PPAR signaling and modulate fatty acid oxidation and lipid metabolism, thereby modifying the normalization of lipid profiles. The increased activity of methanolic extracts, especially from coriander seeds, may be due to a more complex mixture of bioactive compounds that include phytosterols that compete for intestinal absorption of dietary cholesterol and play a role in the modulation of hepatic lipid homeostasis [12, 72]. This may explain the notable difference in the reduction obtained in the LDL and cholesterol concentrations in the group treated with methanol and ethanol. These findings continue to be significant for the application of coriander as a natural agent against aluminum-induced oxidative stress and liver dysfunction. These data illustrate a wide-spectrum mechanism of action of the coriander extracts: (1) direct scavenging of ROS; (2) stimulation of transcription of antioxidants and detoxifying enzymes; (3) reversing alteration of lipid profiles; and (4) limiting the inflammatory process. Given the range of this combined effect, coriander may represent a promising adjunct or complementary agent for continued research regarding oxidative stress-related liver diseases. These results indicate a number of constraints of the

study, albeit with positive outcomes. One limitation in the present study is the lack of histopathological evaluation of liver tissues. Even though the biochemical, antioxidant, molecular, and phytochemical results were consistent and consistently demonstrated protective effects of *Coriandrum sativum* extracts against aluminum induced hepatic damage, a histopathological examination would provide additional evidence of tissue level protection and overall structural integrity. Future studies should include histological analyses, like H&E staining and a quantitative readout of hepatic lesions, to further validate the hepatoprotective benefits that were observed in this study. Second, while the mRNA levels of the antioxidant genes were reported, protein levels and/or enzymatic activity were not examined. In order to confirm transcriptional alterations at the protein level, future studies would benefit from Western blots or activity assays. Third, this study dealt with acute aluminum exposure, therefore the chronic toxicity situation highlighted the potential of coriander extracts to be protective; this remains to be explored. Fourth, the GC-MS analysis carried out in this study gave semi-quantitative information, mostly as relative peak area percentages, not true absolute quantification of each phytochemical. So, when we compare the relative presence of compounds between the methanolic and ethanolic extracts, the results should be read with caution, because it can be potentially misleading. Going forward, future work that uses authentic reference standards along with calibration curves for absolute determination would be more appropriate, it would measure phytochemical concentrations more reliably, and then, that should further reinforce the comparisons among different extract types.

Fifth, besides oxidative stress, aluminum-induced hepatotoxicity appears to engage a set of linked cellular pathways, such as mitochondrial malfunction, an inflammatory switch, ferroptotic processes, and apoptosis. When aluminum builds up inside the cells, it can damage mitochondrial structure, and that, in turn, promotes excessive reactive oxygen species (ROS) and disturbs normal energy production. This oxidative mismatch can also intensify lipid peroxidation and may steer the cell toward ferroptosis via iron-dependent mechanisms. On top of that, aluminum has been described as boosting inflammatory mediators, including IL-1β, TNF-α, and NF-κB, which then may worsen liver damage. Also, when oxidative stress occurs together with inflammatory pressure, apoptotic signaling in hepatocytes can get turned on. Even though these exact routes were not directly checked in the current work, the antioxidant responses that were seen, and the changes in antioxidant-related gene expression after Coriandrum sativum extract treatment, hint that such pathways could partly explain the hepatoprotective action. More mechanistic research will be needed to truly verify whether these pathways are involved, and how strongly they contribute. Furthermore, the Pearson correlation analysis carried out in this study ought to be viewed as exploratory, kind of a first sweep. Even though significant statistical associations were noticed among multiple biochemical and molecular parameters, using correlation by itself cannot really show causality or prove mechanistic relationships. So, the patterns that were observed should be taken with a bit of caution and later confirmed through additional future mechanistic studies. Finally, the study utilized whole-tissue homogenates, which may mask cell-type specific gene regulation; therefore, future studies may have to use single-cell or immunohistochemistry. In conclusion, this study clearly states that extracts of *Coriandrum sativum* L., especially the methanol fraction containing either the seeds and/or aerial parts, significantly ameliorated aluminum-induced hepatotoxicity and oxidative stress via biochemical improvements and modulation of antioxidant-related gene expression, which may help contribute to strengthened cellular defense against oxidative stress, sort of indirectly, even if it is not always so straightforward, and return lipid metabolism to normal. These results give experimental backing for how coriander is traditionally used, and yeah, they sort of point to its possible role as a nutraceutical option for dealing with oxidative stress linked hepatic dysfunction, so in the end it looks promising and not just theoretical, and thus suggests the use of coriander as a nutraceutical agent in the treatment of acute liver and metabolic disorders associated with exposure to oxidative stress.

CONCLUSION

The current study demonstrated significant hepatoprotective effects, antioxidant, and lipid-lowering profiles of *Coriandrum sativum* L. aerial part extracts, especially the methanol extracts. Various bioactive compounds, including phytosterols, fatty acids, and terpenes were detected which was shown by GC-MS analysis that showed differences in the composition of methanol and ethanol extracts. The methanol extract, which contained abundant Stigmastan-3,5-diene, Neophytadiene, and Oleic acid compounds, exhibited the highest biological activity, likely due to its composition with a more diverse mixture of lipophilic and antioxidant compounds. The biochemical and molecular analyses showed significant modifications of the hepatic laboratory indicators, increased oxidative stress, and lipid abnormalities after exposing the animals to HD, followed by raised levels of liver enzymes, LDL cholesterol, and total cholesterol, while antioxidant enzymes were suppressed. *Coriandrum sativum* L.: methanolic aerial and seed extracts significantly reduced the pathological modifications. Notably, the amelioration of pathological conditions correlated with increased antioxidant gene expression (*SOD2*, *CAT*, *GPX1*) and enhanced DPPH scavenging activity, as well as improvement in hepatic and lipid profiles. Flower plot visualizations and hierarchical clustering revealed partial recovery of physiological parameters toward control values in the extract-treated groups, with only ALT, LDL, and antioxidant markers returning to control levels, while ALP and total cholesterol remained elevated. The correlation analysis, regarding physiological parameters alone, supported the mechanistic relationship between enhanced antioxidant defense and improved metabolic status, illustrating the ability of these natural extracts to modulate oxidative stress pathways. Thus, the extracts of aerial parts of *Coriandrum sativum* L., particularly methanolic extracts, may be well considered as natural therapeutic agents for oxidative liver damage and hyperlipidemia. These findings enhance the evaluation of the further pharmacological activity of coriander-based formulations for the prevention and treatment of hepatic and metabolic disorders.

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Authors' contributions

The study was conceptualized and designed by M.R., with S.B. contributing equally to the experimental design and analysis. Laboratory experiments were conducted collaboratively by A.M., and M.N., with equal contributions. All authors reviewed and approved the final manuscript.

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Data availability
Data are provided within the manuscript information files.

Competing Interests
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Clinical Trial Number
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