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Research Article

The Potential of Coriander Leaf Extract in Amelioration of Arsenic-Induced Toxicity in Swiss Albino Mice and Phytochemical Characterization by GC-MS

Raushanara Akter,[1] **Luluel Maknun Fariha**,[1] **Fouzia Noor**,[1] **Hasan Shahriyer Tonmoy**,[1] **Asef Raj**,[1] **and Masum Shahriar**[2]

[1]*School of Pharmacy, BRAC University, Dhaka, Bangladesh*
[2]*Department of Pharmacy, Jahangirnagar University, Savar, Dhaka, Bangladesh*

Correspondence should be addressed to Raushanara Akter; raushanara@bracu.ac.bd

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Arsenic intoxication is a serious public health concern that causes several diseases, urging the discovery of new therapeutics. Coriander (*Coriandrum sativum*) has been reported to demonstrate its ameliorating effect on lead, cadmium, and carbendazim-induced toxicity. However, no studies have investigated the arsenic (As)-induced toxicity protective effect of coriander leaf extract and its phytochemical characterization by GC-MS. The study aimed to investigate the protective role of the methanolic extract of coriander leaves against arsenic-induced toxicity in Swiss albino mice and characterize its phytoconstituents by GC-MS. Mice were divided into three groups, control, As-treated, and As plus extract–treated groups, and treated with three different doses of extract (150, 300, and 500 mg/kg body weight) for 8 weeks. The arsenic-intoxicated mice that received coriander extract doses showed an increased body weight compared to the As-treated group. This extract protected arsenic-intoxicated mice by restoring hematological and biochemical parameters and improving histopathological changes. GC-MS analysis of the extract identified 59 compounds that fall under diverse phytochemical classes, and the major compounds characterized were 13-docosenamide, (Z)-, gamma-sitosterol, phthalic acid, di(2-propylpentyl) ester, stigmasterol, vitamin E, neophytadiene, gamma-tocopherol, and phytol. Incorporating an appropriate amount of coriander into the diet or using it as an adjuvant alongside chelating therapies may offer health benefits in mitigating arsenic-induced toxicity.

Keywords: arsenic; biochemical parameters; coriander; heavy metals; histopathological examination; Swiss albino mice

1. Introduction

It is acknowledged that arsenic is a major environmental contaminant that significantly affects the health of humans and other organisms [1]. In recent times, arsenic has caused serious health issues for the general public in developed nations such as the USA, Romania, and Hungary as well as developing nations including Bangladesh, India, Nepal, China, Taiwan, Thailand, Argentina, Chile, Mexico, Ghana, and Vietnam [2]. Millions of people in different countries including Bangladesh are under constant exposure to arsenic due to the consumption of arsenic-contaminated groundwater supplied especially by tube wells, and it is worrisome that 50% of the tube wells' water contains arsenic at a level of 0.05 mg/L which exceeds the safety level [3]. About 50 million people are projected to be in danger of exposure, making Bangladesh as one of the most severely afflicted countries in the world in terms of population exposure to arsenic-contaminated water [4]. It is known that inorganic arsenic is more hazardous than organo-arsenicals due to which the toxicity of arsenic is dependent on its chemical speciation. As a strong carcinogen, inorganic arsenic promotes tumors in the liver, kidneys, skin, lungs, and urinary bladder [1]. Arsenicosis is the result of repeated exposure to arsenic by consumption, breathing, or physical contact,

subsequently resulting in poisoning [3]. Up to 200 enzymes are rendered inactive by arsenic poisoning, including those involved in DNA replication and repair, cellular energy pathways, and high-energy molecules like ATP where arsenic takes the place of phosphate [5]. One key contributing factor to the increased risk of noncancerous diseases such as pigmentation, hyperkeratosis, hypertension, cardiovascular diseases, neurological, liver, and renal problems, diabetes, fertility issues, and cognitive impairment in children is drinking water that is contaminated with arsenic over an extended period of time [1, 2]. Association of arsenic toxicity and cancer has also been reported where breast cancer, leukemia, stomach cancer, liver cancer, and brain cancers exhibited the uppermost correlation with arsenic contamination [6].

Due to industrialization and environmental contamination, persistent exposure to hazardous heavy metals through food or surroundings is unavoidable. The potential of certain nutrients in plant-based diets such as coriander, to detoxify, can provide remedial and protective benefits against the poisonous effects of heavy metals [7]. For ages, people have been growing coriander (*Coriandrum sativum*), a herb that grows annually and belongs to the Apiaceae family. It can be found in warmer regions such as the Middle East, Latin America, Africa, and Asia, and traditionally, it is used for flavoring and healing purposes [8, 9]. The aerial parts of coriander are rich in essential oil, flavonoids, phenolic acids, and polyphenols, whereas the primary constituents of coriander seeds are essential oil and fatty oil [10, 11]. According to literature studies, coriander leaf and seed extract possess pharmacological properties including antibacterial, antidiabetic, and antioxidant properties [12, 13]. Furthermore, in mice, coriander leaf extract shows benefits for sleep prolongation, memory improvement, and defense against harm to the stomach mucosa [9].

The gas chromatography-mass spectrometry (GC-MS) technique has been widely adopted to analyze mixtures of chemical substances by integrating two analytical methods into one. Mass spectroscopy examines each component independently after gas chromatography isolates the mixture's constituent parts [14]. It is a significant method developed to assess the numerous phytoconstituents and their structures found in different plant extracts. This method produces chemical fingerprints with exceptional accuracy and precision due to its enhanced isolation capability. Moreover, GC-MS not only enables quantitative analysis but also utilizes extensive mass spectral databases, making it particularly effective in correlating identified bioactive compounds with their prospective pharmacological functions [15].

Numerous prior studies have explored the capacity of coriander to reduce heavy metal toxicity, demonstrating its effectiveness in lowering the concentrations of mercury, cadmium, copper, lead, and mercury in organs such as the liver, kidneys, and bones [16–18]. Since no study was conducted previously to evaluate the effect of *Coriandrum sativum* methanolic extract on arsenic-induced toxicity, this study aimed to determine whether *Coriandrum sativum* leaf extract protects Swiss albino mice against arsenic-induced toxicity and to characterize the phytochemicals in the leaf extract using GC-MS which could potentially contribute to this effect.

2. Materials and Method

2.1. Collection and Extraction of Coriandrum sativum. For the current study, fresh coriander herbs were purchased from the local market and leaves were separated and washed thoroughly with clean water to remove soil dirt or other contaminants. Then, they were shade-dried for 7 days and ground into coarse powder. About 500 g of the powder was macerated in 2.5 L of analytical-grade methanol (1:5 w/v) at room temperature for 14 days with intermittent stirring. The mixture was filtered through cotton wool. The filtrate was concentrated under reduced pressure using a rotary evaporator at 40°C to yield a thick, semisolid extract. The extract was stored in an airtight glass container at 4°C until use.

2.2. GC-MS Analysis. GC-MS analysis of the methanolic coriander extract (CE) was carried out following the method described by Akter et al., 2022, and Chikowe et al., 2024 [3, 19]. Coriander extract was analyzed to identify individual compounds present in the extract utilizing a GCMS-TQ8040 instrument (Shimadzu, Japan) with high sensitivity and performance. For analysis, a DAB-5 ms nonpolar INNOWax column having an internal diameter of 30×0.25 mm and film thickness of 0.25 μm was used. The column temperatures were kept at 50°C for 1 min at the beginning, followed by a gradual increase to 300°C for 20 min. A volume of 0.5 μL of the extract was injected as an injection volume for analysis. The noble gas, helium, was used as the carrier gas with a flow rate set at 1 mL/min in the spitless mode. The split ratio was kept at 5, and the temperatures of the sample injector and detector were maintained at 250°C and 230°C, respectively. To perform electron ionization, mass spectrometry electrons with an energy of about +0.50 kV were used. Furthermore, 40 min were spent recording mass spectra in the 50 to 600 m/z region.

Identification and quantification of individual phytoconstituents of the CE were done by comparing retention indices and mass spectra fragmentation patterns of each compound reference sample available in the Wiley database and the National Institute of Standards and Technology (NIST) libraries (ALILOU and AKSSIRA, 2021). Furthermore, the relative proportion of each component was determined by quantifying and relating its % peak area to the overall peak areas.

2.3. Preliminary Phytochemical Screening. Preliminary phytochemical screening of methanolic CE was performed as a qualitative examination in detecting different classes of phytochemicals such as flavonoids, saponins, alkaloids, triterpenes, and steroids present in the extract following established and validated protocols [20].

2.4. Ethical Approval, Animal Maintenance, and Study Design. Ethical approval for conducting experiments was granted from the Biosafety, Biosecurity and Ethical Committee, Faculty of Biological Sciences, Jahangirnagar University,

Savar, Dhaka, Bangladesh (Ref No. BBEC, JU/M 2020 (10)2). Experiments on Swiss albino mice were conducted in the Department of Pharmacy, Jahangirnagar University, Bangladesh. *Swiss albino* mice were housed in individual polypropylene cages in a temperature- and humidity-controlled room (22°C ± 3°C; RH 55%), with a 12 h dark/light cycle. Mice were given free access to standard laboratory feed and water. After acclimation for one week, thirty-six healthy female Swiss albino mice aged 6–8 weeks and weighing 20–25 g were selected for the study. The mice were randomly divided into 6 groups, each containing 6 animals ($n = 6$); group I (As-treated with Na arsenite at the dose of 0.35 mg/kg body weight and concentration 0.0075 mg/mL), group II (treated with coriander at the dose of 500 mg/kg body weight), group III (As plus a CE dose of 150 mg/kg), group IV (As plus a CE dose of 300 mg/kg), group V (As plus a CE dose of 500 mg/kg, and group VI (control group which just had distilled water once in a day). Only female mice were selected for experiments to avoid pregnancy. All the 1 mL doses were administered once/day by oral gavage between the hours of 10–12 at noon and the experiments were conducted for eight weeks. This subclinical dosing strategy of sodium arsenite reflects chronic environmental exposure and is supported by analogous models, including those utilizing low-dose sodium arsenite (0.05–0.2 mg/kg/day) via drinking water or gavage that elicit measurable biological effects without acute toxicity. Moreover, previous studies using 0.0075 mg/mL sodium arsenite have reported biochemical and histological responses under similar exposure duration [3]. The doses of 150, 300, and 500 mg/kg body weight of CE methanolic leaf extract were chosen based on prior experimental studies that demonstrated the extract's antioxidant and hepatoprotective efficacy in murine models [2, 21, 22].

2.5. Measurement of Body Weight. The effect of As and coriander extract on the body weight of mice belonging to control, As-treated, and As-plus CE extract–treated groups was examined in this study. To determine the changes in the body weight of mice, the individual weight of each mouse of each group was recorded using analytical balance throughout the 8-week study period.

2.6. Estimation of Hematological and Biochemical Parameters. After completion of 8-week study, mice were anesthetized by administering ketamine intraperitoneally at a dose of 500 mg/kg of body weight. Blood samples were collected from the posterior vena cava of mice and serum was separated by centrifuging blood at 4000 rpm at 40°C for 10 min. Serum was used for determining different hematological parameters such as red blood cell (RBC), white blood cell (WBC), neutrophil (NEU), lymphocyte (LYM), monocyte (MON), and eosinophil (EOS) count. Biochemical parameters such as serum glutamic pyruvic transaminase (SGPT), creatinine (CRE), and alkaline phosphatase (ALP) levels were measured from plasma and serum obtained from blood. SGPT, CRE, and ALP levels were estimated using a commercial kit specific for the test following the manufacturer's protocol.

2.7. Histopathological Study. Organs such as liver, kidney, skin, and brain were removed from the sacrificed mice and kept in 10% buffered formalin solution, passed through an ascending series of saline baths for further histopathological study. Hematological assay was carried out using the method described by Shrivastava [23].

2.8. Statistical Analysis. Data were expressed as mean ± standard error of the mean (SEM). Statistical analyses were performed using IBM SPSS (version 27). A one-way analysis of variance (ANOVA) was employed for data comparisons followed by an independent sample *t*-test for mean comparison between groups as well as for significance level determination. The sample size for each group was $n = 6$. p value < 0.05 was considered as the level of significance.

3. Results

3.1. Results

3.1.1. GC-MS Analysis. GC-MS analysis of coriander extract identified and quantified a total of 59 compounds belonging to diverse phytochemical classes (Table 1, Figure 1). Based on the % peak area, major compounds characterized in the extract include 13-docosenamide, (Z)- (RT: 23.114 min, 49.82%), gamma-sitosterol (RT: 38.068 min, 8.47%), phthalic acid and di(2-propylpentyl) ester (RT: 25.838 min, 4.28%), decanamide (RT: 20.06 min, 4.16%), stigmasterol (RT: 36.862 min, 3.45%), tetradecanamide (RT: 23.515 min, 3.33%), vitamin E (RT: 34.648 min, 2.58%), 6,9-octadecadienoic acid, methyl ester (RT: 23.002 min, 2.16%), neophytadiene (RT:14.638 min, 1.59%), gamma-tocopherol (RT: 33.467 min, 1.06%), and phytol (RT: 18.904 min, 0.98%). Among these compounds, gamma-sitosterol and stigmasterol fall under the phytosterol class, and phthalic acid and di(2-propylpentyl) ester belong to phthalate esters, 3-docosenamide, (Z)-, decanamide, and tetradecanamide belong to amide class, vitamin E and gamma-tocopherol are fat-soluble vitamins, neophytadiene and phytol fall under diterpenes and the chemical class of 6,9-octadecadienoic acid, and methyl ester is a fatty acid. Table 2 represents 12 compounds out of 59 characterized in this study with huge potential in ameliorating heavy metal toxicities induced by Pb, Cd, Cu, Ni, Cr, As, etc.

3.2. Phytochemical Screening. The preliminary phytochemical screening of the methanolic extract of coriander confirmed the presence of carbohydrates, alkaloids, glycosides, protein, sterol, and triterpenes in the methanolic extract of *C. sativum* (Table 3). However, the extract was devoid of saponins and flavonoids.

Table 1: Phytoconstituents identified from *C. sativum* extract by GC-MS.

Sl. No.	R. time	Area (%)	Compound name	Structure	Phytochemical class
1	3.53	0.22	D-Mannitol, 1-decylsulfonyl-	$C_{16}H_{34}O_7S$	Pentose alcohol
2	3.687	0.15	1,4-Benzenedimethanol	$C_8H_{10}O_2$	Aromatic diol
3	5.887	0.15	Arsenous acid, tris(trimethylsilyl) ester	$C_9H_{27}AsO_3Si_3$	Organosilicon
4	10.658	0.08	Phenol, 2,6-bis(1,1-dimethylethyl)-	$C_{14}H_{22}O$	Phenol
5	11.222	0.07	Heptadecane, 7-methyl-	$C_{18}H_{38}$	Alkanes
6	11.685	0.06	3-Methyl-4-(phenylthio)-2-prop-2-enyl-2,5-dihydrothiophene 1,1-dioxide	$C_{14}H_{16}O_2S_2$	Thiophene
7	12.198	0.23	Octadecane, 1-chloro-	$C_{18}H_{37}Cl$	Alkane
8	12.446	0.2	4-(6,6-Dimethyl-2-methylenecyclohex-3-enylidene)pentan-2-ol	$C_{14}H_{22}O$	Alcohols
9	12.503	0.2	tert-Butyl (2-aminophenyl)carbamate, 2TMS derivative	$C_{11}H_{16}N_2O_2$	–
10	12.84	0.1	Nonadecane	$C_{19}H_40$	Alkane
11	13.174	0.07	1-Dodecanol, 3,7,11-trimethyl-	$C_{15}H_{32}O$	Fatty alcohols
12	13.371	0.12	Eicosane	$C_{20}H_{42}$	Alkane
13	13.628	0.22	Loliolide	$C_{11}H_{16}O_3$	Benzofurans
14	14.18	0.09	Dodecane, 2,6,11-trimethyl-	$C_{15}H_{32}$	Alkane
15	14.638	1.59	Neophytadiene	$C_{20}H_{38}$	Diterpenes
16	14.72	0.35	2-Pentadecanone, 6,10,14-trimethyl-	$C_{18}H_{36}O$	Ketone
17	15.263	0.53	3,7,11,15-Tetramethyl-2-hexadecen-1-ol	$C_{20}H_{40}O$	Diterpenoids
18	15.588	0.12	Glucitol, 6-O-nonyl-	$C_{15}H_{30}O_6$	Alcohol
19	16.68	0.23	Octadecanamide	$C_{18}H_{37}NO$	Amides
20	18.651	0.28	9,11-Octadecadienoic acid, methyl ester, (E,E)-	$C_{19}H_{34}O_2$	Fatty acid
21	18.756	0.79	8,11,14-Docosatrienoic acid, methyl ester	$C_{23}H_{40}O_2$	Fatty acid methyl ester
22	18.904	0.98	Phytol	$C_{20}H_{40}O$	Diterpene
23	19.174	0.4	Methyl stearate	$C_{19}H_{38}O_2$	Fatty acid methyl ester
24	20.06	4.16	Decanamide-	$C_{10}H_{21}NO$	Amides
25	20.384	0.2	Eicosyl isopropyl ether	$C_{23}H_{48}O$	Alkyl ether
26	21.049	0.25	(+/−)-Marmesin	$C_{14}H_{14}O_4$	Furanocoumarins
27	22.2	0.09	Tetrapentacontane, 1,54-dibromo-	$C_{54}H_{108}Br_2$	Halogenated alkanes
28	22.66	0.21	Pentatriacontane	$C_{35}H_{72}$	Alkane
29	22.725	0.35	2-[(2S)-7-Oxo-2,3-dihydro-7H-furo[3,2-g]chromen-2-yl]propan-2-yl acetate or marmesin acetate	$C_{16}H_{16}O_5$	Flavonoids
30	22.899	0.11	4,8,12,16-Tetramethylheptadecan-4-olide	$C_{21}H_{40}O_2$	Terpenoids
31	23.002	2.16	6,9-Octadecadienoic acid, methyl ester	$C_{19}H_{34}O_2$	Fatty acid
32	23.114	49.82	13-Docosenamide, (Z)-	$C_{22}H_{43}NO$	Amides
33	23.515	3.33	Tetradecanamide	$C_{14}H_{29}NO$	Amides
34	23.663	0.18	Tridecane, 3-cyclohexyl-	$C_{13}H_{28}$	Alkane
35	23.735	0.12	Docosane, 1,22-dibromo-	$C_{22}H_{44}Br_2$	Halogenated alkane
36	23.833	0.19	2-Methylhexacosane	$C_{27}H_{56}$	Alkane
37	25.174	0.14	Nonyl tetradecyl ether	$C_{23}H_{48}O$	Alkyl ether
38	25.274	0.24	2,6,10,14-Tetramethyl-7-(3-methylpent-4-enylidene) pentadecane	$C_{25}H_{48}$	Isoprenoids
39	25.344	0.11	Octatriacontyl pentafluoropropionate	$C_{41}H_{77}F_5O_2$	Ester
40	25.838	4.28	Phthalic acid, di(2-propylpentyl) ester	$C_{26}H_{26}O_4$	Phthalate esters
41	27.419	0.12	Dodecyl nonyl ether	$C_{21}H_{44}O$	Alkyl ether
42	28.934	0.1	17-Pentatriacontene	$C_{35}H_7O$	Alkene
43	30.122	0.26	Squalene	$C_{30}H_{50}$	Triterpene

Table 1: Continued.

Sl. No.	R. time	Area (%)	Compound name	Structure	Phytochemical class
44	30.565	0.19	Alpha-tocospiro A	$C_{29}H_{50}O_4$	Terpenoids/alpha-tocopheroids
45	30.795	0.08	Butylphosphonic acid, di(4-octyl) ester	$C_{20}H_{43}O_3P$	Phosphonic acid ester
46	30.876	0.42	Alpha-tocospiro B	$C_{28}H_{48}O_4$	Terpinoids/alpha-tocopheroids
47	31.984	0.09	Delta-tocopherol	$C_{27}H_{46}O_2$	Tochopherol
48	32.225	0.1	Ginsenol	$C_{15}H_{26}O$	Triterpenoid saponins
49	32.533	0.06	Isophthalic acid, allyl isohexyl ester	$C_{17}H_{22}O_4$	Ester
50	33.467	1.06	Gamma-tocopherol	$C_{28}H_{48}O_2$	Tocopherols
51	33.803	0.17	17Alpha(H),21.beta(H)-homohopane	$C_{31}H_{54}$	Pentacyclic triterpenoids
52	34.648	2.58	Vitamin E	$C_{29}H_{50}O_2$	Fat-soluble vitamins
53	34.779	0.7	Alpha-tocopherolquinone	$C_{29}H_{50}O_3$	Vit-E derivatives
54	36.387	0.25	5-Androstenetriol	$C_{19}H_{30}O_3$	3-Hydroxy steroid
55	36.862	3.45	Stigmasterol	$C_{29}H_{48}O$	Phytosterol
56	38.068	8.47	Gamma-sitosterol	$C_{29}H_{50}O$	Phytosterol
57	38.375	0.52	Ergosta-5,24(28)-dien-3-ol, (3.beta.)-	$C_{28}H_{46}O$	Phytosterol
58	39.372	0.85	9,19-Cyclolanostan-3-ol, acetate, (3.beta.)-	$C_{32}H_{54}O_2$	Phytosterol
59	39.52	0.37	1,2-Bis(trimethylsilyl)benzene	$C_{12}H_{22}Si_2$	Organosilicon

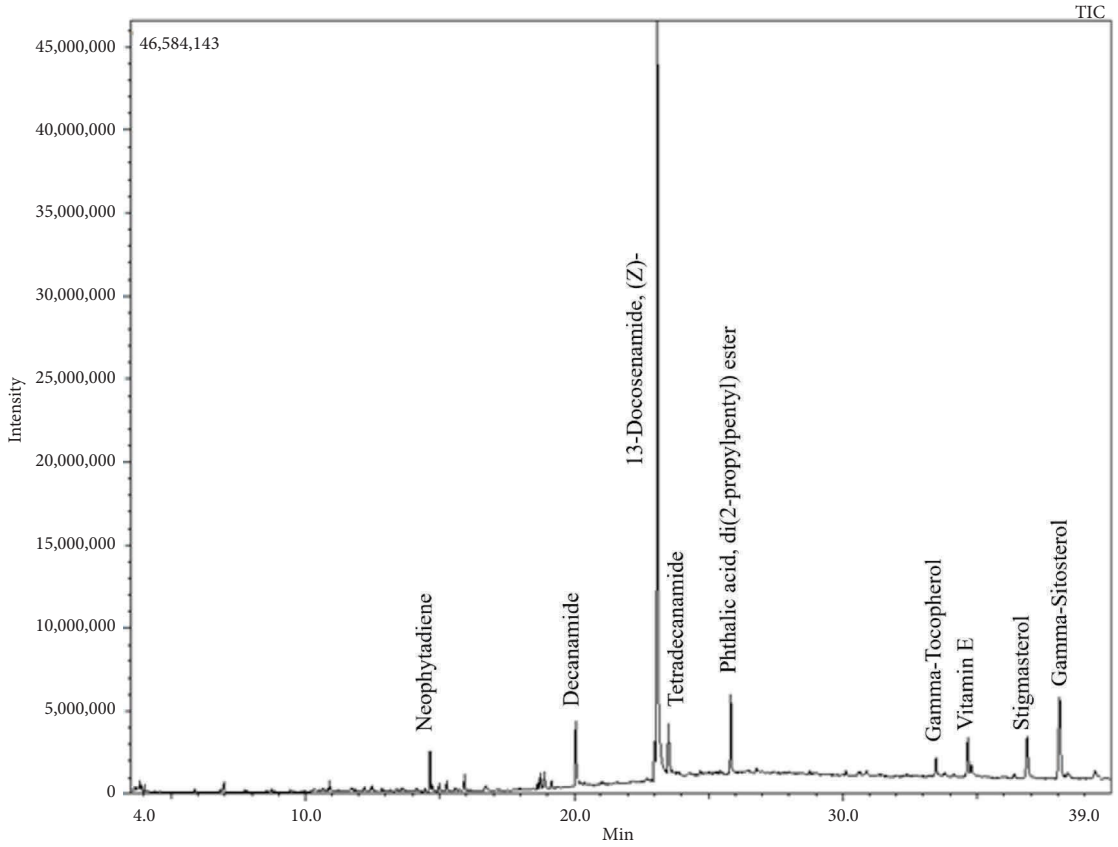


Figure 1: Phytoconstituents of *C. sativum* identified and quantified by GC-MS. Major peaks are annotated with compound names.

3.3. Effect of Extract on Body Weight. The weight variations among six distinct groups of Swiss albino mice subjected to different treatments over 8 weeks are illustrated in Figure 2. The control group exhibited normal weight gain (~13.34 gm), serving as a baseline for comparison. The group exposed to Na arsenite exhibited minimal weight gain (~6.16 gm). In contrast, the group treated solely with 500 mg/kg of coriander extract demonstrated a remarkable increase in body weight (~11.84 gm).

In the arsenite-exposed groups, those treated with different doses of coriander extract showed varying results. The group given As + 150 mg/kg of coriander extract had the least weight gain (~4 gm). The group treated with As + 300 mg/kg extract showed a moderate weight gain (~7.66 gm). Notably, the group with As + 500 mg/kg of coriander extract had the most substantial weight gain (~11.66 gm) over the subsequent weeks among the treatment groups.

3.4. Estimation of Hematological Parameters. Table 4 presents the hematological effects of arsenic (As) + different doses of *Coriandrum sativum* extracts on WBC, RBC, NEU, LYM, MON, and EOS over 8 weeks, compared to control, arsenic-treated, and As + extract-treated groups. A significant decrease in WBC levels was observed in the Na arsenite–treated group (3.30 ± 0.316, $p < 0.05$) compared to the control group (7.83 ± 0.542). In contrast, administering a 500 mg/kg dose of the extract alone significantly increased WBC levels (8.05 ± 0.55, $p < 0.05$) compared to the control group. Among the treatment groups, varying doses of extracts (150, 300, and 500 mg/kg) administered to arsenic-exposed groups resulted in a significant dose-dependent increase in WBC levels (As + extract 150: 6.05 ± 5.53, $p < 0.05$; As + extract 300: 6.20 ± 0.44, $p < 0.05$; As + extract 500: 6.40 ± 4.21, $p < 0.05$) compared to the Na arsenite–treated group.

RBC levels decreased in the arsenic-treated group (4.26 ± 0.06) compared to the control group (5.36 ± 0.098). However, RBC levels increased when varying doses of extracts were administered (As + extract 150: 4.98 ± 0.11; As + extract 300: 5.06 ± 0.12; As + extract 500: 5.06 ± 0.12) compared to the arsenic-treated group, although these changes were not significant.

NEU count was increased in the As-treated group (59.33 ± 1.45) compared to the control group (54.00 ± 3.08). NEU counts significantly decreased in groups treated with arsenic + 150, 300, and 500 mg/kg of the extract (As + extract 150: 51.16 ± 1.83, $p < 0.05$; As + extract 300: 51.83 ± 2.98, $p < 0.05$; As + extract 500: 53.66 ± 2.15, $p < 0.05$) compared to the solely arsenic-treated group (59.33 ± 1.45). Conversely, LYM counts significantly increased in all groups receiving arsenic + extract (As + extract 150: 39.16 ± 1.32, $p < 0.05$; As + extract 300: 41.33 ± 3.40, $p < 0.05$; As + extract 500: 41.83 ± 1.75, $p < 0.05$) compared to the arsenic-treated group (33.16 ± 1.13).

Table 2: Role of some compounds identified by GC-MS analysis of coriander extract in heavy metal toxicity reduction.

Sl. No.	Compound name	Role of compounds in heavy metal poisoning/toxicity	References
1.	Phenol, 2,6-bis(1,1-dimethylethyl)-	Scavenges ROS and lowers oxidative stress; this compound can help protect cells from damage induced by heavy metal toxicity (Cd, Cu, Zn, Ni, Co, Cr, Pb, and As)	[24, 25]
2.	Dodecane, 2,6,11-trimethyl-	An effective adsorbent in removing heavy metal ions from synthetic wastewater, with the following order of concentration: $Zn^{2+} < Cd^{2+} < Fe^{3+}$	[26]
3.	8,11,14-Docosatrienoic acid, methyl ester	Protects against oxidation and free radical damage in tissues by neutralizing excess free radicals that eventually induce oxidative stress and degenerative disease due to Cr toxicity	[27]
4.	Phytol	Exhibits antioxidant properties by reducing oxidative stress due to transition metal Fe^{2+} toxicity	[28]
5.	Nonyl tetradecyl ether	Plays a significant role in decreasing heavy metal toxicity by washing heavy metals like Pb, Zn, Cr, Cu, and Ni	[29]
6.	Dodecyl nonyl ether	Nonionic surfactants play a significant role in decreasing heavy metal toxicity by washing the heavy metals. As a removal of contaminated soil technique, phytoremediation has been gradually applied to heavy metal–contaminated soil.	[29]
7.	Squalene	SQ alleviates the toxicity caused by heavy metals	[30]
8.	Ginsenol	Effectively protects the liver against Cd-induced acute hepatic toxicity in mice by decreasing the elevation of blood biochemical parameters (ALT, AST, and LDH) and restoring the impairments of histopathology. It also ameliorates the depletion of GSH by Cd treatment and inhibits expression of proapoptotic bad protein in liver tissue, indicating the partial involvement of antioxidative and antiapoptotic actions in hepatoprotection	[31]
9.	Gamma-tocopherol	Provides mitochondrial protection against arsenic toxicity	[32]
10.	Vitamin E (alpha-tocopherol)	Reduces Cd-induced oxidative stress in mice by increasing glutathione levels	[33]
11.	Stigmasterol	Increases concentration of stigmasterol and decreases the Cd-induced growth suppression of *Calendula officinalis* plants and hairy root	[34]
12.	Gamma-sitosterol	Increases the concentration of sitosterol and decreases the Cd-induced growth suppression of *Calendula officinalis* plants and hairy root	[34]

Table 3: Preliminary phytochemical screening of *Coriandrum sativum* extract.

Class of compounds	Name of the test(s)	Presence (+)/absence (−)
Carbohydrates	Fehling's test, Benedict's test, and Barfoed's test	+ − −
Alkaloids	Mayer's test and Wagner's test	− +
Glycosides	Borntrager's test and modified Borntrager's test	− +
Saponins	Foam test	—
Flavonoids	Zinc-hydrochloride reduction test and alkaline reagent test	− −
Protein	Biuret test	+
Sterol	Salkowski's test	+
Triterpenes	Salkowski's test and Lieberman–Burchard test	+ +
Acidity	pH meter	6.4



Figure 2: Effects of coriander extract on body weight of Swiss albino mice over 8 weeks. Values are mean ± SEM ($n = 6$ per group). Error bars represent SEM.

MON levels did not undergo any noticeable change in the arsenic + extract–treated groups compared to the arsenic-treated group. Eosinophil counts decreased in the arsenic + extract–treated groups; however, these changes were not statistically significant.

3.5. Estimation of Biochemical Parameters. Table 5 presents the effects of arsenic (As) exposure combined with varying doses (150, 300, and 500 mg/kg) of *Coriandrum sativum* extracts on biochemical parameters such as alanine transaminase (ALT), ALP, and CRE after eight weeks. The results were compared across control, arsenic-treated, and As + extract–treated groups.

A significant increase in ALT levels was observed in the arsenic-only group (66.33 ± 4.07, $p < 0.05$) compared to the control group (56.83 ± 2.98). The administration of arsenic + 150 mg/kg of the extract resulted in a nonsignificant decrease in ALT levels (65.83 ± 4.11). However, higher doses of the extract (300 and 500 mg/kg) significantly reduced ALT levels (As + extract 300: 60.50 ± 3.51, $p < 0.05$; As + extract 500: 51.66 ± 2.18, $p < 0.05$) compared to the arsenic-only group.

Similarly, ALP levels significantly increased in the arsenic-only group (165.74 ± 9.51, $p < 0.05$) compared to the control group (145.02 ± 3.05). The administration of the arsenic + extract at all doses (150, 300, and 500 mg/kg) led to a significant decrease in ALP levels (As + extract 150: 160.66 ± 9.45, $p < 0.05$; As + extract 300: 154.16 ± 7.90, $p < 0.05$; As + extract 500: 150.00 ± 5.09, $p < 0.05$) compared to the arsenic-only group.

Additionally, CRE levels significantly increased in the arsenic-treated group (1.01 ± 0.03, $p < 0.05$) compared to the control group (0.86 ± 0.08). Administering a 150 mg/kg dose of the extract with arsenic nonsignificantly lowered CRE levels (As + extract 150: 0.95 ± 0.08). However, higher doses of 300 and 500 mg/kg led to a significant reduction in CRE levels (As + extract 300: 0.90 ± 0.05, $p < 0.05$; As + extract 500: 0.90 ± 0.08, $p < 0.05$) compared to the arsenic-only group.

3.6. Histopathological Screening. Histopathological examinations were carried out on the liver, kidney, brain, and skin tissues of Swiss albino mice to explore the

Table 4: Effects of coriander extract on hematological parameters in Swiss albino mice.

Parameters	Control	Arsenic-treated	Extract-treated, 500 mg/kg	Arsenic + extract, 150 mg/kg	Arsenic + extract, 300 mg/kg	Arsenic + extract, 500 mg/kg
WBC ($\times 10^9$ cells/L)	7.83 ± 0.54	3.30 ± 0.32*	8.05 ± 0.55*	6.05 ± 0.53*	6.20 ± 0.44*	6.40 ± 0.21*
RBC (cells/μL)	5.36 ± 0.09	4.26 ± 0.06	5.10 ± 0.08	4.98 ± 0.11	5.06 ± 0.12	5.06 ± 0.12
NEU (cells/μL)	54.00 ± 3.08	59.33 ± 1.45	47.16 ± 2.57	51.16 ± 1.83*	51.83 ± 2.98*	53.66 ± 2.15*
LYM ($\times 10^9$ cells/L)	39.50 ± 3.53	33.16 ± 1.13	36.50 ± 1.87	39.16 ± 1.32*	41.33 ± 3.40*	41.83 ± 1.75*
MON (cells/L)	11.66 ± 0.87	3.83 ± 0.65	3.33 ± 0.33	3.16 ± 0.16	3.83 ± 0.90	3.83 ± 0.47
EOS (cells/L)	2.66 ± 0.33	4.16 ± 0.30	3.00 ± 0.36	3.50 ± 0.42	3.16 ± 0.26	3.00 ± 0.36

Note: Values are given as mean ± standard error of mean (SEM) for a group of six animals each.
* $p < 0.05$, significance level compared to control, extract-treated, arsenic-exposed, and As + extract–treated Swiss albino groups.

Table 5: Effects of coriander extract on biochemical parameters in Swiss albino mice.

Parameters	Control	Arsenic-treated	Extract-treated, 500 mg/kg	Arsenic + extract, 150 mg/kg	Arsenic + extract, 300 mg/kg	Arsenic + extract, 500 mg/kg
ALT (U/L)	56.83 ± 2.98	66.33 ± 4.07*	48.50 ± 3.83	65.83 ± 4.11	60.50 ± 3.51*	51.66 ± 2.18*
ALP (U/L)	145.02 ± 3.05	165.74 ± 9.51*	134.66 ± 2.24	160.66 ± 9.45*	154.16 ± 7.90*	150.00 ± 5.09*
CRE (mg/dL)	0.86 ± 0.08	1.01 ± 0.03*	0.75 ± 0.07	0.95 ± 0.08	0.90 ± 0.05*	0.90 ± 0.08*

Note: Values are given as mean ± standard error of mean (SEM) for a group of six animals each.
* $p < 0.05$, significance level compared to control, extract-treated, arsenic-exposed, and As + extract–treated *Swiss albino* groups.

consequences of arsenic exposure on tissue abnormalities. CE treatment at its different doses demonstrated remarkable amelioration of As-induced hepatotoxicity and brain injury, but no significant changes were observed in the kidney and skin tissues.

3.6.1. Histopathology of Liver Tissue. In liver histopathology, diverse treatments reveal discernible alterations, as shown in Figure 3. The findings of the current histopathological study on liver tissue represent that the control group manifests a typical liver architecture (A), while the mice subjected to arsenic exposure present indications of liver impairment, including sinusoidal dilation, and infiltration of inflammatory cells, which are indicative of hepatotoxic effects induced by arsenic (B). Administering coriander extract at different dosages (150, 300, and 500 mg/kg) showcases varying degrees of amelioration, featuring diminished fatty changes and reduced infiltration of inflammatory cells in comparison to the group exposed solely to arsenic. The administration of coriander extract of 150 mg/kg body weight to the As-treated mice exhibited improvement in arsenic-induced liver injury, which was indicated by the regeneration of hepatocytes along with reduced fatty changes (C). Mice treated with coriander 300 mg/kg body weight showed a moderate improvement in arsenic-induced liver injury characterized by normal hepatic structures and the absence of necrosis, along with reduced fatty changes (D). Treatment of As-treated mice with coriander 500 mg/kg body weight demonstrated significant improvement in arsenic-induced liver injury indicated by further reduction of fatty changes and diminished infiltration of inflammatory cells (E).

3.6.2. Histopathology of Brain Tissue. The findings of the brain histopathology examinations (Figure 4) displayed normal histoarchitecture of the brain section obtained from mice belonging to the control group (A), but the arsenic-treated group showed increased intracellular space and edematous change in the brain tissues (B). CE treatment on As-treated mice at 150 mg/kg dose exhibited slight amelioration of brain tissue injury which was evident by reduced intracellular space and edema (C). As-treated mice received a 300 mg/kg dose showing further improvement in their brain injury which was advocated by further reduction of intracellular space with no edema (D). As-treated mice that received the 500 mg/kg dose of CE revealed no edematous change in the brain tissue (E).

4. Discussion

GC-MS analysis is commonly used in drug discovery from medicinal plants to identify individual phytoconstituents. It has been a well-established approach to characterize unknown compounds due to its sensitivity, efficiency, and reproducibility [19, 35]. The sensitivity and specificity of GC-MS make this technique ideal for identifying minute quantities of metabolites (less than 650 Da) present in a complex mixture including fatty acids, sterols, amino acids, sugars, catecholamines, toxins, and drugs. This unique technique permits the discovery of novel compounds by integrating targeted absolute quantitative analysis of specific compounds with untargeted metabolomics [36]. Therefore, GC-MS was employed to characterize and quantify the phytoconstituents present in the coriander extract.

GC-MS analysis of coriander leaf extract is a novel study and enabled the identification and quantification of 59

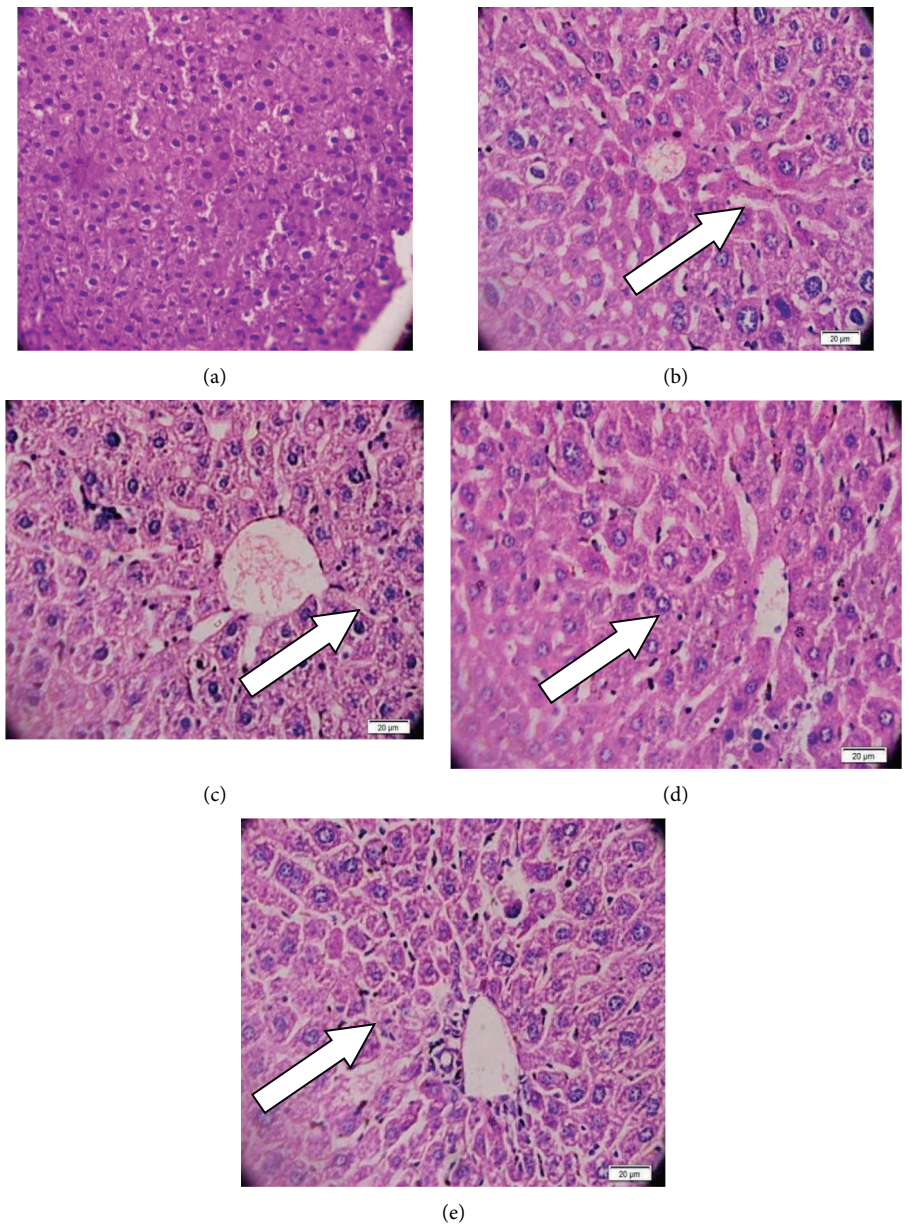


Figure 3: Microphotograph of liver sections taken from mice stained with hematoxylin and eosin (H&E; × 400). (a) Screening of liver tissue (control group); (b) screening of liver tissue (arsenic-treated); (c) screening of liver tissue (150 mg dose extract); (d) screening of liver tissue (300 mg dose extract); and (e) screening of liver tissue (500 mg extract).

compounds belonging to diverse phytochemical classes such as fatty acids, amides, diterpenes, alkanes, halogenated alkanes, fatty alcohols, benzofurans, triterpenes, tocopherols, phytosterols, organosilicons, steroids, terpenoid saponins, and flavonoids (Table 4). Based on % peak area, principal compound identified was 13-docosenamide, (Z)- (49.82%), followed by gamma-sitosterol (8.47%), phthalic acid, di(2-propylpentyl) ester *(4.28%),* decanamide (4.16%), stigmasterol (3.45%), tetradecanamide (3.33%), vitamin E (2.58%), 6,9-octadecadienoic acid, methyl ester (2.16%), neophytadiene *(1.59%),* gamma-tocopherol (1.06%), and phytol (0.98%) (Table 4). Compared to our study, prior research on GC-MS analysis of coriander seed extract (essential oil) identified 48 compounds, and linalool was the principal compound that contributed to 69.60%. Other major compounds characterized were geranyl acetate (4.99%), γ-terpinene (4.17%), α-pinene (1.63%), anethole (1.15%), and p-cymene (1.12%) [37]. This comparison clarifies that different parts of the coriander herb are rich in different types of phytoconstituents.

It is noteworthy to mention that literature search revealed a significant number of identified compounds with heavy metal toxicity ameliorating the potential and the crucial compounds are phenol, 2,6-bis(1,1-dimethylethyl)-, dodecane, 2,6,11-trimethyl-, 8,11,14-docosatrienoic acid, methyl ester, phytol, nonyl tetradecyl ether, dodecyl nonyl

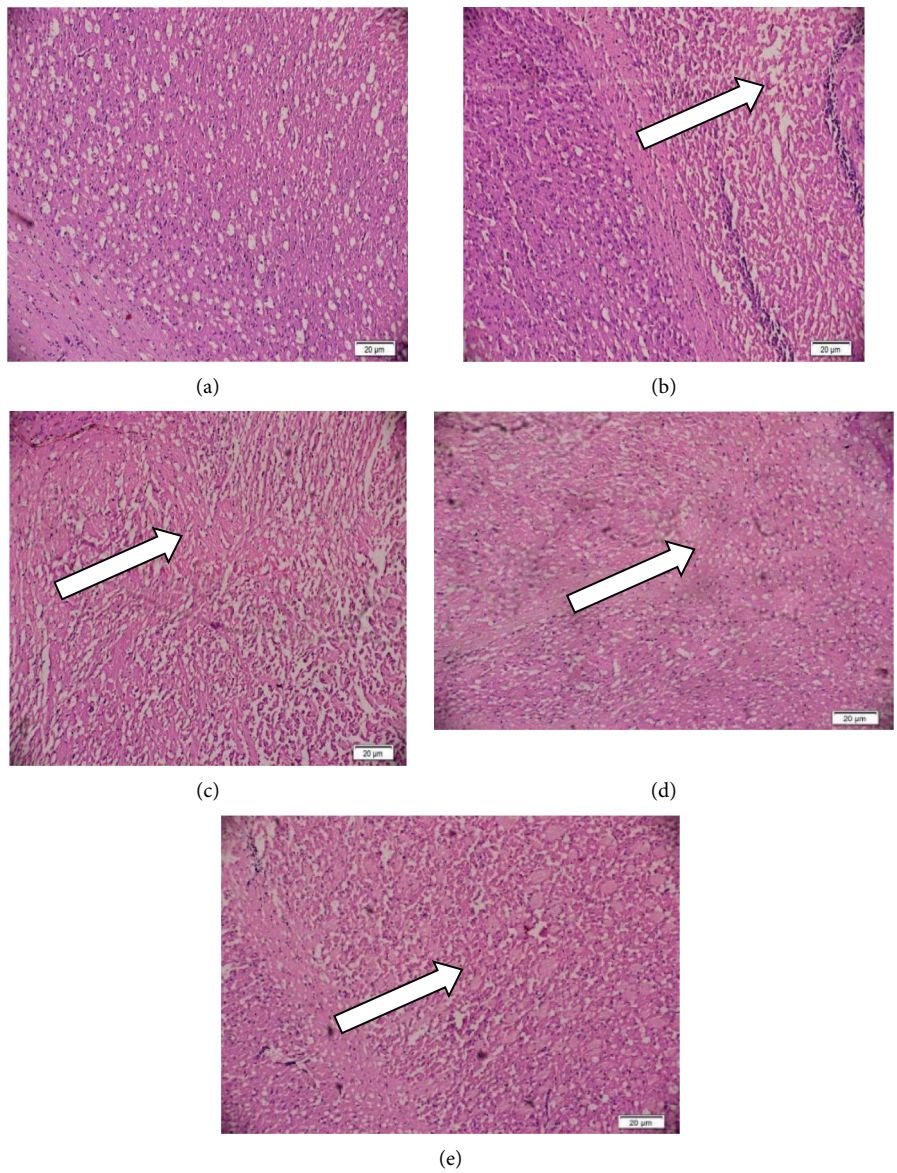


Figure 4: Microphotograph of brain sections taken from mice stained with hematoxylin and eosin (H&E; × 400). (a) Screening of brain tissue (control group); (b) screening of brain tissue (arsenic-treated); (c) screening of brain tissue (150 mg dose extract); (d) screening of brain tissue (300 mg dose extract); and (e) screening of brain tissue (500 mg extract).

ether, ginsenol, squalene, gamma-tocopherol, vitamin E (alpha-tocopherol), stigmasterol, and gamma-sitosterol (Table 5). The principal mechanisms of these compounds' heavy metal toxicity reduction involve metal-chelating effect, ROS scavenging, glutathione (GSH) level enhancement, and augmentation of biochemical parameters such as ALT, AST, and LDH (Table 3). Stigmasterol identified from coriander leaf in our study has been reported to be an effective metal chelator and lipid peroxide scavenger [38]. Squalene, another major compound characterized in coriander leaf extract, was found to act as a metal chelator against Cu^{+2} in a previous study conducted on zebrafish [39]. Ginsenol depleted heavy metal–induced toxicity in mice exposed to Cd by increasing ALT, AST, LDH, and GSH levels [31]. Therefore, the amelioration of As-induced toxicity in mice by coriander extract may be attributed to all these compounds identified in the GC-MS analysis.

Our research findings demonstrated the significant ameliorating effect of coriander leaf extract in arsenic-induced toxicity, evidenced by improvement of body weight, and hematological and biochemical parameters in mice. The coriander extract was found to protect against liver and brain toxicity developed in As-intoxicated mice by restoring normal histology of the liver and brain. GC-MS analysis of the extract identified and quantified 59 compounds belonging to different phytochemical classes, with several compounds reported for their heavy metal toxicity reduction effect. For instance, phytol, alpha-tocopherol, gamma-tocopherol, ginsenol, etc., have already been found effective in Cd-, Pb-, Cu-, and As-induced toxicity depletion

in prior research. Thus, it would benefit an individual's health if a certain quantity of coriander is taken as a dietary supplement or employed as an adjuvant with chelators in treating arsenic-induced toxicity.

Arsenic is one of the most common toxic metals in the environment, and its poisoning has become a major public health concern, affecting over 200 million people globally, particularly in Latin America and Asia [40]. Due to geological activities, arsenic is naturally found in groundwater; however, its use in pesticides and herbicides further increases health risks [41]. Millions of people are exposed to hazardous levels of arsenic via contaminated drinking water and food sources. Chromated copper arsenate was used to preserve wood due to its antifungal properties, but it was outlawed in the United States and Europe in 2004 for arsenic toxicity. Likewise, until recently, dimethyl arsenates and other chemicals containing arsenic were extensively employed as pesticides on fruit trees [42]. Arsenic contamination has impacted the health of millions of people in more than 70 countries, including Asia: China, India, Bangladesh, Taiwan, Nepal, and Vietnam. In South America, Chile and Argentina are among the most affected, while in Europe, Austria and Hungary have been significantly impacted [43]. In Bangladesh, arsenic contamination of groundwater has been the most extensive mass poisoning in history as per WHO reports. Chronic exposure to water with 500 mg/L of arsenic can lead to 1 in 10 individuals developing cancers such as skin, bladder, and lung cancer, posing a critical public health threat [44].

Arsenic significantly affects the respiratory and digestive systems as it enters the body through contaminated water, food, air, and skin. Once absorbed, it circulates through the bloodstream, reaching various tissues and leading to both acute and chronic diseases [40]. Recent research has linked arsenic exposure to the production of reactive oxygen species (ROS), which leads to DNA damage and tumor development. However, the complete mechanisms of disease initiation and progression are still not fully understood [5].

Among others, some common ROS refers to superoxide anion ($O_2\cdot-$), hydrogen peroxide (H_2O_2), hydroxyl radical ($\cdot OH$), peroxyl radical ($LOO\cdot$), and singlet oxygen ($^1O_2$). It is proposed that arsenic exposure disrupts the ubiquitin pathway in oxidative phosphorylation within mitochondria, leading to increased production of free radicals [45]. Diseases occur when the balance between the formation and elimination of ROS is disrupted and the progression of several diseases such as diabetes, cancer, metabolic disorders, and cardiovascular diseases are found to be linked to this imbalance producing oxidative stress in the body [46, 47]. One of the key approaches is to identify agents with strong antioxidant properties to reduce the ROS generation caused by arsenic exposure in the human body. In this study, methanolic extraction of coriander leaf revealed the presence of key phytochemicals such as alkaloids, carbohydrates, proteins, glycosides, and triterpenes (Table 1), which are crucial for mitigating the harmful effects of ROS and provide substantial health benefits. These phytochemicals are recognized for facilitating cell differentiation, stimulating detoxifying enzymes, promoting and accelerating DNA repair, increasing cancer cell apoptosis, and, particularly, providing antioxidant benefits [48]. Literature review indicated that the 14 examined phytochemicals have been identified for their antioxidant traits, among them are ascorbic acid and alpha-tocopherol [49]. Nevertheless, their effectiveness at the cellular level concerning chronic arsenic toxicity requires further validation. A significant number of previous studies identified heavy metal toxicity reduction by coriander extract. For instance, coriander reduced the toxicity caused by Pb, Cu, and Hg in rod shellfish [50]. The coriander seed extract has been reported for reducing lead levels in rat blood and leaves and stalks of this herb attenuated lead accumulation in the brain of mice and protected Pb-induced renal injury [51, 52]. Knowing the tremendous potential of coriander in other heavy metal toxicity amelioration, we attempted this study to evaluate its effect on As-induced toxicity in mice.

This research highlights weight variation observed across six distinct groups of Swiss albino mice subjected to different treatments over 8 weeks (Figure 1). The arsenic-treated group experienced weight loss, while the control group and the group administered 500 mg/kg of coriander extract showed a significant weight gain of approximately 13.34 and 11.84 g, respectively. Weight loss signifies a general deterioration in health. Messarah et al. previously mentioned that arsenic intoxication induces oxidative damage, primarily impacting essential organs such as the liver and kidneys in rats, accompanied by a significant elevation in liver enzymes [53]. The toxic effects of arsenic disrupt normal metabolism and growth, leading to excessive tissue protein breakdown and, consequently, weight loss in rats. The fall in body weight as seen in arsenic exposure mice may be linked to the deleterious effects of arsenic, which interfered with many metabolic processes, hindering growth and development [54]. The results of this study show that animals exposed to both arsenic and coriander extract experienced less weight loss compared to those exposed to arsenic alone. This suggests that the antioxidant properties of coriander had a protective effect, helping to prevent further deterioration of body mass. This perspective is reinforced by prior research indicating turmeric and *Phyllanthus emblica* leaf extracts, and both are recognized for possessing antioxidant and ROS-scavenging capabilities, effectively reducing arsenic-induced weight loss and developmental retardation [55, 56].

In this study, mice were treated with varying concentrations of coriander extract to assess its effect on ALT, ALP, and CRE levels after 8 weeks (Table 3). Arsenic administration resulted in elevated ALT and ALP levels, indicating liver damage. It hampers the integrity of the liver's plasma membrane, resulting in the leakage of ALT, AST, and ALP into the circulation [57]. A similar pattern was observed in previous studies, where arsenic-induced toxicity led to an increase in liver enzyme levels, weight loss, and kidney damage [53, 58]. This research indicates that the combined administration of coriander extract decreased the activity levels of ALT and ALP, simultaneously improving antioxidant capacity and CRE levels. ALT, AST, and ALP are evaluated to examine hepatic function and the other

complications related to kidneys, bones, gallbladder, and heart. ALT and AST are predominantly located in hepatic cells, and ALP is distributed across various tissues, including bones, intestines, kidneys, liver, and other organs [2]. Arsenic (iAsIII) undergoes biotransformation in the liver by binding to the thiol groups of liver enzymes and proteins, thereby disrupting the integrity of the plasma membrane and causing the release of AST, ALT, and ALP into the systemic circulation [57]. The findings from this study indicate that coriander extract mitigates the toxic effects of arsenic-induced hepatic injury, most likely due to its antioxidant properties. Arsenic administration also elevated serum CRE levels, indicating potential kidney damage and an increase in metabolic waste from skeletal muscles due to impaired glomerular filtration [59]. As indicated by this study, following the administration of a higher dose of coriander extract in arsenic-treated mice significantly reduced CRE levels.

Table 2 highlights the blood parameters, including WBCs, RBC, LYMs, NEUs, MONs, and EOSs count, and examines the hematological effects of arsenic and coriander extract over 8 weeks. The arsenic-treated group showed a significant decrease in WBC levels (3.30 ± 0.316, $p < 0.05$) compared to the control (7.83 ± 0.542). However, a 500 mg/kg dose of the extract alone significantly increased WBC levels (8.05 ± 0.55, $p < 0.05$). This finding is consistent with data reported by Ferzand et al. which indicate that the arsenic-treated group had a reduced level of WBC compared to the control group [60]. The proposed mechanism behind this could be the binding ability of arsenic to hemoglobin; consequently, arsenic accumulates in the bloodstream and causes significant changes in hematological parameters [58, 61]. Additionally, this buildup of arsenic in the blood may induce apoptosis in plasma cells, as examined by Rousselot et al., further contributing to the observed changes in hematological parameters [62]. On the contrary, several research studies reported that arsenic exposure treatment for arsenicosis is often associated with increased levels of RBCs, EOSs, NEUs, and MONs [63–65]. Our investigation revealed that RBC, NEU, and LYM counts diminished after arsenic treatment; however, subsequent administration of coriander extract increased these levels.

Histopathological analysis revealed arsenic-exposed mice with distinct liver damage, including sinusoidal dilation, edema, and inflammation in our study (Figure 2), and our results align with prior studies [66, 67]. In the current study, arsenic-intoxicated mice had brain injury which was evident by increased intracellular space and edematous condition in the brain tissue (Figure 3). Administration of coriander extract at different doses in As-exposed mice showed that coriander extract can reduce arsenic-induced toxicity. The liver and brain histopathology suggested that coriander extract helped restore normal liver and brain structure and function by stabilizing the membrane integrity. In a previous study, administration of coriander seed extract in arsenic-intoxicated mice showed significant restoration of the normal architecture of liver tissue [2]. The current study's findings are congruent with prior research indicating arsenic accumulation in essential organs such as the liver, kidneys, lungs, and blood [54, 68, 69]. These improvements in liver and brain could be attributed to the high antioxidant levels in coriander extract, its ROS scavenging property, and its ability to form chelate complexes with arsenic, promoting its rapid elimination from the body [2, 70]. Methanolic extract of *Coriandrum sativum* displayed a suppressive effect on lead deposition in mice and subsequent amelioration of lead-induced toxicity in prior research due to its antioxidant and metal-chelating properties [71]. GC-MS analysis identified a wide range of compounds present in coriander with potential for heavy metal–induced toxicity reduction and the compounds include phenol, 2,6-bis(1,1-dimethylethyl)-, 8,11,14-docosatrienoic acid, methyl ester, phytol, vitamin E (alpha-tocopherol), gamma-tocopherol, squalene, and ginsenol (Table 5). The mechanism of heavy metal–induced toxicity reduction by these compounds involves free radical scavenging, reducing ROS, and oxidative stress (Table 5). In prior research, ginsenol effectively protected the liver against Cd-induced acute hepatic toxicity in mice through antioxidative and anti-apoptotic actions via decreasing the elevation of blood biochemical parameters and restoring the impairments of histopathology, ameliorating the depletion of GSH and inhibiting the expression of proapoptotic bad protein in liver tissue [31]. Alpha-tocopherol reduced Cd-induced oxidative stress in mice by increasing glutathione levels in a study conducted before [33]. Gamma-tocopherol provided mitochondrial protection against oxidative damage induced by arsenic in Wister rats [32].

Although the observed improvements in hepatic architecture and hematological parameters suggest a protective role of *C. sativum*, the specific mechanistic involvement of oxidative stress modulation remains speculative. Quantitative assessment of oxidative stress biomarkers such as MDA, SOD, CAT, GPx, GSH, and TAC was not conducted in this study and represents a limitation. The current study did not include direct quantification of arsenic accumulation in blood or tissues using techniques such as inductively coupled plasma mass spectrometry (ICP-MS) or atomic absorption spectroscopy (AAS). As a result, it is not possible to conclusively determine whether the observed protective effects of *C. sativum* were mediated through reduced arsenic uptake, increased chelation, or enhanced excretion.

While the current findings highlight the protective potential of *C. sativum* leaf extract against arsenic-induced toxicity, future studies should aim to substantiate the underlying mechanisms more directly. This includes incorporating quantitative assessments of oxidative stress biomarkers such as MDA, SOD, CAT, GPx, GSH, and TAC to validate antioxidant-mediated protection. Additionally, direct measurement of arsenic accumulation in blood and target organs using techniques like ICP-MS or AAS is recommended to evaluate the extract's pharmacokinetic or chelating activity. Such studies will provide stronger mechanistic insight and translational relevance for the therapeutic use of *C. sativum* in heavy metal toxicity. Moreover, the GC-MS-based compound identification in this study relied on spectral library matching (Wiley and

NIST databases) without MS/MS fragmentation or standard compound confirmation. Although the similarity indices were ≥ 90%, future work incorporating MS/MS confirmation and standard validation is warranted to ensure structural accuracy and quantification reliability.

5. Conclusion

The present study demonstrates that *C. sativum* (coriander) leaf extract exerts a protective role against arsenic-induced toxicity in Swiss albino mice. Phytochemical screening and GC-MS analysis revealed a diverse array of bioactive compounds, including fatty acids, sterols, and triterpenes, many of which are known to possess antioxidant and detoxifying properties. Administration of coriander extract at doses of 150, 300, and 500 mg/kg ameliorated arsenic-related alterations in hematological and biochemical parameters, with significant improvements observed at higher doses. The extract also improved histopathological architecture of liver and brain tissues, evidenced by reduced necrosis, fatty degeneration, inflammatory cell infiltration, and edema. Collectively, these findings suggest that coriander mitigates arsenic-induced systemic and organ-specific damage in a dose-dependent manner. The observed protective effects are likely mediated by the antioxidant capacity of coriander, which may counteract ROS generated during arsenic exposure, and potentially by chelation or reduced uptake of arsenic compounds. While these mechanisms remain plausible, they were not directly quantified in this study. Future work should include assessments of oxidative stress biomarkers (MDA, SOD, CAT, GPx, GSH, and TAC) and measurement of arsenic accumulation in tissues using ICP-MS or AAS to elucidate the precise pathways involved. Such studies would provide mechanistic clarity and further strengthen the translational potential of coriander as a therapeutic or adjunctive agent.

Data Availability Statement

Data supporting the findings of this study are available from the corresponding author upon reasonable request.

Ethics Statement

All experimental protocols were approved by the Biosafety, Biosecurity and Ethical Committee, Faculty of Biological Sciences, Jahangirnagar University, Savar, Dhaka, Bangladesh (Ref No. BBEC, JU/M 2020 (10)2).

Conflicts of Interest

The authors declare no conflicts of interest.

Author Contributions

The study was conceptualized and designed by Raushanara Akter; analysis of samples was performed by Masum Shahriar, and Asef Raj; data were analyzed and interpreted by Masum Shahriar and Raushanara Akter; the manuscript was written by Raushanara Akter, Luluel Maknun Fariha, Fouzia Noor, and Hasan Shahriyer Tonmoy; the manuscript was revised by Raushanara Akter, Luluel Maknun Fariha, and Asef Raj.

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