J.Agric.Chem.and Biotechn., Mansoura Univ.Vol. 6 (10): 393 - 405, 2015

PROTECTIVE EFFECT OF GINGER OIL AGAINST THIOACETAMIDE- INDUCED LIVER CIRRHOSIS IN MALE RATS.

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ABSTRACT

The viability and efficiency of ginger oil against liver cirrhosis caused by Thioacetamide (TAA) in rats were investigated. Ginger oil having phenolic content of 85.3 mg g^{-1} as gallic acid, was extracted from dried ginger rhizome. Chemical composition of the oil was identified by GC-Ms .The major compounds were 1,8-Cineole 16.58% , Zingiberene 10.94%, α- Curcumen 7.66%, β-Sesqu-iphellandrene 6.26%, Camphene 5.93%, Farnesene 4.54%, Linalool 2.76% and β-Myrcene 2.18%. A biological experiment was done at the end. Evaluation of liver and kidney functions were measured. Protein and lipid profiles as well as antioxidant markers were also studied. The obtained results revealed that administration with ginger oil caused positive protective effect against the TAA-induced oxidative damage in liver.
keywords:Zingiber *officinale* Roscoe;Ginger oil,Antioxidant,Thioacetamide, cirrhosis

INTRODUCTION

Many natural plant products are used to remedy several diseases, oil extracted from ginger (Zingiber *officinale* Roscoe) , is one of them used for disease treatment (El-Sharaky et al., 2019). It is obtained from ginger stems or rhizomes, each of which is consumed by humans (Altman and Marcussen, 2001). Ginger oil had phenolic content of 120 g kg-1 (El-Baroty et al., 2010). According to El-Ghorab et al. (2010), dried ginger essential oil showed antioxidant activity by (DPPH method) of 83.87% while fresh ginger essential oil showed activity of 83.03. They added that volatile oils of fresh and dried ginger contain major compounds as camphene, ρ-cineole, α- terpineol, zingiberene and pentadecanoic acid .

Liver injury is caused by different agents, such as virus, chemicals, alcohol, and auto-immune diseases. According to Poli (2000) hepatotoxicity of thioacetamide (TAA) results from metabolic conversion to free radicals: thioacetamide sulfoxide and thioacetamide-S, S-dioxide which attacks mircrosomal lipids leading to their peroxidation, and production of reactive oxygen species (ROS), such as the H_2O_2, super oxide anion O_2^- ; ROS affects the antioxidant defense mechanisms, activity of SOD, CAT and GPX that cause liver injury, cirrhosis, and hepato-carcinoma. Bassiouny et al. (2011) stated that part of liver injury occurs by TAA and is mediated through oxidative stress and free radicals released as a consequence of TAA metabolism in liver cells activated by fibroblasts which secrete fibrinogen and growth factors. Salama et al. (2013) noted protective effects of antioxidant against induced liver injury by reducing oxidative stress in cell

The present study was aimed at assessing the antioxidant effect of ginger oil, its chemical composition and hepato-protective effect through biological tests on liver and kidney functions, lipid profile and antioxidant status against TAA harmful effects.

MATERIALS AND METHODS

Materials

Ginger powder obtained from local market at Zagazig city , Egypt.

2,2-diphenylpicrylhydrazyl (DPPH) chemical reagent (Aldrich®), Folin-ciocalteu reagent (Sigma®) and diagnostic kits from Bio Meriêuex Laboratory Reagents and Products, France.

Methods

Oil extraction. The volatile oil was extracted using hydro-distillation (El-Ghorab, 2010**)**. A sample 100 g was placed in a Clevenger apparatus followed by adding distilled water, then heated for 3 h. Oil was collected after drying over anhydrous sodium sulphate and stored at -18 °C.

Determination of total Phenolic Contents. Total phenolic content was determined using Folin-Ciocalteu reagent with gallic acid as stander (Singleton and Rossi, 1965).

Determination of antioxidant activity: Free radical activity of the ginger oil was determined using 2, 2-diphenylpicrylhydrazyl (DPPH) (Lee *et al.*, 2002).

Gas Chromatographic-mass Spectrometric Analysis (GC-MS):

GC/MS analysis was carried out using a Thermo Scientific gas chromatograph Model TRACE 1310 coupled with a mass spectrometer (ISQ LT Single Quadrupole, Thermo Scientific. An aliquot of 1.0 µl of oil was injected into GC-MS and separation was performed under the following conditions: DB-1 column (30 m×0.25 mm, film thickness 0.25 µm); helium as a carrier gas, at a constant flow rate of 10 mL min^{-1}; ionization potential 70 eV; and scan range 40 to 300 amu. Oven temperature was 40 to 250°C at a rate of 5°C min^{-1} and injector temperature of 250°C. Identification of essential oil was made by comparison the mass spectra with NIST and WILEY Mass Spectral Databases as well as comparison of the retention indices with those given in literature and those of authentic samples **(**Adams, 2009).

Experimental animals

The experimental animals were healthy adult male albino rats (Wister Strain) of approximately same age, each weighing 120 to 140 g, purchased from the farm of the Organization of Biological Products and Vaccines, Helwan , Egypt. Experimental procedures were conducted in conformity with the institutional guidelines and the Guidelines for Care and Use of Laboratory Animals in Biomedical Research of the WHO (World Health Organization). Animals were housed under ambient temperature of 25°C with 50 % relative humidity and a 12-h light-dark cycle ; and were allowed free access to water and fed on standard diet (Reeves et al., 1993).

Treatments of the experiment:

Twenty eight rats were used. There were 4 treatments each treatment group consisted of 7 rats. Group 1, a negative control received basal diet .

Group 2, a positive control was injected intra-peritoneally (ip) with TAA at a 200 mg kg-$^{-1}$ b.w . Group 3 was injected (ip) with TAA at 200 mg kg^{-1} b.w, along with 100 mg ginger oil kg^{-1} b.w. given orally through gastric gavages. Group 4 was injected (ip) with TAA at 200 mg kg^{-1} b.w. along with 200 mg kg-1 b.w. given orally. Duration of experiment was 8 weeks.

Biochemical determination in blood.

Blood samples were collected at end of experiment obtained from the retro-orbital plexus veins from individual rats by means of fine capillary heparinized tubes, and were allowed to clot. Serum was separated by centrifugation at 3000 rpm for 15 min. and was used to investigate the biochemical parameters including function tests of liver and kidney tests and serum lipid profile. Determinations were done on activities of liver enzymes of alanine transaminase (ALT), aspartate transaminase (AST), alkaline phosphatase *(*ALP*)* as well as serum total protein and serum albumin (Reitman and Frankel, 1957, Tietz., 1983, Doumas. (1975), and Doumas et al., 1971). Globulin was calculated by subtracting the albumin from serum total protein. Kidney function parameters of urea, uric acid and creatinine were determined (Tabacco et al., 1979). Lipid profile of total lipids (TL), triglycerides (TAG), total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), and very low density lipoprotein cholesterol (VLDL-C) were all determined (Fossati and Precipe, 1982).

Antioxidant markers

Liver samples were washed immediately with ice-cold saline to remove excess blood. Liver tissue was homogenized in cold 0.1M potassium phosphate saline (pH =7.4) at extraction ratio of 1:9 w/v. The homogenate was centrifuged at 5000 rpm for 10 min at 4 ˚C, then the supernatant was analyzed for antioxidant markers. Determinations were done on lipid peroxides "malondialdehyde, MDA" (Uchiyama and Mihara, 1978), glutathione peroxidase (GPx), measured spectro-photometrically using Ellman's reagent "DTNB" (Moron et al., 1979) .Glutathione-S-transferase (GST) activity was determined using aromatic substrate by monitoring change in absorbance due to thioether formation (Habig et al., 1974).

Histopathological examination

Small tissue specimens were collected from fresh liver and rapidly fixed in 10% neutral buffered formalin. After proper fixation, thin paraffin sections were routinely prepared and stained with Hematoxylin and Eosin stain (H&E) for the histopathological lesions in hepatic and renal tissues. The liver sections were graded numerically to assess the degree of histopathological features of acute hepatic injury. Hepatocyte necrosis, fatty change, hyaline degeneration, ballooning degeneration, and infiltration of Kupffer cells and lymphocytes were prominent in the histological findings (Drury and Wallington, 1986).

Statistical analysis

All data were analyzed by one-way ANOVA and statistical analyses were performed using the statistical software SPSS 11.0 (SPSS Ltd., Surrey, UK). Ratio values were not arcsine transformed before statistical analysis (SAS, 1996).

RESULTS AND DISCUSSION

Oil yield and phenolic content

Data of total volatile essential oil content of dried ginger extracted by hydrodistillation are shown in Table 1 .The 1.73 % of essential oil content in dried ginger in the current study agree with content reported by El-Ghorab, (2010) who found 1.1% in dried ginger. Total phenolic contents of ginger oil was 85.3 mg g^{-1} as gallic acid. These results are in agreement with those obtained by El-Baroty et al. (2010**)**.

Table 1: Oil and phenolic content and antioxidant activity of ginger oil.

Material / Parameter	Ginger powder
% Oil	1.73±0.09
*Total phenolic content mg g^{-1}	85.3±0.45
*%Antioxidant activity	75.61+0.93

***Results calculated on the dry weight basis**

Antioxidant capacity

Antioxidants react with DPPH (a stable free radical) to convert it into 1.1-diphenyl-2- picrylhydrazine. The degree of discoloration indicates the radical scavenging potential of the antioxidant (Maizura et al., 2010). Ginger oil contained high antioxidant activity of 75.61% which agree with those reported by El-Ghorab et al. (2010) who reported activity of 83.87%.

Chemical analysis of ginger essential oil

Chemical composition of oil (Table 2 and Figure1) shows 77chemical constituents identified by GC-MS analysis .Six major components are identified i.e 1,8- Cineole (16.58%), Zingiberene (10.94%), α- Curcumen (7.66%), β-Sesquiphellandrene (6.26%), Camphene (5.93%) and Farnesene (4.54%). They form 51.91 % of total oil constituents. These results are in agreement with those mentioned by Singh et al., (2008) and Rashidian et al., (2014) who gave approximate values for zingiberene 9.50% and α-Curcumen 6.6%. However, they found higher value for farnesene 7.6%

Table 2 : Chemical composition of ginger essential oil (analyzed by GC-Ms)

peak	R.Time	I.Time	F.Time	Area%	Name
1	8.180	8.085	8.440	2.51	alpha.-Pinene
2	8.695	8.500	9.325	5.93	Camphene (CAS)
3	9.481	9.325	9.730	0.77	2-.BETA.-PINENE
4	9.959	9.730	9.980	3.61	6-Methyl-5-hepten-2-one
5	10.027	9.980	10.135	2.18	.beta.-Myrcene
6	10.206	10.135	10.280	0.17	6-Methyl-hept-5-en-2-ol
7	10.388	10.280	10.505	1.71	.ALPHA.-PINENE, (-)-
8	10.550	10.505	10.670	0.16	.DELTA.3-Carene
9	10.750	10.670	10.945	0.50	(+)-2-CARENE
10	11.447	10.945	11.540	16.58	1,8-Cineole
11	11.589	11.540	11.720	0.27	Acetic acid, sec-octyl ester (CAS)
12	11.937	11.880	11.985	0.28	Melonal
13	12.090	12.045	12.140	0.36	.gamma.-Terpinene
14	12.508	12.450	12.605	0.20	LINALOOL OXIDE CIS
15	13.013	12.900	13.070	1.05	.ALPHA.-TERPINOLENE
16	13.136	13.070	13.220	0.64	2-Nonanone (CAS)
17	13.341	13.220	13.365	2.26	2-Cyclohexen-1-one,
18	13.376	13.365	13.390	0.34	2-pentyl 2-cyclopentene-1-one 2
19	13.473	13.390	13.505	2.76	Linalool
20	13.526	13.505	13.555	0.10	Nonanal (CAS)
21	13.793	13.755	13.815	0.15	METHYL BORNYL ETHER
22	13.871	13.815	13.930	0.40	Geranyl nitrile
23	14.063	13.995	14.155	0.16	cis-2-Pinanol
24	14.584	14.515	14.685	0.15	Bicyclo[3.1.1]heptan-3-ol,
25	14.748	14.685	14.820	0.48	Camphor (CAS)
26	14.880	14.820	14.945	0.35	exo-methyl-camphenilol
27	14.986	14.945	15.030	0.15	CITRONELLA
28	15.072	15.030	15.105	0.09	Bicyclo[2.2.1]heptane,
29	15.140	15.105	15.315	0.19	Camphol
30	15.540	15.315	15.640	3.99	endo-Borneol
31	15.702	15.640	15.735	0.35	2-(2\',3\'-Epoxy-3\'-methylbutyl)-3- methylfuran
32	15.791	15.735	15.855	0.54	1-Terpinen-4-ol
33	16.042	15.990	16.115	0.20	5,5,8-Trimethyl-nona-3,6,7-trien-2-one
34	16.243	16.115	16.295	1.74	p-Menth-1-en-8-ol
35	16.349	16.295	16.445	0.27	1-Iodo-2-methylundecane
36	16.566	16.485	16.625	0.45	Decanal (CAS)
37	16.909	16.830	16.950	0.09	3,5-Dimethylpyrazole-1-methanol
38	17.280	17.170	17.370	0.75	Citronellol
39	17.456	17.370	17.545	0.12	Acetic acid, sec-octyl ester (CAS)
40	17.630	17.545	17.680	0.45	.beta.-Citral
41	18.053	17.915	18.160	1.08	GERANIOL
42	18.484	18.380	18.530	0.50	Z-Citral
43	18.611	18.530	18.700	0.16	PHELLANDRAL
45	19.094	19.015	19.180	0.57	2-Undecanone (CAS)
46	19.293	19.240	19.360	0.11	2-Tridecanol (CAS)
47	20.338	20.275	20.425	0.16	.delta.-Elemene (CAS)
48	20.684	20.605	20.930	0.62	.alpha.-Cubebene
49	21.148	21.075	21.250	0.64	1,2.ALPHA.,4-METHENOINDAN,
50	21.438	21.345	21.490	1.16	.alpha.-Copaene
51	21.552	21.490	21.610	0.53	Geranyl acetate

ContinueTable 2 : Chemical composition of ginger essential oil (analyzed by GC-Ms)

peak	R.Time	I.Time	F.Time	Area%	Name
52	21.861	21.720	21.915	1.35	.BETA. ELEMENE
53	22.170	22.045	22.510	0.82	Zingiberene (CAS)
54	22.591	22.510	22.680	0.20	trans-Caryophyllene
55	22.835	22.680	22.870	0.13	.beta.-Cubebene
57	23.219	23.185	23.290	0.12	HEPTANE, 6-METHYL-2-P-TOLYL-
58	23.340	23.290	23.355	0.11	(-)-.alpha.-Selinene
59	23.370	23.355	23.410	0.11	1-ISOPROPYL-4-METHYL-7-METHYLENE-1,2,3,4,4A,5,6
60	23.483	23.410	23.595	0.85	Farnesol
61	23.687	23.595	23.825	0.68	NEOALLOOCIMENE
62	24.071	23.915	24.115	0.87	Valencene (CAS)
63	24.341	24.115	24.475	7.66	ALPHA.-CURCUMEN
64	24.751	24.475	24.790	10.94	Zingiberene (CAS)
65	24.828	24.790	24.865	1.17	Naphthalene,
66	24.973	24.865	24.995	4.54	Farnesene (CAS)
67	25.022	24.995	25.075	1.93	.beta.-Bisabolene (CAS)
68	25.131	25.075	25.205	0.37	.gamma.-Cadinene (CAS)
69	25.413	25.205	25.460	6.26	.beta.-Sesquiphellandrene (CAS)
70	25.500	25.460	25.545	0.30	.alpha.-Patchoulene (CAS)
71	25.895	25.840	25.990	0.22	Elemol
72	26.137	26.045	26.215	0.44	Nerolidol
73	27.349	27.285	27.415	0.11	zingiberenol
74	27.757	27.655	27.815	0.14	sesquisabinene hydrate
75	29.239	29.170	29.315	0.12	6,10-Dodecadien-1-yn-3-ol,
76	34.671	34.465	35.645	0.17	Pentadecanoic acid (CAS)
77	38.457	38.175	38.920	0.37	9-Octadecenoic acid, (E)-

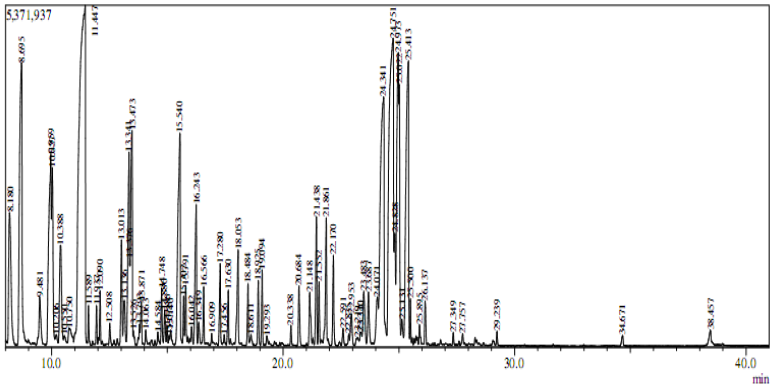


Fig1: Gas Chromatographic-mass Spectrometric Analysis (GC-MS) of ginger oil.

Effect of ginger oil on liver function and protein profile of rats having induced liver cirrhosis with thioacetamide .

Table 3 shows the effect of ginger oil on serum liver enzymes in TAA-intoxicated rats. Activities of AST, ALT, and ALP enzymes in groups 2 were significantly greater as compared with the normal diet rats (group 1). The TAA was reported to metabolize to thioacetamide-s-oxide by cytochrome-P450 enzyme system in liver (Mehul and varsha , 2012). Thioacetamide-s-oxide is responsible for the change in cell permeability. It increases

intracellular Ca^{++} nuclear volume, enlarges the nucleoli and inhibits of mitochondrial activity all of which leads to cell death

The marker enzymes leaked in to the serum resulting in elevation of their contents in serum. This would cause parenchymal damage. The release of ALT from mitochondrial and cytosolic localizations from membranal sites would cause cellular rupture which allows the enzyme to escape into the blood (Amin et al., 2012). Administration of ginger oil must have attenuated the elevated levels of AST, ALT, and ALP enzymes and therefore caused a recovery to normalization. Treatment with both doses of ginger oil significantly reduced the activities of serum AST, ALT, and ALP enzymes as compared with the positive control group of TAA. These results indicate that ginger oil nullified the toxicity of TAA in a dose-dependent manner. The 200-mg dose showed greater efficiency than the 100-mg of dose. The protective activity of ginger oil may have been due to its polyphenols which have many biological activities, such as scavenging free radicals and inhibiting lipid peroxidation.

Table 3: Effect of ginger oil on liver function of rats having TAA-induced liver cirrhosis

Group	Treatment	ALT (U mL^{-1})	AST (U mL^{-1})	ALP (U L^{-1})
1	Negative control (Normal Diet)	45.00 c	23.33 c	81.86 b
2	Positive control (TAA 200 mg/kg)	160.00 a	151.67 a	335.86 a
3	Ginger oil 100mg/kg +TAA 200mg/kg	58.33 b	45.00 b	99.64 b
4	Ginger oil 200mg/kg +TAA 200 mg/kg	45.00 c	30.00 bc	78.97 b

Notes: 1- No significant difference (P>0.05) between any two means with the same letter in each column. 2- ALT: alanine transaminase; AST: aspartate transaminase ; ALP: alkaline phosphatase

Effect of ginger oil on the protein profile.

Rats treated with TAA showed decreased total protein, albumin, globulin and A/G ratio (Fig 2). Treatment with 200-mg ginger oil showed protection against TAA due to changes in total protein, albumin, globulin and A/G ratio level. The decrease in total protein, albumin, globulin and A/G ratio may be due to disturbance in metabolism of carbohydrates, proteins and lipids or due to perturbed protein biosynthesis in the cirrhotic liver (Low et al., 2004).

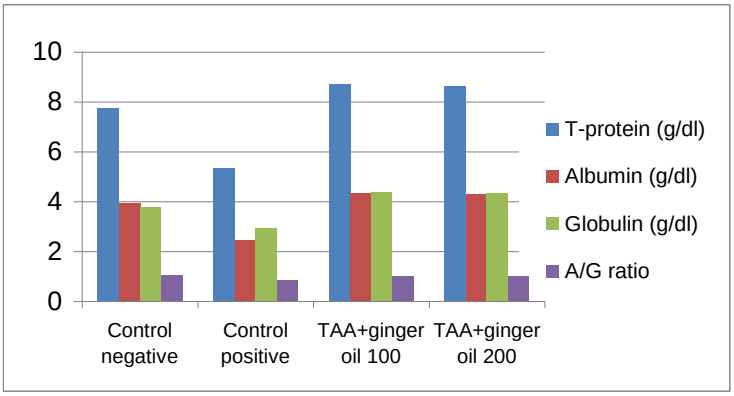


Fig 2 : Effect of ginger oil on protein profile of rats induced liver cirrhosiswith TAA.

Effect of ginger oil on kidney function.

Levels of urea, creatinine and uric acid in the negative control of group 1 were higher than those of the positive control of group 2 (Table 4) after 8 weeks of experiments. The ginger oil treatments showed levels lower than those of the positive control.

Table (4): Effect of ginger oil on kidney function of rats induced liver cirrhosis with TAA.

Group	Treatment	Urea (mg/dL)	creatinine (mg/100 mL)	Uric acid (mg/100mL)
1	Negative control (Normal Diet)	39.93 ^{b}	1.08 ^{b}	5.70 ^{b}
2	Positive control (TAA 200 mg/kg)	68.52 ^{a}	3.25 ^{a}	9.09 ^{a}
3	Ginger oil 100mg/kg +TAA 200mg/kg	48.95 ^{b}	1.36 ^{b}	5.63 ^{b}
4	Ginger oil 200mg/kg +TAA 200 mg/kg	41.68 ^{b}	1.25 ^{b}	6.62 ^{ab}

Notes: No significant difference (P>0.05) between any two means with the same letter in each column.

Effect of ginger oil on lipid profile.

TAA- treatment increases parameters of all lipids (TL, TAG, TC, LDL-C and VLDL-C) except that of HDL-C (Table 5 and Figure 3). Such values for the 200- mg oil treatment were lower as compared with the positive control treatment . The TAA-treatment decreased the level of HDL-C while treatment with both doses of ginger oil increased them. The hyperlipidaemia and hypercholesterolaemia of the rats treated with TAA caused by extrahepatic lipogenesis could be directly related to failure of TAA to induce cirrhosis. (David et al., 2002). A report by (Esteban et al., 1999) mentioned that PUFA or cholesterol dietary supplementation or enteral or pareteral lipids emulsion administration prevented necrosis and inflammation due to TAA.

Table 5: Effect of ginger oil on lipid profile of rats having TAA-induced liver cirrhosis

Group	Treatment	Total lipid (mg dL^{-1})	Triglyceride (mg dL^{-1})	Total cholesterol (mg dL^{-1})	HDL-Cholesterol (mg dL^{-1})	LDL-Cholesterol (mg dL^{-1})	VLDL-Cholesterol (mg dL^{-1})
1	Negative control (Normal Diet)	491.83 ^{b}	151.56 ^{bc}	193.56 ^{b}	39.38 ^{a}	123.87 ^{b}	30.31 ^{cb}
2	Positive control (TAA 200 mg/kg)	663.25 ^{a}	274.83 ^{a}	294.06 ^{a}	27.03 ^{b}	212.07 ^{a}	54.97 ^{a}
3	Ginger oil 100mg/kg +TAA 200mg/kg	538.77 ^{b}	170.74 ^{b}	197.95 ^{b}	43.46 ^{a}	120.33 ^{b}	34.15 ^{b}
4	Ginger oil 200mg/kg +TAA 200 mg/kg	495.91 ^{b}	142.85 ^{c}	203.68 ^{b}	42.05 ^{a}	133.05 ^{b}	28.57 ^{c}

Notes: No significant difference (P>0.05) between any two means with the same letter in each column.

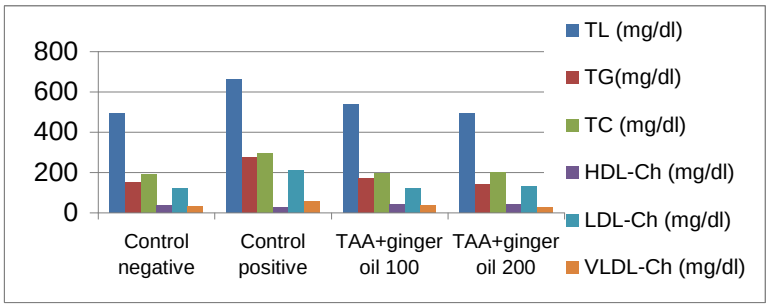


Fig 3: Effect of ginger oil on lipid profile of rats induced liver cirrhosis with TAA.

Effect of ginger oil on Antioxidant markers.

Levels of GPX and GST in the liver homogenates of TAA- rats given two doses of ginger oil were higher than in rats of the positive group . Oxidative stress parameters (liver tissue homogenate MDA) indicate that cirrhosis of the TAA- rats (with no ginger oil), had higher levels of oxidative stress biomarkers the others rats treated with two doses of ginger oil had lower levels of liver MDA compared with the cirrhosis rats of the positive control. Such results indicate that ginger oil can protect hepatic cells from further damage during cirrhosis.

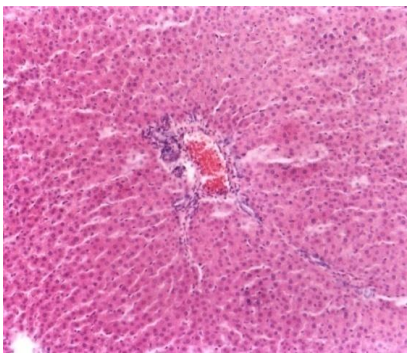
Table 6: Effect of ginger oil on antioxidant markers of rats having TAA-induced liver cirrhosis

Group	Treatment	MDA (mol/mg)	Glutathione peroxidase	glutathione-S-transferase
1	Negative control (Normal Diet)	3.55 ^{b}	2.64 ^{a}	3.50 ^{ab}
2	Positive control (TAA 200 mg/kg)	10.21 ^{a}	0.98 ^{c}	2.55 ^{c}
3	Ginger oil 100mg/kg +TAA 200mg/kg	3.95 ^{b}	1.97 ^{b}	3.26 ^{b}
4	Ginger oil 200mg/kg +TAA 200 mg/kg	3.78 ^{b}	2.70 ^{a}	3.61 ^{a}

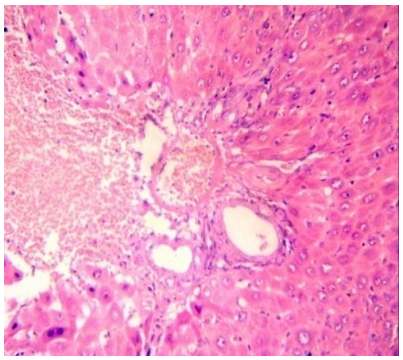
Notes: No significant difference (P>0.05) between any two means with the same letter in each column.

Liver histopathology

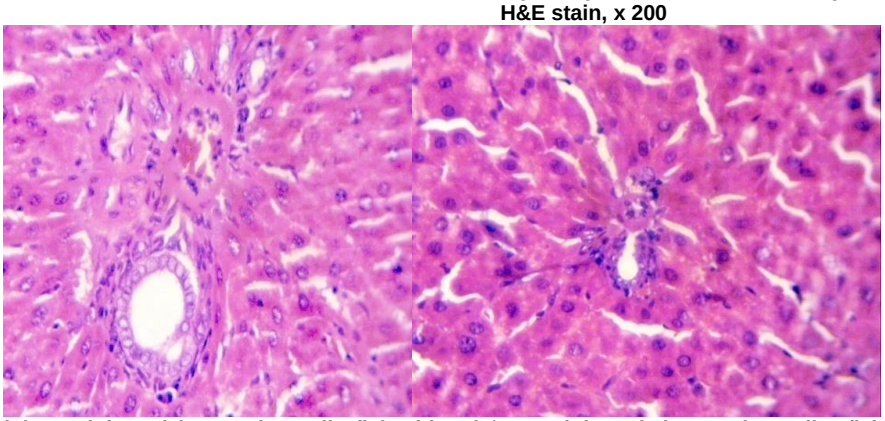
Histopathological analysis of the liver tissue (Figure 4 A-D) support the positive results exhibited by other data. Liver sections of the negative control rats showed nearly histologic hepatic tissues. Normal hepatocytes were arranged in cords around central vein. Small bile ducts lined by cuboidal epithelium with portal vessels were observed in the portal arid (Fig. 4 a). Compared with the normal liver tissues of vehicle controls, liver tissue in the TAA- rats revealed focal area of lytic necrosis characterized by loss of hepatic cord architecture and replacement of vacant space by aggregates of erythrocytes with few leukocytes (Figure 4B). Liver of rats injected ip with 200 mg TAA along with 100 mg ginger oil exhibited congestion of portal vein and mild hyperplasia of the lining epithelium of the bile ducts with newly formed bile ductless (Figure 4C). The liver of rats injected ip with 200 mg TAA and 200 mg ginger oil showed near normal hepatic parenchyma (Figure 4D).



(A) control rats showing normal histobasoligical structure (H&E x 200)



(B)rats injected intraperitoneally (ip) with TAA 200 mg kg^{-1} b.w showing focal area of lytic necrosis characterized by loss of hepatic cord architecture and replacement of vacant space by aggregates of erythrocytes with few leukocytes. H&E stain, x 200



(C)Rats injected intraperitoneally (ip) with TAA 200 mg kg^{-1} b.w and ginger oil 100 mg kg^{-1} b.w showing congestion of portal vein and mild hyperplasia of the lining epithelium of the bile ducts with newly formed bile ductless. H&E stain, x 400

(D)Rats injected intraperitoneally (ip) with TAA 200 mg kg^{-1} b.w and ginger oil 200 mg kg^{-1} b.w showing nearly normal hepatic parenchyma. H&E stain, x 400

Figure 4 :Histopathological photomicrographs of liver of rats.

CONCLUSION

Ginger oil with its high poly phenol content showed effective scavenging of DPPH. Treatment of 100 mg kg $^{-1}$ and 200 mg kg $^{-1}$ b.w exhibited a protective effect against TAA-induced liver cirrhosis indicating antioxidant effect of the oil causing inhibition of the deleterious effect of free radicals generated by TAA. Phenolics compounds and other constituents of the oil may have been responsible for such effects. Histological observations

support the biochemical data. Such results may lead to a recommendation of clinical use of ginger oil in treatment of hepatic disorders. The results suggest ginger oil ameliorate TAA-induced liver injury due to its antioxidant and radical scavenging characteristics.

Acknowledgements

Great thanks due to Prof. Dr. Abd el-basset El-mashed for his kind help and suggestion during the course of the histophathological part of this investigation.

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التأثير الوقائى لزيت الزنجبيل للتليف الكبدى الناتج من معاملة فئران التجارب بالثيواسيتاميد

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قسم الكيمياء الحيوية الزراعية – كلية الزراعة – جامعة بنها

تمت دراسة مدى فعالية زيت الزنجبيل ضد التليف الكبدى الناجم عن معاملة فئران التجارب بمركب الثيواسيتاميد. وجد ان زيت الزنجبيل المستخلص من جذور الزنجبيل الجافة يحتوى على 85,3 ميللجرام /جرام من المركبات الفينولية كحامض جاليك. كما تم التعرف على التركيب الكيميائى لزيت الزنجبيل بواسطة جهاز التحليل الكروماتوجرافى الغازى المزود بمطياف الكتلة وكان محتوى الزيت من المركبات الرئيسية كما يلى 1,8 سينول 16,58 % ، زنجبرين 10,94% ، الفا كيركيومين 7,66% ، بيتا سيسكو فيناندرين 6,26% ، الكامفين 5,93% ، الفارنسين 4,54% ، الليناالول 2,76% والبيتا ميرسين 2,18%. أستخدم ثمانية وعشرين من فئران التجارب لاجراء التجربة البيولوجية تم تقسيمها الى اربعة مجموعات كل مجموعة تحتوى على سبعة فئران . المجموعة الاولى وهى المجموعة الضابطة، المجموعة الثانية تم حقنها بـ 200 ملليجرام /كيلو جرام من وزن الجسم بالثيواسيتاميد، المجموعة الثالثة تم حقنها بـ 200 ملليجرام /كيلو جرام من وزن الجسم بالثيواسيتاميد والمعالجة بتجريعها 100 ملليجرام /كيلو جرام من وزن الجسم بزيت الزنجبيل. المجموعة الرابعة تم حقنها بـ 200 ملليجرام /كيلو جرام من وزن الجسم بالثيواسيتاميد والمعالجة بتجريعها 200 ملليجرام /كيلو جرام من وزن بزيت الزنجبيل.وفى نهاية التجربة (بعد 8 اسابيع) أظهرت نتائج وظائف الكبد والكلى وبرفيل الدهون والبروتين ونشاط إنزيمات مضادات الأكسدة على الدور الوقائى لزيت الزنجبيل ضد التليف الكبدى الناتج عن الثيواسيتاميد.