

ANTIOXIDANT AND HEPATOPROTECTIVE EFFECT OF *Cinnamomum zeylanicum* OIL AGAINST PARACETAMOL INDUCED HEPATOTOXICITY IN MALE RATE.

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ABSTRACT

The main objective of this study is a trial to investigate the efficiency of *cinnamomum zeylanicum* (cinnamon) oil against paracetamol induced hepatotoxicity in male rats. The bark cinnamon was hydrodistilled using Clevenger's apparatus to yield essential oil 2.3%. Total phenolic compounds and scavenging radical effect on 2, 2-diphenylpicrylhydrazyl (DPPH) were investigated. Gas chromatographic- mass spectroscopy fractionated cinnamon bark volatile oil to six components accounting for 100% of total amount. Cinnamaldehyde was found as the major component 68.01% followed by Eugenol 11.47, Cinnamyl alcohol 6.52% , α -murolene 5.24%, δ -cadinen 4.41% and Methyl eugenol 4.35%. Biological experiment was carried out for eight weeks. Results revealed that liver marker enzymes alanine transaminase (ALT) aspartate transaminase (AST), alkaline phosphatase (ALP) were increased. On the other hand serum total protein and serum albumin were decreased as well as, lipid profile of total lipids (TL), triglycerides (TAG), total cholesterol (TC), low density lipoprotein cholesterol (LDL-C) were increased and high density lipoprotein cholesterol (HDL-C) was decreased in paracetamol treated rats (group 2). Lipid peroxidation malondialdehyde (MDA) was increased in liver tissue of rats treated with paracetamol, while activities of glutathione peroxidase (Gpx) and Glutathione-S-transferase (GST) were decreased. Cinnamon oil both dose and silymarin showed remarkable hepatoprotective and antioxidant activity against paracetamol induced hepatotoxicity as judged from the serum marker enzymes and antioxidant in liver tissue.

keywords: *Cinnamomum zeylanicum*; cinnamon oil, phenolic, Antioxidant, hepatoprotective, paracetamol,

INTRODUCTION

Liver is a chief organ which regulates several metabolic drugs. The metabolism of these drugs caused excessive free radical. Hence, this organ is subject to variety of diseases and disorders. Iqbal Dar et al. (2012).

Paracetamol (acetaminophen, 4-hydroxy acetanilide) is a widely used as analgesic and anti-pyretic drug. However, drug is generally considered to be safe for use when administered in doses recommended, at over doses paracetamol produces acute liver damage (Parmar et al., 2010).

Paracetamol hepatotoxicity is caused by the reaction metabolite N-acetyl-p-benzoquinoneimine (NAPQI), which causes oxidative stress and glutathione depletion. (Mandade, 2011). Normally, toxic oxidation metabolites generated in the liver are converted into non-toxic metabolites excreted in urine via conjugation with glutathione (GSH) containing sulphhydryl groups. However, high doses of paracetamol limit the ability of GSH to detoxify

NAPQI and result in the consumption of liver GSH stores (Savides and Oehme, 1983).

Cinnamon (*cinnamomum zeylanicum*) belongs to family *Lauraceae* is one of the most important herbal drugs and has been widely used in Africa and Asia for more than 4000 years. It is obtained from the inner bark of several tree, cinnamon has been traditionally applied to the treatment of inflammatory disorders and gastric diseases (Shen et al., 2012).

(Singh et al., 2007) found that cinnamon leaves and barks were hydrodistilled and yield essential oils 3.1% and 2.5% respectively. El-Baroty et al. (2010) reported that cinnamon oil extracted from bark contains high amount of phenolic compounds (18.2 % of oil, expressed as eugenol equivalent). (Singh et al., 2007) examined the DPPH and hydroxyl radical scavenging activity of leaf and bark volatile oils with several concentrations and found DPPH radical 73.9% and 83.6% respectively and hydroxyl radical 72.2% and 79.6%, respectively.

(Kwshvari et al. 2013) analyzed chemical composition of cinnamon essential oil and they found the major compound was cinnamaldehyde (96.8%), while the minor component were α -capaene (0.2%), α -murolene (0.11%), *p*-methoxycinnamaldehyde (0.6%) and δ -cadinen (0.4%). Also El-Baroty et al. (2010) mentioned that cinnamon oil contained approximately 45.13% cinnamaldehyde, 8.21% cinnamyl alcohol, 7.47% Eugenol and 5.23% methyl eugenol.

Therefore, this research aims to evaluate the antioxidant effect of cinnamon oil, its chemical composition and hepatoprotective effect through biological tests on liver function, lipid profile and antioxidant status against high doses of paracetamol induced harmful effects.

MATERIALS AND METHODS

Materials

Cinnamon bark were obtained from local market at Zagazig governorate, Egypt.

Paracetamol and silymarin were provided as gifts from Sedico Pharmaceutical Company, Egypt.

2, 2-diphenylpicrylhydrazyl (DPPH) chemical reagent (Aldrich[®]), Folin-ciocalteu reagent (Sigma[®]). Diagnostic kits were obtained from Bio Meriêux 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).

Antiradical scavenging 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 \times 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 were allowed free access to water and fed on standard diet (Reeves et al., 1993).

Experimental design:

Thirty five rats were used. There were divided into 5 treatments each treatment group consisted of 7 rats. Group 1, a negative control received basal diet .Group 2, a positive control was received paracetamol (three times a week) at a dose 750 mg kg⁻¹ b.w. Group 3 was received paracetamol at a 750 mg kg⁻¹ b.w, simultaneously administered 50 mg silymarin kg⁻¹ b.w. given orally through gastric gavages (three times a week) for 56 days. Group 4 was received paracetamol at a 750 mg kg⁻¹ b.w, simultaneously administered 150 mg cinnamon oil kg⁻¹ b.w. given orally through gastric gavages (three times a week) for 56 days. Group 5 was received paracetamol at a 750 mg kg⁻¹ b.w, simultaneously administered 300 mg cinnamon oil kg⁻¹ b.w. given orally through gastric gavages (three times a week) for 56 days. Duration of experiment was 8 weeks.

Biochemical blood analyses.

Blood samples were collected at the 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. Lipid profile of total lipids (TL), triglycerides (TAG), total

cholesterol (TC), high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C) were determined according to the methods reported by Fossati and Precipe, 1982.

Assessment of hepatic oxidative stress biomarkers

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), was measured spectro-photometrically by using Ellman's reagent "DTNB" (Moron et al., 1979). Glutathione-S-transferase activity was determined using aromatic substrate by monitoring change in absorbance due to thioether formation (Habig et al., 1974).

Histopathology study.

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

Yield, total phenolic compounds and antioxidant activity of cinnamon

The data given in Table (1) show essential oil content in cinnamon bark (g/100g) , total phenolic content in volatile oil of cinnamon bark and antioxidant activity. The yield of cinnamon oil was found 2.3%. This result was nearly agreed with that obtained by Singh et al., 2007. Who found that cinnamon barks was hydrodistilled and yield essential oil 2.5%.

Total phenolic content in cinnamon oil manifested higher amount of phenolic content 15.18% as gallic acid. These results are in agreement with those obtained by El-Baroty et al. (2010) who reported that cinnamon oil extracted from bark contains high amount of phenolic compounds (18.2 % of oil, expressed as eugenol equivalent).

Table (1) shows the DPPH• scavenging activity of bark essential oil. The antioxidant activity of bark volatile oil was 83.44%. The obtained results are in agreement with (Singh et al., 2007).They reported that antioxidant activity of bark volatile oil was 83.6%

Table (1): Oil content of dried cinnamon bark and phenolic content and DPPH scavenging activity of cinnamon oil.

Parameter	Material	Cinnamon bark powder
% Oil		2.3 ±0.09
*%Total phenolic content		15.18±0.45
*%Antioxidant activity		83.44±0.93

*Results calculated on the dry weight basis

Chemical composition of cinnamon essential oil

GC-MS analysis of cinnamon bark volatile oil showed the presence of six components (Table 2). The main component was cinnamaldehyde 68.01%, followed by Eugenol 11.47, Cinnamyl alcohol 6.52% , α - murelone 5.24%, δ - cadinen 4.41% and Methyl eugenol 4.35%. The achieved results are in agreement with that mentioned by (Elshafie et al., 2012) who reported that GC-MS analysis of cinnamon oil indicated that cinnamaldehyde is main compound in concentration of 64.21%.

Table (2): Chemical composition of cinnamon oil analyzed by GC-MS

Peak	I.Time	F.Time	Area%	Name
1	5.23	7.56	68.01	Cinnamaldehyde
2	27.61	27.72	5.24	α - murelone
3	28.92	29.02	4.41	δ - cadinen
4	34.25	34.87	11.47	Eugenol
5	45.76	46.22	6.52	Cinnamyl alcohol
6	46.22	46.67	4.35	Methyl eugenol

Effect of Silymarin and different doses of cinnamon oil on the activities of serum AST, ALT and ALP levels in rats treated with paracetamol

Aspartate transaminase (AST) predominantly found in mitochondria of hepatocytes. Alanine transaminase (ALT) is more specific to liver, is one of the most sensitive tests employed in diagnosis of hepatic diseases and thus is a better parameter for detecting liver injury. Serum alkaline phosphatase (ALP) concentration is related to the functioning of hepatocytes, high level of Alp in the blood serum is related to increased synthesis of it by cell lining bile canaliculi usually in response to cholestasis and increased billiary pressure.The ALT, AST and ALP activity are largely used as most common biochemical markers to evaluate liver injury (Ayaz et al., 2012). Administration of paracetamol (750 mg/kg) to rats (Group2) exhibited abnormally higher activities of serum ALT,AST and ALP 156.00±2.64, 141.52 ±4.42 and 338.22±17.03, respectively when compared with negative control (Group1). These observations may be attributed to damage structural integrity of liver, because they are cytoplasmic in location and released into circulation after cellular damages indicating development of hepatotoxicity (Ayaz et al., 2012). Whilst, treatment with both doses of cinnamon oil 150 mg.kg⁻¹ and 300 mg.kg⁻¹ (group 4&5) or silymarin 50 mg.kg⁻¹ for 56 days led to prevent paracetamol-induced elevation in ALT, AST and ALP levels if compared to group 2 (Table3).This evidently suggests the protective effect of

cinnamon oil in improving the functional integrity of liver cells as a result containing high amount of phenolic compounds and antioxidant activity.

Table (3): Effect of Silymarin and different doses of cinnamon oil on liver function in rats treated with paracetamol .

Group	Treatment	ALT (U/ml)	AST (U/ml)	ALP (U/L)
1	Negative control (Normal Diet)	40.00±2.64 c	28.32±4.42 c	85.72±17.03 b
2	Positive control (paracetamol 750 mg.kg ⁻¹)	156.00±2.64 a	141.52 ±4.42 a	338.22±17.03 a
3	Silymarin 50 mg.kg ⁻¹ + paracetamol 750 mg.kg ⁻¹	46.00±2.64 bc	34.00 ±4.42 b	92.88±17.03 b
4	Cinnamon oil 150 mg.kg ⁻¹ + paracetamol 750 mg.kg ⁻¹	51.67±2.64 b	35.00±4.42 b	90.92±17.03 b
5	Cinnamon oil 300 mg.kg ⁻¹ + paracetamol 750 mg.kg ⁻¹	42.00±2.64 bc	30.00±4.42 bc	87.31±17.03 b

a, b, c, etc. There is no significant difference (P>0.05) between any two means with the same letter in each column.

Effect of Silymarin and different doses of cinnamon oil on total proteins, albumin and globulin content in rats treated with paracetamol

In the present investigation it was observed that the administration of paracetamol significantly (p< 0.05) decreased the levels of total proteins, albumin and globulin 5.43 ±0.31 g.dl⁻¹, 2.65 ±0.18 g.dl⁻¹ and 2.78 ±0.18 g.dl⁻¹, respectively if compared with normal control group1. Reduction of serum proteins, albumin and globulin in paracetamol treated group may be due to formation of protein adduct. Toxic metabolites NAPQI leads to covalent modification of cellular target protein, cell death and organ damage Parmar et al., 2010. However, treatment with both doses of cinnamon oil 150 mg.kg⁻¹ and 300 mg.kg⁻¹ (group 4&5) or silymarin 50 mg.kg⁻¹ for 56 days increased total proteins, albumin and globulin comparing with group 2 (Table 4) .

Table (4): Effect of Silymarin and different doses of cinnamon oil on protein profile in rats treated with paracetamol.

Group	Treatment	T-protein (g/dl)	Albumin (g/dl)	Globulin (g/dl)	A/G ratio
1	Negative control (Normal Diet)	8.25 ±0.31 a	4.05 ±0.18 a	4.20±0.18a	0.96±0.13a
2	Positive control (paracetamol 750 mg.kg ⁻¹)	5.43 ±0.31 b	2.65 ±0.18 b	2.78 ±0.18b	0.95±0.13a
3	Silymarin 50 mg.kg ⁻¹ + paracetamol 750 mg.kg ⁻¹	8.20 ±0.31 a	4.02 ±0.18 a	4.18 ±0.18b	0.96±0.13a
4	Cinnamon oil 150 mg.kg ⁻¹ + paracetamol 750 mg.kg ⁻¹	8.26 ±0.317 a	4.10 ±0.18 a	4.16 ±0.18a	0.99±0.13a
5	Cinnamon oil 300 mg.kg ⁻¹ + paracetamol 750 mg.kg ⁻¹	8.35 ±0.31 a	4.13 ±0.18 a	4.22 ±0.18a	0.98±0.13a

a, b, c, etc. There is no significant difference (P>0.05) between any two means with the same letter in each column.

Effect of Silymarin and different doses of cinnamon oil on lipid profile in rats treated with paracetamol

Data presented in Table (5) reveals that significantly (p< 0.05) increased in total lipid, triglyceride, total cholesterol and LDL-cholesterol 670.15±10.44, 276.25±7.51, 296.27±8.56, 214.95±6.63, respectively. In contrast significantly (p< 0.05) decreased HDL-cholesterol levels with rats treated paracetamol group 2 (Table5), thereby suggesting severe hepatic injury and confirming the hepatotoxic nature of paracetamol or may be due to the inhibition or destruction of triglycerides secretory mechanism by liver. But treatment with both doses of cinnamon oil was successful in blunting this paracetamol-induced increase in serum lipid profile (lipid, triglyceride, total

cholesterol and LDL-cholesterol) and a decrease in HDL-cholesterol level was observed when compared with silymarin as reference drug.

Table (5): Effect of Silymarin and different doses of cinnamon oil on lipid profile in rats treated with paracetamol.

Group	Treatment	Total lipid (mg/dl)	Triglyceride (mg/dl)	Total cholesterol (mg/dl)	HDL-Cholesterol (mg/dl)	LDL-Cholesterol (mg/dl)
1	Negative control (Normal Diet)	494.57±10.44 ^b	153.13±7.51 ^b	198.34±8.56 ^b	40.29±1.73 ^b	127.43±6.63 ^b
2	Positive control (paracetamol 750 mg.kg ⁻¹)	670.15±10.44 ^a	276.25±7.51 ^a	296.27±8.56 ^a	26.07±1.73 ^c	214.95±6.63 ^a
3	Silymarin 50 mg.kg ⁻¹ + paracetamol 750 mg.kg ⁻¹	491.22±10.44 ^b	153.15±7.51 ^b	200.51±8.56 ^b	41.12±1.73 ^b	128.76±6.63 ^b
4	Cinnamon oil 150 mg.kg ⁻¹ + paracetamol 750 mg.kg ⁻¹	498.46±10.44 ^b	154.89±7.51 ^b	206.51±8.56 ^b	41.30±1.73 ^b	134.23±6.63 ^b
5	Cinnamon oil 300 mg.kg ⁻¹ + paracetamol 750 mg.kg ⁻¹	490.81±10.44 ^b	147.34±7.51 ^{bc}	204.83±8.56 ^b	46.78±1.73 ^a	128.58±6.63 ^b

a, b, c, etc. There is no significant difference (P>0.05) between any two means with the same letter in each column.

Effect of Silymarin and different doses of cinnamon oil on lipid peroxidation and Antioxidant enzyme activities in rats treated with paracetamol.

Data found in Table (6) revealed, an elevation in the levels of MDA in liver tissue of rats treated with paracetamol was observed 11.05 ±0.46 mol.mg⁻¹(group 2) when comparing with control negative group (group1) The increase in lipid peroxidation (MDA) levels in liver induced by paracetamol suggests enhanced lipid peroxidation leading to tissue damage and failure of antioxidant defense mechanism to prevent formation of excessive free radical Kumar et al., 2010l. Treatment with both doses of cinnamon oil 150 and 300mg.kg⁻¹ (group 4 and5) significantly (p< 0.05) reduced the levels of MDA 4.57 ±0.46 and 3.92 ±0.46 mol.mg⁻¹ comparing with silymarin as reference drug. The observed protective effect of silymarin against lipid peroxidation could be related to its antioxidant effects which assist in the preservation of membrane integrity. Galal et al., 2012

Glutathione (GSH) is one of the most abundant tripeptide, non-enzymatic biological antioxidants present in liver. It removes free radical species such as hydrogen peroxide, superoxide radical and maintains membrane protein thiols. Decreased levels of glutathione peroxidase(Gpx) and glutathione-s-transferase (GST) were associated with an enhanced lipid peroxidation in paracetamol treated rats 0.98 ±0.07(U/g tissue) and 2.43±0.09(U/g tissue), respectively in group2. Moreover administration with both doses of cinnamon oil 150 and 300mg.kg⁻¹ (group 4 and5) significantly (p< 0.05) increased the level of GPX and GST.

At therapeutic levels, paracetamol is primarily metabolized in the liver by glucuronidation and sulphation; whilst, a small proportion undergoes cytochrom p 450 (CYP450)-mediated bioactivation to N-acetyl-p-benzoquinimine (NAPQI) , which is rapidly quenched by glutathione. After an over dose of paracetamol , elevated levels of toxic NAPQI metabolite are generated, which extensively deplete hepatocellular GSH and covalently modify cellular proteins resulting in hepatocyte death Ghosh et al., 2007

Table (6): Effect of Silymarin and different doses of cinnamon oil on Antioxidant markers in rats treated with paracetamol.

Group	Treatment	MDA (mol.mg ⁻¹)	Glutathione peroxidase (U/g tissue)	glutathione-S-transferase (U/g tissue)
1	Negative control (Normal Diet)	3.65 ±0.46 ^b	2.64 ±0.07 ^a	3.46±0.09 ^a
2	Positive control (paracetamol 750 mg.kg ⁻¹)	11.05 ±0.46 ^a	0.98 ±0.07 ^c	2.43±0.09 ^b
3	Silymarin 50 mg.kg ⁻¹ + paracetamol 750 mg.kg ⁻¹	4.11 ±0.46 ^b	1.95 ±0.07 ^b	3.49±0.09 ^a
4	Cinnamon oil 150 mg.kg ⁻¹ + paracetamol 750 mg.kg ⁻¹	4.57 ±0.46 ^b	1.97 ±0.07 ^b	3.48±0.09 ^a
5	Cinnamon oil 300 mg.kg ⁻¹ + paracetamol 750 mg.kg ⁻¹	3.92 ±0.46 ^b	2.70 ±0.07 ^a	3.54±0.09 ^a

a, b, c, etc. There is no significant difference (P>0.05) between any two means with the same letter in each column.

Histopathological findings

Microscopical examination of liver was carried out to compare with the effect of cinnamon oil treatments (Figure 1 a-e) the observations which could support the obtained positive results exhibited by other data. Liver sections from the negative control rats showed nearly normal histological 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 area (Fig. 1 a). Compared with the normal liver tissues of vehicle controls, liver tissue in the paracetamol revealed hyperplasia of the lining epithelium of the bile ducts with periportal mononuclear leukocytic cellular infiltration mainly lymphocytes (Figure 1b). Liver of rats received paracetamol 750 mg.kg⁻¹ showing congestion of theportal vein and mild hyperplasia of the lining epithelium of the bile ducts. Notice also, few periportal leukocytic cellular infiltration mainly lymphocytes (Figure 1c). The liver of rats received paracetamol at a 750 mg kg⁻¹ b.w, simultaneously administered 150 mg cinnamon oil kg⁻¹ b.w. given orally through gastric gavages (three times a week) for 56 days. Showing focal areas of coagulative necrosis of hepatocytes which was characterized by loss of cellular details, hypereosinophilic cytoplasm, with pyknotic nuclei. (Figure 1d). The liver of rats received paracetamol at a 750 mg kg⁻¹ b.w, simultaneously administered 300 mg cinnamon oil kg⁻¹ b.w. given orally through gastric gavages (three times a week) for 56 days, showing congestion of the portal vein with mild periductal fibrosis (Figure 1e).

Figure 1 (a, b, c,d & e): Photomicrographs showing histopathological effects of different treatment on liver of rats.

CONCLUSION

Generally, the result of the present study demonstrates that cinnamomum zeylanicum oil has potent hepatoprotection against paracetamol hepatic toxicity comparable to the standard drug silymarin 50 mg.kg⁻¹. The accomplished results illustrates that hepatoprotective effects of cinnamomum zeylanicum oil may be due to its antioxidant and free radical scavenging properties which attributable to the ability of its phenolic component to quench free radical.

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التأثير الوقائي والمضاد للاكسدة لزيت القرفة للتسمم الكبدى المحدث بواسطة الباراسيتامول فى ذكور الفئران عبدالله السيد عبدالله الحضري قسم الكيمياء الحيوية الزراعية - كلية الزراعة - جامعة بنها

الهدف الرئيسى من هذا البحث هو دراسة التأثير الوقائى لزيت القرفة للتسمم المحدث بواسطة الباراسيتامول فى ذكور الفئران. تم استخلاص زيت القرفة من القلف بواسطة جهاز كلفينجر وكانت نسبة الزيت 2,3%. تم تقدير نسبة الفينولات والنشاط المضاد للاكسدة فى زيت القرفة. كما تم التعرف على التركيب الكيمايى لزيت القرفة بواسطة جهاز التحليل الكروماتوجرافى الغازى المزود بمطياف الكتلة و يحتوى الزيت ستة مركبات رئيسية بنسبة 100% كما يلى. السينامالدهيد بنسبة 68,01%، الايجينول 11,47%، سيناميل الكحول 6,52%، الفا ميرولين 5,24%، دلتا كاديين 4,41% و ميتايل ايجينول 4,35%. استخدمت خمسة وثلاثون من فئران التجارب لاجراء التجربة البيولوجية تم تقسيمها الى خمسة مجموعات كل مجموعة تحتوى على سبعة فئران. المجموعة الاولى وهى المجموعة الضابطة، المجموعة الثانية تم تجريعها بـ 750 ملليجرام /كيلو جرام من وزن الجسم بالباراسيتامول ثلاث مرات اسبوعيا، المجموعات الثالثة والرابعة والخامسة تم تجريعها بـ 750 ملليجرام /كيلو جرام من وزن الجسم بالباراسيتامول بالاضافة الى علاجها بالسيلمارين بتركيز 50 ملليجرام /كيلو جرام من وزن الجسم ثلاث مرات اسبوعيا (المجموعة الثالثة) و زيت القرفة بتركيز 150 ملليجرام /كيلو جرام من وزن الجسم ثلاث مرات اسبوعيا (المجموعة الرابعة) و زيت القرفة بتركيز 300 ملليجرام /كيلو جرام من وزن الجسم ثلاث مرات اسبوعيا (المجموعة الخامسة) وبعد نهاية التجربة (56 يوم) تم الحصول على عينات الدم وتم تقدير نشاط انزيمات الكبد وانزيمات الاكسدة فى انسجة الكبد. وتم تدعيم ذلك بدراسة تشريح انسجة الكبد. وقد وجد زيادة مفرطة فى نشاط انزيمات الكبد الانين ترانس امينيز، اسبارتيت ترانس امينيز، والالكلين فوسفاتيز، وعلى النقيض نقص فى تركيز بروتينات الدم الكلبية والاليومين الكلى كما وجد زيادة مفرطة ايضا فى بروفيل الدهون (الدهون الكلبية، الجليسيريدات الثلاثية، الكليسترول الكلى، الليبوبروتينات منخفضة الكثافة) ونقص الليبروتينات عالية الكثافة فى الفئران المعاملة بالباراسيتامول (المجموعة الثانية) كما وجد زيادة فى فساد الدهون فى انسجة الكبد ونقص تركيز انزيمات الاكسدة فى انسجة الكبد (الجلوتاتيون بيروكسيدز والجلوتاتيون اس ترانس فيريز) وذلك فى الفئران المعاملة بالباراسيتامول. وأظهرت النتائج أنه يمكن استخدام السيلمارين او زيت القرفة بأى من التركيزين كمضاد أكسده ومصدر وقائى ضد التسمم المحدث بواسطة الباراسيتامول.

