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Original article

Protective Effect of Cinnamon and Ginger on Acrylamide Induced Hepatotoxicity in Adult Male Albino Rats

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ABSTRACT

Background: Acrylamide [ACR] is an essential chemical that is extensively used in many industries and also in laboratories such as treatment of drinking water, wastewater, and soil, production of paper, petroleum, mine, asphalt, dyes, adhesives, and polishes; it is known as a possible carcinogenic compound. Ginger oleoresin's hepatoprotective role can be attributed to its free radical scavenging, anti-inflammatory, and hypolipidemic activities and is partially mediated by the 6-gingerol, shogaol, and zingerone of its active component. Cinnamomum zeylanicum is used to treat many diseases and to prevent such diseases.

Aim of the work: Evaluation of the effects of cinnamon and ginger extracts administration on Hepatotoxicity adult male albino rats.

Materials and Methods: The groups were divided into five adult male albino rats of a local strain: Group I: acted as a control group with regular salines, group II: hepatotoxicity caused by acrylamid, groups III: hepatotoxicity plus cinnamon, group IV: hepatotoxicity plus ginger, group V: hepatotoxicity plus cinnamon and ginger. Samples for alanine aminotransferase [ALT], aspartate transfers of amino [AST], urea, creatinine, blood urea nitrogen (BUN), total cholesterol, triglyceride [TG], malondialdehyde, tumor necrosis factor α [TNF], superoxide dismutase [SOD], glutathione peroxidase [GPX], as well as total antioxidant [TAC] levels were obtained at the end of the experimental period.

Results: Administration of cinnamon and ginger to hepatotoxic rats led to a significant decrease in the mean value of ALT, AST, urea, creatinine, BUN, MDA, and TNF. In addition, it is associated with a significant increase of SOD, GPX, and TAC.

Conclusion: Cinnamon and ginger have a protective effect against abnormalities in hepatotoxic rats due to its several protective properties.

Keywords: Chronic Rhinosinusitis; Eustachian tube dysfunction; Balloon dilatation; Function endoscopic sinus surgery; Eustachian Tube Dysfunction Questionnaire.

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* Main subject and any subcategories have been classified according to the research topic.

INTRODUCTION

The liver is a major body detoxifying organ. It plays an important role in controlling most concentrations of metabolites, particularly serum ASTs and ALTs, which are used in the diagnosis of liver disease as sensitive biomarkers [1].

Acrylamide [ACR] is a hydrophilic molecule that's very small. In other words, passive diffusion conveniently takes it across the body. Oxidative stress, genotoxicity, neurotoxicity, cancer, developmental disorders, and reproductive toxicity have been shown to trigger [2]. The vinyl monomer of acrylamide is water-soluble and has a range of chemical and industrial uses, e.g., wastewater management or treatment. It is present in a high concentration in potatoes, rice, carrots, Chinese cabbage, lettuce, parsley, onions, spinach, and sugar and olives [3].

Ginger [*Zingiber officinale*] of the Zingiberaceae family is generally recognized as a medicinal herb as anti-inflammatory, antioxidant, antimicrobial, antiviral and antifungal, cardioprotective, anti-cancer, and chemopreventive. Gingerol and shogaol are the main ginger-derived components that display lipid peroxidation and metabolic changes. For obese women, weight loss and insulin, leptin resistance, and glucose decreases were promoted[4]. Oleoresin Ginger is dark golden brown viscous oil. It consists of essential oils, organic soluble resins, and other non-volatile pungent components, which comprised homologous series of phenolic ketones. 6-gingerol was effective in preventing hepatic and renal damage by reducing oxidative stress. 6-Gingerol-rich fraction lowers total bilirubin and further restores alanine aminotransferase [ALT], aspartate aminotransferase [AST], alkaline phosphatase [ALP], and gamma glutaryl transpeptidase [GGT] enzymes that sensitive indicators of hepatocellular damage and dysfunction[5].

Cinnamon, an everlasting tropical herb, belongs to the family of Lauraceae. It consists mainly of essential oils, cinnamic acid, cinnamaldehyde and cinnamate, and others. In order to use them in the prevention and treatment of different diseases, the beneficial medicinal properties of cinnamon are measured. These characteristics can be applied to treat some inflammatory conditions thanks to vast

therapeutic strategies such as antioxidant, anti-inflammatory, antidiabetic, anticancer, cardiovascular, and lipid-lowering properties for cinnamon. By eliminating oxidative stress, cinnamon reduces organ harm[6].

AIM OF THE WORK

The purpose of this research was to evaluate the impact on acrylamide-induced hepatotoxicity of cinnamon and ginger in adult male albino rats.

MATERIALS AND METHODS

Animals and experimental design: Fifty adult male local strain albino rats were brought from the center of the Nile, Mansura, Egypt, and selected to be the current research model. All of the rats were around the same age and healthy, ranging in weight from 140 to 160 g [150 g average weight]. At room temperature and good ventilation, they were housed in stainless steel cages [25 × 35 × 25 cm for every five rats], and they also earned diet water and commissural rat chow. They were left in the laboratory room for 14 days before the beginning of the acclimatization experiment. Rats were split into equal groups of 5 as follows;

The control group (Group I): Rats fed on normal rat chow diet for four weeks.

Hepatotoxic group (Group II): Rats received oral administration of acrylamide at a dose of 50 mg/kg body weight for four weeks[7].

Hepatotoxic plus Cinnamon group (Group III): Rats received oral administration of acrylamide at a dose of 50mg/kg body weight for four weeks after the 4th week, rats received 400mg/kg cinnamon extract by oral gavage daily for five weeks[8].

Hepatotoxic plus ginger group (Group IV): Rats received oral administration of acrylamide at a dose of 50 mg/kg body weight for four weeks. After the 4th week, rats received 400 mg/kg ginger extract by oral gavage daily for five weeks[9].

Group V [hepatotoxic plus cinnamon and ginger group]: Rats received oral administration of acrylamide at a dose of 50 mg/kg body weight for four weeks. After the 4th week, rats received cinnamon and ginger extracts by oral gavage daily for five weeks

Induction of hepatotoxicity: Pure [99%] acrylamide was brought from Sigma Chemical Company [Egypt, Eltayaran st., Nasr city-Cairo]. Acrylamide [50 mg/kg/day] was dissolved in 10 ml distilled water, so each 1 ml distilled water contained 5 ml acrylamide. Each rat was given distilled water containing acrylamide by weight and orally administered to rats for four weeks via gastric intubation^[7].

Cinnamon extract preparation: Cinnamon powder was obtained in New Damietta from a local market. 10 g were then weighed and mixed with 100 ml of water and held for two hours in a water bath at 60°C, then filtered by 150 µ Capron silica fabric. These diluted extracts [water 1:10] were administered by a feeding needle [oral gavage] to rats. The philtre was kept in the dark bottles at [4°C] in the refrigerator. Such procedures are replicated every week^[10].

Ginger extract preparation: Ginger powder was purchased from a local market in New Damietta. Then 10 g was soaked for 6 hours in 100 ml [10 percent] hot water [88 C] in a water bath. Then 150 µ of Capron silica cloth is filtered. These diluted extracts [water 1:10] were administered by a feeding needle [oral gavage] to rats. The philtre was kept in the dark bottles at [4°C] in the refrigerator. Such procedures are replicated every week^[11].

Ethyl Ether [Analar, Nile Pharmaceutical]: For anesthesia

Collection of Blood and tissue: All rats were fasted overnight at the end of the experimental duration and anesthetized by putting them in an anesthetic box filled with ether vapor. Ether vapor was preserved by adding liquid ether to the cotton wool at the box's base periodically. Using the heparinized capillary tube, blood was extracted from the retro-orbital plexus, left to clot to acquire serum, and centrifuged for 10 minutes at a rate of 5000 per minute. Serum in Eppendorf tubes was sucked out and deposited frozen at -20°C^[12]. Aspartate aminotransferase [AST], alanine aminotransferase [ALT]^[13], urea^[14], creatinine^[15], blood urea nitrogen [BUN]^[16], total cholesterol^[17], TG^[18], malondialdehyde^[19], tumor necrosis factor-α [TNF-α]^[20], superoxide dismutase [SOD]^[21], glutathione peroxidase [GPX]^[22] and total antioxidant capacity [TAC]^[23] were assayed.

Statistical analysis: The data's input and analysis was performed using the computer program Statistical Package for Social Sciences [SPSS] version 19. All outcomes were expressed as mean ± standard deviation. The mean values of the various groups were compared using one-way variance analysis or student t-tests. A post hoc analysis of the least significant difference [LSD] was used to identify significantly different mean values. To denote a significant difference, a p-value < 0.05 was accepted.

RESULTS

Results of the current study showed that oral administration of acrylamide at a dose of 50 mg/kg body weight for four weeks resulted in a statistically significant increase in ALT, AST, urea, creatinine, BUN, MDA, TNF associated with a significant decrease in SOD, GPX, and TAC, as well as an insignificant increase in TC and TG in Group II compared to Group I [Table 1]. Cinnamon administration to hepatotoxic rats resulted in a significant decrease in mean ALT, AST, urea, creatinine, BUN, MDA, TNF values associated with a significant increase in SOD, GPX, and TAC, in addition to an insignificant decrease in TC and TG levels in group III compared to group II. Ginger administration to hepatotoxic rats also resulted in a significant reduction in mean ALT, AST, urea, creatinine, BUN, MDA, TNF values associated with a significant increase in SOD, GPX, and TAC, in addition to an insignificant decrease in TC and TG levels in group III compared to group III [Table 2]. Administration of cinnamon plus ginger to hepatotoxic rats led to a significant decrease in the mean value of ALT, AST, urea, creatinine, BUN, MDA, TNF associated with a significant increase of SOD, GPX, and TAC, in addition to the insignificant decrease of TC and TG in group III when compared with group II [Table 2].

In the present study, when group IV compare with group III, there were insignificant changes between all parameters. In addition to when group V compared with group III and with group IV, there was a significant decrease in the mean value of ALT, AST, urea, creatinine, BUN, MDA, TNF associated with the significant increase of SOD, GPX, and TAC, in addition to the insignificant decrease of TC and TG [Table 3].

Table [1]: Comparison between group I and II [Mean \pm SD].

	Group I	Group II	t-test	P-value
	Mean \pm SD	Mean \pm SD		
ALT [U/L]	33.39 \pm 1.7	53.82 \pm 2.3	22.52	<0.001*
AST [U/L]	29.3 \pm 0.84	48.46 \pm 1.44	36.36	<0.001*
Urea [mg/dl]	22.42 \pm 1.86	34.15 \pm 3.14	10.16	<0.001*
Creatinin [mg/dl]	0.55 \pm 0.18	2.01 \pm 0.27	14.03	<0.001*
BUN [mg/dl]	7.3 \pm 0.55	14.7 \pm 0.71	25.98	<0.001*
TC [mg/dl]	111.1 \pm 4.25	118.7 \pm 10.57	2.11	0.06
TG [mg/dl]	103.4 \pm 7.5	107.4 \pm 3.27	1.55	0.14
MDA [nmol/L]	17.1 \pm 1.52	30.7 \pm 4.11	9.81	<0.001*
TNF [pg/ml]	57.3 \pm 5.48	94.3 \pm 5.17	15.54	<0.001*
SOD [mg/dl]	72.0 \pm 4.27	52.4 \pm 4.29	10.23	<0.001*
GPX [mg/dl]	23.6 \pm 1.78	15.2 \pm 2.15	9.52	<0.001*
TAC [nmol/L]	9.26 \pm 0.47	7.15 \pm 0.29	11.93	<0.001*

* = Significant difference between the two groups; **Group I:** Control group; **Group II:** Hepatotoxic group; **SD** = Standard deviation.

Table [2]: Effects of cinnamon and ginger on the hepatotoxic group [Mean \pm SD].

Parameters	Group II	Group III	Group IV	Group V
ALT [U/L]	53.82 \pm 2.3	47.94 \pm 1.5*	46.57 \pm 1.95*	41.2 \pm 1.63*
AST [U/L]	48.46 \pm 1.44	43.38 \pm 3.8*	41.79 \pm 3.72*	36.3 \pm 2.25*
Urea [mg/dl]	34.15 \pm 3.14	29.0 \pm 1.26*	29.54 \pm 0.98*	27.8 \pm 1.15*
Creatinin [mg/dl]	2.01 \pm 0.27	1.63 \pm 0.16*	1.48 \pm 0.17*	1.25 \pm 0.14*
BUN [mg/dl]	14.7 \pm 0.71	12.63 \pm 0.47*	12.3 \pm 0.49*	10.53 \pm 0.33*
TC [mg/dl]	118.7 \pm 10.57	113.0 \pm 5.87	111.9 \pm 7.46	111.7 \pm 5.14
TG [mg/dl]	107.4 \pm 3.27	105.9 \pm 2.33	104.9 \pm 3.07	104.8 \pm 2.7
MDA [nmol/L]	30.7 \pm 4.11	24.4 \pm 1.89*	22.5 \pm 2.17*	20.5 \pm 1.43*
TNF [pg/ml]	94.3 \pm 5.17	84.8 \pm 3.35*	81.3 \pm 4.55*	62.8 \pm 4.42*
SOD [mg/dl]	52.4 \pm 4.29	57.0 \pm 2.75*	59.3 \pm 2.71*	63.9 \pm 3.07*
GPX [mg/dl]	15.2 \pm 2.15	17.6 \pm 1.07*	18.2 \pm 1.14*	20.14 \pm 1.01*
TAC [nmol/L]	7.15 \pm 0.29	8.4 \pm 0.32*	8.49 \pm 0.35*	9.14 \pm 0.18*

* = Significant difference compared to group II; **Group II:** Hepatotoxic group; **Group III:** Hepatotoxic plus cinnamon group; **Group IV:** Hepatotoxic plus ginger group; **Group V:** hepatotoxic plus cinnamon and ginger group

Table [3]: Comparison between groups III, IV and V [Mean \pm SD].

Parameters	Group III	Group IV	Group V
ALT [U/L]	47.94 \pm 1.5	46.57 \pm 1.95	41.2 \pm 1.63*#
AST [U/L]	43.38 \pm 3.8	41.79 \pm 3.72	36.3 \pm 2.25*#
Urea [mg/dl]	29.0 \pm 1.26	29.54 \pm 0.98	27.8 \pm 1.15*#
Creatinin [mg/dl]	1.63 \pm 0.16	1.48 \pm 0.17	1.25 \pm 0.14*#
BUN [mg/dl]	12.63 \pm 0.47	12.3 \pm 0.49	10.53 \pm 0.33*#
TC [mg/dl]	113.0 \pm 5.87	111.9 \pm 7.46	111.7 \pm 5.14
TG [mg/dl]	105.9 \pm 2.33	104.9 \pm 3.07	104.8 \pm 2.7
MDA [nmol/L]	24.4 \pm 1.89	22.5 \pm 2.17	20.5 \pm 1.43*#
TNF [pg/ml]	84.8 \pm 3.35	81.3 \pm 4.55	62.8 \pm 4.42*#
SOD [mg/dl]	57.0 \pm 2.75	59.3 \pm 2.71	63.9 \pm 3.07*#
GPX [mg/dl]	17.6 \pm 1.07	18.2 \pm 1.14	20.14 \pm 1.01*#
TAC [nmol/L]	8.4 \pm 0.32	8.49 \pm 0.35	9.14 \pm 0.18*#

* = Significant difference compared to group III; # = Significant difference compared to group IV; **Group III:** Hepatotoxic plus cinnamon group; **Group IV:** Hepatotoxic plus ginger group; **Group V:** hepatotoxic plus cinnamon and ginger group

DISCUSSION

This study was designed to evaluate cinnamon's effect and ginger on hepatotoxicity induced by acrylamide in adult male albino rats. Results of the

present study showed that oral administration of acrylamide [AA] at a dose of 50 mg/kg body weight for four weeks resulted in a statistically significant increase in ALT, AST, urea, creatinine, BUN, MDA, TNF- α , associated with a significant decrease of

SOD, GPX, and TAC in group II compared to group I. Elhelaly *et al.* [24] asserted that oral administration of acrylamide, due to severe damage to the tissue membranes, resulted in significantly increased levels of ALT and AST. The viability of hepatic tissue was impaired by acrylamide, damaging its architecture through the loss of phenotypic features. In hepatic tissue, it produced an acute time-dependent toxic response[25].

Acrylamide produced free radicals, which can cause liver damage. Free radicals have already been reported to be more reactive than molecular elements and cause serious harm to nucleic acids, proteins, and cell membranes[26].

These results also agreed with Hamdy *et al.*[27] and Belhadj Benziane *et al.*[28], who reported that blood urea, creatinine, and BUN levels increased after acrylamide administration.

Urea is filtered by the renal glomerulus from plasma. Via renal tubules, it returns to the blood, but much of it is excreted via urine. However, if the kidney does not function correctly, plasma does not expel adequate urea, leading to higher BUN levels. To show the origin of the renal form, BUN should be considered along with serum creatinine[29].

The present study compatible with Elhelaly *et al.*[24], who indicated that acrylamide oral administration led to significantly increased TNF and MDA levels. In addition to a significant decreased in GPX and SOD.

Abdel-Daim *et al.*[30] reported that acrylamide has dangerous effects on liver and kidney tissues due to it raises the incidence of lipid peroxidation and changes antioxidant enzyme systems. Such a drastic increase in the incidence of lipid peroxidation, especially in the liver and kidneys, has a potential role to play in altering the state of an organism's oxidative stress and has seen a decrease in the concentration of GSP and SOD.

Acrylamide resulted in excessive levels of reactive oxygen species [ROS]. It can reduce the level of antioxidant protection in the organ, resulting in an imbalance between the production and removal of free radicals by antioxidant defense[31].

The present study showed insignificant increased TC and TG and these similar to Akram *et al.*[32], who reported that low doses of acrylamide cause significant increases in total cholesterol and triglyceride levels compared with controls due to lipid peroxidation, the liver has a vital role in the synthesis of whole-body lipids and the metabolism of carbohydrates[25].

The present work resulted in substantial decreases in the mean value of ALT and AST, in addition to a small reduction of TC and TG, for cinnamon and ginger or both to hepatotoxic rats. These findings are consistent with Ismail *et al.* [33] that indicated the substantial decrease in AST, ALT, cholesterol, and triglyceride in cinnamon administration to hepatotoxic rats. Shalaby and Saifan[34] found that in diabetic obese rats, cinnamon aqueous extract developed hypo-lipidemic, diabetic, hepatoprotective, and anti-oxidant effects.

The hepatoprotective effect of cinnamon extract through stimulation of liver synthesis of protein helps speed up recovery and the development of the liver cells[35]. It helps to regenerate the injured liver cells by stimulating the enzyme RNA polymerase I, which increases the synthesis of ribosomal proteins and helps regenerate hepatocytes[36].

Dietary cinnamon inhibits the hepatic activity of hydroxy-3-methylglutaryl-coenzyme A [HMG Co-A] reductase, which decreases lipids and improves lipid peroxidation by enhancing the function of the lipid antioxidant enzyme[35]. The development of LDL-R gene expression is also a mechanism for lowering the lipid activity of cinnamon [37]. Cinnamon did not alter the rats' lipid profile in the proportion of the human diet, but it increased the lipolysis of the fatty tissue of the dietary mouse[38].

Ginger enhances lipid profile because of ginger's pharmacological effects that enhance the hepatic cholesterol activity -7 α -hydroxylase, which is the rate-limiting enzyme for bile acid biosynthesis and stimulation cholesterol conversion into bile acids[39]. In addition, ginger antihypercholesterol may be caused by cellular cholesterol inhibition because of niacin, lower TG levels, hepatic

absorbance of LDL, and cholesterologenesis inhibition. Ginger antioxidants can prevent membranous lipid peroxidation leading to cell damage and necrosis^[40].

The ginger components demonstrated significant inhibition of the mediated cytochrome P450 metabolism of marker substrates and blocked the cytochrome P450-2E1 enzyme to prevent bromobenzene-induced hepatotoxicity^[41].

This also led to a consensus with Shatwan *et al.*^[42], who noted that the high levels of TC and TG in men and rats are decreased by cinnamon and ginger. Due to their large polyphenols [cinnamon], gingerols, and shogaols [ginger] that inhibit intestinal absorption by hypocholesterol activity, the probable potential mechanism underlies its hypolipidemic effects may also have a high content of polyphenols.

The present paper shows a substantial decrease in the mean urea, creatinine, and BUN value when administering cinnamon and ginger or both to hepatotoxic rats. These findings were consistent with Ismail *et al.*^[33], which recorded substantial decreases of urea and creatinine in cinnamon administration to hepato-intoxicated rats. Mani *et al.*^[43] indicated that Ginger's administration resulted in a major reduction in urea, creatinine, and BUN, which is consistent with our findings because Zingerone maintains the kidneys' toxicity functional capability. Shanmugam *et al.*^[44] indicated that ginger extracts' supplementation prevented large renal tubules and changes in renal tissue degenerative.

In the present work, cinnamon and ginger administration or both to hepatotoxic rats led to a significant decrease in the mean value of MDA, TNF associated with a significant increase of SOD, GPX, and TAC. These results are in-agreement with Qin *et al.*^[45], who asserted that cinnamon extract supplementation reduced tumor necrosis factor- α and MDA. Gholamreza *et al.*^[46] assured that cinnamon administration led to a significant decrease of MDA and TNF associated with increased TAC and SOD due to high phenol contents and excellent potential in scavenging free

radicals.

Cinnamon extract existed in liposomes and tapering hydroxyl radicals [OH•] and H₂O₂ for protective capacities against radiation-induced lipid peroxidation phase [LPO]. Cinnamon's protective effect on ROS development is due to its suppressive effect against irradiation-induced oxidative and inflammatory harm. Apart from altering gene expression by inhibiting nuclear factor kappa B [NF-Kb] activation, this is due to its phenolic and flavonoid content. Polyphenols serve as scavengers for reactive oxygen and nitrogen, transitional redox metal chelators, and modulators for the enzyme^[47].

Administration of liver damage to the Cinnamon Oils led to a substantial increase of hepatic GSP and SOD by lipid peroxidation suppression and protein oxidation, ROS development inhibition in conjunction with radical activity, and decreased DNA carcinogenic interaction showing the restoration of the liver's antioxidant system^[48].

Ginger extract can reduce the elevated expression of TNF- α in rats. Ginger significantly inhibits serum TNF alpha levels. Since the inflammatory response is an inducer of reactive oxygen species [ROS] generation. Ginger has the spectrum of activity as an anti-inflammatory and antioxidant agent^[49].

Zhang *et al.*^[50] noticed a substantial increase in total SOD, TAC, and GPX by dietary supplementation with ginger powder but reported a decrease in the MDA level. These findings relate to the results of our study.

Malondialdehyde is formed as an end product of lipid peroxidation, and so, the extent of lipid peroxidation by ROS can be determined according to MDA levels. The decrease in MDA and TAC increase indicates decreased oxidative stress or an increase in total antioxidant defense. Accordingly, reduction in MDA level in treatments containing cinnamon and ginger compared to control proved that lipid peroxidation was reduced by consuming medical plants and increasing the antioxidative activities. Besides, a decrease in the concentration

of MDA in serum can be related to an increase in antioxidant enzyme activity because of the use of medicinal herbal supplements [51].

Conclusion: Administration of Cinnamon and ginger to liver toxicity led to a significant decrease in liver enzymes and considerable improvement of oxidative stress that led to many diseases.

Financial and Non-financial Relationships and Activities of Interest

None

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