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Hepatoprotective Potentials of Onion and Garlic Extracts on Cadmium-Induced Oxidative Damage in Rats

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Abstract The hepatoprotective effect of onion and garlic extracts on cadmium (Cd)-induced oxidative damage in rats is reported. Control group received double-distilled water alone. Cd group was challenged with $3CdSO_4 \cdot 8H_2O$ (as Cd; 1.5 mg/kg bw per day per oral) alone, while extract-treated groups were pretreated with varied doses of onion and/or garlic extract (0.5 and 1.0 ml/100 g bw per day per oral) for a week and thereafter co-treated with Cd (1.5 mg/kg bw per day per oral) for 3 weeks. Cd caused a marked ($p<0.001$) increase in the levels of lipid peroxidation and glutathione S-transferase, whereas glutathione, superoxide dismutase, and catalase levels were decreased in the liver. We also observed a decrease in hepatic activities of alanine transaminase (ALT), aspartate transaminase (AST), and alkaline phosphatase and a concomitant increase in the plasma activities of ALT and AST. Onion and garlic extracts significantly attenuated these adverse effects of Cd. Onion extract proffered a dose-dependent hepatoprotection. Our study showed that Cd-induced oxidative damage in rat liver is amenable to attenuation by high dose of onion and moderate dose of garlic extracts possibly via reduced lipid peroxidation and enhanced antioxidant defense system that is insufficient to prevent and protect Cd-induced hepatotoxicity.

Keywords Hepatoprotection · Cadmium · Onion · Garlic · Oxidative damage · Antioxidant

Introduction

The incidence of liver intoxication has increased in recent years as a result of exposure to high levels of environmental toxicants such as cadmium (Cd). Cd has been involved in historic poisoning episodes of human and animal populations as a result of increased anthropogenic activities including industrial activities and lifestyles (smoking) [1, 2].

Among the many organ systems that mediate the effects of Cd on the human body and health, the liver plays a crucial role. Particularly for enteral exposure route, the liver is the

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primary site of metabolism and accumulation/deposition of Cd, thus rendering hepatic tissues more susceptible to hepatic injuries and necrosis [2]. One identifiable factor that plays a central role in Cd-induced hepatotoxicity and which has been the focus of much research is the excessive generation of reactive oxygen species (ROS). Chronic exposure to Cd not only enhances ROS generation but also depletes antioxidant levels, resulting in a state of oxidant/antioxidant imbalance. The overall biochemical implications of the deleterious effects of excessive ROS on hepatic tissues are mainly the composite pathobiochemical signs of Cd-induced hepatotoxicity [3].

The pivotal roles of nutritional/dietary antioxidants in hepatotoxicity are still under intense research. Studies have been conducted in search of protective agents for Cd-induced hepatotoxicity. Treatment strategies for acute exposure to Cd include chelation therapy using 2,3-dimercaptopropanol and penincillamine to enhance its biliary excretion [4]. For chronic exposure to Cd, antioxidant therapies in addition to mild chelation therapy are often employed. In recent times, however, doubts about the efficacy and safety of these chelating agents as well as cost have prompted the search for alternative, safer, and affordable therapy in medicinal plants. This has potentiated the quest for the evaluation of functional vegetables as a phytotherapeutic approach in curbing the menace of environmental toxicants. In this regard, the chemoprotective potential of onion and garlic on Cd-induced hepatotoxicity is of special interest.

Apart from culinary purposes, onion (*Allium cepa* L.) and garlic (*Allium sativum* L.) have received considerable attention for their functional health benefits. The functional health benefits elicited by onion and garlic have been largely attributed to (1) modulation of risk factors for cardiovascular diseases, diabetes, and cancer; (2) stimulation of immune function; (3) enhanced detoxification of xenobiotics; (4) hepatoprotection; (5) antimicrobial effect; and (6) antioxidant effect [5–8].

In view of this consideration, it seems reasonable to hypothesize that the functional effects of onion and garlic may be particularly important in preventing and diminishing Cd-induced hepatotoxicity. As such, this study was focused on evaluating the protective potentials of onion and garlic on Cd-induced hepatotoxicity.

Materials and Methods

Preparation of Extract

Fresh bulbs of onion and garlic were purchased from the local Sasa Market in Ibadan, Nigeria. Their botanical identification and authentication were confirmed at the Department of Microbiology and Botany, University of Ibadan and National Institute of Horticultural Research (NIHORT), Ibadan. Among the three local varieties of onion identified at NIHORT, the Kano Red was preferably selected because of its reported high antioxidant potentials and pungency [9]. Only a single variety of garlic was identified. The bulbs were carefully dressed and frozen (0°C and 4°C). About 100 ml of chilled distilled water per 100 g of onion and/or garlic was added and crushed in a mixing machine. The resultant slurry was squeezed and filtered through a fine cloth, and the filtrate was quickly frozen until used.

Animals and Treatments

The study was performed on 56 healthy adult male Wistar rats weighing 210±5 g. They were purchased from the Institute of Medical Research and Training (IMRAT), University

College Hospital, Ibadan. The animals were housed in well-ventilated plastic cages in an environment at 25±2°C with a 12-h dark/light cycle. They were allowed access to rat chow (Bendel Feed and Flour Mill, Edo State) and water ad libitum. All animal experiments were conducted without anesthesia and according to the *Ethical Norms on Animal Care and Use* approved by IMRAT and Faculty of Basic Medical Sciences, College of Medicine, University of Ibadan.

After 2 weeks of acclimatization, animals were divided into nine groups of six rats each and treated accordingly (Table 1). The extract-treated groups were pretreated with their respective extracts for 1 week and continued for additional 3 weeks during which Cd, CdOn, CdOnOn, CdGa, CdGaGa, and CdOnGa groups were simultaneously treated with cadmium solution. The control and Cd groups were similarly pretreated with vehicle (distilled water) for a week. More so, Control, On, and Ga groups received vehicle during the last 3 weeks of treatment. The extracts and cadmium solution were administered by gavage, respectively, 2 h apart. At the end of the experimental period of 4 weeks, the animals were weighed and fasted overnight for 12 h prior to when they were killed.

Collection and Preparation of Tissue

Blood was collected by cardiac puncture into heparinized tubes and plasma was obtained from the blood after centrifugation at 5,000 rpm for 5 min at 4°C. Whole liver was quickly excised, weighed, minced in ice-cold 1.15% KCl, blotted on filter paper, and 10% *w*/*v* homogenate was made in ice-cold Tris buffer (0.1 M, pH 7.4) using a Potter–Elvehjem type homogenizer. The homogenate was centrifuged at 5,000 rpm for 15 min at 4°C, and aliquots of the supernatant were used for biochemical assays.

Biochemical Analysis

The total protein content of the homogenized testes was determined by the method of Lowry et al. [10]. Lipid peroxidation, as measured by malondialdehyde (MDA) content, was assayed by the thiobarbituric acid method of Beuge and Aust [11]. Reduced glutathione level was assayed by the method of Jollow et al. [12] based on the reaction with 5,5′-dinitro bis(2-nitrobenzoic acid). Superoxide dismutase (SOD; EC 1.15.1.1) activity was measured by the method of Misra and Fridovich [13]. Catalase (CAT; EC 1.11.1.6) activity was estimated by the method of Sinha [14] by measuring the rate of

Table 1 Experimental Design

Group	Treatment
Control	Distilled water (0.5 ml/100 g bw per day)
Cd	Cadmium only (1.5 mg/kg bw per day)
On	Onion extract (0.5 ml/100 g bw per day)
Ga	Garlic extract (0.5 ml/100 g bw per day)
CdOn	Cd (1.5 mg/kg bw per day) + Onion extract (0.5 ml/100 g bw per day)
CdOnOn	Cd (1.5 mg/kg bw per day) + Onion extract (1 ml/100 g bw per day)
CdGa	Cd (1.5 mg/kg bw per day) + Garlic extract (0.5 ml/100 g bw per day)
CdGaGa	Cd (1.5 mg/kg bw per day) + Garlic extract (1 ml/100 g bw per day)
CdOnGa	Cd (1.5 mg/kg bw per day) + Onion extract (0.5 ml/100 g bw per day) + Garlic extract (0.5 ml/100 g bw per day)

n=6 for each treatment group

decomposition of hydrogen peroxide (H_2O_2). The method of Habig et al. [15] was followed to assay the activity of glutathione S-transferase (GST; EC 2.5.1.18) based on the rate of increase in the conjugate formation between glutathione (GSH) and 1-chloro-2,4-dinitrobenzene. The activities of alanine transaminase (ALT) and aspartate transaminase (AST) were estimated according to the method of Reitman and Frankel [16]. The activity of alkaline phosphate (ALP) was estimated spectrophotometrically by a kinetic method (based on the liberation of *p*-nitrophenol from *p*-nitrophenyl phosphate) using a commercially available diagnostic kit (Randox Lab, Ardmore, UK).

Statistical Analysis

All results are expressed as mean ± SD. Data were analyzed by one-way analysis of variance (ANOVA) followed by Fischer's least significant difference (LSD) post hoc test using SPSS 13 software (SPSS, Chicago, USA). Statistical significance was considered at $p<0.05$.

Results

The effect of onion and garlic extracts on the level of hepatic MDA in Cd-exposed rats is presented in Fig. 1. Cd exerted a significant ($p<0.001$) increase in hepatic MDA level, whereas treatment with extract of onion or garlic alone caused significant ($p<0.001$) decrease on hepatic MDA level. Treatment of Cd-intoxicated rats with varied doses of onion and/or garlic extract significantly ($p<0.05$) reduced hepatic MDA level. While a dose-dependent decrease in hepatic MDA level was evident in Cd-intoxicated rats treated with onion extract, the high dose of garlic administered caused a dose-dependent increase in hepatic MDA level, though not significant.

The effect of onion and garlic extracts on the level of hepatic GSH in Cd-exposed rats is presented in Fig. 2. Cd administration significantly ($p<0.001$) depleted hepatic GSH level, while treatment with extracts alone caused an increase in GSH level, which was only significant ($p<0.001$) in rats treated with onion extract alone (Fig. 2). Treatment of Cd-challenged rats with varied doses of onion and/or garlic extract significantly ($p<0.001$) enhanced the level of GSH. Although a significant ($p<0.001$) dose-dependent increase in hepatic GSH level was evident in Cd-intoxicated rats treated with onion extract, there was a slight decrease in Cd-intoxicated rats treated with high dose of garlic extract, though not significant.

The effect of onion and garlic extracts on the activity of hepatic SOD in Cd-exposed rats is presented in Fig. 3. The activity of hepatic SOD was significantly ($p<0.001$) reduced in rats treated with Cd alone. Treatment with extract of onion or garlic alone enhanced the activity of SOD, with the activity of the former treatment only significant ($p<0.05$). Treatment of Cd-challenged rats with varied doses of onion and/or garlic extract significantly ($p<0.001$) improved the activity of SOD except in the CdOn-treated and CdGaGa-treated groups. While administration of onion extract to Cd-challenged rats exerted a significant ($p<0.001$) dose-dependent increase in SOD activity, high dose of garlic extract caused a significant ($p<0.05$) dose-dependent decrease in SOD activity.

The effect of onion and garlic extracts on the activity of hepatic CAT in Cd-exposed rats is presented in Fig. 4. The activity of hepatic CAT was significantly ($p<0.001$) reduced in rats treated with Cd alone. Treatment with extract of onion or garlic alone enhanced the activity of CAT, with the activity of the former treatment only significant ($p<0.01$). Treatment of Cd-challenged rats with varied doses of onion and/or garlic extract improved

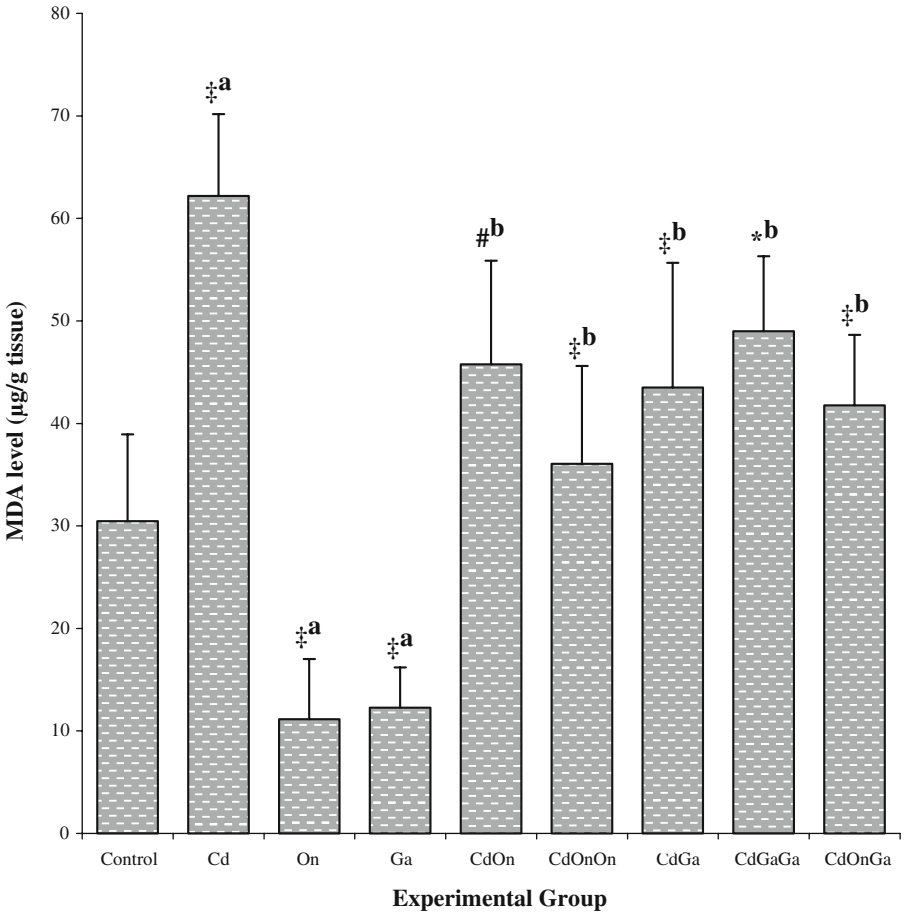

Fig. 1 Effects of onion and garlic extracts on the level of hepatic lipid peroxidation in cadmium-exposed rats. *Bars* are means ± SD. $n=6$ for each treatment group. Data were analyzed by one-way ANOVA followed by Fischer's LSD post hoc test. *$p<0.05$, #$p<0.01$, and †$p<0.001$. *a* = control vs Cd, On, Ga; *b* = Cd vs CdOn, CdOnOn, CdGa, CdGaGa, CdOnGa; *c* = CdOn vs CdOnOn; *d* = CdGa vs CdGaGa

the activity of CAT, but this was only significant in CdOnOn-treated group. Treatment of Cd-intoxicated rats with high dose of garlic extract failed to exert any dose-dependent changes in hepatic CAT activity.

The effect of onion and garlic extracts on the activity of hepatic GST in Cd-exposed rats is presented in Fig. 5. Cd intoxication caused a marked ($p<0.001$) increase in hepatic GST activity. While treatment with onion extract alone caused a significant ($p<0.01$) increase in GST activity, treatment with garlic extract alone exerted a mild increase. Hepatic GST activity, which was heightened by Cd intoxication, was significantly ($p<0.05$) reduced upon treatment with varied doses of onion and/or garlic extract. A significant dose-dependent decrease in GST activity was observed in Cd-intoxicated rats upon treatment with onion ($p<0.001$) or garlic ($p<0.01$) extract.

The effects of onion and garlic extracts on the hepatic and plasma activities of ALT and AST in Cd-exposed rats are presented in Figs. 6 and 7. Cd intoxication caused a significant ($p<0.001$) decrease in the activities of hepatic ALT and AST with a concomitant significant

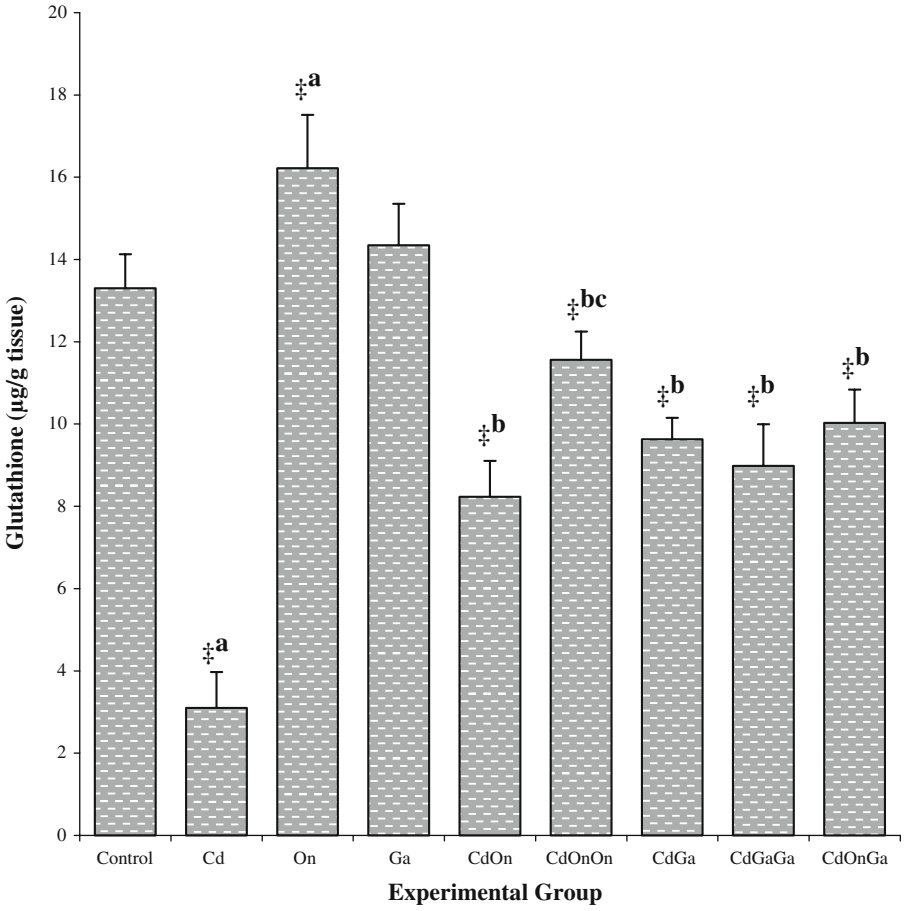

Fig. 2 Effects of onion and garlic extracts on the level of hepatic glutathione in cadmium-exposed rats. *Bars* are means ± SD. $n=6$ for each treatment group. Data were analyzed by one-way ANOVA followed by Fischer's LSD post hoc test. *$p<0.05$, #$p<0.01$, and †$p<0.001$; *a* = control vs Cd, On, Ga; *b* = Cd vs CdOn, CdOnOn, CdGa, CdGaGa, CdOnGa; *c* = CdOn vs CdOnOn; *d* = CdGa vs CdGaGa

($p<0.001$) increase in their plasma activities. Treatment with onion or garlic extract alone failed to exert any effect on the hepatic and plasma activities of ALT and AST. Co-administration of varied doses of extracts with Cd significantly ($p<0.001$) enhanced hepatic activities of ALT and AST with a concomitant significant ($p<0.001$) reduction in their plasma activities, except plasma ALT activity in CdGaGa-treated rats. While a trend toward a dose-dependent increase was observed in hepatic ALT and AST activities, a concomitant significant ($p<0.05$) reduction was evident in their plasma activities.

The effect of onion and garlic extracts on the activity of hepatic ALP in Cd-exposed rats is presented in Fig. 8. Although rats treated with Cd alone had significantly ($p<0.001$) reduced hepatic ALP activity, those treated with onion or garlic extract alone showed a trend to high ALP activity, though not significant. Treatment of Cd-challenged rats with varied doses of onion and/or garlic extract significantly ($p<0.001$) enhanced the activity of ALP. Also, the activity of ALP in Cd-challenged rats followed a significant dose-dependent increase upon treatment with high dose of onion ($p<0.001$) or garlic ($p<0.05$) extract.

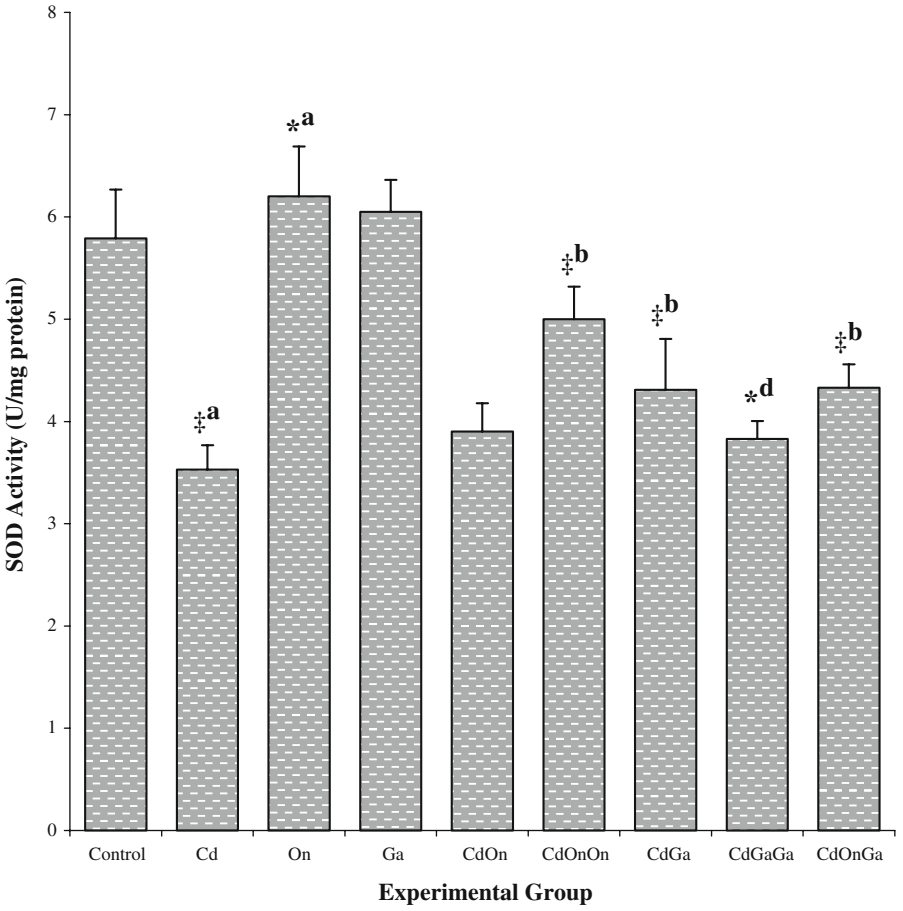

Fig. 3 Effects of onion and garlic extracts on the activity of hepatic superoxide dismutase in cadmium-exposed rats. *Bars* are means ± SD. $n=6$ for each treatment group. Data were analyzed by one-way ANOVA followed by Fischer's LSD post hoc test. *$p<0.05$, #$p<0.01$, and †$p<0.001$; *a* = control vs Cd, On, Ga; *b* = Cd vs CdOn, CdOnOn, CdGa, CdGaGa, CdOnGa; *c* = CdOn vs CdOnOn; *d* = CdGa vs CdGaGa

Discussion

The hepatotoxic action of Cd is mediated mainly via induction of early oxidative stress. Cd has been reported to interact with critical subcellular sites such as the cytosol, mitochondria, peroxisomes, and microsomes, resulting in the excessive generation of free radicals [3]. The free radicals in turn may induce oxidative stress, causing oxidative damage to biomolecules such as lipids, proteins, and DNA, which may culminate in various pathological conditions [3, 17, 18]. Biomembrane lipid peroxidation is an early sign of Cd-induced oxidative damage [19, 20]. Our results indicate that Cd intoxication caused a tremendous increase in the levels of hepatic lipid peroxidation (LPO) and GST as well as significant decrease in the levels of hepatic GSH, SOD, and CAT. Other investigators have reported similar findings [19–23].

Studies on mechanisms of antioxidant defense in Cd-induced hepatotoxicity have shown that endogenous GSH is highly sensitive to Cd-induced oxidative stress, acting as the first line of antioxidant defense, and that liver necrosis begins when hepatic GSH stores are

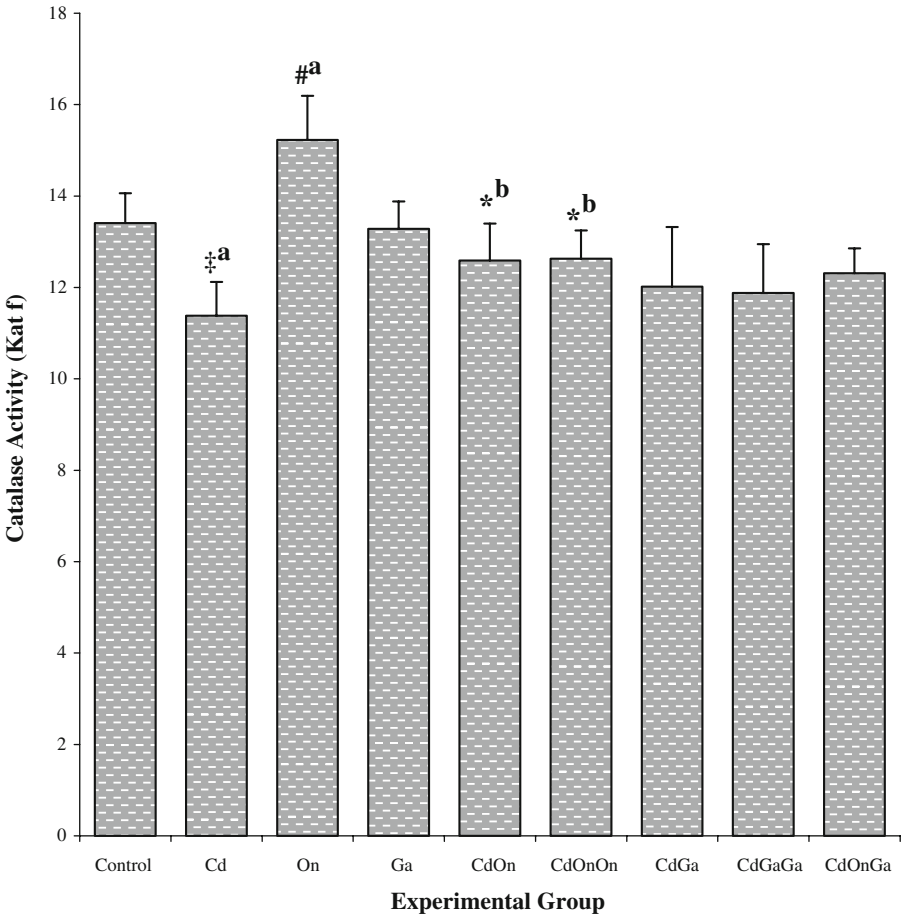

Fig. 4 Effects of onion and garlic extracts on the activity of hepatic catalase in cadmium-exposed rats. *Bars* are means ± SD. n=6 for each treatment group. Data were analyzed by one-way ANOVA followed by Fischer's LSD post hoc test. *p<0.05, #p<0.01 and †p<0.001; a = control vs Cd, On, Ga; b = Cd vs CdOn, CdOnOn, CdGa, CdGaGa, CdOnGa; c = CdOn vs CdOnOn; d = CdGa vs CdGaGa. Catalase feiahigkeit or "Kat f" is equivalent to micromole H_2O_2 consumed per minute per milligram protein

markedly depleted [24, 25]. Therefore, the observed GSH depletion in our study may be due to enhanced utilization to conjugate Cd, counteract ROS and lipid peroxidative products as well as inhibition of GSH biosynthesis [24, 26]. GST enzyme plays an important role in the detoxification of xenobiotics, drugs, and carcinogens and thus protects hepatic tissues against oxidative insults [21]. Thus, the significant increase in hepatic GST activity may reflect its protective role.

SOD and CAT are essential parts of cellular antioxidant defense system, and they play a crucial role in circumventing oxidative stress. The significant decrease in the activities of SOD and CAT in the Cd group may be attributed, in part, to an overwhelming oxidative modification of enzymatic proteins and biomembrane lipids by ROS as evident by heightened level of LPO [27]. More so, Cd has been reported to exert a direct inhibitory effect on SOD and CAT activities via Cd–enzyme interaction with a resultant perturbation of enzyme topography critical for catalytic activity [27, 28].

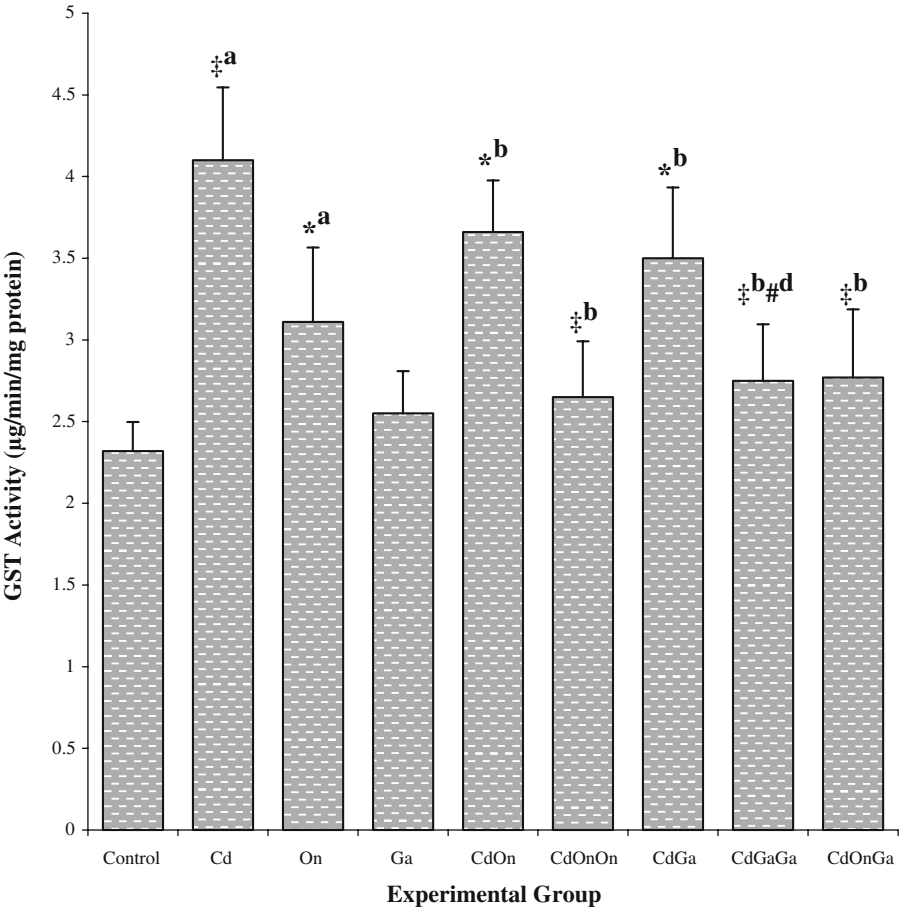

Fig. 5 Effects of onion and garlic extracts on the activity of hepatic glutathione S-transferase in cadmium-exposed rats. *Bars* are means ± SD. $n=6$ for each treatment group. Data were analyzed by one-way ANOVA followed by Fischer's LSD post hoc test. *$p<0.05$, #$p<0.01$ and †$p<0.001$; *a* = control vs Cd, On, Ga; *b* = Cd vs CdOn, CdOnOn, CdGa, CdGaGa, CdOnGa; *c* = CdOn vs CdOnOn; *d* = CdGa vs CdGaGa

Our data confirm that Cd intoxication causes a significant decrease in the activities of hepatic ALT and AST with a concomitant increase in their plasma activities. This observation is in consonance with the findings of other investigators [18, 29, 30]. ALT and AST are cytoplasmic in location and are released into circulation sequel to hepatocellular damage [31]. Hence, the observed increase in the plasma activities of these enzymes may be the consequence of Cd-induced hepatocellular damage. Also, it may, in conjunction with enzyme inhibition and disturbances in hepatic biosynthesis, explain the decrease in hepatic activities of ALT and AST [18]. Besides, Cd-induced hepatic damage was evident as decrease in hepatic activity of ALP. This decrease may be attributed to Cd-induced inhibition, alterations in the balance between synthesis, and degradation of enzyme and changes in the permeability of plasma membrane [18, 29, 32].

The extent to which oxidative damage may occur in the liver is highly dependent on its antioxidant defense status. There is accumulating evidence that Cd toxicity is strongly associated with marked decrease in several dietary antioxidants [33]. More so, it has been

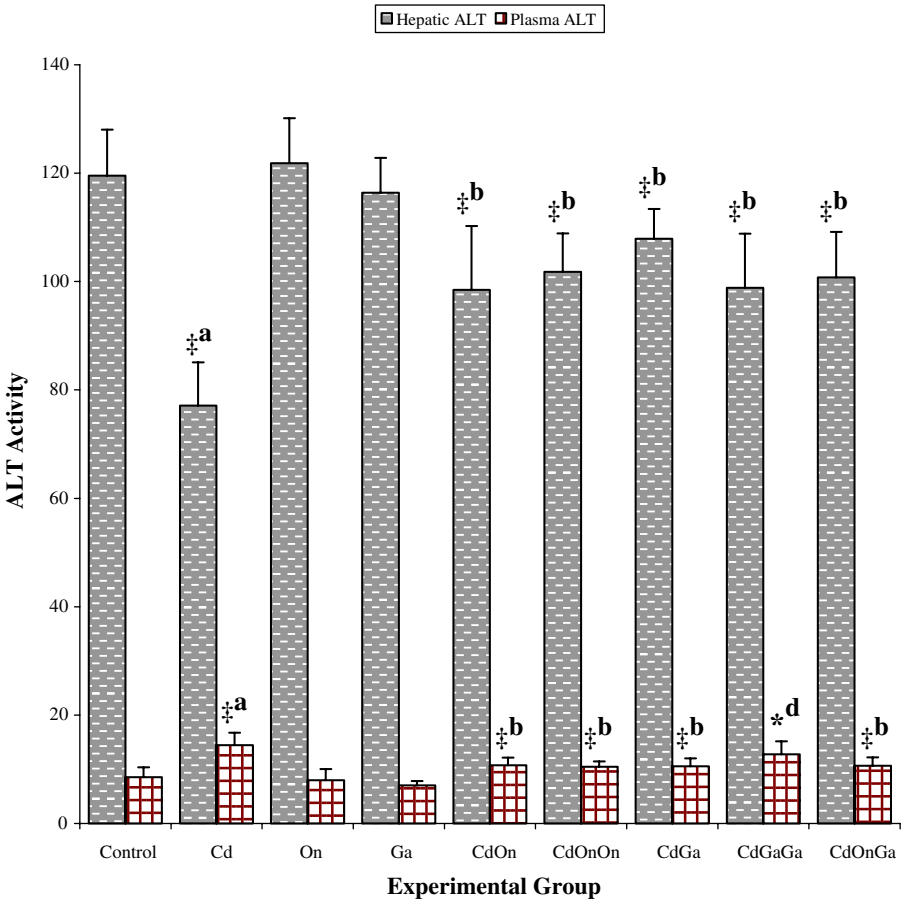

Fig. 6 Effects of onion and garlic extracts on hepatic and plasma activity of alanine transaminase in cadmium-exposed rats. *Bars* are means ± SD. $n=6$ for each treatment group. Data were analyzed by one-way ANOVA followed by Fischer's LSD post hoc test. *$p<0.05$, #$p<0.01$, and †$p<0.001$; *a* = control vs Cd, On, Ga; *b* = Cd vs CdOn, CdOnOn, CdGa, CdGaGa, CdOnGa; *c* = CdOn vs CdOnOn; *d* = CdGa vs CdGaGa

reported that Cd-induced lipid peroxidation was diminished by plant extracts such as anthocyanins, quercetin, and diallyl tetrasulphide [30, 34–36]. This, therefore, implies that ingestion of vegetables containing these and other bioactive compounds may also offer direct chemoprotective roles and help reduce oxidative stress associated with Cd toxicity.

In the present study, onion and garlic extracts partially protected the rats against Cd-induced hepatic injury as evident from decreased levels of LPO and enhanced antioxidant defense status. Hepatic GSH biosynthesis is known to be dependent on dietary amino acid supplies [25]. Dietary cysteine, a sulfur-containing amino acid, is a rate-determining substrate in hepatic GSH biosynthesis [25]. Onion and garlic are endowed with cysteine-containing bioactive compounds (alk(en)yl cysteine sulphoxides) and flavonoids (quercetin, allixin, anthocyanins, and kaempferol), which are known to exert antioxidant effects [5, 7]. Therefore, the enhanced hepatic GSH level in Cd-challenged rats fed extracts could have resulted, in part, from increased bioavailability and utilization of cysteine-containing compounds for biosynthesis of GSH. Singhal et al. [24] reported that administration of cysteine prior to Cd intoxication reduced hepatic Cd toxicity.

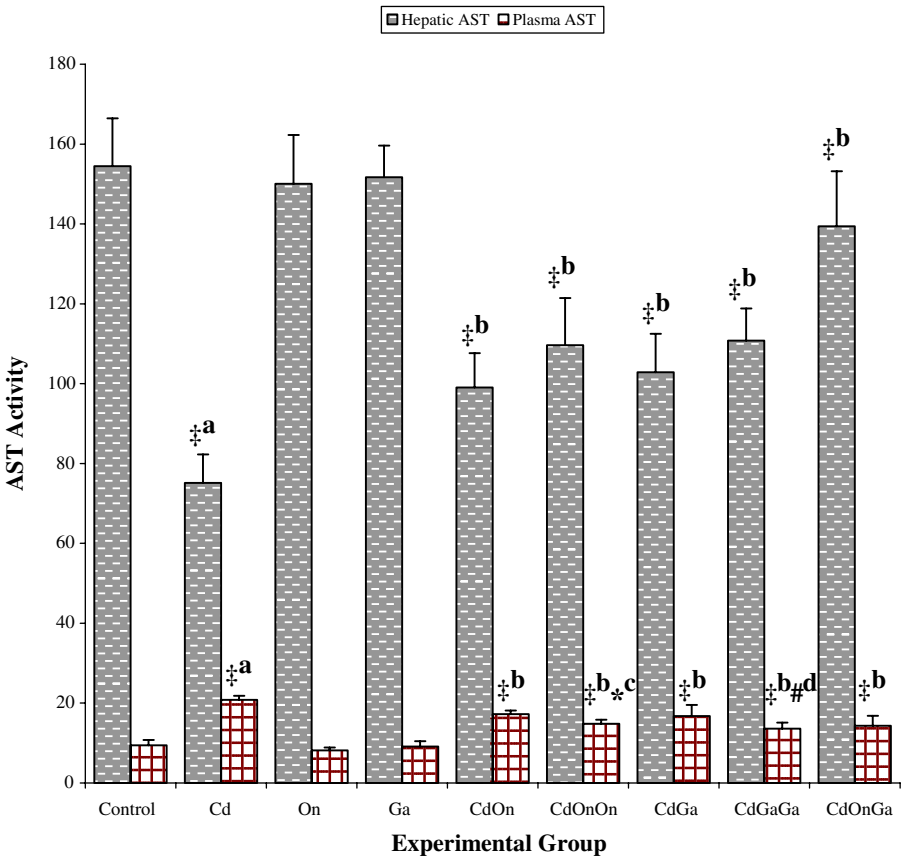

Fig. 7 Effects of onion and garlic extracts on hepatic and plasma activity of aspartate transaminase in cadmium-exposed rats. *Bars* are means ± SD. $n=6$ for each treatment group. Data were analyzed by one-way ANOVA followed by Fischer's LSD post hoc test. *$p<0.05$, #$p<0.01$, and †$p<0.001$; *a* = control vs Cd, On, Ga; *b* = Cd vs CdOn, CdOnOn, CdGa, CdGaGa, CdOnGa; *c* = CdOn vs CdOnOn; *d* = CdGa vs CdGaGa

Cd is a well-known selenium (Se) antagonist and has been reported to reduce the activities of Se-dependent enzymes such as GST and GSH-Px [23, 37, 38]. Both onion and garlic are rich in seleno-compounds [39]. The bioavailability of Se from the extracts could possibly have contributed to the enhancement of the activities of Se-dependent antioxidant enzymes. Several studies have suggested that relief of Cd-imposed inhibition of GST and GSH-Px is partly dependent on the dietary and tissue levels of reduced GSH and Se [23, 37].

Moreover, since onion and garlic extracts have been reported to exhibit antioxidant properties [40–42], it is possible that both extracts could have spared the consumption of GSH, SOD, and CAT occasioned by Cd-induced oxidative stress. Also, the protective effects of these extracts may be related to their ability to chelate/sequester Cd via formation of Cd–flavonoid complexes [43, 44]. Another possible intriguing mechanism may be that onion and garlic initiated hepatocytes to produce their own chemical oxidative defense mechanisms via induction of metallothionein and phase II drug-metabolizing enzymes such as GST and *p*-nitrophenol UGT [45–47].

Furthermore, treatment of Cd-intoxicated rats with onion and/or garlic extract also enhanced the hepatic activities of ALT, AST, and ALP and reduced the plasma activities of ALT and AST.

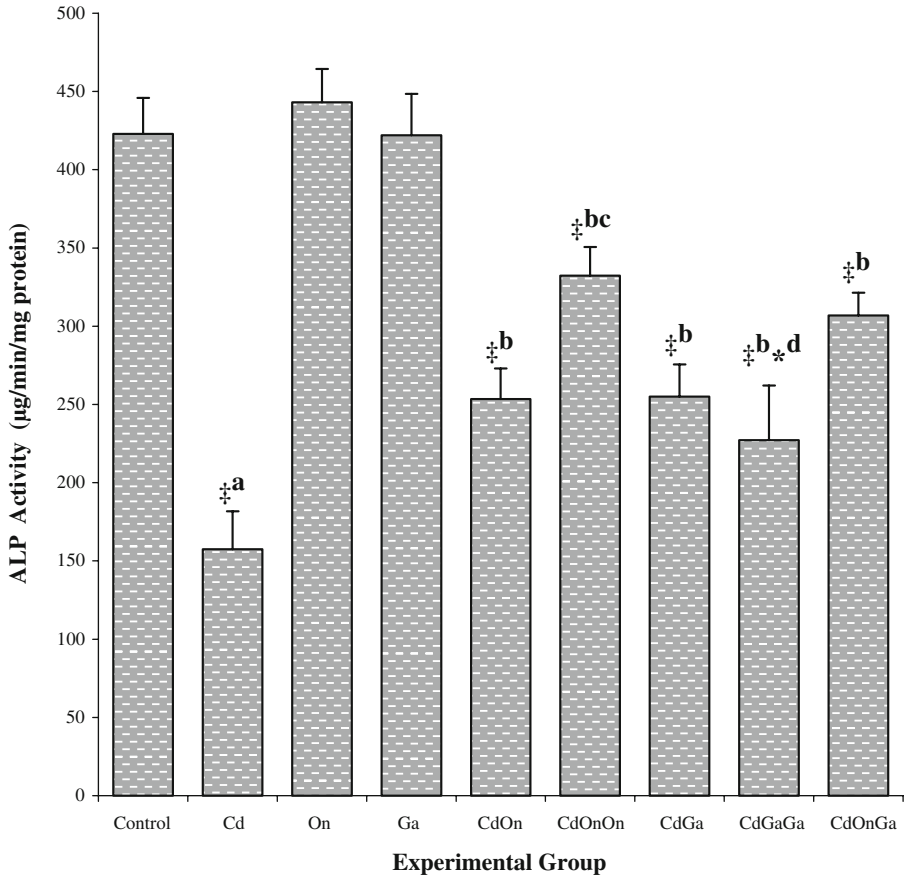

Fig. 8 Effects of onion and garlic extracts on the activity of hepatic alkaline phosphatase in cadmium-exposed rats. *Bars* are means ± SD. $n=6$ for each treatment group. Data were analyzed by one-way ANOVA followed by Fischer's LSD post hoc test. *$p<0.05$, #$p<0.01$ and †$p<0.001$; *a* = control vs Cd, On, Ga; *b* = Cd vs CdOn, CdOnOn, CdGa, CdGaGa, CdOnGa; *c* = CdOn vs CdOnOn; *d* = CdGa vs CdGaGa

These findings are indications that onion and garlic extracts possibly protect hepatic tissues against Cd-induced hepatocellular damage. Our findings are compatible with the reports of other authors that onion and garlic enhanced the hepatic antioxidant status of rats [29, 40, 48].

Based on our study, it can be presumed that onion and garlic possibly exerted their chemopreventive and protective effects via, at least, one of these mechanisms: (a) by acting as sacrificial antioxidants, thus sparing the consumption of endogenous antioxidants; (b) by stimulating the antioxidant defense system; and (c) possibly by synergic induction of metallothionein and some phase II detoxification enzymes.

It should be noted, however, that while onion failed to exert toxic effect at the varied doses co-administered with Cd in this study for 4 weeks, high dose of garlic elicited a pro-oxidant effect. This suggests that onion, at both doses, and garlic in low dose have the potential to enhance the endogenous antioxidant status and thus protect against Cd hepatotoxicity. Besides, the study highlights the potential ability of high dose of garlic to elicit pro-oxidant effect and thus reverse its beneficial effects. Hence, onion may be more protective than garlic in this regard.

While it may not be assured that the protective effect of both extracts stems from antioxidant actions alone, the findings of this study support the conclusion that high onion and modest garlic intake may have a measure of hepatoprotective potentials against Cd-induced oxidative damage.

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