

# Protective Value of *Aloe vera* against Some Toxic Effects of Arsenic in Rats

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Concomitant oral supplementation of *Aloe vera*, (1, 2 or 5% w/v in drinking water) during arsenic exposure (0.2 mg/kg, intraperitoneally, once daily for 3 weeks) was investigated in rats for its protective value. Animals exposed to arsenic (III) showed a significant inhibition of  $\delta$ -aminolevulinic acid dehydratase (ALAD) activity, a marginal decrease in glutathione (GSH) and an increase in zinc protoporphyrin (ZPP) level in blood. White blood corpuscles (WBC) level decreased while most of the other clinical blood parameters like red blood cells count, haemoglobin, MCV, MCH, MCHC ratio and platelet number, etc. remained unaltered on arsenic exposure. Hepatic reduced GSH, oxidized glutathione (GSSG) level remained unaltered, thiobarbituric acid reactive substance (TBARS) level increased significantly while the activity of alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine aminotransferase (ALT) and catalase decreased on arsenic exposure. Renal GSH contents decreased while superoxide dismutase (SOD) activity decreased significantly on arsenic exposure. Concomitant administration of *Aloe vera* had remarkable protective action on inhibited blood ALAD activity and restored blood GSH level while most of the other blood biochemical parameters remained unchanged on *Aloe vera* supplementation. Interestingly, most of hepatic biochemical variables indicative of oxidative stress showed protection; no effect of *Aloe vera* on blood and liver arsenic concentration was noted. Also, no effect of *Aloe vera* on most of the altered renal biochemical parameters were noticed. The results thus lead us to conclude that simultaneous supplementation of *Aloe vera* protects against arsenic induced oxidative stress but does not influence the arsenic concentration in these organs. Copyright © 2005 John Wiley & Sons, Ltd.

**Keywords:** *Aloe vera*; protection against arsenic exposure: oxidative stress; haematological and hepatic effects.

## INTRODUCTION

Arsenic (As) is a ubiquitous element present in low concentrations in air, soil and water. Arsenic compounds may represent a concern to environmental and occupational health becoming concentrated in the environment as a result of natural or anthropogenic source (ATSDR, 1993). Arsenic (III) has a century long history as a human poison and cases of acute intoxication due to its oral ingestion (e.g. in order to commit suicide) may still be observed nowadays (Moore *et al.*, 1994; Wax and Thornton, 2000; Blythe and Joyee, 2001). Exposure to arsenic causes melanosis, depigmentation and ultimately cancers of the skin and various organs including liver and lungs (Waalkes, 1995). Abnormalities of the peripheral vasculature, diseases of cerebrovascular and reproductive failure have also been reported in people chronically exposed to arsenic (Neiger and Osweiler, 1989; Chiou *et al.*, 1995). Acute arsenic exposure may lead to immediate gastrointestinal symptoms and subacute sequela resulting in polyneuropathy (Jolliffe *et al.*, 1991; Dong *et al.*, 1993). The heme metabolism pathway is known to be highly susceptible to alterations induced by drugs and environmental chemicals, offering the chance to use these changes as indicator of damage caused by arsenic.

The clinical therapy of choice is the administration of sulfhydryl-containing chelators such as meso 2,3-dimercaptosuccinic acid (DMSA), 2,3-dimercaptopropane 1-sulfonate (DMPS) or formerly British Anti Lewisite (BAL; 2,3-dimercaprol). These dimercapto chelators compete with sulfhydryl groups in tissues or enzymes for binding arsenic (III), which results in the elimination of arsenic. Since most conventional metal chelating agents have toxic side effects or disadvantages (Mehta and Flora, 2001), the possibility of dietary intervention or supplementation with naturally occurring dietary nutrients, to prevent the effects of arsenic in populations of risk, is of interest. A positive correlation has also been established between dietary supplementation with certain of vegetables and plants and the reduction of toxic effects of various toxicants, environmental agents including heavy metals (Nandi *et al.*, 1997).

*Aloe vera* (*Aloe barbadensis*) is used in the traditional medicine of many cultures and said to be beneficial in the treatment of disorders such as arthritis, gout, dermatitis etc and wounds such as peptic ulcer and burns (Grindlay and Reynolds, 1986). The fresh gel, juice and formulated products have long been used for medical and cosmetic purposes and general health (Chithra *et al.*, 1998; Reynolds and Dweck, 1999). In spite of its wide use in folk remedies any influence on various heavy metals/metalloid induced altered biochemical and physiological processes have not yet been described in detail.

In the present study, we report the influence of concomitant administration of *Aloe vera* on arsenic induced haematological and hepatic disorders in male

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rats. Blood and liver arsenic concentration too were determined.

## MATERIALS AND METHODS

**Chemicals.** Sodium arsenite ( $\text{NaAsO}_2$ , Sigma Chemical, St Louis, MO, USA),  $\delta$ -aminolevulinic acid (Sigma Chemical, St Louis, MO, USA). All other chemicals were of analytical grade and were purchased from Merck (Darmstadt, Germany), BDH Chemical (Mumbai, India) or Sigma (USA). *Aloe vera* gel was purchased from a local source (Safed Musali Farms, Gwalior, India), ground in a blender, and centrifuged at 10 000 g to remove the fibres. The supernatant was lyophilized and stored at room temperature. A small quantity of water was added to each of portion of lyophilized *Aloe vera* powder and the gel was prepared.

**Test system.** All animals were obtained from the animal house facility of Defence Research and Development Establishment (DRDE), Gwalior. The animal ethical committee of DRDE, Gwalior, India approved the experimental protocol. All experiments were performed on male Wistar rats weighing  $120 \pm 10$  g housed in stainless steel cages in an air-conditioned room with temperature maintained at  $25 \pm 2$  °C and 12 h alternating day and night cycles. Rats were allowed standard rat chow diet (Amrut Feeds, Pranav Agro, New Delhi, India; Metal contents of diet, in p.p.m. dry weight, Cu 10.0, Zn 45.0, Mn 55.0, Co 5.0, Fe 75.0) throughout the study.

**Experimental protocol.** Forty animals were randomized into eight groups of five rats each and were treated as below for the period of three weeks: Group I – normal animals; Group II – *Aloe vera*, 1% in drinking water (w/v); Group III – *Aloe vera*, 2% in drinking water (w/v); Group IV – *Aloe vera*, 5% in drinking water (w/v); Group V – Arsenic, 0.2 mg/kg, intraperitoneally (i.p.), once daily; Group VI – Arsenic (as in group V) plus *Aloe vera*, 1% in drinking water; Group VII – Arsenic (as in group VI) plus *Aloe vera*, 2% in drinking water; Group VIII – Arsenic (as in group VII) plus *Aloe vera*, 5% in drinking water.

The food and water intake was recorded and rats were weighed every week. One day after the administration of the last dose, the animals were sacrificed under light ether anesthesia. Blood was collected by cardiac puncture in heparinized tubes. Liver and kidneys were removed, rinsed in cold saline, blotted, weighed and used for various biochemical variables and metal analysis. Half portion of the liver and one of the kidneys from each rat were processed immediately for biochemical estimation and the remaining was stored at  $-20$  °C before wet acid digestion with  $\text{HNO}_3$  for estimation of arsenic contents.

## BIOCHEMICAL ASSAY

### Blood $\delta$ -aminolevulinic acid dehydratase (ALAD)

The activity of blood ALAD was assayed according to the procedure of Berlin and Schaller (1974). 0.2 ml of

heparinized blood was mixed with 1.3 ml of distilled water and incubated for 10 min at 37 °C for complete hemolysis. After adding 1 ml of standard  $\delta$ -ALA, the tubes were incubated for 60 min at 37 °C. The reaction was stopped after 1 h by adding 1 ml of 10% trichloroacetic acid (TCA). To the supernatant, an equal volume of Ehrlich reagent was added and the absorbance was recorded at 555 nm after 5 min.

### Blood zinc protoporphyrin

Zinc protoporphyrin (ZPP) was determined in a drop of blood with the help of a haematofluorometer (Model 2060, Aviv, Lakewood, USA) and a calibrated glass slide (Grandjean, 1979).

### Blood glutathione (GSH), hepatic reduced GSH and oxidized glutathione (GSSG)

For the determination of blood GSH concentration (Ellman, 1959), 200  $\mu$ l of whole blood was added to 200  $\mu$ l of 10 mM solution of 5,5' dithiobis-(2 nitrobenzoic acid) (DTNB) in phosphate buffer (pH 7.5) containing 17.5 mM  $\text{Na}_2\text{EDTA}$ . Samples were centrifuged at  $2000 \times g$  for 6 min and the supernatant used for assay.

For tissue GSH and GSSG estimation, 0.25 g of tissue sample was homogenized on ice with 3.75 ml of phosphate-EDTA buffer and 1 ml of 25%  $\text{HPO}_3$  which was used as a protein precipitant. The total homogenate was centrifuged at 100 000 g for 30 min at 4 °C. For the GSH assay in soft tissues (Hissin and Hilf, 1973), 0.5 ml supernatant and 4.5 ml phosphate buffer (pH 8.0) were mixed. The final assay mixture (2.0 ml) contained 100  $\mu$ l supernatant, 1.8 ml phosphate-EDTA buffer and 100  $\mu$ l O-phthaldehyde (OPT; 1000  $\mu$ l/ml in absolute methanol, fresh prepared). After mixing, fluorescence was determined at 420 nm with an excitation wavelength of 350 nm using a spectrofluorometer (Model RF 5000, Shimadzu, Japan).

For the GSSG assay, 0.5 ml supernatant was incubated at room temperature with 200  $\mu$ l of 0.04 mol/L N-ethylmaleimide solution for 30 min. To this mixture 4.3 ml of 0.1 mol/L NaOH was added. A 100  $\mu$ l sample of this mixture was taken for the measurement of GSSG using the procedure described above for GSH assay except that 0.1 mol/L NaOH was used as the diluent instead of phosphate buffer (Hissin and Hilf, 1973).

**Superoxide dismutase.** Superoxide dismutase (SOD) activity in brain was assayed spectrophotometrically as described by Durak *et al.* (1996). Briefly, 2.8 ml of reactive mixture (xanthine 0.3 mM, EDTA 0.67 mM, 150  $\mu$ M nitrotetrazolium blue chloride (NBT), sodium carbonate 0.4 M, bovine albumin 30 mg/30 ml) is added to 0.1 ml sample and 50  $\mu$ l xanthine oxidase (10  $\mu$ l in 2 M ammonium sulphate), incubated at 25 °C for 20 min and mixed with 0.1 ml 8 M copper chloride. The colour reaction was measured at 560 nm.

**Catalase.** Catalase activity in brain was assayed following the procedure of Aebi (1984) at room temperature. 100  $\mu$ l of tissue extract was placed on ice bath for 30 min and then for another 30 min at room temperature. 10  $\mu$ l

Triton-X 100 was added to the each tube. In a cuvette containing 200  $\mu\text{l}$  phosphate buffer and 50  $\mu\text{l}$  of tissue extract, was added 250  $\mu\text{l}$  of 0.066 M  $\text{H}_2\text{O}_2$  (in phosphate buffer) and decrease in optical density was measured at 240 nm for 60 s. The molar extinction coefficient of 43.6  $\text{M cm}^{-1}$  was used to determine CAT activity. One unit of activity is equal to the moles of  $\text{H}_2\text{O}_2$  degraded/min/mg protein.

**Thiobarbituric acid reactive substances (TBARS).** Tissue lipid oxidizability was measured by shaking the liver homogenate in 150 mM KCl, 0.025 M Tris-HCl buffer (pH 7.5) for 30 min at 37 °C and measuring the lipid oxidation formed with the thiobarbituric acid reaction (Wilber *et al.*, 1949). The incubation is interrupted by adding 0.1 ml of 10% TCA. 2 ml of reaction mixture was then mixed with 2 ml of 30% TCA, 0.2 ml of 5M HCl and 2 ml of 0.75% TBA. The mixture is then kept in a boiling water bath for 15 min to get the red colour of various compounds that react to TBA. After centrifugation the colour is measured at 535 nm. The amount of TBARS was calculated using a molar extinction coefficient of  $1.56 \times 10^5/\text{M per cm}$ .

#### Alkaline and acid phosphatase

The activities of alkaline (ALP) and acid phosphatase (ACP) liver tissues were determined as described by Halk *et al.* (1954). A total of 0.5 ml of the homogenate was reacted with 0.1 ml of Triton X-100. A 4 ml quantity of alkaline or acid buffer was added to this and the mixture was incubated at 37 °C for 1 h. After incubation, 0.5 ml of 30% trichloroacetic acid (TCA) was added to stop the reaction. The mixture was centrifuged, 2 ml of the supernatant were mixed with 6.6 ml of distilled water, 1 ml of 2.5% ammonium molybdate and 0.4 ml of amino-naphthol sulfuric acid. Optical density was measured at 620 nm and the phosphorus liberated was calculated.

#### Alanine and Aspartate Transaminases

Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities were measured in liver (Reitman and Frankel, 1957). The assay system contained 1 ml of buffer/substrate solution, 0.2 ml of liver homogenate (10% w/v) and incubated for exactly 60 min (for ALT) and 30 min for AST at 37 °C in water bath. 1 ml of chromogen solution was added, mixed and allowed to stand for 20 min at room temperature and

10 ml of 0.4N NaOH was added subsequently. The extinction was read at 505 nm against blank. The controls were run in parallel, the substrate being added after deproteinization.

#### Arsenic estimation

Arsenic concentration in blood, liver and kidneys were measured after wet acid digestion using a Microwave Digestion System (CEM, Matthews, NC, USA, model MDS-2100). Arsenic was estimated using a Hydride Vapour Generation System (Perkin Elmer model MHS-10) fitted with an atomic absorption spectrophotometer (AAS, Perkin Elmer model AAnalyst 100; Überlingen, Germany).

#### Statistical analysis

Data are expressed as means  $\pm$  SEM. Data comparisons were carried out using one way analysis of variance followed by Tukey's post test to compare the means of the different treatment groups. Differences between unexposed (with or without chelation) with a *p*-value < 0.05 were considered significant.

## RESULTS AND DISCUSSION

The animals did not show any abnormal behaviour and none died during the experimental period. The difference in body weight at the end of experiment between the groups was not statistically significant (Table 1). There was a marginal (non significant) reduction in fluid intake in *Aloe vera* treated rats but no change in food intake was noted. Liver weight although increased marginally in all the three arsenic exposed rats, was not statistically different from control (Table 1), while, kidney weight remained unchanged in the groups.

Blood arsenic concentrations and changes in some biochemical variables in the blood of arsenic exposed animals are presented in Table 2. Blood arsenic concentrations increased significantly whilst activity of blood ALAD decreased significantly on arsenic exposure. Blood GSH concentration also showed a significant depletion while blood ZPP level increased moderately but not significantly. No effects of *Aloe vera* in normal animals at any dose level, on these parameters, was noted. Co-administration of *Aloe vera* with arsenic produced a significant protective effect on blood ALAD

**Table 1. Body weight, liver and kidneys weight and average food and water intake in different groups**

	Body weight gain	Liver	Kidneys	Mean food intake	Mean water intake
Normal animals	71.3 $\pm$ 4.71	7.2 $\pm$ 0.32	2.1 $\pm$ 0.03	24.2 $\pm$ 2.3	28.1 $\pm$ 1.3
<i>Aloe vera</i> , 1%	73.5 $\pm$ 5.65	7.3 $\pm$ 0.18	2.4 $\pm$ 0.25	25.3 $\pm$ 3.4	26.2 $\pm$ 2.2
<i>Aloe vera</i> , 2%	68.3 $\pm$ 5.64	6.7 $\pm$ 0.67	2.3 $\pm$ 0.06	22.8 $\pm$ 3.2	24.1 $\pm$ 2.1
<i>Aloe vera</i> , 5%	68.5 $\pm$ 4.64	7.6 $\pm$ 0.12	2.4 $\pm$ 0.09	22.5 $\pm$ 4.3	22.5 $\pm$ 1.6
Arsenic	63.7 $\pm$ 5.46	8.3 $\pm$ 0.56	2.2 $\pm$ 0.32	25.3 $\pm$ 5.1	26.1 $\pm$ 2.2
Arsenic + <i>Aloe vera</i> , 1%	57.8 $\pm$ 1.37	8.1 $\pm$ 0.21	2.4 $\pm$ 0.06	26.1 $\pm$ 3.4	25.1 $\pm$ 3.2
Arsenic + <i>Aloe vera</i> , 2%	64.7 $\pm$ 5.94	7.7 $\pm$ 0.34	2.7 $\pm$ 0.06	25.3 $\pm$ 1.5	22.1 $\pm$ 3.6
Arsenic + <i>Aloe vera</i> , 5%	64.7 $\pm$ 4.58	8.4 $\pm$ 0.74	2.5 $\pm$ 0.03	26.1 $\pm$ 3.7	21.1 $\pm$ 2.4

Values are mean  $\pm$  SE; *n* = 5.

**Table 2. Protective effects of *Aloe vera* against arsenic induced changes in few biochemical variables in rat blood**

	ALAD nmol/min/ml erythrocyte	ZPP µmol/mol heme	GSH mg/ml	Blood As µg/100 ml
Normal animals	7.69 ± 0.71	62.0 ± 4.32	5.8 ± 0.03	0.07 ± 0.01
<i>Aloe vera</i> , 1%	8.01 ± 0.65	66.3 ± 6.18	5.4 ± 0.25	0.05 ± 0.00
<i>Aloe vera</i> , 2%	6.83 ± 0.86	63.4 ± 2.87	5.3 ± 0.06	0.08 ± 0.01
<i>Aloe vera</i> , 5%	8.80 ± 0.40	69.4 ± 6.02	6.1 ± 0.09	0.08 ± 0.01
Arsenic	2.57 ± 0.68*	77.7 ± 3.21	4.2 ± 0.32*	2.81 ± 0.10*
Arsenic + <i>Aloe vera</i> , 1%	3.87 ± 0.13	71.2 ± 2.67	4.6 ± 0.06	2.26 ± 0.12
Arsenic + <i>Aloe vera</i> , 2%	5.07 ± 1.68 <sup>†</sup>	65.1 ± 3.01	4.7 ± 0.06	2.53 ± 0.51
Arsenic + <i>Aloe vera</i> , 5%	5.57 ± 0.74 <sup>†</sup>	67.3 ± 5.32	5.0 ± 0.03	2.16 ± 0.23

Values are mean ± SE;  $n = 5$ ; \*  $p < 0.05$  compared to normal animals; <sup>†</sup>  $p < 0.05$  compared to arsenic exposed animals.

**Table 3. Protective effects of *Aloe vera* on some hepatic biochemical parameters in arsenic exposed animals**

	GSH	GSSG	TBARS	ALP	ACP
Normal animals	12.86 ± 2.3	1.24 ± 0.05	5.04 ± 0.51	1.88 ± 0.19	2.64 ± 0.09
<i>Aloe vera</i> , 1%	12.82 ± 1.21	1.17 ± 0.06	3.25 ± 0.19	1.59 ± 0.13	2.47 ± 0.10
<i>Aloe vera</i> , 2%	12.17 ± 1.40	1.05 ± 0.18	3.24 ± 0.14	1.41 ± 0.08	2.73 ± 0.14
<i>Aloe vera</i> , 5%	13.76 ± 0.67	1.24 ± 0.09	3.67 ± 0.26	1.08 ± 0.08	2.74 ± 0.08
Arsenic	10.73 ± 1.75	1.19 ± 0.02	7.72 ± 0.19*	0.67 ± 0.08*	2.56 ± 0.21
Arsenic + <i>Aloe vera</i> , 1%	12.16 ± 0.54	1.39 ± 0.08	5.91 ± 0.24 <sup>†</sup>	0.73 ± 0.03	2.79 ± 0.29
Arsenic + <i>Aloe vera</i> , 2%	12.62 ± 1.16	1.26 ± 0.07	5.43 ± 0.19 <sup>†</sup>	0.83 ± 0.06	3.38 ± 0.07
Arsenic + <i>Aloe vera</i> , 5%	13.79 ± 0.49	0.97 ± 0.10	5.81 ± 0.33 <sup>†</sup>	1.44 ± 0.05 <sup>†</sup>	3.18 ± 0.11

GSH and GSSG = reduced and oxidized glutathione as µg/g; TBARS = thiobarbituric acid reactive substances as µg/g; ALP and ACP = alkaline and acid phosphatase as mgP/g/hr; values are mean ± SE;  $n = 5$ ; \*  $p < 0.05$  compared to normal animals; <sup>†</sup>  $p < 0.05$  compared to arsenic exposed animals.

activity, particularly at the two higher dose levels (2 and 5%), but failed to influence any of the other biochemical variables in blood. This is an interesting observation as there are no previous reports available where *Aloe vera* affects heme synthesis pathway. Herlihy *et al.* (1998), however, reported the long term effects on growth, food intake and serum chemistry, indicating no adverse effects of *Aloe vera* on growth and pathology in rats when given as 1% in the diet. A major enzyme system affected by arsenic involves heme synthesis and inhibition of ALAD by arsenic has been associated with decreased heme synthesis. A small increase in blood ZPP indicates that arsenic also interferes with ferrochelatase. When ferrous ions are not available for incorporation into protoporphyrin to produce heme, protoporphyrin starts chelating other cations such as zinc to produce ZPP, which is then incorporated into RBC instead of heme. Inhibition of blood ALAD and elevation of urinary ALA accompanied by an increase in ZPP correlates well with blood arsenic levels, and has been proposed as an early indicator of arsenic exposure (Flora, 2000; Kannan *et al.*, 2001, 2003). There was a notable trend towards recovery in blood ALAD activity in this study.

The changes in some hepatic biochemical variables indicative of oxidative stress following exposure to arsenic either alone or in combination with different doses of *Aloe vera* are reported in Table 3. A marginal decrease in reduced GSH contents was noted while oxidized glutathione contents remained unchanged on arsenic exposure. Hepatic TBARS content increased significantly on arsenic exposure. Hepatic ALP activity decreased while no significant change in ACP activity was noted. Co-administration of *Aloe vera* (particularly

at the higher dose level) during arsenic exposure led to significant protection in all these changes. Lipid peroxidation has been widely reported to be one of the mechanisms of arsenic toxicity (Ramos *et al.*, 1995; Flora, 1999). A decrease in cellular GSH concentration has been inversely correlated with lipid peroxidation in the liver (Maiti and Chatterjee, 2000, 2001), therefore an increased GSH concentration could presumably protect the organ from arsenic induced lipid peroxidation. A marginal decrease in hepatic GSH correlated well with an increase in hepatic arsenic concentration and TBARS levels.

A significant decrease in AST and ALT activities suggests hepatic injury following arsenic exposure, while depletion of catalase and SOD activity further supports arsenic induced oxidative injury (Table 4). Co-administration of *Aloe vera*, particularly at the highest dose, provided a significant recovery in the depleted AST, ALT, catalase and SOD activities while the two lower doses had no effect. Maiti and Chatterjee (2000) reported a significant and apparent role for decreased activity of catalase and SOD in the induction of lipid peroxidation in liver. The antioxidant potential of *Aloe vera* has not been described in details. Yagi *et al.* (2002) recently reported antioxidant components in *Aloe vera*. They examined lipid peroxidation using rat liver microsomal and mitochondrial enzymes. Among the aloesin derivatives examined, isorabaichromone showed a potent antioxidative activity. Some of other derivatives showed potent DPPH and superoxide anion scavenging activities.

Kidney GSH levels decreased significantly, GSSG levels remained unchanged, while a significant depletion of renal SOD activity was noted, suggesting

**Table 4. Protective effects of *Aloe vera* on some hepatic biochemical parameters in arsenic exposed animals**

	AST	ALT	Catalase	SOD	As
Normal animals	4.66 ± 0.01	6.19 ± 0.40	79.9 ± 3.42	1.83 ± 0.13	0.21 ± 0.01
<i>Aloe vera</i> , 1%	4.46 ± 0.63	5.68 ± 0.34	78.2 ± 10.11	1.58 ± 0.15	0.23 ± 0.03
<i>Aloe vera</i> , 2%	4.46 ± 0.69	4.65 ± 0.18	79.4 ± 2.72	1.63 ± 0.34	0.17 ± 0.03
<i>Aloe vera</i> , 5%	4.03 ± 0.70	5.49 ± 0.24	74.3 ± 3.56	1.77 ± 0.20	0.22 ± 0.04
Arsenic	3.53 ± 0.23*	4.33 ± 0.24*	40.8 ± 5.73*	1.50 ± 0.05*	21.5 ± 0.04*
Arsenic + <i>Aloe vera</i> , 1%	3.17 ± 0.08	4.19 ± 0.22	46.5 ± 5.47	1.62 ± 0.19	23.1 ± 0.26
Arsenic + <i>Aloe vera</i> , 2%	3.55 ± 0.25	5.27 ± 0.12 <sup>†</sup>	49.8 ± 6.24	1.60 ± 0.21	19.1 ± 1.98
Arsenic + <i>Aloe vera</i> , 5%	4.32 ± 0.49 <sup>†</sup>	5.53 ± 0.17 <sup>†</sup>	72.1 ± 8.45 <sup>†</sup>	1.83 ± 0.13 <sup>†</sup>	20.1 ± 2.01

AST and ALT = aspartate and alanine aminotransferase as nmol/min/ml; Catalase as  $\mu$ moles/min/mg protein; SOD = superoxide dismutase as U/mg protein; Liver As =  $\mu$ g/g. values are mean  $\pm$  SE;  $n = 5$ ; \*  $p < 0.05$  compared to normal animals; <sup>†</sup>  $p < 0.05$  compared to arsenic exposed animals.

**Table 5. Protective effects of *Aloe vera* on some renal biochemical parameters in arsenic exposed animals**

	GSH	GSSG	SOD	As
Normal animals	8.16 ± 0.07	0.38 ± 0.03	2.13 ± 0.10	0.18 ± 0.04
<i>Aloe vera</i> , 1%	7.71 ± 0.72	0.35 ± 0.03	1.81 ± 0.10	0.21 ± 0.03
<i>Aloe vera</i> , 2%	7.83 ± 0.12	0.39 ± 0.01	1.98 ± 0.17	0.19 ± 0.04
<i>Aloe vera</i> , 5%	7.07 ± 0.70	0.37 ± 0.01	1.96 ± 0.22	0.23 ± 0.02
Arsenic	6.72 ± 0.33*	0.32 ± 0.02	1.67 ± 0.20*	15.1 ± 1.21*
Arsenic + <i>Aloe vera</i> , 1%	6.93 ± 0.61	0.37 ± 0.02	1.89 ± 0.46	13.1 ± 1.98
Arsenic + <i>Aloe vera</i> , 2%	5.90 ± 0.62	0.30 ± 0.01	2.31 ± 0.38	13.7 ± 0.87
Arsenic + <i>Aloe vera</i> , 5%	7.36 ± 0.97	0.33 ± 0.01	2.56 ± 0.14	11.1 ± 0.67

GSH and GSSG = reduced and oxidized glutathione as  $\mu$ g/g; SOD = superoxide dismutase as U/mg protein; Kidney As as  $\mu$ g/g; values are mean  $\pm$  SE;  $n = 5$ ; \*  $p < 0.05$  compared to normal animals.

arsenic-induced oxidative stress (Table 5). Simultaneous supplementation of *Aloe vera* prevented the appearance of signs of arsenic induced oxidative stress in kidneys but had no effect on kidney arsenic concentration at any of the three dose levels.

The above results suggest that *Aloe vera* has some protective value against arsenic-induced oxidative stress,

but these effects are independent of arsenic depletion from these organs.

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