Bull Environ Contam Toxicol (2012) 89:61–66
DOI 10.1007/s00128-012-0620-0

Effect of Vitamin E Supplementation on Arsenic Induced Oxidative Stress in Goats

T. K. Das · V. Mani · H. Kaur · N. Kewalramani · S. De · A. Hossain · D. Banerjee · B. K. Datta

Received: 10 January 2012 / Accepted: 21 March 2012 / Published online: 3 April 2012

Abstract The present study was designed to assess whether supplementation of different levels of vitamin E to long-term arsenic exposed goats affords protection against the oxidative stress caused by the metalloid. Twenty-four crossbred lactating goats were distributed randomly into four groups (control, T_1, T_2 and T_3) of six in each. The animals in T_1, T_2 and T_3 were given 50 mg/kg DM arsenic daily, while in T_2 and T_3, vitamin E @100 IU and 150 IU/kg DM, respectively, was also supplemented additionally for the period of 12 months. Compared to control, significant ($p < 0.05$) decline in SOD (45 %), CAT activities of erythrocytes (63 %), plasma total Ig (22 %) and total antioxidant activity (24 %) was observed in only arsenic treated groups and vitamin E supplementation in both doses produced partial mitigation effect against SOD (23 %, 20 %) and CAT (39 %, 48 %) while complete mitigation against total Ig (16 %, 7 %) and antioxidant activity (10 %, 8 %) was found. Average lymphocyte stimulation index at the end of experiment was ($p < 0.05$) lower in arsenic exposed groups (1.003 ± 0.01) and significant ($p < 0.05$) recovery was observed in response of vitamin E supplementation at higher doses (1.138 ± 0.03). So, vitamin E is helpful in reducing the burden of arsenic induced oxidative stress and activities of antioxidant enzymes in goats.

Keywords Arsenic · Oxidative stress · Goat · Vitamin E

T. K. Das (✉) · V. Mani · H. Kaur · N. Kewalramani · S. De · A. Hossain · D. Banerjee
National Dairy Research Institute, Karnal 132001, Haryana, India
e-mail: tapanndri@gmail.com

B. K. Datta
West Bengal University of Animal and Fishery Sciences, Kolkata 700037, West Bengal, India

Arsenic pollution in the environment is becoming a major concern for environmental and occupational health, owing to its widespread toxic effects on humans, animals, birds, aquatic life and plants through polluted ground water and food chains. Many countries of the world including Argentina, Bangladesh, India, Mexico, Thailand, and Taiwan have documented acute and chronic arsenic toxicity both in man and animals due to drinking of contaminated water (Tchounwou et al. 1999). Various livestock animals are also victims of such catastrophes arising from arsenic pollution. The health effects of toxic level of arsenic are multi-dimensional in both human and animal population. Arsenic has been reported to be responsible for defective cell mediated immunity and decreased percentage of T helper cells in the body (Yu et al. 2002). Arsenic toxicity involves oxidative damage that plays a vital role for biochemical and molecular alteration. Decreased level of antioxidants, increased levels of oxidation products in blood were reported in human population exposed to arsenic (Wu et al. 2001). Various studies reported that arsenic could participate in the cellular oxidation–reduction reactions resulting with the formation of excess ROS such as superoxide anion (O_2^-) and hydroxyl radical (OH^-) via a chain reaction (Garcia-Shavez et al. 2006). The potential role of oxidative stress in the injury associated with arsenic poisoning suggests that antioxidants may enhance the efficacy of treatment protocols designed to mitigate arsenic induced toxicity.

Beneficial role of antioxidants have been reported earlier against arsenic toxicity using rat model (Nandi et al. 2006). Basic information on arsenical poisoning in cattle and small ruminants are meagre, except a report of development of anemia, gastrointestinal signs, toxic nephropathy and mortality in goats with induced chronic arsenic toxicity (Biswas et al. 1998).

For cattle, sheep and swine maximum tolerable dietary arsenic recommended is 50 mg/kg (inorganic) and 100 mg/kg (organic). The purpose of this study was to evaluate the cumulative effect of recommended inorganic arsenic in a long term exposure. Role of vitamin E which acts as first line of defence in ruminants against pro-oxidant not much reported. As the vitamin E requirement of all classes goat is 100 IU/kg (Morand-Fehr 1981), therefore 100 and 150 IU/kg doses were decided to see the antioxidant effect. Keeping in view the above facts, the present investigation was undertaken to assess the protective effect of different doses of vitamin E against arsenic induced changes in the oxidative stress indices and immunological variables in lactating goats.

Materials and Methods

Twenty-four crossbred lactating goats (2nd to 3rd stage of lactation) were selected from the Cattle Yard of National Dairy Research Institute, Karnal, India and distributed randomly into four groups of six each. The distribution of the animals was done on the basis of average milk yield (1.61 ± 0.004 kg/day) and body weight (36.10 ± 0.11 kg). The experimental feeding period was of 12 months. The details of the treatments were as follows.

Group I(C): Control Animals

Group II (T_1): Arsenic as sodium arsenite (s-d-Fine-Chem Limited, India, purity 98.5 %), 50 mg/kg dry matter (DM).

Group III (T_2): Arsenic as in group II plus vitamin E as dl-α-tocopheryl acetate (Lutavit, BASF, Germany, minimum assay 50%), 100 IU/kg DM.

Group III (T_3): Arsenic as in group II plus vitamin E, 150 IU/kg DM.

The goats were housed in well ventilated pens having facilities for individual feeding. The experiment was performed with approval from Institute (NDRI, India) Animal Ethics Committee (IAEC No. 28/09-21/11/2009). The animals were fed as per NRC (1981) feeding standard to meet their nutrient requirements. The concentrate mixture was offered in the morning in the plastic tubs whereas, the chaffed green fodder was offered at 11:00 am. As it was a long term study, two types of fodder either berseem or maize depending upon the season/availability was provided. Concentrate mixture (maize 33 %, groundnut cake (oiled) 21 %, mustard oil cake (oiled) 12 %, wheat bran 20 %, deoiled rice bran 11 %, mineral mixture 2 % and common salt 1 %) having CP 19.81 % and TDN 70 % was procured from Godrej Agrovet Pvt Ltd. Content of arsenic in concentrate mixture, berseem and maize was 1.116, 0.10 and 0.09 mg/kg and content of vitamin E was 10.72, 24.86 and 5.99 mg/kg respectively. Arsenic containing capsules were administered orally to each goat of T_1, T_2 and T_3 groups whereas vitamin E capsules of 100 and 150 IU doses were given to each goat of T_2 and T_3 groups, respectively.

Blood samples were collected aseptically from jugular vein of each animal using heparinized vacuitainer tube. Immediately after collection plasma was separated by centrifuging the whole blood samples at 840 g for 15 min in a portable centrifuge machine. The heparinized plasma samples were stored at −20°C in storage vials and analyzed subsequently. Total immunoglobulin was estimated by precipitation method of McEwan and Fisher (1970). Total antioxidant activity was measured by ferric reducing antioxidant power assay of Benzie and Strain (1999) and values were expressed as μmol/l of plasma. Erythrocyte superoxide dismutase (SOD) activity was estimated in haemolysate 1:10 as per the method described by Madesh and Balasubramanian (1998) and expressed as U. Catalase (CAT) activity in RBC hemolysate was estimated following the method of Aebi (1984) and the values were expressed as μmoles of H_2O_2 consumed/min/g Hb in blood. Cell mediated immunity was calculated as stimulation index (SI) during blastogenic response of lymphocyte culture on stimulation with mitogen. The proliferative response of lymphocyte was estimated using the colorimetric 3-(4-5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) according to the procedure given by Mosmann (1983).SI is a ratio of optical density of stimulated and unstimulated cells. The data were analyzed using two way repeated measures ANOVA with two factors i.e., different treatment groups and periods. All results were compared to control animals, as well as to the arsenic exposed animals, in order to elucidate the possible protective effect of vitamin E supply on arsenic toxicity. Tukey's all pair wise multiple comparison tests have been used to compare means between the different treatment groups. The results were represented as mean ± SE in the treatment and period interaction table mentioning the significance level. The specific p values were mentioned in the text for where significant difference was found.

Results and Discussion

Data revealed that after 3 months of dietary exposure of arsenic, there was significant ($p < 0.05$) increase in SOD activities in the supplemented groups followed by gradual decrease in the activities in comparison to un-supplemented groups (Table 1). Up to 9 months, activity in T_2 and T_3 was statistically comparable with control values. But after that values were less than control but higher than T_1. From the critical perusal of the data, it was cleared that

Table 1 Effect of vitamin E supplementation on erythrocytic activity of superoxide dismutase and catalase in arsenic fed goats

Group	Months													Mean
	0	1	2	3	4	5	6	7	8	9	10	11	12	
Superoxide dismutase (Units/g Hb/min)														
C	85.16 ± 1.08	87.94 ± 2.77	87.57 ± 1.74	86.41^{a} ±1.47	88.42^{b} ± 1.14	88.68^{b} ± 1.19	84.85^{b} ± 1.54	85.45^{b} ± 1.69	84.95^{b} ± 1.48	84.98^{c} ± 1.80	85.42^{c} ± 1.82	84.63^{c} ± 1.53	85.07^{c} ± 1.57	86.12^{b} ± 0.41
T_1	85.16 ± 1.35	88.74 ± 2.76	93.83 ± 2.17	85.34^{a} ±1.64	70.41^{a} ± 2.12	58.00^{a} ± 2.32	51.02^{a} ± 2.36	54.02^{a} ± 1.38	53.51^{a} ± 1.94	53.45^{a} ± 2.28	51.05^{a} ± 1.77	50.56^{a} ± 2.14	46.81^{a} ± 1.89	64.76^{a} ± 4.81
T_2	87.25 ± 2.41	90.03 ± 2.95	94.97 ± 2.58	100.67^{b} ±3.58	95.03^{bc} ± 1.98	85.01^{b} ± 1.17	84.05^{b} ± 1.89	80.03^{b} ± 1.81	78.03^{b} ± 2.10	77.00^{b} ± 2.03	74.01^{b} ± 1.67	69.98^{b} ± 2.09	66.99^{b} ± 1.92	83.31^{b} ± 2.83
T_3	87.29 ± 2.05	91.22 ± 2.80	93.80 ± 2.74	98.61^{b} ±3.51	95.99^{c} ± 2.38	89.98^{b} ± 2.59	87.97^{b} ± 1.94	83.99^{b} ± 2.09	81.99^{b} ± 1.38	79.03^{bc} ± 2.02	77.03^{b} ± 1.93	73.98^{b} ± 1.36	70.18^{b} ± 1.89	85.46^{b} ± 2.41
Mean	86.21 ± 0.61	89.48 ± 0.72	92.54 ± 1.68	92.76 ± 4.00	87.46 ± 5.93	80.42 ± 7.55	76.97 ± 8.69	75.87 ± 7.37	74.62 ± 7.18	73.61 ± 6.93	71.88 ± 7.35	69.79 ± 7.12	67.26 ± 7.87	
Catalase (μmol of H_2O_{2-} consumed/min/mg Hb)														
C	116.88 ± 8.99	110.75 ± 4.62	111.80 ± 6.23	118.44 ± 7.43	118.25 ± 7.49	110.52^{a} ± 4.93	127.60 ± 6.40	121.35 ± 7.16	123.08 ± 5.30	126.66^{b} ± 4.77	129.98^{b} ± 6.51	120.18^{b} ± 8.74	117.03^{c} ± 6.40	119.43^{b} ± 1.75
T_1	120.03 ± 4.22	105.60 ± 6.80	107.29 ± 5.45	123.75 ± 6.85	128.43 ± 7.41	136.94^{b} ± 3.38	114.82 ± 5.20	110.02 ± 5.50	104.69 ± 6.99	97.00^{a} ± 8.00	83.41^{a} ± 7.26	64.39^{a} ± 5.04	44.72^{a} ± 5.02	103.16^{a} ± 7.18
T_2	113.17 ± 7.26	99.89 ± 5.38	105.45 ± 6.13	110.23 ± 5.30	109.53 ± 5.79	118.36^{ab} ± 6.62	110.29 ± 5.44	104.07 ± 6.56	100.79 ± 5.95	93.00^{a} ± 6.20	82.17^{a} ± 6.89	65.49^{a} ± 6.05	68.87^{b} ± 6.38	98.56^{a} ± 5.44
T_3	115.56 ± 9.26	96.42 ± 5.13	98.87 ± 5.78	104.18 ± 6.07	106.62 ± 6.74	119.71^{ab} ± 5.27	116.59 ± 6.72	110.53 ± 4.96	107.24 ± 5.61	99.32^{a} ± 6.07	90.73^{a} ± 7.33	76.32^{a} ± 7.40	59.13^{ab} ± 7.72	100.10^{a} ± 4.71
Mean	116.41 ± 1.43	103.17 ± 3.16	105.85 ± 2.68	114.15 ± 4.33	115.71 ± 4.91	121.38 ± 5.57	117.33 ± 3.67	111.49 ± 3.60	108.95 ± 4.89	104.00 ± 7.67	96.57 ± 11.30	81.59 ± 13.14	72.44 ± 16.53	

Mean within the column bearing different superscript (a, b, c) are significantly different ($p < 0.05$)

Table 2 Effect of vitamin E supplementation on plasma total Ig concentration and total antioxidant activity in arsenic fed goats

Group	Months													Mean
	0	1	2	3	4	5	6	7	8	9	10	11	12	
Total Ig concentration (mg/ml)														
C	23.53 ± 0.57	22.27 ± 1.51	23.26 ± 1.07	24.52 ± 0.55	23.46^{a} ± 0.68	25.91 ± 0.81	23.84 ± 0.97	24.24 ± 0.95	23.46 ± 1.14	26.29^{b} ± 1.20	23.77^{b} ± 0.93	23.14^{b} ± 1.06	22.28^{b} ± 0.83	23.84 ± 0.33
T_1	22.60 ± 1.40	22.43 ± 1.55	25.32 ± 1.16	28.48 ± 1.18	28.01^{b} ± 0.53	27.06 ± 1.12	22.16 ± 1.19	20.51 ± 1.05	19.44 ± 1.00	19.12^{a} ± 1.34	18.87^{a} ± 1.30	18.06^{a} ± 1.18	17.61^{a} ± 0.78	22.28 ± 1.06
T_2	22.98 ± 1.49	22.67 ± 1.54	23.99 ± 1.17	27.87 ± 1.50	27.44^{ab} ± 1.08	26.67 ± 1.38	22.52 ± 0.97	20.55 ± 0.96	20.31 ± 0.82	20.07^{a} ± 1.01	20.04^{ab} ± 1.07	19.51^{ab} ± 0.94	19.33^{ab} ± 0.85	22.61 ± 0.85
T_3	22.67 ± 0.98	22.62 ± 1.62	23.87 ± 1.27	27.76 ± 2.14	27.35^{ab} ± 1.51	26.64 ± 1.72	24.46 ± 1.02	24.14 ± 1.10	23.40 ± 0.74	23.12^{ab} ± 0.95	22.80^{ab} ± 0.95	21.91^{ab} ± 1.15	21.14^{ab} ± 1.20	23.99 ± 0.57
Mean	22.95 ± 0.21	22.50 ± 0.09	24.11 ± 0.43	27.16 ± 0.89	26.56 ± 1.05	26.57 ± 0.24	23.24 ± 0.54	22.36 ± 1.06	21.65 ± 1.04	22.15 ± 1.62	21.37 ± 1.15	20.65 ± 1.15	20.09 ± 1.03	
Total antioxidant activity (μmol/l)														
C	1,114.8 ±31.2	1,157.9 ±34.15	1,209.8^{a} ±22.50	1,235.7^{a} ±23.75	1,204.6^{a} ±36.25	1,140.9^{a} ±43.14	1,159.8^{a} ±43.97	1,184.5 ±37.67	1,220.4 ±48.44	1,160.2 ±43.11	1,165.2 ±44.33	1,173.2^{b} ±42.20	1,157.4^{b} ±44.17	1,171.6 ±8.06
T_1	1,206.1 ±44.82	1,244.0 ±29.67	1,422.6^{b} ±38.38	1,632.8^{c} ±37.44	1,420.1^{b} ±47.74	1,333.6^{b} ±56.10	1,265.6^{ab} ± 43.30	1,205.9 ±54.02	1,116.6 ±39.12	1,073.6 ±42.37	1,050.6 ±58.62	994.3^{a} ±49.44	915.3^{a} ±50.81	1,221.6 ±55.01
T_2	1,134.6 ±46.99	1,160.1 ±34.49	1,310.3^{ab} ±58.10	1,397.7^{b} ±55.70	1,436.1^{b} ±39.95	1,397.8^{b} ±34.74	1,330.0^{b} ±44.20	1,258.3 ±38.98	1,218.3 ±46.66	1,179.9 ±49.34	1,140.7 ±45.73	1,069.0^{ab} ±39.21	1,016.9^{ab} ± 21.18	1,237.1 ±35.65
T_3	1,116.3 ±30.82	1,150.5 ±37.33	1,203.3^{a} ±27.63	1,281.9^{ab} ±42.81	1,300.2^{ab} ±45.32	1,344.0^{b} ±46.67	1,300.9^{ab} ±52.49	1,240.5 ±42.57	1,197.6 ±46.01	1,160.2 ±46.62	1,030.4 ±33.83	1,079.7^{ab} ±40.86	1,019.8^{ab} ±33.34	1,192.8 ±28.49
Mean	1,142.9 ±21.53	1,178.2 ±22.05	1,290.8 ±51.29	1,380.7 ±92.51	1,340.6 ±54.17	1,304.1 ±56.18	1,264.1 ±37.17	1,222.3 ±16.63	1,188.2 ±24.41	1,143.5 ±23.76	1,096.7 ±33.11	1,079.1 ±36.67	1,044.3 ±49.65	

Mean within the column bearing different superscript (a, b) are significantly different ($p < 0.05$)

vitamin E supplementation in both the doses (100 IU and 150 IU/kg) were able ($p < 0.001$) to mitigate the adverse effect as the values were statistically similar to control. A significant treatment x period interaction ($p < 0.001$) was observed for SOD activity during the study. Up to 5 months of experimental feeding, there was gradual increase of CAT activity in all arsenic supplemented groups compared to control but after that decline trend in activity was observed (Table 1). After 11 months, T_2 groups showed significant ($p < 0.001$) increase in CAT activity compared to T_1 though between the two doses of vitamin E there was no difference. Treatment × period interaction ($p < 0.001$) was found during the experimental period. Overall vitamin E supplementation in both the doses was not sufficient to increase the CAT activity.

In the present investigation, reduced SOD and CAT activity might be due to an enhanced production of superoxide radicals or down-regulation in the synthesis of antioxidant enzymes by persistent toxic insult (Irshad and Chaudhuri 2002). The sharp rise of SOD and CAT activity at 3 months and 5 months, respectively of arsenic feeding might be attributed to up-regulation in the synthesis of SOD or CAT, as a self protective response against oxidative stress (Pi et al. 2002). The decline in activities following an initial enhanced activity during the course of arsenic exposure in present study might be due to down regulated synthesis or over-utilization of antioxidant enzymes resulting failure of adaptive mechanism. Similar to our findings, Rana et al. (2010) also reported significant decreased SOD and CAT activity in cattle of arsenic prone zone. Nandi et al. (2008) reported that the administration of ascorbic acid as an antioxidant restored blood SOD and CAT activities towards near normalcy in arsenic exposed rats after 8 weeks.

Up to 3 months, there was no significant effect of dietary treatments on total Ig but at 4th months there was significant ($p < 0.05$) increase in three arsenic supplemented groups compared to control (Table 2). From 5th months onwards, there was a declining trend in total Ig in T_1, T_2 and T_3 groups. In vitamin E supplemented groups, final values were more than T_1 but comparable to control, which indicated the ameliorating potential of vitamin E. There was also treatment x period interaction ($p < 0.001$) during the study i.e. the effect of different levels of treatment depends on what level of period was present. Initial increase in total Ig during this study may be due to adaptive mechanism to counteract the oxidative stress induced by arsenic. Besides, arsenic has a cytotoxic effect on the antibody producing cells as it causes necrosis and depletion of lymphoid cells in the immuno-biological organs so that immune-suppression may be a possible outcome of chronic arsenic intoxication (Biswas et al. 2000). In the present study, major impact of vitamin E on immunity most probably attributable to their antioxidant properties and may due to their enhanced neutrophil function which minimizes the free radical induced damage (Politis et al. 1995).

T_1 groups showed an initial significant ($p < 0.001$) increase in total antioxidant activity at 3 months which was followed by gradual decrease up to the end of the experiment (Table 2). In groups T_2 and T_3, total antioxidant activity increased up to 4 months and after that values were decreased as the days of supplementation increases. Whereas, the final values were higher than T_1 but statistically equal to control group. So, vitamin E supplementation in both the doses produced significant mitigating effect. There was a significant treatment x period interaction ($p < 0.001$) observed during the study. The decrease in total antioxidant activity in arsenic supplemented group might be due to increased production of reactive oxygen metabolites. Initial elevation of total antioxidant activity due to adaptation on part of the system to counteract the

Table 3 Effect of vitamin E supplementation on lymphocyte stimulation index in arsenic fed goats

Days	Group				Mean
	Control	T_1	T_2	T_3	
0	1.273 ± 0.09	1.250 ± 0.06	1.270 ± 0.04	1.283 ± 0.02	1.269 ± 0.01
45	1.271 ± 0.07	1.240 ± 0.06	1.264 ± 0.05	1.280 ± 0.03	1.264 ± 0.01
90	1.255 ± 0.06	1.202 ± 0.02	1.230 ± 0.05	1.260 ± 0.02	1.237 ± 0.01
135	1.263 ± 0.05	1.167 ± 0.05	1.202 ± 0.05	1.228 ± 0.04	1.215 ± 0.02
180	1.270 ± 0.05	1.156 ± 0.01	1.172 ± 0.03	1.218 ± 0.03	1.204 ± 0.03
225	1.263 ± 0.05	1.130 ± 0.02	1.176 ± 0.03	1.200 ± 0.01	1.192 ± 0.03
270	1.278^{b} ± 0.07	1.090^{a} ± 0.01	1.145^{ab} ± 0.03	1.167^{ab} ± 0.03	1.170 ± 0.04
315	1.280^{b} ± 0.04	1.050^{a} ± 0.03	1.101^{a} ± 0.02	1.150^{ab} ± 0.02	1.145 ± 0.05
360	1.286^{b} ± 0.03	1.003^{a} ± 0.01	1.086^{a} ± 0.04	1.138^{ab} ± 0.03	1.128 ± 0.06
Mean	1.271 ± 0.03	1.143 ± 0.03	1.183 ± 0.02	1.214 ± 0.02	

Mean within the row bearing different superscript (a, b) are significantly different ($p < 0.05$)

oxidative stress. Further feeding of arsenic caused failure of adaptive mechanism towards chronic arsenic exposure. It has been reported that the free radicals attack the double bonds of polyunsaturated fatty acids and thereby initiating a chain reaction which affect membrane integrity and cellular function. This chain reaction is inhibited by vitamin E by reacting with free radicals and converting itself into α-tocopheroxyl radical which is not harmful, thus vitamin E checks lipid peroxidation (Bueter 1993).

Average lymphocyte SI at the end of experiment was significantly ($p < 0.001$) lower in T_1 group as compared to control and T_3 due to arsenic supplementation (Table 3). Compared to initial values, there was gradual decline trend in SI in T_1 (19.76 %), T_2 (14.48 %) and T_3 (11.30 %) at the end of dietary treatments. T_1 group showed it's significant ($p < 0.05$) adverse effect from 270 days onwards. Arsenic with vitamin E supplementation at higher doses (T_3) produced ameliorating effect in improving SI compared to T_1 groups from 315 days of supplementation. Treatment x period interaction ($p < 0.001$) was encountered for SI during the study. Inorganic arsenicals by inhibiting DNA synthesis of lymphocytes affected the cellular immunity adversely because of decreased lymphocyte proliferation (Meng and Meng 2000). Increased SI in vitamin E supplemented group might be due to enhance chemotaxis by increasing receptor-bound urokinase-plasminogen activator in neutrophils and production of interleukin-1 (IL-1) and major histo-compatibility (MHC) class II antigen expression by blood monocytes (Politis et al. 1995).

So, specific protection of vitamin E from oxidative damage may be helpful to combat arsenic-associated adverse effect in animal.

Acknowledgments Authors would like to express their sincere thanks to the Director of the Institute for providing necessary helps.

References

Aebi H (1984) Catalase in vitro. In: Packer L (ed) Methods in enzymology, vol 105. Academic press, New York, pp 121–126

Benzie EFI, Strain JJ (1999) Ferric reducing antioxidant power assay: direct measurement of total antioxidant activity of biological fluids and modified version for simultaneous measurement of total antioxidant and ascorbic acid concentration. Methods Enzymol 299:15–27

Biswas U, Sarkar S, Bhowmik MK, Roy S (1998) Clinicopathological profile of induced chronic arsenic toxicity in goats. Indian J Anim Sci 68:320–323

Biswas U, Sarkar S, Bhowmik MK, Samanta SK, Biswas S (2000) Chronic toxicity of arsenic in goats: clinicobiochemical changes, pathomorphology and tissue residues. Small Rumin Res 38:229–235

Bueter GR (1993) The pecking order of free radicals and antioxidants. Lipid peroxidation, α-tocopherol and ascorbate. Arch Biochem Biophys 300:535–543

Garcia-Shavez E, Jimenez I, Segura B, Razo LMD (2006) Lipid peroxidative damage and distribution of inorganic and its metabolite in the rat nervous system after arsenite exposure: influence of alpha tocopherol. Neurotoxicol 27:1024–1031

Irshad M, Chaudhuri PS (2002) Oxidant-antioxidant system: role and significance in human body. Indian J Exp Biol 40:1233–1239

Madesh M, Balasubramanian KA (1998) Microtiter plate assay for superoxide dismutase using MTT reduction by superoxide. Indian J Biochem Biophys 35:184–188

McEwan AD, Fisher EW (1970) A turbidity test for the estimation of immunoglobulins levels in neonatal calf serum. Clin Chim Acta 17:155

Morand-Fehr P (1981) Nutrition and feeding of goats: application to temperate climate. In: Gall G (ed) Goat production. Academic Press, London, pp 193–233

Meng ZQ, Meng NY (2000) Effects of arsenic on blast transformation and DNA synthesis of human blood lymphocytes. Chemosphere 41(1–2):115–119

Mosmann T (1983) Rapid colorimetric assay for cellular growth and survival application to proliferation and cytotoxicity assay. J Immunol Methods Des 65:55–63

Nandi D, Patra RC, Swarup D (2006) Oxidative stress indices and plasma biochemical parameters during oral exposure to arsenic in rats. Food Chem Toxicol 44:1579–1584

Nandi D, Patra RC, Ranjan R, Swarup D (2008) Role of co-administration of antioxidants in prevention of oxidative injury following sub-chronic exposure to arsenic in rats. Veterinarski Arhiv 78:113–121

NRC (1981) Nutrient requirements of goats: Angora, dairy and meat goats in temperate and tropical countries. National Academy Press, Washington

Pi J, Yamauchi H, Kumagai Y (2002) Evidence for induction of oxidative stress caused by chronic exposure of Chinese residents to arsenic contained in drinking water. Environ Health Persp 110:331–336

Politis I, Hidiroglou M, Batra TR, Gilmore JR, Gorewit RC, Scherf H (1995) Effects of vitamin E on immune function of diary cows. Am J Vet Res 56:179–184

Rana T, Bera AK, Das S, Bhattacharya D, Bandyopadhyay S, Pan D, Das SK (2010) Effect of chronic intake of arsenic-contaminated water on blood oxidative stress indices in cattle in an arsenic-affected zone. Ecotoxicol Environ Safety 73:1327–1332

Tchounwou PB, Wilson B, Ishaque A (1999) Important considerations in the development of public health advisories for arsenic and arsenic containing compounds in drinking water. Rev Environ Health 14:211–219

Wu MM, Chiou HY, Wang TW, Hsues YM, Wang IH, Chen CJ, Lee TC (2001) Association of blood arsenic levels with increased reactive oxidants and decreased antioxidant capacity in a human population of northeastern Taiwan. Environ Health Perspect 109:1011–1017

Yu HS, Liao WT, Chang KL, Yu GL, Chen GS (2002) Arsenic induces tumor necrosis factor alpha release and tumor necrosis factor receptor 1 signaling in T helper cell apoptosis. J Invest Dermatol 119:812–819