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RESEARCH ARTICLE

Role of zinc on lipid peroxidation and antioxidant status in liver and muscle tissues of rats under ammonium sulfate-induced stress

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ABSTRACT

Background: Ammonia is an important source of nitrogen metabolism and it is necessary for the synthesis of protein and amino acids. An excessive level of ammonia leads to disturbing the physiological functions of the body, causes to increase the free radical levels in tissues and body fluids, its reveal that damage of tissue by reactive oxygen species (ROS) changes of acid-base lances in the body fluid's causes' physiological disturbance and damage of organs. **Aims and Objectives:** The present study was conducted to assess the mitigating role of zinc on ammonium sulfate (AS)-induced biochemical alterations (lipid peroxidation [LPO] and antioxidants) in rat liver and muscle. **Materials and Methods:** Rats were divided into four groups (six animals in each group). Group I (GI) served as control and rats provided with normal diet and water, Group II (AS) rats treated intraperitoneal (i.p) with 18.3 mg/kg b.w of AS, Group III (zinc chloride [Zc]) rats administered with Zc (4 mg/kg b.w i.p), and Group IV (AS + Zc) treated with both of AS (18.3 mg/kg bwi.p) plus Zc (4 mg/kg b.w i.p) Zc given after 1 h dosage of AS. **Results:** In the present investigation, AS-treated animals showed a significant increase in the levels of LPO and declined levels of selected antioxidants such as superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and glutathione reductase (GR) levels in the liver and muscle of rats. On the other hand, only zinc-treated rats (Zc) showed no significant variations in LPO and antioxidants in the liver and muscle. However, in AS + Zc treated animals, elevated levels of LPO and decreased SOD, CAT, GPx, and GR antioxidant levels were reversed to normal when compared with AS-treated rats. **Conclusion:** Accordingly, these findings suggest that zinc supplementation significantly inhibits the oxidative stress in hepatic cells and muscle cells in AS toxicity.

KEY WORDS: Ammonium sulfate; Zinc chloride; Lipid peroxidation; Antioxidants; Liver; Muscle

INTRODUCTION

Ammonium compounds are actually commercial inorganic fertilizers used for high yield of crops in the agriculture area as well as household gardening. They are also used as coproduct in the production of formic acid, acrylamide, and synthetic fiber intermediates (methyl methacrylate, acrylonitrile, and caprolactam).[1] Ammonium compounds are also used in food and beverage industries and printing industries. Utilization in excess of nitrogenous pollutants actually enters the aquatic ecosystem and runoff from land and industrial sewage, results in toxic effect on living forms as well as terrestrial forms through the food chain, and gets accumulated in their body.[2,3] In the present days, most of the ammonium compounds used as a quaternary form in commercial products such as shampoos, bathroom cleaners, cosmetics, hair color dyes, and food additives.[4-6] The overexposure of these products in our daily purpose enters into our body and accumulates in body fluid causes the multiorgan dysfunctions through disturbing the urea

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cycle pathway. The liver is the one and only chief organ for metabolization of excessive levels of ammonium levels through ornithine cycle and excreted by the kidneys in the form of urine. However, excessive levels of ammonium levels in the body fluids lead to hyperammonemia condition, which is responsible for the functional impairment of hepatocytes in the liver causing hepatic encephalopathy.[7] Ammonia noxiousness arises essentially by redox homeostasis, which leads enhanced, increasing lipid peroxidation (LPO) and the generation of free radicals and reactive oxygen species (ROS) in the liver and muscle tissues by the oxidative stress. Oxidative stress aggravated by ammonia causes unnecessary synthesis of superoxide radical (O2-) and hydrogen peroxide (H_2O_2).[8] Excessive ROS damage the lipids, proteins, and DNA by decreasing levels of antioxidant enzyme levels and enhanced levels of stress markers malondialdehyde (MDA).[9]

In the present study, ammonium sulfate (AS) toxicity on the antioxidant enzymes superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and glutathione reductase (GR). The stress marker LPO MDA levels were investigated in male albino rat liver and muscle tissue.

Zinc is a multipurpose trace element required for different metabolic functions in all living organisms.[10] A sufficient intake of zinc is important for as it supports the body to regulate the key functions including cell proliferation, immune functions, protein synthesis, cell division, carbohydrate metabolism, DNA synthesis, and reproductive functions.[11-13,3] Zinc is considered as an endogenous antioxidant for the stress suppressor for the many toxicants because zinc is one of the internal parts of Cu-zinc SOD antioxidant and regulates the SOD depended on CAT levels.[14] The excessive free radicals are consumed by zinc and control the internal metabolic stress for proper functioning of organs.[15] Previously, several reports reported on the beneficial (antioxidant) role of zinc, against the toxic chemicals.[16] Zinc deficiency leads to hepatic encephalopathy and muscle fatigue.[17,18] Zinc supplementation protects the liver by increasing of antioxidants under the cadmium,[19] aluminum sulfate,[20] nickel,[21] and arsenic toxicants.[22]

Consequently, the intention of the present work is to study whether the zinc might chelate the ammonia generating oxidative radicals and reduce the ammonia stress by enhancing of antioxidants and reduction of MDA levels in the liver and muscle tissue of the male rat.

MATERIALS AND METHODS

Procurement of Chemicals

AS and zinc chloride (Zc) (analar grade) chemicals were used for the present study and purchased from Molychem, Mumbai (Maharashtra).

Animal's Acclimatization and Maintains

In the present study, 24 animals are used for experimental purpose. Healthy Wistar strain male albino rats are obtained from authorized scientific company Sree Raghavendra Enterprises, Bangalore. Before experimentation, all animals are acclimatized to laboratory condition and fed with proper healthy rat chew purchased from the same scientific company mentioned above. The laboratory conditions are maintained at 27 ± 2°C temperature, humidity 45 ± 3, and sterile paddy husk used as bed material in the cages. Animals are fed with standard rat chew and water *ad libitum*, and maintained with 12 h light and 12 h dark cycle during the entire experimental period. The experiments were carried out in accordance with the guidelines of the Institutional Animal Ethical Committee, Sri Venkateswara University, Tirupati, India (Resolution No. 10/(i)/a/CPCSEA/IAEC/SVU/ZOOL/PN/Dt. July 08, 2012).

Experimental Design

In the present study, a total of 24 healthy 80-day-old male Wistar strain rats, weighing 220 ± 10 g were used. The 24 animals were randomly divided into four groups, six animals in each group. Group I (G1) animals taken as a control, Group II (G2) animals treated with 18.3 mg/kg b.w of AS through intraperitoneally to 24 h time interval, Group III (G3) animals supplanted with Zc (4 mg/kg b.w i.p), and Group IV (G4) animals administered with both of AS and Zc, after 1 h time interval of AS injected. After the end of the experimental period (7 days), all animals were sacrificed by cervical dislocation and selected tissues, namely liver and muscle (thigh), were isolated, quickly washed in 0.9% saline. The liver and muscle tissues were kept in deep freeze at −40°C for the analysis of biochemical parameters.

Biochemical Assays

Oxidative stress marker lipid peroxidation (MDA) levels in liver and muscle tissues were assayed by previously described method by Ohkawa *et al*.[23] The antioxidant enzyme levels such as SOD, CAT, GPx, and GR activity were estimated according to the methods of Misra and Fridovich,[24] Aebi,[25] Flohé and Günzler,[26] and Carlberg and Mannervik.[27] The total protein levels were estimated by the method of Lowry *et al*.(1951).[28]

Statistical Analysis

Data were statistically analyzed using one-way analysis of variance followed by Dunnett's multiple comparison test. $P < 0.05$ was considered statistically significant. The results were represented as mean ± standard deviation (SD) of six observations. % - Percent change over control and AS, [a]Values are significantly over control at $P < 0.05$, [b]Values are significantly over ammonium sulfate at $P < 0.05$. [#]Not significant over control. All statistical tests performed using the Statistical Package for the Social Sciences, Version 16.0.

RESULTS

AS-treated rats showed the decreased levels of antioxidant enzyme levels in liver tissue [Table 1] such as SOD (9.77 ± 1.265), CAT (0.498 ± 0.016), GPx (0.98 ± 0.046), and GR (0.458 ± 0.030) when compare with control (15.48 ± 0.535, 0.835 ± 0.018, 1.79 ± 0.067, and 0.817 ± 0.053) (G1) animals. Oxidative stress marker LPO (MDA) [Figure 1] content levels were significantly increased in AS rats compared with control. AS + Zinc parallel treated rats (G4) showed increased levels all antioxidant enzymes (13.13 ± 0.705, 0.738 ± 0.017, 1.57 ± 0.064, and 0.716 ± 0.033) and restored levels of MDA when compare with AS rats (G2). Zinc (G3) supplementation animals did not show any significant variation of antioxidant and stress marker levels compared with control.

In the muscle tissue, antioxidant enzyme levels [Table 2] (SOD [5.55 ± 0.315], CAT [0.43 ± 0.028], GPx [0.88 ± 0.060], and GR [0.388 ± 0.035]) were notably decreased in AS (G2) injected animals when compared with normal animals (G2) (9.23 ± 0.531, 0.79 ± 0.016, 1.29 ± 0.059, and 0.602 ± 0.028), whereas MDA levels [Figure 1] were significantly increased. AS + Zinc treated animals (G4) showed considerable increased levels of SOD (8.22 ± 0.251), CAT (0.70 ± 0.018), GPx (1.12 ± 0.040), and GR (0.526 ± 0.016) and decreased levels of MDA when compared with AS (G2)-treated rats. Zinc-treated animals (G3) showed some alteration levels, but these are NS when compared with control (G1) animals biochemical parameters.

DISCUSSION

In the present study, the aim of this study was to investigate the zinc potential role against AS-induced oxidative stress in the liver and muscle tissues of rats. Oxidative stress is a pathological phenomenon due to excessive generation of ROS by free radicals and increasing levels of LPO status in body fluids and organs. Increasing levels of stress markers (LPO) are responsible for the declined production of antioxidant enzyme levels and diminish of antioxidant defense system.[29] In the present study observations, AS-treated rats (GII) showed that significant increased levels of LPO [Figure 1] and decreased levels of antioxidant enzymes such as SOD, CAT, GPx, and GR in the liver and muscle tissues of rats [Tables 1 and 2]. The similar findings also found with ammonium chloride, acetate, and nitrate administered rats. Kanimozhi *et al*.[8] reported that ammonium chloride treatment (100 mg/kg bw i.p) in rats showed biochemical and histological alterations in liver and brain tissue. Oxidative stress markers thiobarbituric acid reactive substances and hydroperoxides (HP) levels increased and decreased levels of antioxidants SOD, CAT, GPx, and reduced glutathione (GSH) in plasma and tissues (liver and brain) reported. Ammonium acetate treatment (AMA; 100 mg/kg daily; orally) also causes the elevated MDA content and decreased levels of CAT, SOD, GPx, GR, and reduced GSH in liver and brain tissue of rat.[30]

Table 1: Changes in the activity of levels of liver antioxidant enzymes in control and experimental animals

Group/parameter	SOD (units of superoxide anion reduced/mg protein/min)	CAT (µ moles of H_2O_2 degraded/mg protein/min)	GPx (µ moles of NADPH oxidized/mg protein/min)	GR (µ moles of NADPH oxidized/mg protein/min)
Control	15.48±0.535	0.835±0.018	1.79±0.067	0.817±0.053
AS (percent change over control)	9.77±1.265 [a](−36.88%)	0.498±0.016 [a](−40.35%)	0.98±0.046 [a](−45.25%)	0.458±0.030 [a](−43.94%)
Zc (percent change over control)	15.59±0.488 [#](0.71%)	0.845±0.015 [#](1.19%)	1.81±0.087 [#](1.11%)	0.821±0.042 [#](0.48%)
AS+Zc (percent change over AS)	13.13±0.705 [b](34.39%)	0.738±0.017 [b](48.19%)	1.57±0.064 [b](60.20%)	0.716±0.033 [b](56.33%)

All the values are mean of six individual observations. [a]Values are significantly over control at $P<0.05$, [b]Values are significantly over ammonium sulfate at $P<0.05$. [#]Not significant over control. AS: Ammonium sulfate, Zc: Zinc chloride, H_2O_2: Hydrogen peroxide

Table 2: Zinc impact on antioxidant enzyme levels of muscle tissue in control, AS, and AS+Zc treated rats

Group/parameter	SOD (units of superoxide anion reduced/mg protein/min)	CAT (µ moles of H_2O_2 degraded/mg protein/min)	GPx (µ moles of NADPH oxidized/mg protein/min)	GR (µ moles of NADPH oxidized/mg protein/min)
Control	9.23±0.531	0.79±0.016	1.29±0.059	0.602±0.028
AS (percent change over control)	5.55±0.315[a] (−66.30%)	0.43±0.028 [a](−45.56%)	0.88±0.060 [a](−31.78%)	0.388±0.035 [a](−35.54%)
Zc (percent change over control)	9.53±0.909[#] (3.25%)	0.80±0.025 [#](1.26%)	1.30±0.039 [#](0.77%)	0.622±0.056 [#](3.32%)
AS+Zc (percent change over AS)	8.22±0.251 [b](48.10%)	0.70±0.018 [b](62.79%)	1.12±0.040 [b](27.27%)	0.526±0.016 [b](35.56%)

All the values are mean of six individual observations. [a]Values are significantly over control at $P<0.05$, [b]Values are significantly over ammonium sulfate at $P<0.05$. [#]Not significant over control. AS: Ammonium sulfate, Zc: Zinc chloride, H_2O_2: Hydrogen peroxide

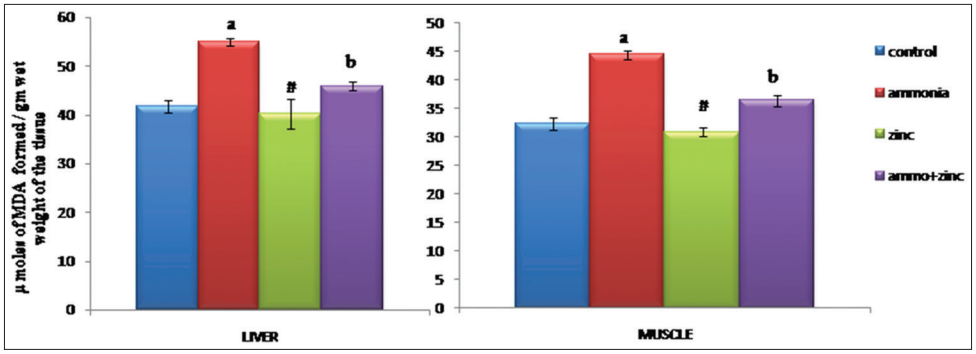

Figure 1: Effect of zinc lipid peroxidation changes in the liver and muscle tissue of rats on control and experimental rats. Each value is mean±standard deviation for six rats in each group, a – Values are significantly over control at $P<0.05$, b – Values are significantly over ammonium sulfate at $P<0.05$. #Not significant over control

Related findings found allied to ammonium nitrate (220 mg/kg b.wt orally) and AS (18.3 mg/kg b.w i.p) treatment in liver and kidney tissues of the rat.[31,32] Hence, these findings support the AS treatment leads to alteration in the antioxidant defense mechanism by the excessive generation ROS and oxidative stress, whereas AS + Zc parallel treated rats (GIV) showed that stabilized levels of LPO and increased levels of antioxidants (SOD, CAT, GPx, and GR) in the liver and muscle when compared with AS-treated animals. Group III animals (treated with Zc) showed that slightly increased levels of antioxidants and decreased content of LPO when compared with control, these slightly increased and decreased levels of antioxidant, LPO levels were not significantly over control.

Antioxidant defense system plays an important role in the response of LPO by depleting of ROS or free radicals. SOD and CAT are considered as the first line of defense antioxidants against consumption of ROS.[33] SOD catalyzes the conversion of O_2- to H_2O and H_2O_2, and the later H_2O_2 further degraded into H_2O and O_2 by CAT, GPx, GR, and many types of peroxides.[9] GPx is a major defense system against oxidative damage of essential intracellular compounds (e.g., proteins and polyunsaturated fatty acids), particularly by reducing HP to water. GR or reduced GSH is the natural antioxidant of the cell. It has a key role in the detoxification process by destroying the formed free radicals in the cells. Therefore, deficiency of the GR causes greater LPO leading to cell damage.[34] Zinc is one of the exogenous antioxidant, considered as multipurpose trace element and it is suggested for remedial supplementation of many stressful and pathological conditions.[35] Zinc deficiency has a significant impact on different aspects in the human health as well as animal health.[36] Present observations showed that the SOD, CAT, GPx, and GR levels were significantly increased with zinc supplementation under the AS stress in the liver and muscle. Increased levels of selected antioxidant in the liver and muscle tissue denote that zinc treatment chelating the AS generated ROS and prohibits the oxidative stress as the role of antioxidant nutrition. Previous research reports also have suggested on zinc as having antioxidant role of toxic chemicals. Zinc supplementation protects heavy metals (cadmium, aluminum, nickel and arsenic, etc.) induced oxidative stress in liver, kidney, and brain.[19-22] Zinc supplementation enhances the endogenous antioxidant enzyme levels; these enhanced elevated enzymes work together to eliminate ROS and alleviate the harmful substances in the liver and muscle of rats.[37] Hence, in the present investigation, it is clearly indicated that zinc supplementation successfully inhibits the AS toxicity by diminished levels of LPO and restores the levels of antioxidants in the parallel administration animals (AS + Zc) when compared with AS-treated animals. Zinc supplementation may be reduced the hepatic and muscle stress by enhancement of antioxidants and prevent of LPO under the AS toxicity. The limitation of the present study is inability to estimate the liver marker enzymes, components in carbohydrate metabolism in the liver and muscle tissue to identify the exact constituents. Further, planning to identify the AS toxicity causes any histological changes in the liver and muscle; zinc might be protecting the liver and muscle histomorphological changes.

CONCLUSION

AS toxicity causes the oxidative stress in liver and muscle tissues of rat evidenced by reduced levels of SOD, CAT, GPx, and GR antioxidant enzymes and increased levels of stress marker levels (MDA), so ammonia toxicity leads to antioxidant functional disturbance. Zinc successfully diminishes the ammonia oxidative stress and significantly increased the defense enzyme levels by reduction of stress markers in both the tissues of albino rat, so that zinc might be used as a remedial therapeutic drug for ammonium exposed chemicals or liver dysfunctional patients.

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