Camel's Milk Protects Against Aluminum Chloride-Induced Normocytic Normocromic Anemia, Lipid Peroxidation and Oxidative Stress in Erythrocytes of White Albino Rats

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Abstract: Problem statement: Aluminum (Al) is an indifferent element from a toxicological point of view. In recent years, however, Al has been implicated in the pathogenesis of several clinical disorders. One of the most frequently described problem in aluminum toxicity is anemia. The present study was carried out to determine the effectiveness of Camel's milk in alleviating the toxicity of aluminum chloride ($AlCl_3$) on certain hematological parameters, lipid peroxidation and oxidative stress enzyme in the RBC's of white albino rats. **Approach:** Ten rats per group were divided into three treatment groups: Group one were rats given normal saline and served as control group, group two were rats treated with 1 ml of $AlCl_3$ (0.5 mg kg^{-1} body weight) and named $AlCl_3$ treated rats, group 3 were rat treated with 1ml fresh camel's milk 10 min before the administration of $AlCl_3$ (0.5 mg kg^{-1} body weight) and named Camel's milk and $AlCl_3$ treated rats. Rats were orally administered their respective doses every day for 30 days. Evaluations were made for hematological parameters in the blood and for lipid peroxidation and oxidative stress enzymes activities in the RBC's. **Results:** Results obtained showed that oral $AlCl_3$ treatment caused a significant decrease ($p<0.05$) in total erythrocytes count, blood Hemoglobin (Hb), hematocrite (PCV) and Serum iron levels, where as the values of Mean Corpuscular Volume (MCV), Mean Hemoglobin Concentration (MHC), Mean Corpuscular Hemoglobin Concentration (MCHC) and Total Ion Binding Capacity (TIBC) didn't change. Also oral administration of $AlCl_3$ induced free radicals and as a result caused an increase the concentration of Thiobarbituric Acid Reactive Substances (TBARS) and decreased activities of Superoxide Dismutase (SOD) and Catalsae (CAT) in the RBCs homolysate. The oral administration of Camel's milk before the administration of $AlCl_3$, alleviated it's toxic effect. Camel's milk administration resulted in a significant increase ($p<0.05$) in the in total erythrocytes count, blood hemoglobin (Hb), hematocrite (PCV) and Serum iron with No change in the values of MCV, MHC, MCHC and TIBC when compared to $AlCl_3$ treated rats. Camel's milk reduced free radicals production and oxidative stress status in the RBC's noticed by the significant decreased levels of TBARS and increased activities of SOD and CAT when compared to $AlCl_3$ treated rats. **Conclusion:** our data proved that there is an alternation in the hematological parameters and antioxidant system in the red blood cells of rats administered aluminum chloride orally, whereas oral administration of Camel's milk prior the administration of Aluminum chloride protects the red blood cell form toxic effect of aluminum.

Key words: Aluminum chloride, red blood cells, erythrocytes, oxidative stress, camel's milk

INTRODUCTION

Aluminum (Al), the third most abundant element of the Earth's crust, is a nonessential and toxic metal in humans[1]. Due to its abundance, every organism contains small quantities of aluminum and it can be found in practically all of the tissues of mammals, including the brain, liver, kidney, heart, blood and bones[2].

Particular sources of Aluminum include corn, yellow cheese, salt, herbs, spices, tea, cosmetics and Aluminum cooking utensils and containers[3]. Also,

Aluminum is widely used in food additives and toothpaste[4]. Aluminum compounds are widely used in medicine e.g., antacids, phosphate binders, buffered aspirins, vaccines and allergen injections[5-7]. In addition, aluminum is added to drinking water for purification purposes[8].

Aluminum has the potential to be toxic for humans and animals The human toxicological effects include encephalopathy[9], bone disease[10], Skeletal system disease[11], Alzheimer's disease[12]. And blood problems[13].

The most frequently described blood problem in aluminum toxicity is anemia. It is important to note, that this disorder was observed in patients after dialyses when aluminum ions were present in dialysis fluid[14]; also in the case of laboratory animals that were administered solutions of aluminum salts orally, intraperitonally, or intravenously[13,15,16].

Aluminum has a direct effect on hematopoiesis[17]. Excess aluminum has been shown to induce anemia[13,15,16]. Daily injections of aluminum into rats produced severe anemia within 2-3 weeks[16]. Previous investigators proposed that Aluminum may cause anemia through decreased heme synthesis[18,19], decreased globulin synthesis and increased hemolysis[20,21]. Patients with anemia from aluminum toxicity often have increased reticulocyte counts, decreased hemoglobin concentration, decreased hematocrite value, decreased mean corpuscular volume and decreased mean corpuscular hemoglobin[16].

These toxic effects of aluminum on RBCS have been suggested to be due to the generation of reactive oxygen species and induction of oxidative stress[22], which results in the oxidative deterioration of cellular lipids (through lipid peroxidation), proteins and DNA[22,23]. So, these toxic effects of aluminum appear to be mediated, at least in part, by free-radical generation[24,25]. Recent research shows that aluminum may induce changes in the activity of a number of antioxidative enzymes (xanthine oxidase, glutathione peroxidase, superoxide dismutase)[24].

Several studies have implicated lipid peroxidation as one of the molecular mechanisms underlying aluminum toxicity *in vitro* and *in vivo*[26-30]. A recent report suggests that aluminum can induce morphological and functional alterations in erythroid cells by a direct action on circulating erythrocytes[31], suggesting membrane alterations due to lipid peroxidation mechanisms. In line with this, aluminum decreases erythrocytic membrane fluidity[32]. However, a casual link between hematological changes and lipid peroxidation after exposure to aluminum is lacking in the literature.

Also, Aluminum may have a direct effect on iron metabolism; it influences absorption of iron via the intestine, it hinders iron's transport in the serum and it displaces iron's binding to transferring[33,34]. The major forms of aluminum in blood circulation has been reported to be in complex with transferring using immuno-affinity chromatography and spectrophotometric titration techniques[35]. Transferrin is known primarily for its role in the transport and cellular uptake of iron but is also the major serum binding protein for aluminum[36,37]. previous studies showed that aluminum bound to serum transferring and the complex of aluminum-transferrin interacts with the same receptors as iron-transferrin[38], this receptor-mediated cellular uptake appears to be an important factor in the uptake of aluminum by the tissues[39].

Several studies suggest that chronic exposure to relatively high doses of aluminum can change iron metabolism in different animal species[15,40,41]. However, the findings are not always in the same direction. In fact, some studies have indicated a reduction in iron in serum of rats exposed to high levels of aluminum[40,41], whereas other investigators have found no alterations or an increase in iron stores after exposure to aluminum[10,42].

Consequently, data concerning the mechanism of Aluminum toxicity on hematological system and iron metabolism after administration of aluminum are contradictory and seem to depend on the conditions of toxicity, which refer mainly to different doses and different routes of administration.

Most of experimental studies on aluminum toxicity in an animal model have been preformed with the use of this metal in a soluble form[43] or with certain metal[44]. However, in has been reported that in most of studies done to demonstrate the toxic effect of aluminum chloride in animals, the chosen routes of Al administration (i.e., intraperitoneal, intravenous and parenteral) do not simulate the main route by which human population is exposed to Aluminum. There are two main routes by which aluminum enters the body: Pulmonary and oral. The bulk of inhaled contaminants in the air are aluminosilicates. Although only a small portion of aluminum is absorbed by the gastrointestinal tract, oral intake represents the route with greatest toxicological implications[45]. For this reason our current study was carried out by oral administration of Aluminum chloride.

It was reported that the components of camel's milk is different from any other milk and play a major role in its therapeutic action. Camels milk is rich in minerals such as potassium, zinc and magnesium and has high concentrations of vitamin B2, C and E[45].

In India, camel's milk is used therapeutically against dropsy, jaundice, problems of the spleen, tuberculosis, asthma, anemia, piles and diabetes[47].

The conflicting data related to the basis of the toxic effects of orally low dose of aluminum chloride administration on erythropoiesis and on iron metabolism do not allow definitive conclusions. Also, in our survey, we didn't find any study dealt with the therapeutic effect of Camel's milk against the bad effect of Aluminum chloride on blood of rats. Therefore the aim of our study was to investigate a possible protective

influence of Camel's milk treatment on some hematological parameters, Iron metabolism, lipid peroxidation and antioxidant defense system in the blood of rats treated with Aluminum. The following parameters were determined in the blood to confirm anemia: Red Blood Cells count (RBCs), Hematocrite value (Hct), Hemoglobin (Hb), blood indices, iron concentration, Total Iron Binding Capacity (TIBC). TBARS in the RBC's was determined as an indicator of lipid peroxidation status. Also, the following antioxidant enzymes activities were determined in the RBC's: Superoxide Dismutase (SOD) and Catalase (CAT).

MATERIALS AND METHODS

Preparing of Camel's milk Samples: Milk samples were supplied every day early morning by farmers from Abha region (Southeastern province of Saudi Arabia), Milk was collected into sterile bottles and then transported in cool boxes to our laboratory. Farmers collected the milk by hand milking.

Chemicals: Aluminum chloride chloride [$AlCl_3 \times 6 H_2O$] was purchased from Aldrich chemical Company (USA).

Experimental design: Rats weighing between 230-250 g were supplied from the animal house at College of Medicine at King Khalid University. The rats were divided into 10 rats/cage, the rats were housed in plastic cages at a temperature regulated (22°C) and humidity (55%) controlled room with a 12 h light/12 h dark cycle. A water and standard pellet diet were available *ad libitum* throughout the experimental period.

For determination of daily orally aluminum chloride dose we followed the guidelines of drug institute in which they recommend that the drug applied to rodents must be 4 to 10 folds of daily human intake[48]. In reference to FAO/WHO expert committee on food additives, they reported that the daily intake of aluminum in adult human is 6-14 mg/70 kg[49-51], which gives a daily intake of aluminum ranges between 0.0857-0.2 mg kg^{-1}. In our experiment the daily dose of aluminum chloride given to rats was 0.5 mg kg^{-1} which exceeded 6 folds of minimal dose and 2.8 fold of maximal dose on average of 4 fold the daily aluminum intake in human.

Rats were divided into three group each of ten rats, all treatments were carried out for 30 days and they were given orally by using special cavage needle. Rats received a single dose of the selected treatment every day and were treated as follows:

Group 1: were control rats received a single daily dose of normal saline orally.

Group 2: were rats given a daily single dose of aluminum chloride at a final concentration of 0.5 mg kg^{-1} and name ALCL$_3$-treated rats.

Group 3: were rats given a single dose of 1 mL Camel's milk 10 min before the oral administration of Aluminum chloride with same concentration used in group two and named Camel's milk-ALCL$_3$ treated group.

Physiological and biochemical analyses: After the treatment (24 h after the last administration), the animals were sacrificed by decapitation always between 8:00 and 10:00 am and fresh blood was immediately collected into heparinized test tubes for routine hematological analysis. A second blood fraction was collected without anticoagulant and centrifuged at 5000 rpm for 10 min and used for determination of iron content and TIBC in serum. Some heparinized Blood were used for preparation of homolysate for determination of TBARS, SOD and CAT activities. The following hematological parameters were determined in the plasma: Erythrocytes were counted on hemocytometer using a light microscope at 40×10 magnification. Blood samples were diluted to 200 times by physiological saline (0.9% sodium chloride solution) before counting. Hematocrite value were determined by the method of Strumia *et al.*[52]. The hemoglobin concentration was determined by the cyanmethemoglobin method[53]. Mean Corpuscular Volume (MCV), Mean Hemoglobin Concentration (MHC) and Mean Corpuscular Hemoglobin Concentration (MCHC) were calculated in accordance with the models given by Wintrobe[54]. Iron and TIBC in serum were determined using cell using Commercial available kits (human), according to manufacture's instruction.

Preparation of hemolysate: After collecting blood samples in heparinized tubes, centrifugation was performed at 1000 g for 15 min to remove the buffy coat. The packed cells obtained at the bottom were washed thrice with phosphate buffer saline (0.9% NaCl

in 0.01 M phosphate buffer, pH 7.4). A known amount of erythrocytes was lysed with hypotonic phosphate buffer. The hemolysate was obtained after removing the cell debris by centrifugation at 3000 g for 15 min and used for determination of reduced glutathione levels.

Measurement of TBARS levels SDO and CAT activity: Superoxide dismutase activity in the red blood cells homolysate was measured by using commercial kits (Biovision, K335-100). The activity was expressed as U mL^{-1}.

Catalase Activity (CAT) in the red blood cells homolysate was determined by using commercial kit (biovision K773-100. Cat activity was expressed as U/ml. One unit of catalase is the amount of catalase decomposes 1.0 μmol of H_2O_2 per min at pH 4.5 at 25°C.)

The concentration of Thiobarbituric Acid Reactive Substances (TBARS) in the RBCs homolysate was determined by the method of Okhawa[55]. In brief, the reaction mixture contained 0.1 mL of homolysate, 0.2 mL of sodium dodecyl sulfate, 1.5 mL of acetic acid and 1.5 mL of aqueous solution of thiobarbituric acid. The pH of 20% acetic acid was pre-adjusted with 1 M NaOH to 3.5. The mixture was made up to 4 mL with distilled water and heated at 95°C for 1 h, in a water bath. After cooling, 1 mL of distilled water and 5 mL of mixture of n-butanol and pyridine (15:1) were added and mixture was shaken vigorously on a vortex mixer. The absorbance of the upper organic layer was read at 532 nm. The values were expressed as mM mL^{-1}.

Statistical analysis: Data are expressed as mean ± SD. Student's t-test was used to determine the difference between groups. Statistical significance was considered at p<0.05.

RESULTS

Table 1 shows the results of total RBC's count, hemoglobin concentration (Hb), Hematocrite value (PCV) and red blood cell indices (MCV, MCH and MCHC) in all groups of rat. Rats administered Aluminum chloride orally showed decreased values of erythrocyte counts, hemoglobin and hematocrite, but the levels of MCV, MCH and MCHC didn't change when compared to rats received normal saline orally. On other hand oral administration of Camel's milk before the administration of aluminum chloride to rats significantly increased these parameters to their normal levels when compared to rats administered cadmium only.

Table 1: Red Blood Cells (RBC's) count, Hematocrite (Hct) value and Hemoglobin (Hb) concentration in the blood of control rats, aluminum treated-rats and Camel's milk and aluminum treated rats

Parameters	Control	AlCl$_3$	AlCl$_3$+milk
RBC's (X10^6 mm^3)	7.02±0.089	4.89±0.284*	7.25±0.093*
Hematocrite (%)	57.76±0.856	37.68±0.633*	58.40±0.432*
Hemoglobin (g dL^{-1})	16.73±0.335	9.78±0.509*	16.78±0.944*
MCV (fl)	82.20±0.85	77.10±0.56	80.46±0.63
MCH (pg)	23.83±0.94	20.01±1.1	23.12±0.98
MHCH (g dL^{-1})	28.97±1.1	25.97±0.88	28.97±1.3

Values are given as mean ± SD for groups of six animals each. Values are statistically significant at *p<0.05. Cadmium treated rats were compared with control rats; Camel's milk cadmium treated rats were compared with Cadmium treated rats

Table 2: Iron and TIBC levels in the serum of control rats, Aluminum treated-rats and camel's milk and cadmium treated rats

Parameters	Control	AlCl$_3$	AlCl$_3$ + milk
Iron	291.6±5.41	202.1±4.44*	287.00±2.87*
TIBC	607.0±8.78	602.0±5.94	605± 6.18

Values are given as mean ± SD for groups of six animals each. Values are statistically significant at *p<0.05. Cadmium treated rats were compared with control rats; Camel's milk Cadmium treated rats were compared with Cadmium treated rats. Fe concentration and TIBC are expressed as μg dL^{-1}

Table 3: TBARS levels and SDO and CAT activities in the RBC's of control rats, Aluminum treated-rats and Camel's milk and aluminum treated rats

Parameters	Control	AlCl$_3$	AlCl$_3$+milk
SOD (U mL^{-1})	8.37±0.25	2.22±0.16*	8.110±0.28*
CAT (U mL^{-1})	94.40±2.3	57.0±3.7*	95.10±4.05*
TBARS (mM mL^{-1})	28.20±2.10	31.3± 1.01*	27.36±065*

Values are given as mean ± SD for groups of six animals each. Values are statistically significant at *p<0.05. Cadmium treated rats were compared with control rats; Camel's milk Cadmium treated rats were compared with Cadmium treated rats

The levels of serum Iron and TIBC are presented in Table 2. There was a significant decrease in the levels of iron in the rats treated with aluminum chloride. Administration of camel's milk into rats before the administration of aluminum chloride caused a significant increase in the levels of serum iron. TIBC levels didn't change in all treated group.

Table 3 shows the activity of Superoxide Dismutase (SOD) and Catalase (CAT) as well as Thiobarbituric Acid Reactive Substances (TBARS) concentration in RBC's of all groups of rats. Aluminum chloride treated-rats showed a significant decrease in the activity of these enzymes (SOD and CAT) and a significant increase in the level of TBARS. Rats treated with Camel's milk and aluminum chloride showed a significant increase in the activity of SOD and CAT as well as a significant decrease in the levels TBARS in the RBC's of the treated rats when compared to cadmium treated rats.

DISCUSSION

The present results show hematological modifications associated with orally administered aluminum chloride in rats and show the reversible effect of Camel's milk on these modifications.

Several lines of evidence have confirmed the fact that aluminum can induce anemia[13,15,16,31]. It is essential to evaluate the level of anemia by the following measurements: RBC's count hemoglobin concentration, hematocrite and red blood cell indices (MCV, MCH and MCHC).

In aluminum chloride oral administered rats, a reduction in erythrocyte count, haemoglobin levels and hematocrit values were observed which is in agreement with Vittori *et al.*[27] and other authors[15,16,18,56] who found similar effect following aluminum administration with different routs. Values of Mean Corpuscular Volume (MCV), Mean Corpuscular Hemoglobin (MCH) and Mean Corpuscular Hemoglobin Concentration (MCHC) were not affected by Aluminum chloride indicating that, in our experimental rats, the rats developed normocytic and normocromic anemia.

The reduction in RBC's, hematocrit value indicate to possible hemolytic effect of aluminum chloride. Hemolytic activity of this element is due to changes in cell membrane of red blood cells. It has been reported that in the presence of Aluminum ions, human erythrocytes lost their typical biconcave shape, turning into acanthocytes and stomatocytes[27]. Aluminum, like other xenobiotics, generates free radicals and reactive oxygen species in cells, which cause fatty acid superoxides and oxidation of cellular membrane proteins, which leads to reduction of membrane fluidity and in consequence to damage of cellular membrane[57]. Also, It has been reported that aluminum intoxication lead to a decrease in activity of membrane ATPases and in effect to cellular accumulation of adenylates and to reduce rate of ATP: ADP converting. This lead to diminish of energy essential to maintenance of membrane integrity and to red blood cells dysfunction and so haemolysis[58,59].

The reduced level of hemoglobin can be associated with hemolysis or disturbances in heme biosynthesis as a result of inhibit linking of iron with heme and drop in activity of enzymes taking part in heme biosynthesis, mainly dehydratase of delta-aminolevulonic acid (ALA-D)[5,32,59,60], also, Ganchev *et al.*[61] reported that aluminum treatment in rat hinderes hemoglobin synthesis and erythroid cell maturation.

Aluminum can interact with iron metabolism[5,15,41,42,62] and iron depletion may occur after aluminum intoxication[63,64]. There are contradictory data in literature about transferrin saturation after Al exposure. Some studies have reported that Aluminum intoxication decreases the percentage of transferrin saturation (15). Furthermore, iron seems to influence the transferrin binding of Al by lowering the affinity of transferrin for Aluminum[65]. Conversely, Vittori *et al.*[31] did not observe changes in plasma iron concentration, TIBC or transferrin saturation in rats chronically exposed to Al, although anemia signs were observed in these animals. In our study, the oral exposure to Aluminum chloride decreased serum iron but TIBC (the amount of Fe^{3+} needed to fully saturate plasma transferrin) did not change after aluminum treatment. It would be speculated that Aluminum is present at transferring sites in iron-deficient (Al-exposed) rats.

Previous research has indicated that oxidative stress has an important role in aluminum toxicity[26-30]. In agreement with this is the hypothesis by Joshi[66] that aluminum interferes with iron ions, particularly with trivalent iron ions which participate in oxidoreduction. This results in increased production of free radicals. Kong *et al.*[67] proposed that aluminum builds a complex with O_2.

In this study, oral administration of aluminum chloride produced a significant increase in TBARS in the RBC's suggesting increased lipid peroxidation as a result of generation of free radicals, TBARS has been associated with reduction of membrane fluidity[68] The presence of such changes suggests that the alterations on erythrocyte parameters in this group are due to an enhanced intravascular hemolysis as a result of oxidative stress and lipid peroxidation in the circulating erythrocytes. Our result are in agreement with Vittori *et al.*[15] who reported that aluminum exposure can induce alterations of erythroid cells by a direct action on circulating erythrocytes suggesting membrane alterations due to lipid peroxidation. Our result is the first one that shows an increase in the levels of TBARS in Aluminum chloride intoxicated rats.

Maintenance of normal cellular functions in the presence of oxygen largely depends on the efficiency of the defense mechanisms against free-radical mediated oxidative stress. To prevent biological macromolecules from oxidative damage, antioxidant enzymes are considered to be the first line of cellular defense. The most common antioxidant enzymes in the biological systems are SOD and CAT. Our results indicate that

erythrocyte catalase activity is significantly lower in the aluminum-exposed group. Mena *et al.*[69] had a similar finding of reduced catalase activity in subjects with increased amounts of free radicals during an intensive physical strain. This inhibition in CAT activity could be a result of the oxidation of catalase sulfhydryl groups[70] or due to decrease synthesis.

Previous reports on SOD activity and aluminum exposure are controversial. Shainkin Kestenbaum *et al.*[71] and Hasanoglu *et al.*[72] reported significantly decreased SOD activity in erythrocytes of haemodialysed patients, but Abd-Elghaffar *et al.*[73] reported decreased brain SOD activity in rabbits exposed to aluminum chloride. In contrast, an other *in vitro* study[74] reports unchanged SOD activity. Our study show a significant decrease in erythrocyte SOD activity in rats exposed to aluminum chloride.

Camel's milk oral administration 10 min before the administration of aluminum chloride in resulted in a reverse action of aluminum chloride, it maintain all the above altered hematological parameters within their normal levels. Camel's milk protect the rats from anemia induced by aluminum chloride; in rats treated with 1 ml camel's milk with aluminum chloride orally, a significant increase in the RBC's count, hemoglobin, hematocrite and iron serum levels were shown when compared to rats administered aluminum chloride only. Also, oral administration of camel's milk resulted in a significant decrease in the levels of TBARS and an increase in the enzymatic components of oxidative system: SOD and CAT in the RBC's, when compared to rats received aluminum chloride only.

According to our result, it could be concluded a protective effect of Camel's milk against aluminum chloride induced toxicity in the red blood cells of rats. Camel's milk reduced the risk of anemia and oxidative stress in the erythrocytes of rats. Such effect could be due to a possible chelating effect of aluminum chloride but further studies are required.

Camel's milk Contain high levels of Vitamins including Vitamin C and E which considered potent antioxidants that help in preventing tissue damage caused by production of free radicals. These vitamin may play a major role in the protective effect of Camel's milk against aluminum chloride induced oxidative stress[46]. Also Knoss[46] have reported that camel's milk is very rich in trace elements such as Magnesium (Mg) and zinc (Zn). Mg was found to protect cells form the toxic effect of heavy metals such as aluminum and cadmium. It was reported that there is an increase production of reactive oxygen species in patients with magnesium deficiency[75]. Also, Mg play a major role in the absorption and metabolism of Vitamin

C and E[76]. Thus helping our bodies in cell protection against free radicals.

Also, it has been reported that Vitamin E enhances the levels of reduced glutathione[76]. Recent study showed that magnesium is essential for synthesis of glutathione[77] and this may gives an idea about increased glutathione levels and in rats treated with camels milk.

Zn is an essential element required for many enzymes in our biological system. Zinc is very important in DNA replication, protein synthesis and cell division[77]. Recent studies showed a relationship between antioxidant enzymes and zinc levels[78-81], One study has shown that Zn deficiency in the diet paves the way for cell damage in the rat testis[82]. It has been reported that zinc deficiency resulted in increased lipid peroxidation in various rats tissues[81-84].

Finally, we made a similar study to investigate the role of camel's milk in preventing the oxidative stress produced by oral administration of cadmium chloride and we found similar finding to our current study[85].

CONCLUSION

Our results showed that oral administration of aluminum chloride into rats caused a marked alternation in hematological parameters such as decreased total erythrocytes count, decrease hemoglobin and hematocrite values and decreased serum iron levels. Also aluminum chloride led to increase lipid peroxidation and oxidative stress in the rats. Whereas the oral administration of Camel's milk prior the administration of aluminum chloride protect ed the erythrocytes from aluminum chloride induced oxidative damage and lipid peroxidation and maintained the mentioned hematological parameters within their normal levels.

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