Impact of Camel’s Milk on Aluminum Chloride (AlCl₃) Induced Toxicity in Rats

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The present study was carried out to evaluate the efficiency of camel’s milk to ameliorate the toxicity of aluminum chloride AlCl₃ on some hematological parameters; hepatic and renal functions and lipids profile; as well as histopathological alterations of some organs. Forty rats (8 / group) were divided into 5 treatment groups: Group 1: Normal rats (negative control); Group 2: AlCl₃ induced toxicity rats (positive control); Group 3: AlCl₃ induced toxicity rats fed with raw camel milk; Group 4: AlCl₃ induced toxicity rats fed with heat treated camel milk; and Group 5: AlCl₃ induced toxicity rats fed with sweet acidophilus camel milk. Rats were treated by 5ml camel’s milk 10 min before the administration of 1 ml AlCl₃ (0.5 mg / kg body weight); and had their respective doses daily for 30 successive days orally. AlCl₃ oral administration resulted in a significant decrease in red blood cells count (RBC’s), significant increase in mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH); while hemoglobin (Hb), hematocrite (Hct), platelets(plt), reticulocytes (Ret), mean corpuscular hemoglobin concentration (MCHC) did not reveal significant changes; the obtained anemia was macrocytic normochromic. The lipids profile; hepatic and renal functions showed non significant changes between different groups; however, histopathological examination showed variable alterations of varying severity in some organs; besides their response to camel’s milk administration. Camel’s milk administration in groups 3, 4, 5 alleviated the toxic effect of AlCl₃ with variable degrees between different groups.

Keywords: Aluminum chloride, Camel’s milk, Kidney and liver functions, Lipids profile, Macrocytic normochromic anemia, Red blood cells.

Aluminum (Al), the third most abundant element of the Earth’s crust, is found in combination with oxygen, silicon, fluorine and other elements in the soil, rocks, clays and gems; nonessential and toxic metal in humans¹. With the industrialization and consequent pollution, Al is increasingly taken into our bodies through foods, air, water, and even drugs². Food is the primary common source of Al; include yellow cheese, salt, herbs, spices, tea leaves, food additives³. The use of Al and its

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compounds in processing (cooking utensils and containers); wrappings, packaging (Al foil); storage of food products (almost 95% beverage cans); cosmetics; and toothpaste may contribute to its presence. Al compounds are widely used in medicine e.g., antacids, phosphate binders, buffered aspirins, vaccines and allergen injections. Al has been recognized to be toxic for humans and animals, and is involved in the etiology of some diseases. Chronic exposure to Al ions may result in mood changes, dysmnesia, convulsions, muscular weakness, pathological fractures of bones. Al accumulates mainly in bones, spleen, liver and lungs. Daily injections of Al into rats produced severe anemia within 2-3 weeks. Al has a direct effect on hematopoiesis. Al-induced damage to body organs has been reported in several studies which pointed out the toxic effect of AlCl₃, such as hepatotoxicity, nephrotoxicity, and neurotoxicity. AlCl₃ administration lead to increased activity of liver enzymes in the serum; accumulating of Al in the kidneys destroys renal tubular cells, causing renal toxicity; and Al-induced LPO “peroxidation of lipid” could arise from alteration of lipid metabolism.

Fresh camel milk and its products are a good bioactive adjuvant for the people living in the arid and semiarid areas. Awareness and utilization of camel milk as health adjuvant are gradually increasing as the camel milk has been found to have unique properties of its proteins, fatty acids, richer in microminerals and vitamin C compared to milks of other animal species, such as bovine milk. Fresh and fermented camel milk are reported for improving immunity and provides particular health benefits to the consumer depending on the unique bioactive substances in milk. It has high concentration of iron which makes it panacea for those who have iron deficiency anemia. Camel milk is unique from other ruminant milk in terms of composition as well as functionality. Moreover, it is also used for its potential therapeutic properties such as efficacy against diabetes and cancer as well as having anti-hypertensive properties.

This study aims to evaluate the effectiveness of camel’s milk towards the toxicity of aluminum chloride AlCl₃ on some hematologic parameters; hepatic, renal functions; lipids profile; and histopathology of some organs.

MATERIALS AND METHODS

Materials
- Aluminium Chloride anhydrous 98% ; M.W. 133.34 (Alpha Chemika India)
- Dromedary camel milk fresh and frozen (Animal Production Research Institute, Agricultural Research Center, Dokki-Giza).
- AST/ GOT- ALT/ GPT, total and direct bilirubin, total cholesterol, triglycerides, HDL- cholesterol, urea, creatinine, and haemoglobin kits of Biolabo, France.
- Lactobacillus acidophilus DSM 20079 (Egypt Microbial Culture Collection, Microbiological Resource Centre [Cairo Mircen] Faculty of Agriculture, Ain Shams University, Shobra Khayma, Cairo, Egypt).

Animals And Experimental Diets
Forty male Sprague – dawely albino rats were obtained from Animal House at Food Technology Research Institute, Agricultural Research Center, Giza, Egypt. Rats weighing (200-250 g) were housed in plastic cages under standard condition temperature (25-27°C), humidity 30-70% and 12 hour light / dark cycles) and fed with standard pellet diet and water ad libitum. All rats were fed on basal diet for one week before starting the experiment (acclimatization period). The basal diet consisted of corn starch (60%), casein (20%), corn oil (10%), cellulose (5%), salt mixture (4%), and vitamin mixture (1%)17.

Animal Grouping And Experimental Design:
The rats were divided into five groups comprising 8 rats in each group as follows:
Group 1: Negative control rats (normal, fed with basal diet only).
Group 2: Positive control rats (AlCl₃ induced toxicity).
Group 3: AlCl₃ induced toxicity rats fed with raw camel milk.
Group 4: AlCl₃ induced toxicity rats fed with heat treated camel milk (72°C for 15 sec. and cooling).
Group 5: AlCl₃ induced toxicity rats fed with sweet acidophilus camel milk (10% of starter culture added to heat treated camel milk).

Daily AlCl₃ oral dose given to rats was 1 ml AlCl₃ (0.5 mg / kg body weight)18. Rats were treated by 5ml camel’s milk 10 min before
the administration of 1 ml AlCl₃. Rats received a single dose of the selected treatment daily; and had their respective doses for 30 successive days orally by using a cavage needle. Twenty Four hours after the last administration fresh blood samples were collected from orbital plexus venous into heparinized test tubes for hematological analysis. A second blood fraction was collected without anticoagulant into centrifuge tubes and the serum was separated into eppendorf tubes and stored at -20°C for analysis. Then the animals were sacrificed and some organs were collected for histopathological examination.

All the experiment was approved by the ethical committee of Cairo University in accordance with the guidelines of the National Institute of Health (NIH) for the care and use of laboratory animals in scientific investigations.

**Haematological Studies**

The evaluated hematological parameters in this study included estimation of red blood cells (RBC’s), hemoglobin concentration (Hb), hematocrite (Hct), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), reticulocytes counts (Retics), and blood platelets (plt). These parameters were performed according to the adopted routine hematological procedures ¹⁹.

**Serum Biochemical Studies**

Serum sample was examined for the enzymatic activities of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and the concentration of serum bilirubin, blood urea, creatinine, and lipid profile (cholesterol, triglycerides and HDL cholesterol). These parameters were determined by spectrophotometric method using commercial diagnostic kits supplied by Biolabo, France and followed the manufacturer’s instructions.

**Histopathological studies**

For histopathological sections, the liver, kidney were carefully dissected and collected as whole organ specimens for all experimental
### Table 1. Results of erythrogram, blood platelets and reticulocyte count in control and treated Rats

<table>
<thead>
<tr>
<th>Biochemical test</th>
<th>G1</th>
<th>G2</th>
<th>G3</th>
<th>G4</th>
<th>G5</th>
<th>Prop.</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>17.05±0.389</td>
<td>17.31±0.611</td>
<td>15.55±0.455</td>
<td>15.33±0.374</td>
<td>15.53±0.348</td>
<td>0.0292S at p ≤ 0.05</td>
<td>1.470</td>
</tr>
<tr>
<td>RBC's (X10^6 mm³)</td>
<td>8.33±0.327</td>
<td>5.76±0.152</td>
<td>7.52±0.455</td>
<td>6.04±0.262</td>
<td>7.06±0.678</td>
<td>0.0104S at p ≤ 0.05</td>
<td>1.410</td>
</tr>
<tr>
<td>Hematocrite (%)</td>
<td>39.13±0.848</td>
<td>39.90±1.408</td>
<td>35.88±1.050</td>
<td>35.33±0.974</td>
<td>36.70±0.734</td>
<td>0.0424S at p ≤ 0.05</td>
<td>3.351</td>
</tr>
<tr>
<td>MCV (fl)</td>
<td>47.20±2.077</td>
<td>69.34±2.152</td>
<td>48.40±3.884</td>
<td>52.17±6.018</td>
<td>53.16±4.115</td>
<td>0.0064S at p ≤ 0.01</td>
<td>11.10</td>
</tr>
<tr>
<td>MCH (pg)</td>
<td>20.57±0.917</td>
<td>30.08±0.882</td>
<td>20.98±1.699</td>
<td>25.58±1.474</td>
<td>22.46±1.595</td>
<td>0.0031S at p ≤ 0.01</td>
<td>4.493</td>
</tr>
<tr>
<td>MCHC g/dL</td>
<td>43.57±0.055</td>
<td>43.38±0.126</td>
<td>43.34±0.193</td>
<td>43.41±0.252</td>
<td>42.32±0.436</td>
<td>0.0250S at p ≤ 0.05</td>
<td>0.767</td>
</tr>
<tr>
<td>Platelets(x10^9/L)</td>
<td>409.75±22.95</td>
<td>292.25±19.598</td>
<td>384.50±58.506</td>
<td>399.00±29.637</td>
<td>318.00±56.006</td>
<td>0.2587ns</td>
<td>130.9</td>
</tr>
<tr>
<td>Reticulocyte count</td>
<td>3.00±0.220</td>
<td>2.90±0.058</td>
<td>3.20±0.071</td>
<td>3.13±0.155</td>
<td>3.08±0.103</td>
<td>0.4054ns</td>
<td>0.341</td>
</tr>
</tbody>
</table>

### Table 2. Results of kidney and liver functions in control and treated rats

<table>
<thead>
<tr>
<th>Biochemical test</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>Prop.</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea mg/dl</td>
<td>68.15±6.400</td>
<td>71.15±6.313</td>
<td>66.05±3.650</td>
<td>67.28±5.726</td>
<td>72.30±6.261</td>
<td>0.2762ns</td>
<td>6.75</td>
</tr>
<tr>
<td>Creatinine mg/dl</td>
<td>1.73±0.333</td>
<td>1.80±0.252</td>
<td>1.73±0.189</td>
<td>1.74±0.314</td>
<td>1.65±0.328</td>
<td>ns</td>
<td>0.41</td>
</tr>
<tr>
<td>AST/GOT U/ml</td>
<td>142.92±14.282</td>
<td>143.15±9.971</td>
<td>127.15±13.779</td>
<td>128.28±19.305</td>
<td>69.33±0.394</td>
<td>0.0171S</td>
<td>43.68</td>
</tr>
<tr>
<td>ALT/GPT U/ml</td>
<td>52.32±9.021</td>
<td>50.09±3.499</td>
<td>34.95±7.804</td>
<td>45.48±7.212</td>
<td>34.74±4.974</td>
<td>0.3362ns</td>
<td>22.70</td>
</tr>
<tr>
<td>Total bilirubin mg/dl</td>
<td>0.20±0.033</td>
<td>0.20±0.018</td>
<td>0.18±0.015</td>
<td>0.18±0.026</td>
<td>0.18±0.034</td>
<td>ns</td>
<td>0.05</td>
</tr>
</tbody>
</table>

### Table 3. Results of lipogram in control and treated rats

<table>
<thead>
<tr>
<th>Biochemical test</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>Prop.</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chol. mg/dl</td>
<td>57.64±10.121</td>
<td>85.91±21.465</td>
<td>80.72±20.327</td>
<td>68.14±5.194</td>
<td>68.08±18.329</td>
<td>ns</td>
<td>54.61</td>
</tr>
<tr>
<td>TG mg/dl</td>
<td>66.14±7.050</td>
<td>73.94±18.827</td>
<td>77.72±20.266</td>
<td>42.84±4.741</td>
<td>71.69±15.483</td>
<td>47.84</td>
<td></td>
</tr>
<tr>
<td>HDL mg/dl</td>
<td>46.21±23.918</td>
<td>48.58±19.935</td>
<td>59.28±16.866</td>
<td>83.19±5.656</td>
<td>70.35±6.718</td>
<td>ns</td>
<td>53.68</td>
</tr>
</tbody>
</table>
animals. The organs were fixed in 10% neutral buffered formalin solution and paraffin sections were prepared and stained with hematoxylin and eosin.

**Statistical analysis**

Data were analyzed by means of one way (ANOVA) using the software statistical program (SPSS, ver.16, USA). Data are expressed as the mean ± SE, and results were statistically significant at p<0.05.

**RESULTS**

**Clinical Signs**

Rapid heartbeat, rat hair loss, outer ear hematoma, blood thinning, laziness, weight loss and diarrhea.

**Hematological Parameters**

Results of erythrogram, blood platelets and reticulocytes as shown in Table 1 and Figs 1-3, revealed a significant difference in Hb, RBC’s, Hct, MCV, MCH, MCHC between different groups, while platelets and reticulocyte count showed no differences. Referring to the obtained data we can deduce that:

**Relative to control group 1:**
- RBC’s of groups 2, 3, 4, and 5 is lower by 30.9%, 9.7%, 27.5%, 15.2% respectively.
- MCV of groups 2, 3, 4, and 5 is higher by 46.9%, 2.5%, 10.5%, 12.6% respectively.
- MCH of groups 2, 3, 4, and 5 is higher by 46.2%, 2.02%, 24.4%, 9.2% respectively.

**Relative to AlCl₃ group 2:**
- RBC’s of groups 3, 4, and 5 is higher by 30.6%, 4.8%, 22.6% respectively.
- MCV of groups 3, 4, and 5 is lower by 30.2%, 24.8%, 23.3% respectively.
- MCH of groups 3, 4, and 5 is lower by 30.2%, 14.9%, 25.3% respectively.

The following bar charts is showing the degree of alterations in some haematological parameters that induced by oral administration of AlCl₃ (group 2), and the alleviating degree of
different oral camel milk treatments by (groups 3, 4, 5) compared to the control (group 1).

**Biochemical Parameters**

**Renal And Liver Functions**

The results of renal function (urea and creatinine) and liver function (ALT, AST and bilirubin) tests have been summarized in Table 2 and Figs 4-6. The obtained values clarified that there were no significant differences between the different treated groups in comparison with control one.

**Lipogram Test**

Compared to the control group, values of lipogram (total cholesterol, triglyceride and HDL), showed no significant difference in all treated groups as shown in Table 3, Fig. 7.

**Bacterial Count**

Lactobacillus acidophilus count was $1.7 \times 10^7$ (Cfu/ml).

**Histopathological Examination**

Hepatic and renal of control, AlCl$_3$, and AlCl$_3$ + camel milk treated rats showed variable histopathological alterations of varying severity and extent. Histopathological examination of rat’s organs proved that camel milk has the capacity to reduce the aluminum toxicity in liver, kidney.

**Liver**

Microscopical examination of liver sections from the control group showed no evidence of histological abnormalities. The examination revealed regular hepatocytes architecture with distinct central vein, polygonal hepatocytes arranged in strands running radially from the central vein with blood sinusoids in between these hepatic strands (Fig. 8-a). On the other hand, liver sections from rats administered AlCl$_3$ showed distorted liver architecture, focal hepatic necrosis.

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**Fig. 5.** Effect of Aluminum and different oral camel milk treatments on AST/GOT & ALT/GPT

**Fig. 6.** Effect of Aluminum and different oral camel milk treatments on Total Bilirubin. G1 (control group), G2 (AlCl$_3$ treated group), G3 (AlCl$_3$ + raw camel milk group), G4 (AlCl$_3$ + heat treated camel milk group); and G5 (AlCl$_3$ + sweet acidophilus camel milk)
associated with inflammatory cells infiltration and cytoplasmic vacuolization of hepatocytes were observed (Fig.8-b). Administration of camel milk (the 3 different treatments) prior to AlCl₃ improved to a large extent the hepatic damage induced by AlCl₃. The examined sections revealed normal architecture of the liver (Figs 8-c; d; e).

Kidneys

Kidney sections from control group revealed the normal histological structure of renal parenchyma from renal cortex and medulla (Fig.9-a). Microscopical examination of AlCl₃ treated group showed necrobiotic changes of renal tubular epithelium, interstitial inflammatory cells infiltration, atrophy of glomerular tuft and distension of Bowman’s space (Fig.9-b). The different treatments of camel milk restoring the normal histological structure of renal tissue (Figs 9-c; d; e).

**DISCUSSION**

Aluminum in its metallic form has many useful roles, such as in construction, packaging, and transport vehicles. Aluminum salts are included in pharmaceuticals, processed foods, and vaccines to enhance their qualities. Many cities use aluminum sulfate or poly aluminum chloride to clarify their drinking water. According to the World Health Organization, aluminum additives are the main source of exposure for most humans.  

Camel milk is full of balanced nutritional constituents and also displays a wide variety of biological actions that influence growth and development of particular body organs, metabolic responses towards nutrients absorption, digestion and fight against diseases.

The present study was carried out to evaluate the efficiency of camel’s milk to ameliorate the toxicity of AlCl₃ on some hematologic parameters; hepatic, renal functions; lipids profile; and histopathology of liver and kidney.

Our results showed a significant decrease in red blood cells count (RBC’s), significant

![Figure 7](image_url)

**Fig. 7.** Effect of Aluminum and different oral camel milk treatments on Cholesterol (Chol.), Triglyceride (TG) and HDL. G1 (control group), G2 (AlCl₃ treated group), G3 (AlCl₃ + raw camel milk group), G4 (AlCl₃ + heat treated camel milk group); and G5 (AlCl₃ + sweet acidophilus camel milk)

![Figure 8](image_url)

**Fig. 8.** a. Liver of control rat [group1] showing the normal histological structure of hepatic lobule (H & E X 400). b. Liver of AlCl₃ treated rat [group2] showing cytoplasmic vacuolization of hepatocytes (H & E X 400). c. Liver of AlCl₃ + raw camel milk rat [group3] showing no histopathological changes (H & E X 400). d. Liver of AlCl₃ + heat treated camel milk rat [group4] showing no histopathological changes (H & E X 400). e. Liver of AlCl₃ + sweet acidophilus camel milk rat [group5] showing no histopathological changes (H & E X 400).
increase in mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) 5.76±0.152, 69.34±2.152, 30.08±0.882 respectively in AlCl$_3$ group compared with the control group values that were 8.33±0.327, 47.20±2.077, 20.57±0.917 respectively; while hemoglobin (Hb), hematocrite (Hct), platelets (plt), reticulocytes (Ret), mean corpuscular hemoglobin concentration (MCHC) did not revealed significant changes between two groups; the obtained anemia was macrocytic normochromic. Similar results more or less were reported in a previous studies$^{24}$ which denoted that AlCl$_3$ had led to a significant decrease (P< 0.05) in red blood cells count (RBCs), hemoglobin (Hb), mean corpuscular hemoglobin concentration (MCHC), hematocrite (Hct) and iron level, and a significant increase in the mean cell volume (MCV) and no significant change in the platelets (plt); while another study$^{18}$ showed that oral AlCl$_3$ treatment caused a significant decrease (P< 0.05) in red blood cells count (RBCs), blood hemoglobin (Hb), and hematocrite (Hct), whereas the values of mean corpuscular volume (MCV), mean hemoglobin concentration (MHC), mean corpuscular hemoglobin concentration (MCHC) didn’t change. The differences between records may be due to the experimental design adopted by the investigator. Administration of camel’s milk with AlCl$_3$ significantly increased red blood cells count (RBCs), and decreased mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) compared with the results in rats orally administered AlCl$_3$ alone. Thus brought the altered blood parameters to near normal levels. The obtained macrocytic normochromic anemia (megaloblastic – pernicious anemia) may be due to vitamin B$_{12}$ deficiency$^{25}$. The term macrocytosis is a disease in which not enough red blood cells are produced therefore red blood cells (RBC) are larger than normal so macrocytosis is reported in terms of mean corpuscular volume (MCV).

Vitamin B$_{12}$ or cobalamin is important in normal blood formation. It is well established that gastric acid secretion is needed for dietary vitamin B$_{12}$ absorption from foods bound to proteins, and the presence of gastric acid is required for the peptic enzymes, mainly pepsin, allowing its reassociation with intrinsic factor (IF) and eventual absorption in the terminal ileum. Accordingly, alterations in the gastric acid secretion may lead to malabsorption of vitamin B$_{12}$. The origin of these alterations could be either natural, such as ageing process, or iatrogenic, such as partial resection of the stomach or acid suppressive therapy which lead to low stomach acid with a high or alkaline pH thus foods aren’t broken down, B$_{12}$ is not released. That is the way by which aluminum antagonize B$_{12}$; the antagonism may not be direct but, as a result of excessive intake. For example Al- containing antacids are helpful in relieving symptoms of gastritis by neutralizing gastric acids and inhibit smooth muscle contraction, thus inhibiting gastric emptying, hence antacids may lead to malabsorption of vitamin B$_{12}$.\textsuperscript{26}

Our results showed that the hepatic and renal functions; lipids profile had no significant changes between different groups. It has been reported that animals exposed to AlCl$_3$ displayed hepatic necrosis, which was indicated by an increase in serum levels of liver enzymes including
AST and ALT; increase in total bilirubin in the serum increase in serum urea and creatinine; increased serum cholesterol and triglyceride levels. In our study, the lack of significant changes is due to a mild change in organs not lead to significant increase in current laboratory test, so the differences from other studies probably according to the magnitude of AlCl₃ dose: 20 mg/kg body weight; 50 mg/kg body weight; 100 mg/kg body weight. However, histopathological examination showed variable alterations of varying severity in different organs. Disagreement with our results may be attributed to several reasons like experimental design, chemicals, camel milk source, the animal model, dosage and the way by which both AlCl₃ and camel milk was administered, the exposure frequency and duration, environmental conditions and methods of analysis.

Exposure to high concentrations of Al (500 mg/kg body weight) can result in its accumulation in the liver and in turn to alterations in the liver function. Transaminases are intracellular enzymes, released into the circulation after damage and necrosis of hepatocytes, the induced histological changes in the liver are attributed to free radicals production and oxidative stress. Moreover, it was found that the induction rate of serum bilirubin was associated with free radical production.

It was recorded that the kidney may be exposed to high concentrations of Al during the normal process of renal excretion making the kidney vulnerable to Al-mediated toxicity, which is dependent on the route of exposure. Alternatively, a study declared that the increase in urea serum concentration in Al treated animals may be due to Al-mediated changes in liver function.

A study showed that oral administration of AlCl₃ induced a significant increase in plasma level of total lipids, total cholesterol and triglycerides. Accumulation of AlCl₃ in the liver may lead to a disturbance of lipid metabolism and in turn to the elevation in lipid profile. On the other hand, a research demonstrated that treatment of rats with camel’s milk prior to AlCl₃ resulted in decreased cholesterol and triglyceride levels in the serum compared to the AlCl₃-intoxicated group.

In our experiment camel’s milk administration in groups 3, 4, 5 alleviated the toxic effect of AlCl₃ with variable degrees between different groups. This alleviation could be attributed to the positive health effects of camel milk as antioxidant, anti-microbial, antihypertensive or immuno-modulatory and anti-thrombotic. Both caseins and whey proteins of camel milk possess bioactive peptides with significant radical-scavenging activities (e.g. lactoferrin) and thus herald a fascinating opportunity for their potential as nutraceuticals or therapeutic peptides for prevention and treatment of oxidative stress-associated diseases. Numerous vitamins exhibit powerful antioxidant action such as D, E, A, C and vitamins of B group e.g. folic acid and B₁₂ are found in dromedary camel milk. Camel milk is rich in Mg and Zn. Magnesium (Mg) protects cells from heavy metals such as aluminum. Mg protects cells against oxyradical damage; assists in the absorption and metabolism of vitamins B, C and E; and is essential for biosynthesis of glutathione. Additionally, camel’s milk is rich in zinc (Zn). More than 300 enzymes require Zn for activity, and can prevent cell damage through activation of the antioxidant system. Some authors reported that the major nutrients of milk were left unchanged by pasteurization; Suliman et al. (2013) showing that in general heat has very little effect on mineral content with exception of Zn; while another concluded that pasteurization does not destroy zinc in camel milk.

Development of the technology for special purpose dairy products based on camel milk using probiotic starter cultures that have the ability to destroy toxic metabolites and synthesize vitamins. Lactic acid bacteria (LAB) and more particularly lactobacilli were reported to bind heavy metals and thus represent a promising approach for decontamination of heavy metals in food and water and perhaps gastrointestinal tract as well.

**CONCLUSION**

Indeed, it could be concluded that oral administration camel’s milk administration at a dose of 5 ml camel’s milk 10 min before the administration of AlCl₃ alleviated its toxic effect. Moreover, it improved to a large extent the histological changes induced by AlCl₃ in such a way that more or less normal architecture of the liver and kidney was observed. We can suggest that the best treatment was raw camel milk group;
followed by sweet acidophilus milk group; and the last one was thermally treated camel milk. Therefore supplementation with camel milk may be useful as a protective therapy in cases of intoxication with aluminum.

ACKNOWLEDGMENT

The author would thank all participants and their parents

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