

Effect of Treated Oily Waste Water of Khartoum Refinery on Nubian Goat Kids

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ABSTRACT

One of the very many imposed environmental threats of the petroleum industry internationally is its effluent of treated oily waste water (TOWW), which was reported causing mortalities in small ruminants. This study was carried out to investigate the toxic effects of TOWW of Khartoum Refinery Company (KRC) on Nubian goat kids. Twenty-one goat kids were divided into three groups: Group A (control), arranged in 1x3, and B and C (test groups) arranged in 2x3x3. Each test group was divided into three subgroups; B1, B2 and B3 and C1, C2 and C3. Groups B and C were watered for 6 weeks from KRC oxidation ponds II and III, respectively, diluted at ratios of 1:0, 1:1 and 1:2 with tap water respective to subgroups 1, 2 and 3. The control group was tap watered. All groups were fed on a basal ration of 2.86 Mcal ME/kg and crude protein 20.88% energy concentration meeting goat daily requirements. Clinical signs and mortalities were daily observed. Body weights were recorded fortnightly. Blood and serum samples were collected fortnightly for haematological (RBCs, Hb, PCV, WBCs and their differentials) and serum metabolites (glucose, total protein, albumin and globulin), enzymes (AST and ALP) and electrolytes (Ca, P, Co, Cu, Fe and Mn) examinations. Organs samples for histopathological investigations were collected on post mortem examinations or *in extremis* slaughter. All test groups significantly ($P \leq 0.05$) lost weight with variations in the total water consumption and similar average water consumption for all subgroups except C3 ($P \leq 0.05$). All test groups showed decreased appetite, diarrhoea, weakness and partial paralysis. The mortality was 100%, 66.6% and 33.3% in subgroups C1, C2 and C3, respectively. The mortality was 66.6% in subgroup B3 and 33.3% in each of B2 or B1. Haematologically, test goats were neither anaemic nor infected with haemodilutions ($P \leq 0.05$) in subgroups B3, C2 and C3. All test groups showed congestion or haemorrhage in the intestinal submucosa, with detachment of epithelium or detachment of the villi, in response to the osmotic diarrhoea. All test groups showed no significant ($P > 0.05$) changes in serum metabolites, electrolytes or serum enzymes, but livers were congested, stressed and engorged. Bile and kidney showed shrinkage, necrosis of the glomeruli, congestion and haemorrhage in the cortical and medullary tubules. These signs were indications of intensive mineral excretion. It is concluded that TOWW from KRC has toxic effects on Nubian goat kids judged by the early mortalities and by the clinical signs

of diarrhoea and recumbency, metabolic disturbances in the liver with histopathological changes in various body systems. It is recommended that TOWW initial concentration be restored by addition of equivalent water to reduce or abolish its toxic manifestations.

Key words: Treated petroleum effluent water; Nubian goat kids; refinery

INTRODUCTION

Petroleum in the Sudan was first exploratory-drilled in Abugaabra 1979 till commercially produced and exported later in 1999 (MEM, 2006) during which three local refineries were established. The Khartoum Refinery Company (KRC) (25 km north-east to Gaili) the biggest to produce 50-70 thousand barrels/day (El Amin, 2004 and MEM, 2006). The petroleum industry, though strategic and vital to the modern civilizations, imposes a real threat to the local, regional or global environment. Elements of soil, water and air pollution exist throughout all activities from exploration to final exhaust from internal combustion (MEM, 2006). The KRC water effluent of treated oily waste water (TOWW) was the major environmental pollution source to man and animals. Water pollution is a major problem and has been suggested as the leading worldwide cause of deaths and diseases (Helmer and Hespanhol, 1997) as contaminants may include a wide spectrum of organic and inorganic substances (Hogan, 2010). Nevertheless, MEM (2006) enforced environmental health and safety and prevention of pollution by law Petroleum Wealth Act 1998, and the bylaws on environment protection in the petroleum industry 2002.

The refinery main solid wastes are either oiled or non-oiled materials. Waste oiled water (hydrocarbon polluted water) is drawn from the bottom of the crude oil tank (mechanical separation) into the grid pond and then with further physical treatment (precipitation) to pond 1 when biologically treated (aerobic and anaerobic bacteria) to pond 2 and finally by oxidation to the third and last pond in an attempt to minimize pollutants to the lowest harmful level (El Amin, 2004 and Obeadalla, 2005). Finally, treated oily waste water from the third and last oxidation pond is then transferred to external sequential storage ponds I, II and III. Analyses of the first oxidation pond water revealed no oil, pH 8.3, sulphide: 0.09mg/L, ammonia nitrogen 38.9 mg/L, Chemical oxygen demand (COD) 290 mg/L and biological oxygen demand (BOD) 28 mg/L. Minerals Cr, Cd, Cu, Co, Fe, Ni, Pb, and Zn were also analyzed at 0.09, 0.02, 0.03, 0.07, 0.12, 0.10, 0.10 and 0.17 ppm respectively (CPL, 2007 and KRC, 2007). Frequent domestic animal mortalities subsequent to erratic watering on a refinery TOWW oxidation ponds were reported (Hamadaniel, 2007). Whether the water pollutants level set above exerts a toxic threat to animals, no research was run to investigate the assumption. The following experiment was designed to study toxicity of TOWW in Nubian goat kids from Khartoum Refinery second and third External oxidation ponds.

MATERIALS AND METHODS

Animals and grouping

Twenty one healthy male Nubian goat kids, 3-5 months old, weighing 16–25 Kg, were launched within the premises of the Department of Pharmacology and Toxicology, Faculty of Veterinary Medicine, University of Khartoum. Animals were kept for a one week adaptation period and then were weight-distributed and allotted randomly to three treatment groups A, B and C. Groups B and C (test groups) were divided each into three subgroups (each of three animals) B₁ (No. 10, 11 and 12), B₂ (No. 16, 17 and 18), B₃ (No. 4, 5 and 6), C₁ (No. 1, 2 and 3), C₂ (No. 13, 14 and 15) and C₃ (No. 7, 8 and 9). Group A goats (No. 19, 20 and 21) were designated as control group.

Feeding and TOWW dosages

All experimental groups were basally fed formula concentrate composed of 54% Dura, 20% groundnut cake, 25% wheat bran and 1% salt (NaCl). Calculated energy concentration (2.86 Mcal ME/kg) and crude protein (20.88%) were made for goat daily requirements (Devendra and Burns, 1983 and NAS, 1980). Daily allowance was 200g per head/day. Groundnut hay as roughage was supplied *ad lib*. TOWW as test material was secured from KRC, Gaili. Groups B and C were watered from oxidation ponds II and III, respectively, diluted at ratios of 1:0, 1:1 and 1:2 with tap water respective to subgroups 1, 2 and 3. The control group was tap watered. Experimental watering continued for 10 weeks.

Data collection

Clinical signs and mortalities were daily observed. Body weights were recorded fortnightly. Blood and serum samples were collected fortnightly for haematological RBCs, Hb, PCV, WBCs values and their differentials (Schalm *et al.*, 1965). Serum metabolites (glucose, total protein, albumin and globulin), enzymes (AST and ALP) values and serum macrominerals were determined using commercial kits. Serum trace elements Cu, Co, Fe and Mn were determined using atomic absorption spectrometry (Sperling and Welz, 1999). At necropsy or *in extremis* slaughter, all goats were examined for gross lesions and samples of intestines, livers, kidneys, hearts and lungs were immediately fixed in 10% neutral buffered formalin and paraffin sections were stained with haematoxylin and eosin (H&E).

Statistical verifications

Mean values obtained in blood and serum parameters were statistically verified using the un-paired Student *t*-test. All means were compared at the 5 or 1% probability

level. Trend in body weight changes were regressed against time and expressed as growth coefficients (Mendehall, 1971).

RESULTS

Clinical signs

Severity of symptoms in goat kids surviving treatment periods with mortalities are shown in Table 1. Goats in subgroups B and C showed dullness, decreased appetite, diarrhea and weakness at varying degrees. Goats in subgroup C₁, two goats in subgroup B₂ and one goat in either of subgroup B₁, C₂, B₃ and C₃ showed marked decrease in appetite. Diarrhoea was severe in two goats in either subgroup C₁ and B₁ and one goat in either subgroup C₂, B₃ and C₃. Goats showing marked decrease in appetite were also markedly weak except for goat 1 (C₁) mild weakness and goat 4 (B₃) severe weakness. Partial paralysis followed the same with goats having marked decrease in appetite except for goat 13 (C₂) and goat 6 (B₃). One goat was recumbent in subgroups B₁, B₂, C₂ and C₃ and two goats in subgroup C₁. The mortality was 100% in subgroup C₁ occurring in weeks 2(G₂), 3(G₃) and 5 (G₁) and the least (33.3%) in subgroups B₁ (G₁₁) and C₂ (G₁₃) occurring in weeks 8 and 6, respectively. No mortalities were recorded in the control group.

Performance values

Goats in the three treatment groups started experimental watering at a similar ($p>0.05$) initial body weights and ended also at a similar ($p>0.05$) final body weights except for subgroup C₁ ($p<0.05$) when all goats died by week 5 before termination of the experiment (Table 2). Goats in subgroups B₃ and C₃ recorded lower ($p>0.05$) final body weights (06.00 ± 1.04 and 06.17 ± 1.07 kg/ head, respectively) compared to the control. Goats in subgroups B and C lost weights significantly ($p<0.05$) with the B₁ subgroup losing most (-5.17 ± 3.33 kg) and C₂ subgroup losing least (-2.67 ± 1.61 kg) compared to the control (Table 3). Subgroup B₃ rate ($- 0.82$) and C₁ ($- 0.92$) were the highest in their respective groups.

Haematological findings

There were no significant ($p>0.05$) differences in the haematological parameters between the control and test goats except in PCV ($p<0.01$) in subgroups B₃, C₁, C₂ and C₃, which were also higher ($p>0.05$) in WBC values (Table 4). WBCs differential count showed lymphocytes ($55.13\pm 8.13\%$) and monocytes ($1.80\pm 0.77\%$) values of subgroup C₂ to be lower ($p>0.05$) and neutrophils ($41.27\pm 8.19\%$) value to be higher ($p>0.05$) than the control group. Eosinophils of subgroups B₁ ($1.71\pm 0.69\%$) and C₁ ($1.75\pm 0.89\%$) were lower ($p>0.05$) than the control group.

Changes in serum metabolites and enzymes

There were no significant ($p>0.05$) differences between control goats and those watered TOWW in serum metabolites glucose, total protein, albumin and globulin and activities of serum enzymes AST and ALP (Table 5). Serum glucose was the highest ($p>0.05$) in subgroup B₂ (51.98 ± 23.73 mg/dl) and the lowest ($p>0.05$) in subgroup C₃ (36.45 ± 18.50 mg/dl) compared to other subgroups. The total protein values in subgroup C₂ (6.25 ± 0.73 mg/dl) were the highest ($p>0.05$) of all subgroups and likewise ($p>0.05$) the globulin value (3.03 ± 0.36 mg/dl) of subgroup C₁. Subgroups C₁, C₂ and C₃, AST values were higher ($p>0.05$) than the control group (124.25 ± 64.22 i.u.) whereas in the subgroups ALP, values were also higher ($p>0.05$) than the control group (63.70 ± 59.58 i.u.).

Changes in serum electrolytes

There were no significant ($p>0.05$) differences between the control goat kids and goat kids groups in the electrolytes parameters except in phosphorus ($p<0.05$) concentration (5.15 ± 1.65 mg/dl) of subgroup C₃ (Table 6). Non-significantly ($p>0.05$) higher values than the control in subgroup B₂ (10.27 ± 1.89 mg/dl) were monitored for Ca and in the subgroups for Cu. Cobalt values (0.06 ± 0.02 , 0.06 ± 0.01 , 0.07 ± 0.02 and 0.06 ± 0.02 mg/dl) were the least ($p>0.05$) of the subgroups B₃, C₁, C₂ and C₃, respectively. Only in B₃ subgroup, Fe and Mn values (2.94 ± 1.74 and 0.14 ± 0.12 mg/dl) were the lowest ($p>0.05$) of the treatment groups.

Post mortem findings

Post mortem findings in G₂ (Fig. 1) showed normal lung and distended rumen with engorged external vessels and severe intestinal flatulent haemorrhagic vessels. G₂ also showed congested liver with round edges and highly engorged gall bladder (Fig. 2).

Histopathological findings

In subgroup C₁ goats, the intestine of goat 1 showed congestion in the submucosa and detachment of the epithelium (Fig. 3 and the the kidney goat showed necrosis and shrinkage of the glomeruli. The lung of goat 1 and goat 2 (subgroup C₁) showed congestion and emphysema, generalized fatty changes and congestion in the liver central vein. Goat 2 (subgroup C₁) kidney showed severe congestion and hemorrhage in the cortical tubules and shrinkage of glomeruli. Goat 3 (subgroup C₁) showed intestinal congestion of the submucosa and sloughing of epithelial membrane and its kidney showed severe shrinkage and necrosis of the glomeruli. In goats of subgroup C₂, the livers of goats 13, 14 and 15 showed severe fatty changes and congestion of the portal tract. Lungs of all of the C₂ goats subgroup showed severe emphysema and haemorrhage while only intestines of goats 13 and 14 showed slight congestion in the submucosa and the intestine of goat 15 revealed no change. In the spleen of goats 13 and 14, there was a

haemosiderin deposit, whereas in goat 15, there was a slight splenic congestion. The kidneys of goats 13 and 14 showed necrosis of the glomeruli and congestion of the medullary tubule but hemorrhaged in G₁₅.

In goats of subgroup C₃, the intestine of G₇ showed slight congestion in the submucosa and sloughing of epithelial membrane and the kidney showed congestion and hemorrhage in the cortical and medullary tubules. The lungs of G₇ and G₈ showed severe hemorrhage and emphysema (Fig. 4), whereas in the same subgroup, G₉ lung showed severe congestion and oedema and its liver showed generalized fatty change and congestion in the central vein (Fig. 5). In goats 8 and 9, the kidneys showed severe central congestion and in the medulla and shrinkage of the glomeruli (Fig. 6). The spleen of all goats in subgroup C₃ showed slight congestion.

In subgroup B₁ goats, the intestine of goat 10 showed slight congestion in the submucosa whereas in goats 11 and 12, no pathological changes were observed. The lungs of goats 10 and 11, showed oedema and emphysema. Livers of goats 10 and 12 showed generalized fatty changes and congestion in the central vein when the liver of goat 11 showed only generalized fatty change. Kidney of goat 12 showed congestion in the medullary tubules and slight necrosis in the glomeruli. The spleen of goat 10 showed slight congestion.

In the goats of subgroup B₂, the lungs showed congestion and emphysema. The liver of goat 17 showed generalized fatty changes and lymphocyte infiltration but liver of goat 18 showed only severe hepatocytic fatty changes. The intestine of goat 16 showed hemorrhage of the submucosa and the intestine of goat 18 showed congestion in the submucosa and detachment of villi. The kidneys of goats 17 and 18 showed shrinkage of the glomeruli and dilatation of the medullary tubules but the kidney of goat 16 showed necrosis of the glomeruli and congestion of the medullary tubules. There were congestion and haemorrhages in the heart of goat 17. In goats subgroup B₃, the kidney of G₅ and G₆ showed necrosis and shrinkage of the glomeruli and congestion in the medullary tubules whereas in G₄ the kidney had severe congestion and hemorrhage in the medullary tubules (Fig. 7).

Goats of the subgroup B₃ showed severe congestion, haemorrhage and oedema of the lungs. The livers of goats 4 and 5 showed generalized fatty changes and congestion in the hepatocytes whereas the congestion in goat 6 liver was in the portal tract (Fig. 8). Goat 5 showed sloughing of the epithelial membrane of the intestines whereas in goat 6 there was detachment of the villi and congestion in the submucosa (Fig. 9).

DISCUSSION

Khartoum refinery is a complex one that generates waste water in big quantities. The origin of this water comes from the processing units which involve large numbers of heavy metals and other contaminants from the crude oil, catalysts and chemicals; hence it

produces water with a potential toxicity. There is a system of treatment in Khartoum refinery for the produced waste water, however it is not certain that this treatment is enough for this water to be safely used by animals and in particular the Nubian goats which are raised in the area. Analyses of the third pond water revealed minerals Cr, Cd, Cu, Co, Fe, Ni, Pb and Zn at 0.09, 0.02, 0.03, 0.07, 0.12, 0.10, 0.10 and 0.17 ppm, respectively (CPL, 2007 and KRC, 2007).

The goats in groups B and C showed a decrease in appetite which was well marked in subgroups B₂ and C₁ of the highest average daily water intake from the two ponds waters *per se* and decreases with the dilution in other subgroups. High ruminal salinity delays the microflora and microfauna enzymatic digestion of feed and hence reduces appetite (McDonald *et al.*, 1988 and Church, 1993). Diarrhoea and weakness were shown in all goat subgroups being severer with goats watered whole ponds water i.e. subgroups B₁ and C₁ and decreased with the dilution in other subgroups. Varying dullness signs dominated over all goat groups. Pariesis and recumbency were well marked in goats of subgroup C₁ (whole pond III water) and decreased with ponds water dilution in other subgroups. Earlier mortalities (all goats within 5 weeks) occurred with whole pond III water compared to a later mortality (one goat within 8 weeks) watered from whole pond II water. Diarrhoea likely happened as osmotic one, due to the high ionic concentration in the intestinal tract beside enteric effects of heavy metal toxicants like lead, mercury, arsenic or cadmium. The post mortem picture of the alimentary tract showed distension of rumen (bloat) and intestine (flatulence). Ruminal gas production always occurs when gas producing microflora are favoured due to nutritional factors that alter their environment, as when changing pH. The pathological changes in the intestine, like congestion in the submucosa and detachment of epithelium in the intestine and the villi, confirmed the histological picture in osmotic diarrhoea (Radostits *et al.*, 1994). Consequences of diarrhoea are dehydration, weakness, partial paralysis and recumbency (Radostits *et al.*, 1994 and Merck, 2004).

Mortalities with higher diluted pond waters occurred earlier than the lower dilutions. This can be attributed to the fact that reduction of water salinity induces higher consumption. The mortalities evoked were not well correlated to ponds and concentrations. Pond III water induced 100% mortality (C₁) when pond II water induced 33.3% mortality (B₁). Disparities also showed lower concentrations (1:2 from either pond) causing 66.6% mortality (B₃ and C₃) and a higher one (1:1 from pond III) causing a lower mortality (33.3% in C₂). Factors contributing to this irregularity varied with the amount of water consumed which was in turn governed by pond water palatability, individual animal tolerance to water salinity and the degree of thirst relative to feed dry matter consumption. It is likely to consider pond III water more toxic to goats due to evaporative concentration of salts.

The goats experienced significant ($p < 0.05$) weight losses being higher with whole pond waters (the least total water consumption) and at the higher dilutions (high

total water consumption). Though average water consumption remained similar ($p>0.05$) for all the subgroups, only C₃ subgroup consumed a higher ($p<0.05$) average. Oral saline water, as toxicant, may exert its effect on the ruminal microflora and microfauna, thus, affecting the microbial digestion and consequently energy and protein substrates. Hence, oral TOWW retards significantly the growth rate of small ruminants (goats).

Haematologically, the test goats experienced similar ($p>0.05$) red cell values i.e. no anaemia. Normal WBCs and their differential counts pointed to non-infectious disease. PCV values were significantly ($p<0.05$) lower with higher water consumption (higher ponds water dilutions) with subgroups B₃, C₂ and C₃ being more haemodiluted.

The normal serum enzyme AST and ALP activities, normal serum total proteins and its fractions (active synthesis) in all test goat groups refer to normal liver function but stressed at high blood supply demand (congestion of the portal tract). Fatty livers monitored with normal peripheral glucose (no hypoglycaemia) revealed high liver glycogen immobilization for a critical energy demand (Cornelius and Kaneko, 1963). Though drinking water supplied to all test goat subgroups has high mineral content, serum electrolytes (macro and micro) remained at normal values, that is, not following concentration gradients. Intestinal ionic absorption mechanisms may follow active blockade. The engorged gall bladder in the post mortem picture might be a sign of intensive mineral excretion. The kidneys also will be loaded with excretion of minerals derived of the saline water. Varying tissue affections of the kidney depends on the water salinity and the quantity drank by the animal. Affections vary from shrinkage to severe shrinkage and necrosis of the glomeruli. Further, severe congestion and haemorrhage in the cortical tubules down with slight dilatation, congestion to severe central congestion or haemorrhage of the medullary tubules. The lung tissues showed congestion and oedematous changes to severe haemorrhage and emphysema in test goats. Acidosis may arise from osmotic diarrhoea leading to enhanced alveolar ventilation; however, laboured respiration was not noticed.

The present study has clearly shown that TOWW has toxic manifestations as clinical signs, metabolic disturbances and histopathological changes in various Nubian goat kids. When used for animal watering or any other uses, freshly TOWW of ponds I or II were believed to have less toxic or detrimental manifestations than pond III. Equivalent water addition or more restores, at least, initial concentration as in pond I and consequently reduces TOWW toxic properties and toxicity risks. Public awareness is essential for avoidance of the TOWW from refinery oxidation ponds.

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Table 1. Severity of symptoms and mortalities of individual goat kids (G) watered TOWW from KRC for 10 weeks.

Symptoms	Treatment groups						
	A	B ₁	C ₁	B ₂	C ₂	B ₃	C ₃
		pond (II)	pond (III)	(II/1:1)	(III/1:1)	(II/1:2)	(III/1:2)
Decreased Appetite	-	G ₁₀ +	G ₃ ++	G ₁₆ ++	G ₁₃ ++	G ₄ +	G ₇ +
		G ₁₁ ++	G ₂ ++	G ₁₇ +	G ₁₄ +	G ₅ +	G ₈ +
		G ₁₂ +	G ₁ ++	G ₁₈ ++	G ₁₅ +	G ₆ ++	G ₉ ++
Diarrhoea	-	G ₁₀ +	G ₃ ++	G ₁₆ ++	G ₁₃ ++	G ₄ +	G ₇ +
		G ₁₁ +	G ₂ ++	G ₁₇ +	G ₁₄ +	G ₅ +	G ₈ +
		G ₁₂ +	G ₁ +	G ₁₈ ++	G ₁₅ +	G ₆ ++	G ₉ ++
Dullness	-	G ₁₀ +	G ₃ ++	G ₁₆ ++	G ₁₃ +	G ₄ +	G ₇ +
		G ₁₁ +	G ₂ +	G ₁₇ +	G ₁₄ +	G ₅ +	G ₈ +
		G ₁₂ +	G ₁ +	G ₁₈ ++	G ₁₅ +	G ₆ +	G ₉ ++
Weakness	-	G ₁₀ +	G ₃ ++	G ₁₆ ++	G ₁₃ ++	G ₄ ++	G ₇ +
		G ₁₁ ++	G ₂ ++	G ₁₇ +	G ₁₄ +	G ₅ +	G ₈ +
		G ₁₂ +	G ₁ +	G ₁₈ ++	G ₁₅ +	G ₆ ++	G ₉ ++
Partial Paralysis	-	G ₁₀ +	G ₃ ++	G ₁₆ ++	G ₁₃ +		G ₈ +
		G ₁₁ ++	G ₂ ++	G ₁₇ +		G ₄ +	G ₉ ++
		G ₁₂ +	G ₁ ++	G ₁₈ ++		G ₆ +	
Recumbency	-	G ₁₁ +	G ₃ +	G ₁₆ +	G ₁₃ +	-	G ₈ +
			G ₁ +				
Mortality							
	Week 2	-	G ₂				

Week 3	-	G ₃		G ₉
Week 4	-			G ₆
Week 5	-	G ₁		G ₈
Week 6	-		G ₁₆	G ₁₃
Week 7	-			G ₄
Week 8	-	G ₁₁	G ₁₈	

Count of the + sign denotes severity of symptoms.

Table 2. Average (mean \pm SD) performance values of goat kids watered TOWW from KRC for 10 weeks.

Group	Initial weight (kg/ head)	Final weight (kg/head)	Weight gain (kg/head)	Total water intake (lit./head)	Average daily water intake (lit./head/day)
A	20.50 \pm 3.78	2.00 \pm 06.932	04.33 \pm 2.08	10.44 \pm 1.43	0.19 \pm 0.04
B₁ pond (II)	23.17 \pm 0.29	18.00 \pm 03.46	-5.17 \pm 3.33*	7.05 \pm 1.03*	0.13 \pm 0.02
B₂ (II/1:1)	17.83 \pm 1.76	10.50 \pm 09.26	-4.07 \pm 2.65*	9.01 \pm 1.40	0.22 \pm 0.03
B₃(II/1:2)	24.50 \pm 1.80	06.00 \pm 1.04	-5.17 \pm 2.75*	7.81 \pm 1.35	0.16 \pm 0.02
C₁ pond (III)	21.50 \pm 0.01	00.00 \pm 0.00*	-3.50 \pm 1.00*	5.08 \pm 1.01*	0.24 \pm 0.02
C₂ (III/1:1)	19.33 \pm 0.29	12.83 \pm 11.25	-2.67 \pm 1.61*	7.72 \pm 0.86*	0.16 \pm 0.06
C₃ (III/1:2)	21.00 \pm 0.50	06.17 \pm 1.07	-5.17 \pm 2.75*	8.30 \pm 1.20	0.30 \pm 0.04*

Means in a column followed with no asterisks are similar ($p>0.05$). * mean value significant at ($p<0.05$).

Table 3. Regression equations of the growth curves of group A and subgroups B and C

Group/Subgroup	Regression equation
A	$y = 0.27x^* + 19.97$ (SE \pm 0.57)
B₁	$y = -0.60x^* + 22.33$ (SE \pm 0.35)
B₂	$y = -0.51x^* + 18.13$ (SE \pm 0.30)
B₃	$y = -0.82x^* + 23.86$ (SE \pm 0.32)
C₁	$y = -0.92x^* + 21.38$ (SE \pm 0.33)
C₂	$y = -0.07x + 18.71$ (SE \pm 0.27) NS
C₃	$y = -0.52x^* + 20.17$ (SE \pm 0.33)

Table 4. Mean (\pm SD) blood values and differential leucocyte count of goat kids watered TOWW from KRC for 10 weeks.

Group	A	B			C		
		B ₁	B ₂	B ₃	C ₁	C ₂	C ₃
RBCs $\times 10^6$ mm	9.43 \pm 1.24	10.09 \pm 0.68	8.89 \pm 0.79	9.83 \pm 0.62	10.95 \pm 0.78	9.56 \pm 0.76	9.51 \pm 0.66
Hb g/dl	9.07 \pm 1.44	9.67 \pm 1.40	9.66 \pm 1.57	9.93 \pm 2.00	10.88 \pm 1.99	9.33 \pm 1.08	9.14 \pm 1.04
PCV %	27.08 \pm 4.77	29.53 \pm 5.79	27.71 \pm 5.29	9.07 \pm 1.44**	10.88 \pm 1.99**	9.33 \pm 1.08**	9.14 \pm 1.04**
WBCs $\times 10^3$ mm	6.26 \pm 0.82	6.16 \pm 1.28	7.15 \pm 1.24	8.43 \pm 2.11	9.84 \pm 2.59	8.77 \pm 2.04	7.16 \pm 1.56
Lympho. %	62.00 \pm 4.55	64.59 \pm 3.87	63.36 \pm 4.29	60.08 \pm 8.32	61.75 \pm 7.96	55.13 \pm 8.13	62.08 \pm 7.27
Neutro. %	33.46 \pm 4.61	31.94 \pm 3.38	31.64 \pm 4.72	36.33 \pm 7.75	34.50 \pm 8.02	41.27 \pm 8.19	34.00 \pm 7.83
Mono. %	2.25 \pm 0.85	1.94 \pm 0.77	2.57 \pm 1.09	2.25 \pm 0.85	2.00 \pm 0.54	1.80 \pm 0.77	2.08 \pm 0.90
eosino. %	2.33 \pm 0.87	1.71 \pm 0.69	2.43 \pm 1.16	2.42 \pm 0.79	1.75 \pm 0.89	1.93 \pm 0.99	1.83 \pm 0.83

Means in a row bearing no asterisks are not significantly different ($p > 0.05$).

** mean values significantly different at ($p < 0.01$).

Table 5. Mean (\pm SD) serum values of goat kids watered TOWW from KRC for 10 weeks.

Group	A	B			C		
		B ₁	B ₂	B ₃	C ₁	C ₂	C ₃
Glucose (mg/dl)	45.88 \pm 6.59	43.38 \pm 14.25	51.98 \pm 23.73	40.69 \pm 15.12	43.36 \pm 19.60	41.43 \pm 15.35	36.45 \pm 18.50
Total protein (mg/dl)	5.72 \pm 0.72	5.84 \pm 0.69	6.09 \pm 0.79	5.72 \pm 0.49	5.94 \pm 0.44	6.25 \pm 0.73	5.94 \pm 0.93
Albumin (mg/dl)	2.64 \pm 0.57	3.04 \pm 0.43	3.11 \pm 0.48	3.01 \pm 0.51	2.93 \pm 0.56	3.12 \pm 0.32	2.96 \pm 0.54
Globulin (mg/dl)	2.93 \pm 0.45	2.72 \pm 0.42	2.94 \pm 0.61	2.72 \pm 0.33	3.03 \pm 0.36	2.95 \pm 0.57	2.55 \pm 0.35
AST (i.u.)	124.25 \pm 64.2 2	137.11 \pm 58.9 4	109.43 \pm 51.01	138.96 \pm 98.9 2	157.59 \pm 79.13	156.69 \pm 111.5 2	174.21 \pm 15.0 7
ALP (i.u.)	63.70 \pm 59.58	68.97 \pm 37.22	123.51 \pm 104.3 9	69.51 \pm 52.59	115.91 \pm 111.7 5	109.44 \pm 61.62	100.61 \pm 42.2 2

Means in a row are not significantly different ($p>0.05$).

Table 6. Mean (\pm SD) values of serum electrolytes of goat kids watered TOWW from KRC for 10 weeks.

Group	A	B			C		
		B ₁	B ₂	B ₃	C ₁	C ₂	C ₃
Ca (mg/dl)	9.26 \pm 1.27	9.68 \pm 1.04	10.27 \pm 1.89	9.24 \pm 1.10	8.33 \pm 1.29	9.58 \pm 1.03	9.63 \pm 1.27
PO₄ (mg/dl)	9.58 \pm 1.63	6.05 \pm 2.07	7.69 \pm 2.67	6.55 \pm 1.97	8.21 \pm 2.21	7.45 \pm 1.59	5.15 \pm 1.65*
Co (mg/dl)	0.07 \pm 0.02	0.07 \pm 0.0	0.07 \pm 0.02	0.06 \pm 0.02	0.06 \pm 0.01	0.07 \pm 0.02	0.06 \pm 0.02
Cu (mg/dl)	0.95 \pm 0.12	2.38 \pm 1.63	2.16 \pm 1.39	1.72 \pm 1.66	1.23 \pm 1.13	2.02 \pm 1.50	1.71 \pm 1.64
Fe (mg/dl)	3.81 \pm 1.57	3.86 \pm 1.31	3.17 \pm 1.84	2.94 \pm 1.74	3.05 \pm 1.51	3.58 \pm 1.52	3.30 \pm 1.73
Mn (mg/dl)	0.21 \pm 0.20	0.19 \pm 0.18	0.15 \pm 0.13	0.14 \pm 0.12	0.27 \pm 0.14	0.31 \pm 0.21	0.16 \pm 0.13

Means in a row bearing no asterisks are not significantly different ($p > 0.05$).

* mean values significantly different at ($p < 0.05$).



Fig. 1. Normal lung and distended rumen with engorged external vessels of goat kid G2 in subgroup C1.



Fig. 2. Congested liver with round edges and engorged gall bladder of goat kid G2 in

subgroup C1.

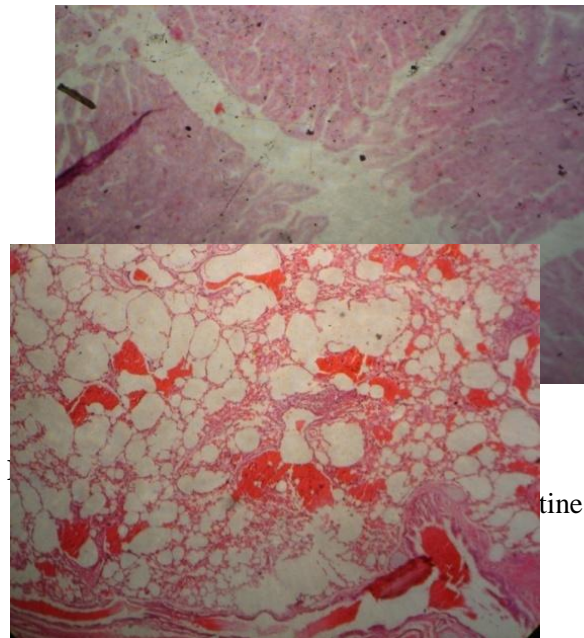


Fig. 4. Severe hemorrhage and emphysema
in the lung of G₈ in subgroup C₃.

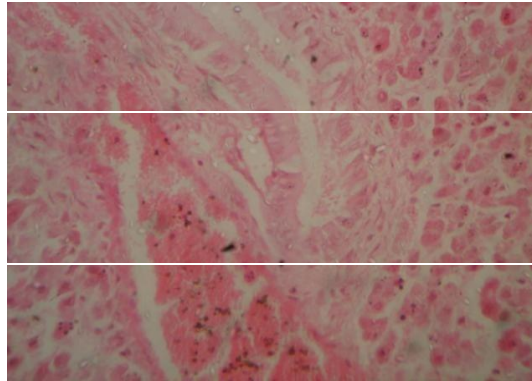


Fig. 5 Generalized fatty changes and Congestion in the central vein in the liver of G9 in the subgroup C3

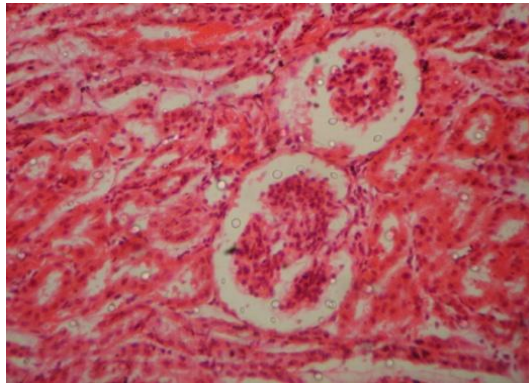


Fig. 6. Severe central congestion in the medulla and shrinkage of glomeruli in the kidney of G₈ in subgroup C₃.

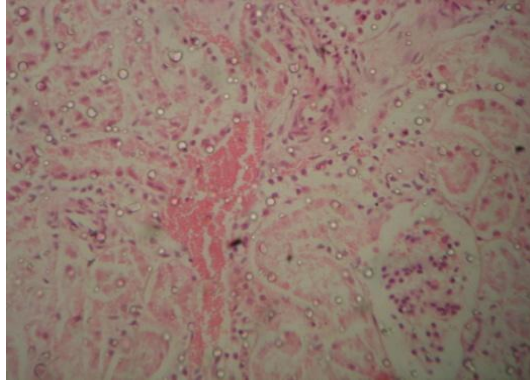


Fig. 7. Severe congestion and hemorrhage in the medullary tubules of the kidney of G₄ in subgroup B₃.

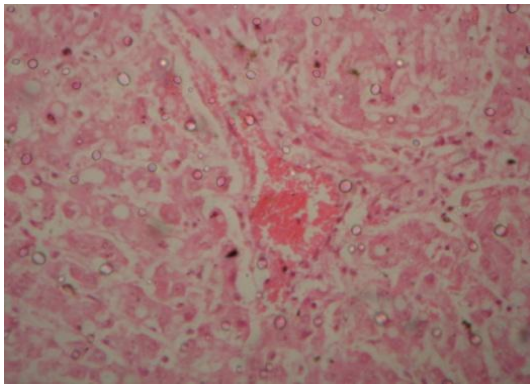


Fig. 8. Generalized fatty change and congestion in the portal tract in the liver of G₆ in subgroup B₃.

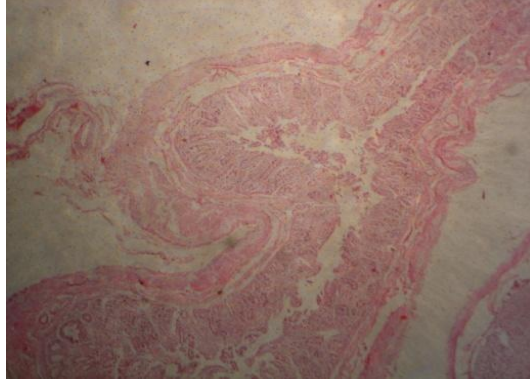


Fig. 9. Detachment of the villi and congestion in the intestinal submucosa of G_6 in subgroup B_3 .