

## Biological Attributes of Unfermented and Fermented Oat Milk Compared to Cow Milk in Alloxan-Induced Diabetic Rats

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### ABSTRACT

The present study aimed to assess the therapeutic effect of the unfermented and fermented cow; oat milk (CM, OM, FCM, and FOM) and fermented oat milk supplemented with 2% whey protein concentrate (FOM+WP) in diabetic rats. Alloxan was applied for inducing diabetes and hyperlipidemia in rats. The rats were randomly divided into two main groups. Control (-) (6 rats) were fed on a standard diet, while the second group (42 diabetics rats) were divided into six groups (6 rats each), and treated by different unfermented and fermented milk types by epigastric tube for 4 weeks. After four weeks, all treatments reduced the level of glucose, the serum levels of TC, TG and LDL-c compared to control (+) group and this was associated with a significant increase ( $p < 0.05$ ) in HDL-c in these groups. The highest significantly ( $p < 0.05$ ) decrease recorded in the groups fed with (OM) and (FCM), respectively. Liver enzymes activity decreased significantly ( $P < 0.05$ ) in FOM + WP, FOM, FCM and OM groups, respectively. Feeding the rats on fermented oat milk fortified with whey protein led to decrease the fecal total anaerobes and fecal enterobacterial counts and stimulate the viability of both fecal lactobacilli and bifidobacteria in the intestinal colon, compared with other treatments. Histopathological examination of the liver sections showed no histopathological alteration in the hepatic parenchyma in (FOM+WP) group. (OM) and (FCM) groups showed no histopathological alteration and the islands of Langerhans cells were in normal size as well as the surrounding acini and ducts. In conclusion, treatment with oat milk led to curing hyperglycemia and hyperlipidemia in diabetic rats. Meanwhile, treatment with fermented oat milk fortified with whey proteins resulted in a better effect on the liver and improved the growth of the health-promoting intestinal microflora.

**Keywords:** Diabetes, Alloxan, Hyperlipidemia, Hypercholesterolemia, Fermented oat milk.

### INTRODUCTION

*Diabetes mellitus* (DM) is epidemic disease suffusion around the world. The global prevalence of the disease is expected to increase from 2.8% in 2000 to 4.4% in 2030 (Peschke, 2007, Yajing *et al.*, 2012 and Shehata and Moussa, 2014).

The  $\beta$ -cell toxin (alloxan) is a model substance in type 1 diabetes. Alloxan resulted in ROS. During this redox reaction, free radicals are formed which results in the beta cell toxic action of alloxan (Tyrberg *et al.* 2001 and Lenzen, 2008).

There is a significant relationship between diabetes and lipid profile abnormalities which may induce a high risk of cardiovascular diseases (Lukic *et al.*, 2014 and Sangwan and Singh, 2018).

Numerous drugs and treatments to diabetes and lower cholesterol concentration have been successfully used but there were many adverse effects associated with synthetic medicine. Oat is effective in relieving the effects of high blood pressure, resulting in lower total serum, and LDL cholesterol, regulation of glucose and insulin levels in the blood, weight control and enhanced gastrointestinal health. Many of these effects are attributed to the presence of dietary fiber, primarily  $\beta$ -glucans.  $\beta$ -glucans are known to show the hypocholesterolemic effect. An increase in bowel viscosity is critical to lowering cholesterol. Concentration, structure, and molecular weight, play important roles in  $\beta$ -glucan functionality.  $\beta$ -glucans decreased serum total cholesterol (18.9%) and LDL-cholesterol (24.3%) in Sprague-Dawley rats. Addition of  $\beta$ -glucan resulted in increased bile acid secretion (Bekers *et al.*, 2001, Lambo *et al.*, 2005, Baroutkoub *et al.*, 2010, Bruckert, 2006 Yang *et al.*, 2003, Ibrugger *et al.*, 2013, Ahmed *et al.*, 2017, and Grundy *et al.*, (2018)).

Probiotics in functional fermented food can improve patient's conditions in medical disorders such as cancer, liver disease, genitourinary tract infections, atopic disease, and infections with *Helicobacter pylori*. Also,

inflammatory intestinal diseases like Crohn's disease and Allergy, ulcerative colitis irritable bowel syndrome, diarrhea, hypercholesterolemia and others; all such findings have been supported by several studied demonstrating improved results using fermented food containing probiotics (Saikali *et al.*, 2004, Sheth, and Garcia-Tsao, 2008, Reid *et al.*, 2001, Kalliomäki *et al.*, 2001, Felley *et al.*, 2001, Furrie *et al.*, 2005, Prantera *et al.*, 2002, and Bibiloni *et al.*, 2005, Fanigliulo *et al.*, 2006, Allen *et al.*, 2010 and Sudha *et al.*, 2009). Probiotics have antidiabetic and antihypertensive

properties by modulating lipid profile and insulin resistance.. Oat is shown as a suitable substrate for various types of lactic acid bacteria and functional fermented products could be improved by the production of oat-based milk or cow-oat milk mixtures (FAO/WHO, 2002; Fernandez-Garcia *et al.*, 1998; Bekers *et al.*, St-Onge *et al.*, 2001, Martensson *et al.*, 2001 and Lye *et al.*, 2009).

Therefore, the recent study aimed to detect the probable anti-diabetic and hypolipidemic effects of unfermented and fermented oat milk compared to cow milk in alloxan-induced diabetic rats. As well as, their abilities to activate the growth of the health-promoting intestinal microflora, with a particular stress on Bifidobacteria and lactic acid bacteria genus.

### MATERIALS AND METHODS

Fresh cow's milk was obtained from Faculty of Agriculture, Cairo University, Cairo, Egypt. Skim milk powder (SMP) (97% DM). Dried whey protein concentrate (DWPC) was purchased from Mullins Whey Company, USA origin. The chemical composition of DWPC was 95.23, 2.74, 87.21 and 0.25 % for dry matter, ash, protein and acidity contents, respectively. Oat flakes purchased from local market.  $\alpha$ -amylase obtained Sigma Aldrich which had an activity of 2000 IU in a powder form.

#### Bacterial starter cultures

The bacterial culture used in this study, ABT-3 DIP 50u consists of *Lactobacillus acidophilus*,

*Streptococcus thermophilus* and *Bifidobacterium bifidum* obtained from Chr. Hansens Laboratories., Denmark and prepared by adding 1.2% of lyophilized cell culture into 11% sterilized reconstituted skim milk powder and incubated at 39°C for 5 hr before 24 hr.

**Preparation of oat milk sample:**

Oat milk was prepared by Deswal et al., (2014).

**Making of fermented milk samples:**

Three experimental fermented milk samples, (labeled as FCM, FOM and FOM+DWPC) were made from cow milk fortified with 2% skim milk powder, oat milk and oat milk fortified with 2% dried whey protein concentrate, respectively. All milk samples were heated at 90°C for 10 min and subsequently cooled to 40°C and inoculated with 3% ABT-3 starter culture (*Streptococcus thermophilus*, *Lactobacillus acidophilus* and *Bifidobacterium bifidum*). All samples were aseptically transferred into 100 ml plastic containers. Inoculated cow milk samples were incubated at 39°C till coagulation (pH 4.6) then cooled to 4°C. However, different inoculated oat milk samples were incubated at 39°C for 16 hrs. The resulting fermented samples were stored at 4°C for 21 days. Samples were taken when fresh, and after 3, 7, 14 and 21 days at 5 °C for analyses.

**Evaluation of the biological attributes of different unfermented and fermented milk types:**

**Animal, housing and diets:**

Forty-two male Albino rats weighing about 150±20 g were obtained from ARC. The animal groups were filtered under atmospheric condition. Pathogen-free water and air, and maintained at a temperature between 20-25°C for

8weeks with a 12 h light/dark cycle and light cycle (8-20 h) and relative humidity of 50%. The animals acclimatized for one week as an adaptation period. The animals were randomly divided into 2 groups. The first group of rats control (-) (6 rats) was fed on standard diet, while the second group (42 rats) were fast 24 hours, injected with prepared alloxan using citrate buffer 0.1M (pH = 4.6) as vehicle, at a dose of 150 mg alloxan/kg body weight (Szkudelski, 2001).At the third day, alloxan injection was carried out, blood glucose was examined and animals with glucose concentration higher than 200 mg/dl were considered as diabetic rats. Then rats were divided into six groups and treated by different unfermented and fermented milk types by epigastric tube as seen in Table(1) for 4 weeks. The rats were weighed weekly and at the end of the experimental feeding period, the animals were fasted overnight, anesthetized with ether and sacrificed for analysis.

**The followed steps mentioned were done in 6 rats after 4 weeks of treatment in each group:**

- 1- Fasting animals for 12 hours, 2- samples of blood were withdrawn from orbital plexus venous, 3- Samples of blood were allowed to clot, 4- serum of blood was obtained by centrifugation at 3500 rpm for 10 min at 4°C., 5- freezing the serum at -18°C until analyzed, 6- anesthetizing the animals with ether and sacrificed, 7- quickly dissecting the animals to excise the liver, kidney, testes, heart, and spleen and 8-weighing these organs and keeping them in 10% formaldehyde until histological investigations (Schermer and Chermner, (1967)

**Table 1. Experimental diets of rats used in the biological study.**

Groups	Experimental diets
Control (-)	basal diet (-)
Control (+)	*Alloxan (control +) + Standard diet
CM	* Alloxan + Standard diet + (5ml cow milk by epi gastric tube)
FCM	* Alloxan + Standard diet + (5 ml fermented cow milk by epi gastric tube)
OM	* Alloxan + Standard diet + (5ml oat milk by epi gastric tube)
FOM	* Alloxan + Standard diet + (5 ml fermented oat milk by epi gastric tube)
FOM+WP	* Alloxan + Standard diet + (5 ml fermented oat milk and whey protein by epi gastric tube)

\*intraperitoneal injection

**Biological Determination:**

Biological evaluation of the various examined diets according to Chapman et al., (1959).

**Serum biochemical Analyses:**

Blood glucose level was estimated as described by . Serum total cholesterol was determined colorimetrically at 500 nm. serum triglycerides was determined at 505 nm b, HDL-c(High-density lipoprotein) cholesterol was determined at 500 nm by, and LDL-c (Low-density lipoprotein) cholesterol were determined at 500 nm as described by (Allain et al. 1974, Trinder, Allain et al. 1974, 1969 Fassati and prencipe, 1982 and Wieland and Seidel, 1983).

**Determination of liver enzymes:**

The determination of liver enzymes described by Sherwin, (1984).

**Histopathology technique**

The tissues of the liver and kidney were fixed immediately after dissection in 10% neutral formalin for 24 h. according to Bancroft and Stevens, (1996).

**Fecal microbiological analysis:**

Rat faces were collected every week in sterile Petri dishes and immediately subjected to microbiological

analysis. Serial dilutions of feces were prepared according to Guerin- Danan and Andrieux, (1998) in pre-reduced liquid casein yeast medium. Total anaerobic bacteria and enterobacteria were counted in brain heart infusion agar (BHI) and Desoxycholate agar (DCA) medium, respectively. Lactobacilli count was determined using Man Rogosa and Sharpe (MRS) agar media according to De Man et al., (1960). Bifidobacteria spp. was counted on Beerens agar media according to Beerens, (1990).

**Statistical analysis:**

Statistical analysis was performed according to SAS, (1999).

**RESULTS AND DISCUSSION**

**Biological evaluation of alloxan-induced diabetic rats supplemented with different unfermented/fermented cow and oat milk samples:**

**General signs in the rats:**

No rats in among groups died during the experimental period (4 weeks) and some of the rats in groups exhibited abnormal signs throughout the test period. The weights of the various organs/body weight % of the

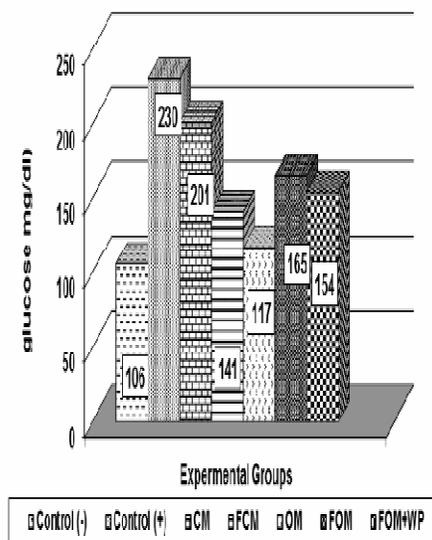
rats treated by different fermented cow and oat milk samples are shown in Table (2). The liver/body weight ratio significantly increased in rats injected by alloxan, compared with the control (-) group. These increases could be attributed to the accumulation of cholesterol in the liver (Jemai *et al.*, 2008). A significant decrease ( $P \leq 0.05$ ) could be observed in the weight of liver in the treatment groups (FOM+WP, FOM, FCM, OM, and CM), respectively compared with control (+) group. These results agree with those shown in Table (4), which resulted in higher significant of liver enzyme (ALT and AST) in control (+) group, and histopathology examination which illustrates liver damage.

**Table 2. Effect of different unfermented/fermented cow and oat milk samples on organs weight of alloxan-induced diabetic rats.**

Groups	Mean $\pm$ SE of Organs Weight	
	Liver	Pancreas
Control (-)	2.60 <sup>c</sup> $\pm$ 1.00	0.17 <sup>a</sup> $\pm$ 1.68
Control (+)	4.04 <sup>a</sup> $\pm$ 1.08	0.09 <sup>b</sup> $\pm$ 0.65
CM	3.58 <sup>b</sup> $\pm$ 1.64	0.14 <sup>ab</sup> $\pm$ 1.42
FCM	2.98 <sup>c</sup> $\pm$ 1.75	0.17 <sup>a</sup> $\pm$ 1.72
OM	3.02 <sup>c</sup> $\pm$ 0.72	0.17 <sup>a</sup> $\pm$ 1.75
FOM	2.86 <sup>c</sup> $\pm$ 1.51	0.13 <sup>ab</sup> $\pm$ 1.06
FOM+WP	2.74 <sup>c</sup> $\pm$ 1.66	0.14 <sup>ab</sup> $\pm$ 1.50

Data are presented as means  $\pm$  SE (n=6). Data in a column with different superscript letters are statistically different ( $P \leq 0.05$ ).

On the contrary, the pancreas /body weight ratio significantly decreased in rats injected by alloxan compared with the control (-) group, after feeding on all groups by different fermented cow and oat milk samples. It could also be observed an increase in the pancreas /body weight ratio in the treatment groups (OM , FCM, FOM+WP, CM and FOM), respectively, compared with control (+) group. These results agree with the data shown in figure (1).



**Fig. 1. Serum blood glucose concentration (mg/dl) in alloxan-induced diabetic rats treated with different unfermented and fermented milk types.**

**Biochemical Analyses:**

**Serum biochemical Analyses:**

The effects of feeding fermented cow milk and fermented oat milk on glucose level in rats are shown in (Fig.1). It could be seen highly increase ( $P \leq 0.05$ ) in the level of glucose (230 mg/dl) of control (+) group, treated with a single dose of alloxan, compared to control (-) group (106 mg/dl), which agrees with Radhika *et al.*, (2011). After four weeks of administration of OM, FCM, FOM + WP, FOM and CM reduce the level of glucose to 117, 141, 154, 156 and 201 mg/dl, respectively. The highest decrease in glucose concentration was observed in the animal groups treated by alloxan and fed with (OM) and (FCM). Little concentrations of  $\beta$ -glucans in oats) was. The viscosity of  $\beta$  -d-glucan plays an important role in post-prandial hyperglycemia and insulin responses (Tosh, 2013, Jenkins *et al.*, 2002 and Tomimatsu and Horie, 2005; Shori & Baba, 2011; Regand *et al.* and Dong *et al.* 2011; Batista *et al.*, 2015 and Pei *et al.*, 2017). Certain chain fatty acids in fermented milk have anti-diabetic proved the hypoglycemic effect of yogurt. This was attributed to its inhibitory effect on  $\alpha$ -glucosidase in milk.

Insulin deficiency as a result of diabetes leads to abnormalities in lipid metabolism (Arkkila *et al.*, 2001 Balku *et al.*, 2004). Alloxan-induced diabetic rats in the present studies showed a markedly increase  $P < 0.05$  in total triglycerides (TG) ,low-density lipoprotein (LDL-c) and total cholesterol (TC) of the diabetic group were detected, compared with control (-) group. While HDL-c decreased significantly  $P < 0.05$  as compared to control (-) group (Table 3). This increase in TG might be due to the shortage of insulin under diabetic condition. The obtained result came in agreement with those of Arkkila *et al.* 2001, Hassan & Emam, 2012 and Isa *et al.*, (2013). These results are in agreement with previous reports (Hassan & Emam, 2012; and Isa *et al.*, 2013) and in the data presented in figure (1).

**Table 3. Effect of different unfermented/fermented cow and oat milk samples on serum Lipid Profile (mg/dL) of alloxan-induced diabetic rats**

Groups	Parameters			
	TC (mg/dL)	LDL-c (mg/dL)	HDL-c (mg/dL)	TG (mg/dL)
Control (-)	93.67 <sup>cd</sup> $\pm$ 1.37	40.33 <sup>c</sup> $\pm$ 0.07	52.00 <sup>b</sup> $\pm$ 1.40	49.67 <sup>bc</sup> $\pm$ 0.06
Control (+)	155.33 <sup>a</sup> $\pm$ 1.37	76.00 <sup>a</sup> $\pm$ 1.22	27.00 <sup>d</sup> $\pm$ 0.71	89.33 <sup>a</sup> $\pm$ 0.14
CM	105.00 <sup>bc</sup> $\pm$ 0.01	55.00 <sup>bc</sup> $\pm$ 0.01	38.33 <sup>c</sup> $\pm$ 1.28	60.67 <sup>b</sup> $\pm$ 1.04
FCM	96.66 <sup>cd</sup> $\pm$ 0.94	47.67 <sup>c</sup> $\pm$ 0.42	68.00 <sup>b</sup> $\pm$ 1.39	35.00 <sup>d</sup> $\pm$ 1.40
OM	90.33 <sup>d</sup> $\pm$ 1.33	41.33 <sup>c</sup> $\pm$ 0.15	60.33 <sup>a</sup> $\pm$ 0.12	32.33 <sup>d</sup> $\pm$ 1.26
FOM	115.33 <sup>b</sup> $\pm$ 0.71	66.33 <sup>ab</sup> $\pm$ 0.54	45.33 <sup>bc</sup> $\pm$ 0.98	50.00 <sup>bc</sup> $\pm$ 0.31
FOM+WP	100.33 <sup>cd</sup> $\pm$ 1.41	56.33 <sup>bc</sup> $\pm$ 1.14	48.67 <sup>b</sup> $\pm$ 1.01	54.33 <sup>b</sup> $\pm$ 0.43

Data are presented as means  $\pm$  SE (n=6). Data in a column with different superscript letters are statistically different ( $P \leq 0.05$ ).

LDL-C: Low density lipoproteins cholesterol; LDL-C: Serum low density lipoproteins cholesterol; TC: Serum total cholesterol; TG: Serum triglyceride.

The present study showed that consumption of different unfermented and fermented milk types reversed the effects of alloxan by reducing the serum levels of TC, TG and LDL-c as compared with control (+) group and this was associated with a significant increase  $p < 0.05$  in HDL-c in these groups and therefore, reducing the risk for atherosclerosis. But, the highest significantly  $p < 0.05$

decreased recorded at the groups affected by alloxan and fed with (OM) and (FCM) respectively.

The above hypolipidemic activity of oat milk might be due to the presence of  $\beta$ -glucan, which inhibits the lipid absorption can enhance fecal cholesterol and bile acid excretion (Tiwari and Cummins, 2011). In addition,  $\beta$ -glucan suppresses the insulin secretion and thus halt the endogenous cholesterol synthesis. Furthermore, the lipid and protein contents in oat might also be responsible for the cholesterol-lowering activity. It could also be suggested that lipid and protein present in the oat could contribute to the hypocholesterolemic activity (Othman *et al.*, 2011, Gunness and Gidley, 2014 and Othman *et al.*, 2011).

The fermented milk administration resulted in an evident decrease of TG, TC, LDL-C, and increase of HDL-C level, indicating that fermented milk alleviated the metabolic disorder of lipids. These results are consistent with previous studies. The cholesterol-lowering effect of probiotics up to a significant concentration of 22–33% and *Lactobacillus rhamnosus* GG also play an active role in lowering of cholesterol in mice as reported by (Pereira and Gibson, 2002, Liang and Shah, 2005, Begley *et al.*, 2006, Kim *et al.*, 2016).

**Table 4. Effect of different unfermented/fermented cow and oat milk samples on Liver enzymes (mg/dl) of alloxan-induced diabetic rats.**

Groups	Parameters	
	ALT(U/I)	AST(U/I)
Control (-)	26.33 <sup>b</sup> ±0.71	43.67 <sup>b</sup> ±0.71
Control (+)	48.33 <sup>a</sup> ±0.12	69.67 <sup>a</sup> ±0.16
CM	38.00 <sup>ab</sup> ±0.46	48.67 <sup>b</sup> ±1.41
FCM	35.00 <sup>b</sup> ±1.22	51.00 <sup>b</sup> ±0.71
OM	36.00 <sup>b</sup> ±1.34	57.00 <sup>ab</sup> ±0.71
FOM	31.00 <sup>b</sup> ±1.02	49.67 <sup>b</sup> ±0.71
FOM+WP	29.00 <sup>b</sup> ±1.22	47.33 <sup>b</sup> ±1.36

Data are presented as means  $\pm$  SE ( $n=6$ ). Data in the same column with different superscript letters are statistically different ( $P \leq 0.05$ ).

Results in Table (4) indicated a remarkable decrease ( $P \leq 0.05$ ) in serum ALT activity for all groups injected by alloxan and fed on different unfermented and fermented milk types as compared with the control group (-) 26.33 U/I, but (FOM + WP, FOM, FCM, and OM) groups recorded high significantly decreased ( $P < 0.05$ ), respectively as compared with the control (+) group this results are consistent with Tameda *et al.*, (2005). However, ALT is more specific for liver damage than AST. And AST recorded a significant decreased in all groups injected by alloxan and fed on different unfermented and fermented milk types as compared with the control group (+) (69.67U/I) in the same Table (4). The death of hepatocytes liver usually results in the leakage of the enzymes in the affected tissue into the blood stream (Obi *et al.*, 2001).

The increase in the activities of serum AST and ALT indicated that diabetes may have induced hepatic dysfunction, that liver cells were necrotized in the diabetic

patient. noted that whey protein could decrease increased levels of AST and ALT in D-galactosamine-induced hepatitis in rats (Larcan *et al.*, 1979 and Villani *et al.*, 2005; Kume *et al.* 2006 and Pei *et al.*, 2017).

## 2. Microbiological analysis of faces:

Since fecal flora populations reflect the microflora of the intestinal colon (Cummings and Macfarlane, 1991), therefore, rat's faces were collected and analyzed for flora analysis. As shown in Table (5), it could be noticed that, no significant differences in fecal total anaerobes and fecal enterobacterial counts were observed for rates feed on the basal diet (negative and positive control), unfermented cow and oat milk samples over the experimental period.

While, there were noticeable decreases in the fecal total anaerobes and fecal enterobacterial counts of rates received different unfermented and fermented milk types (FCM, FOM, and FOM+WP) through the first week and up to the end of the feeding period.

No significant differences in fecal lactobacilli counts of rats fed on the basal diet (negative and positive control) could be observed in unfermented cow and oat milk samples along the experimental period. It could also be seen that the rats received fermented oat milk fortified with whey protein had the highest count of fecal lactobacilli followed by group received unfortified fermented oat and cow milk samples along the experimental period respectively. The feces samples before the feeding trials did not exhibit the presence of bifidobacteria in case of the two control groups and that groups received unfermented cow and oat milk samples over the experimental period.

A significant increase in fecal bifidobacteria of rats receiving different fermented oat milk samples (FOM and FOM+WP) compared with rats received fermented cow milk. This is might be due to the high content of soluble and non-soluble fiber which makes oat a useful product to use in the prevention of different diseases, especially, those affecting the colon. B-glucans, the most prevalent oat soluble fiber, have prebiotic activity. B-glucans are seen to be able to stimulate the growth of the health-promoting intestinal microflora, with a particular effect on lactic acid bacteria and *Bifidobacteria* genus (Mattila-Sandholm *et al.*, 2002; Roberfroid, 2002, Angelov *et al.*, 2006 and Sadiq-Butt *et al.*, 2008).

Generally, feeding the rats on fermented oat milk fortified with whey protein led to stimulating the viability of both fecal lactobacilli and bifidobacteria in the intestinal colon compared with feeding on unfortified fermented oat milk. This is might be due to the whey protein to stimulate the probiotic bacteria in the intestinal tract. Whey proteins are rich in sulfur-containing amino acids, which are liberated during heat treatment. These amino acids containing sulfur reduce oxidative potential and favor the environment for the growth of probiotics (Mc-Comas and Gilliland, 2003).

**Table 5. Effect of feeding different unfermented and fermented milk types on the fecal microorganisms counts (log cfu/ml) in alloxan-induced diabetic rat's faces**

Treatment	Feeding period (week)				
	Initial count	1	2	3	4
<b>Fecal total anaerobes</b>					
Negative control	9.65 <sup>Aa</sup> ±0.70	9.74 <sup>Aa</sup> ±0.34	9.81 <sup>Aa</sup> ±0.65	9.85 <sup>Aa</sup> ±0.70	9.87 <sup>Aa</sup> ±0.12
Positive control	9.51 <sup>Aa</sup> ±0.53	9.73 <sup>Aa</sup> ±0.62	9.86 <sup>Aa</sup> ±0.45	9.90 <sup>Aa</sup> ±0.54	9.94 <sup>Aa</sup> ±0.43
CM	9.63 <sup>Aa</sup> ±0.48	9.60 <sup>Aa</sup> ±0.30	9.57 <sup>Aa</sup> ±0.35	9.58 <sup>Aa</sup> ±0.30	9.65 <sup>Aa</sup> ±0.61
OM	9.60 <sup>Aa</sup> ±0.19	9.54 <sup>Aa</sup> ±0.43	9.31 <sup>Aab</sup> ±0.60	9.11 <sup>Bb</sup> ±0.45	8.94 <sup>Bb</sup> ±0.46
FCM	9.52 <sup>Aa</sup> ±0.43	8.93 <sup>Bb</sup> ±0.47	8.34 <sup>Bc</sup> ±0.11	7.81 <sup>Cd</sup> ±0.20	7.21 <sup>Ce</sup> ±0.15
FOM	9.55 <sup>Aa</sup> ±0.36	8.90 <sup>Bb</sup> ±0.11	8.14 <sup>Bc</sup> ±0.33	7.64 <sup>Cd</sup> ±0.10	7.00 <sup>Ce</sup> ±0.10
FOM+WP	9.49 <sup>Aa</sup> ±0.61	8.91 <sup>Bb</sup> ±0.38	8.03 <sup>Bc</sup> ±0.40	7.59 <sup>Cd</sup> ±0.08	6.95 <sup>Ce</sup> ±0.22
<b>Fecal enterobacteria</b>					
Negative control	9.12 <sup>Ac</sup> ±0.44	9.57 <sup>Ab</sup> ±0.45	9.82 <sup>Aa</sup> ±0.40	9.87 <sup>Aa</sup> ±0.40	9.90 <sup>Aa</sup> ±0.56
Positive control	9.20 <sup>Ac</sup> ±0.50	9.61 <sup>Ab</sup> ±0.38	9.92 <sup>Aa</sup> ±0.30	9.98 <sup>Aa</sup> ±0.50	10.11 <sup>Aa</sup> ±0.60
CM	9.23 <sup>Aa</sup> ±0.36	9.31 <sup>Aa</sup> ±0.40	9.33 <sup>Aa</sup> ±0.52	9.35 <sup>Aa</sup> ±0.28	9.38 <sup>Aa</sup> ±0.34
OM	9.21 <sup>Aa</sup> ±0.50	9.30 <sup>Aa</sup> ±0.41	9.34 <sup>Aa</sup> ±0.20	9.36 <sup>Aa</sup> ±0.30	9.38 <sup>Aa</sup> ±0.37
FCM	9.16 <sup>Aa</sup> ±0.36	8.71 <sup>Bb</sup> ±0.30	7.90 <sup>Bc</sup> ±0.14	7.11 <sup>Bd</sup> ±0.10	6.42 <sup>Bc</sup> ±0.40
FOM	9.21 <sup>Aa</sup> ±0.28	8.64 <sup>Bb</sup> ±0.18	7.20 <sup>Bc</sup> ±0.40	6.70 <sup>Cd</sup> ±0.15	6.26 <sup>Ce</sup> ±0.20
FOM+WP	9.15 <sup>Aa</sup> ±0.35	8.57 <sup>Bb</sup> ±0.50	7.11 <sup>Bc</sup> ±0.36	6.66 <sup>Cd</sup> ±0.10	6.10 <sup>Ce</sup> ±0.10
<b>Fecal lactobacilli</b>					
Negative control	7.04 <sup>Aa</sup> ±0.15	7.00 <sup>ABa</sup> ±0.01	6.98 <sup>Ca</sup> ±0.10	6.96 <sup>Da</sup> ±0.09	6.92 <sup>Ea</sup> ±0.15
Positive control	6.96 <sup>Aa</sup> ±0.25	6.80 <sup>Ba</sup> ±0.09	6.66 <sup>Cb</sup> ±0.15	6.61 <sup>Db</sup> ±0.07	6.56 <sup>Eb</sup> ±0.50
CM	7.11 <sup>Ab</sup> ±0.48	7.27 <sup>Aa</sup> ±0.10	7.31 <sup>Ba</sup> ±0.31	7.35 <sup>Ca</sup> ±0.60	7.41 <sup>Da</sup> ±0.45
OM	7.27 <sup>Ab</sup> ±0.20	7.40 <sup>Aa</sup> ±0.40	7.44 <sup>Ba</sup> ±0.30	7.49 <sup>Ba</sup> ±0.18	7.52 <sup>Da</sup> ±0.55
FCM	7.19 <sup>Aac</sup> ±0.21	7.37 <sup>Ab</sup> ±0.56	7.40 <sup>Bb</sup> ±0.30	7.55 <sup>Bab</sup> ±0.14	7.91 <sup>Ca</sup> ±0.20
FOM	7.20 <sup>Ad</sup> ±0.80	7.66 <sup>Ac</sup> ±0.25	7.81 <sup>Ab</sup> ±0.20	7.93 <sup>Aab</sup> ±0.44	8.14 <sup>Ba</sup> ±0.32
FOM+WP	7.21 <sup>Ac</sup> ±0.20	7.70 <sup>Ad</sup> ±0.08	7.82 <sup>Ac</sup> ±0.25	8.11 <sup>Ab</sup> ±0.70	8.38 <sup>Aa</sup> ±0.10
<b>Fecal bifidobacteria</b>					
Negative control	ND	ND	ND	ND	ND
Positive control	ND	ND	ND	ND	ND
CM	ND	ND	ND	ND	ND
OM	ND	ND	ND	ND	ND
FCM	ND	4.31 <sup>Cd</sup> ±0.01	4.68 <sup>Cc</sup> ±0.09	5.03 <sup>Cb</sup> ±0.15	5.28 <sup>Ba</sup> ±0.09
FOM	ND	5.06 <sup>Bd</sup> ±0.01	5.42 <sup>Bc</sup> ±0.12	6.17 <sup>Bb</sup> ±0.20	6.20 <sup>Aa</sup> ±0.06
FOM+WP	ND	5.21 <sup>Ad</sup> ±0.05	5.63 <sup>Ac</sup> ±0.10	6.21 <sup>Ab</sup> ±0.15	6.35 <sup>Aa</sup> ±0.08

<sup>abc</sup> Values (mean ± SE; n=6) with different superscripts in a row are significantly different at the level of P < 0.05.

<sup>ABC</sup> Values (mean ± SE; n=6) with different superscripts in a column are significantly different at the level of P < 0.05.

**Histopathological Examination:**

Liver and pancreas were examined and the photomicrographs are illustrated in Figs. (2 and 3). Alloxan is diabetogenic chemical, pathological effects and toxic glucose analogs that preferentially accumulates in pancreatic beta cells via the glucose transporter 2. The presence of intracellular thiols, especially, glutathione, alloxan generates reactive oxygen species in a cyclic redox reaction with its reduction product, dialuric acid. Auto-oxidation of dialuric acid generates superoxide radicals, hydrogen peroxide and, in a final iron-catalyzed reaction step, hydroxyl radicals. (Lenzen, 2008, and Minaiyan *et al.*, 2011).

Histopathological examination of the liver sections from control - (normal rats fed on commercial diet only) showing no histopathological alteration and the normal histological structure of the central vein and surrounding hepatocytes in the parenchyma were recorded in (Fig.2A). In contrast, in the control (+) group (normal rats fed on commercial diet + injection with alloxan) showing edema in the portal area, few inflammatory cells infiltration with dilatation and hyperplasia in the bile ducts as well as dilatation in the portal vein (Fig.2B).

There were dilatation and hyperplasia in the bile ducts in the portal area associated with dilatation in the central vein at the animal's group affected by alloxan plus fed on (CM) as seen in (Fig. 2C). While, in the animal's

group affected by alloxan plus fed on (FCM) newly formed bile ductules were detected in the portal area (Fig. 2D).

On other hand, the liver of injected rat by alloxan fed on (OM) showed congestion in the central vein as seen in (Fig.2E). Inflammatory cells infiltration was observed in a focal manner in the portal area associated with congestion in the portal vein at the animal's group affected by alloxan plus fed on (FOM) in (Fig.2F). In contrast, there was no histopathological alteration in the hepatic parenchyma as recorded in (Fig.2G) at the liver of injected rat by alloxan plus fed on (FOM+WP).

**Pancreas**

The rats fed on commercial diet only (control (-)) showing no histopathological alteration and the normal histological structure of the islands of Langerhans cells as the endocrine portion as well as the acini and duct system as exocrine one was recorded in (Fig.3A). Meanwhile, animals fed on commercial diet plus injection with alloxan (control (+)) observed atrophy with the absence of most of the cells of the islands of Langerhans in all of the lobules (Fig.3B). Furthermore, Pancreas of animals affected by alloxan plus fed on (CM) the cells of the islands of Langerhans were histologically intact while the blood vessels were dilated as well as the ducts (Fig.3C). In contrast, animals affected by alloxan and fed (OM) and (FCM) as seen in Fig.(3E and D) respectively, showing no

histopathological alteration and the islands of Langerhans cells were in normal size as well as the surrounding acini and ducts. There was atrophy in the islands of Langerhans cells associated with eosinophilic casts formation in the

duct lumen in the rats affected by alloxan plus fed on (FOM) and (FOM+WP) as recorded in Fig. (3F and G).

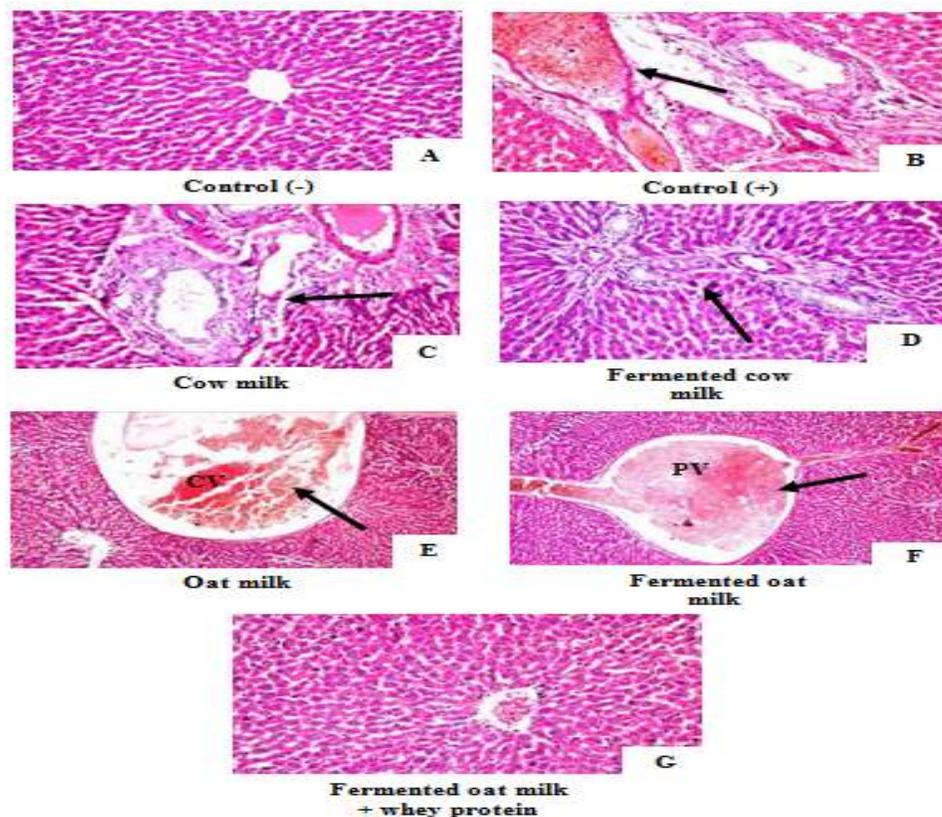


Fig. 2. Photomicrograph of sections of liver of different rats groups, stained with H & E, X 400 Liver

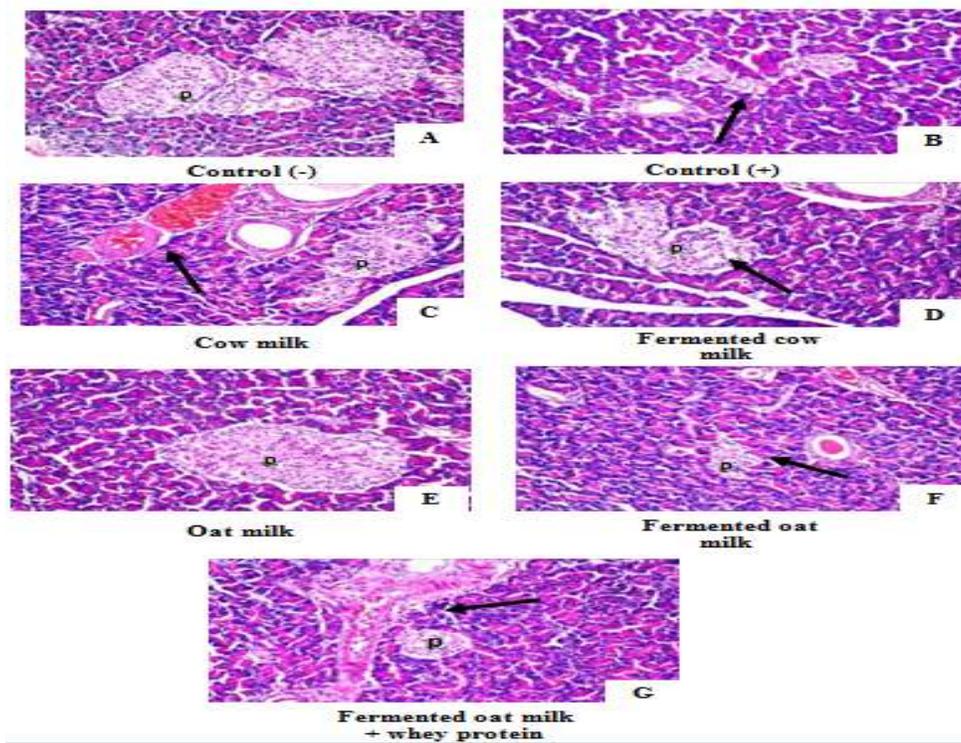


Fig. 3. Photomicrograph of Sections of Pancreas of different rats groups, stained with H & E, X 400.

## CONCLUSION

Based on the aforementioned results, it could be concluded that consumption of oat milk and fermented oat milk products with "health-enhancing" properties as symbiotic and probiotics products play a major role in prevention as well as in treating some health problems such as diabetes, hypercholesterolemia, and hyperlipidemia. Feeding the rats on fermented oat milk fortified with whey protein has a better effect on the liver and led to stimulating the viability of both fecal lactobacilli and bifidobacteria in the intestinal colon.

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## الخصائص البيولوجية لحليب الشوفان غير المتخمّر والمتخمّر مقارنة بحليب البقر في الفئران المصابة بمرض السكري والتي يسببها الألوكسان

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يهدف هذا العمل إلى تقييم التأثير العلاجي لحليب البقر والشوفان غير المتخمّر والمتخمّر (FOM، CM، FCM، OM) وحليب الشوفان المتخمّر والمدعم ب 2٪ من مركز بروتين الشروش (FOM + WP) على الفئران المصابة بالسكري. حيث تم استخدام الألوكسان Alloxan للتحث على الإصابة بمرض السكري وزيادة دهون الدم في الفئران. وقد تم تقسيم الفئران بشكل عشوائي إلى مجموعتين رئيسيتين. مجموعة كترول سالية يتم تغذيتها على نظام غذائي قياسي، في حين تم تقسيم المجموعة الثانية (42 فئران مصاب بالسكري) إلى ست مجموعات (6 فئران لكل منهما)، وقد تمت المعاملة بواسطة أنواع مختلفة من الحليب غير المتخمّر والمتخمّر لمدة 4 أسابيع. وقد أظهرت النتائج أن جميع المعاملات في نهاية فترة التغذية أدت إلى انخفاض مستوى الجلوكوز ومستويات الكوليسترول الكلي، الدهون الثلاثية والليبيدات منخفضة الكثافة في الدم مقارنة بمجموعة الكترول الموجبة (+) وارتبط ذلك بزيادة ملحوظة ( $P < 0.05$ ) في الليبوبروتين مرتفع الكثافة HDL-C في هذه المجموعات. واتضح أن أعلى انخفاض ملحوظ ( $P < 0.05$ ) مسجل في المجموعات التي تم تغذيتها على (OM) و (FCM)، على التوالي بينما انخفض نشاط إنزيمات الكبد بشكل ملحوظ ( $P < 0.05$ ) في مجموعات FOM و FOM + WP و FCM و OM على التوالي. وقد أدى إطعام الفئران على حليب الشوفان المتخمّر المدعم بمركز بروتين الشروش إلى انخفاض في العدد الكلي للبيكتيريا المعوية الضارة (fecal total anaerobes and fecal enterobacterial counts) بينما حفز نمو البكتيريا النافعة (*Lactobacilli* and *Bifidobacteria*) في القولون المعوي مقارنة بالمعاملات الأخرى. أظهر نتائج الفحص الهستوباثولوجي لأقسام الكبد عدم وجود تغيرات هستوباثولوجية في الخلايا البرنشيمية للكبد في مجموعة (FOM + WP) وكذلك لم تُظهر مجموعات (OM) و (FCM) أي تغيير نسيجي، وكانت جزر خلايا لانجرهانس في الحجم الطبيعي وكذلك الحويصلات والقنوات المحيطة. في الختام. أدى المعاملة بحليب الشوفان إلى علاج ارتفاع السكر في الدم و فرط دهون الدم في الفئران المصابة بالسكري. وفي الوقت نفسه، فإن التغذية على حليب الشوفان المتخمّر المدعم ب بروتينات الشروش كان له تأثير أفضل على الكبد كذلك تأثير إيجابي على تحفيز نمو البكتيريا المفيدة في القولون.