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A REVIEW ON CAMEL MILK WITH HEPATOPROTECTIVE EFFECTS

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Abstract:

The largest solid organ in the human body is the liver. Basically, adults weigh about 3 pounds. The liver is important for the body's metabolic functions and the immune system. The survival of a person is not possible without a functioning liver. Liver disease is a major health problem worldwide, which is why there is a need to develop new molecules that can help combat or prevent such diseases. The toxic chemicals called Hepatotoxins damage the liver. It can be a side effect of drugs or found naturally as microcystin or in the laboratory. Current medicine includes reliable hepatoprotective agents for preventing and treating a drug-induced liver injury. there is not. Nature has provided us with many things. Among them, cow's milk is the most functional natural liquid, because it is abundantly produced and has many nutritional values. Camel's milk is a blessing to mankind. Camel milk has been used as a medicinal drink by cultures in the Middle East, Asia, and Africa for hundreds of years. Camel milk is a great substitute for breast milk because it does not contain beta-lactoglobulin. Numerous research results have demonstrated that people with lactase deficiency can easily digest camel milk. It contains small disease-fighting immunoglobulins, allowing antigens to penetrate and the immune system to become more effective. The current review aims to compile data on camel milk with hepatoprotective activity in different models of hepatotoxicity.

Keywords: *Liver disease, Hepatotoxin, hepatoprotective, camel milk, hepatotoxicity.*

1. Background:

The Greek word for liver is hepar, so medical terms referring to the liver often begin with hepato or hepatic. The liver plays an important role in metabolism, secretion, and storage, and we rely on it to store, secrete, purify, and purify many common proteins, nutrients, and chemicals, making it the body's which is sometimes called "grand chemical factory." Eliminates toxins and unwanted substances from the body. Bile secreted by the liver plays an important role, among other things, in digestion. (Sai et al., 2017). The liver performs the body's typical metabolic homeostasis, as well as biotransformation, detoxification, and elimination of many endogenous and exogenous compounds, as well as pharmaceuticals and environmental chemicals. Liver disease is a major public health threat and a global problem. The World Health Organization estimates that chronic diseases account for 46% of morbidity and 59% of deaths worldwide, and the treatment of liver disease continues to be a prominent concern of modern medicine. 2017). Drug-induced hepatotoxicity is a major cause of iatrogenic disease. Consider 1 in 600 to 1 in 3500 of all hospital admissions.

Below are some of the commonly observed liver diseases:

- Necrosis
- Liver cirrhosis
- Hepatitis - can be viral, toxic, or deficient.
- liver failure - acute or chronic;
- Liver disease due to metabolic dysfunction.

In general, disorders related to fat (steatosis) and bilirubin metabolism (jaundice) are very frequently observed.

- Disorders related to lipid metabolism: Fatty liver.
- Disorders related to bilirubin metabolism: This may be due to jaundice or mechanism of action and etiology.

a) Hemolytic /prehepatic jaundice
b) Obstructive (postoperative/cholestatic jaundice)
c) Hepatogenic/hepatic jaundice/cholestasis
d) Hereditary jaundice or pure cholestasis
e) chemical/drug-induced hepatotoxicity: Common hepatitis, jaundice, carcinogenesis

2. Hepatotoxin:

The toxic chemicals that damage the liver are called Hepatotoxins. Toxic liver damage caused by drugs and chemicals can mimic virtually any type of modern disease. Metabolism of hepatotoxins by cytochrome P-450 enzymes can also be a sign of addiction. I have. Therefore, enzyme inhibitors have been shown to limit hepatotoxin-related liver dysfunction. Furthermore, there is substantial evidence that mitochondrial permeability transition (MPT) is involved in hepatocyte reactive oxygen species (ROS) damage, and new findings reduce cellular damage by blocking MPT initiation. offers a completely unique therapeutic approach Therefore, oxidative stress and lipid peroxidation are influential factors leading to hepatotoxin-related liver injury (Sai et al., 2017).

2.1 Hepatotoxins and their mechanism of hepatotoxicity:

Carbon tetrachloride:

The hepatotoxicity of CCl4 is due to the formation of the highly reactive free radical trichloromethyl in the body that attacks polyunsaturated fatty acids in the endoplasmic reticulum membrane. Carbon tetrachloride poisoning rapidly stops the movement of large amounts of triglycerides from the liver to the plasma, causing fatty liver (Richard O. Recknagel., 1967). If the damage is severe, liver enzymes are abnormally increased, followed by hepatocellular necrosis. During acute and chronic CCl4-induced hepatotoxicity,

there is an influx of monocytes into the liver, resulting in increased reactive oxygen species (ROS) production, increased hepatic Kupffer cell leukotriene production, and cytoprotective prostanoids. and cytotoxic prostanoids. (Alric L et al., 2000).

Paracetamol: Paracetamol is metabolically induced by cytochrome P450s to become responsive metabolites that covalently bind to proteins (Mitchell et al., 1973). et al., 2005). Although considered safe in therapeutic doses, overdose causes fatal centrilobular liver necrosisVarious mechanisms leading to acetaminophen toxicity include:

• Increased formation of superoxide anions that cause lipid peroxidation (oxidative stress) via the formation of hydrogen peroxide (Coles et al., 1988).
• Decreased glutathione concentration in centrilobular cells (Nakamura et al., 1997).

3. Camel Milk

This animal has the potential to be a versatile animal with great production capacity. Humans have used it for transportation, milk, meat, hides, and more. Camels produce more milk over a longer period of time than other cows in similarly harsh environments. His daily production of 3-10 kg during lactation from 12-18 months is common. Camel milk is one of the main components of the staple diet of pastoral communities, accounting for up to 30% of the annual caloric intake, and also a major source of essential nutrients and vitamin C (Farah., 1993). Camel milk has been reported to have medicinal properties (Yagil, 1982), suggesting that it contains protective proteins that play a role in enhancing immune defences. Camel milk is also rich in zinc. Rapidly dividing cells of the immune system are sensitive to zinc deficiency. The role of zinc in the development and maintenance of a normally functioning immune system is well known (Hansen et al., 1982). Camel milk exhibits hypoglycemic effects when administered as adjunctive therapy, which may be attributed to the presence of insulin/insulin-like proteins in it, and has beneficial effects in the treatment of diabetic patients. It is also used to treat food allergies, Crohn's disease and autism (Shabo and Yagil, 2005). Surprisingly, although numerous reviews have been conducted on several dairy animals around the world, the importance and uses of camel milk and its products have not been verified, so information in this area is scarce. (Ayele et al., 2014).

3.1. Nutritional Value

3.1.1 Milk protein

This animal has the potential to be a versatile animal with great production capacity. Humans have used it for transportation, milk, meat, hides, and more. Camels produce more milk over a longer period of time than other cows in similarly harsh environments. His daily production of 3-10 kg during lactation from 12-18 months is common. Camel milk is one of the main components of the staple diet of pastoral communities, accounting for up to 30% of the annual caloric intake, and also a major source of essential nutrients and vitamin C (Farah., 1993). Camel milk has been reported to have medicinal properties (Yagil, 1982), suggesting that it contains protective proteins that play a role in enhancing immune defences. The zinc content is rich in camel milk. Rapidly dividing cells of the immune system are sensitive to zinc deficiency. The role of zinc in the development and maintenance of a normally functioning immune system is well known (Hansen et al., 1982). Camel milk exhibits hypoglycaemic effects when administered as adjunctive therapy, which may be attributed to the presence of insulin/insulin-like proteins in it, and has beneficial effects in the treatment of diabetic patients. It is also used to treat food allergies, Crohn's disease, and autism (Shabo and Yagil, 2005). Surprisingly, although numerous reviews have been conducted on several dairy animals around the world, the importance and uses of camel milk and its products have not been verified, so information in this area is scarce. (Ayele et al., 2014).

3.1.2 Milk Lipids:

Fat is a key factor explaining the energy benefits of milk and, along with its technical performance, contributes significantly to its nutritional value. Milk fat globules typically range in diameter from less than 0.1 μm to approximately 18 μm (El-Zeini, 2006) and contain a triglyceride core surrounded by a native biological membrane. Milk fat globule membranes (MFGM) contain typical components of all biological membranes, including cholesterol, enzymes, glycoproteins and glycolipids (Fauquant et al., 2007). Mansson (2008) claims that lipids make up 30% of membranes and can be divided into the following categories:
Phospholipids (25%), Cerebrosides (3%), Cholesterol (2%). The remaining 70% of the membrane contains proteins. Fat globules with the most significant mean diameters are found in buffalo milk (8.7 μm), camel bedrock (2.99 μm), and goat milk (3.19 μm). High circulation of milk fat makes small fat globules (SFGs) more accessible to lipolytic enzymes. This makes goat and camel milk more digestible for humans (D'Urso et al., 2008).

3.1.3 Milk Mineral Components

Milk is an important source of minerals, especially calcium, phosphorus, sodium, potassium, chloride, iodine, magnesium and small amounts of iron. The most mineral-rich compounds in milk are calcium and phosphorus, which are essential for bone growth and proper neonatal development. The high bioavailability of these minerals influences the unique nutritional value of milk. Camel milk is the richest source of these minerals (Al-Wabel, 2008). Different camel breeds have different amounts of minerals in their milk (Wangoh et al., 1998). The concentrations of Fe, Zn and Cu were therefore 1.00012, 2.00002 and 0.44004 mg/dl. Levels of trace elements or Fe, Zn, and Cu were significantly higher in camel milk than in cow's milk (Singh et al., 2006).

3.1.4 Milk Vitamins

Milk is a useful source of both water-soluble and fat-soluble vitamins. Camel milk is an exception due to its high vitamin C content. Camel milk contains 30 times more vitamin C than cow's milk and 6 times more than breast milk. This is very important in desert areas where fruits and vegetables are scarce. Therefore, camel milk is usually the only source of vitamin C in the diets of people living in these regions (Haddadin et al., 2008). The levels of vitamins A, E, and B1 in camel milk have been reported to be lower than in cow's milk. Cow's milk contains 99.6 ± 62.0 µg% β-carotene, which is not detected in camel milk. Vitamin C concentrations in early and late lactation camel milk were declared to be 5.26 ± 0.47 and 4.84 ± 0.20 mg%, respectively. The vitamin C content in camel milk is two to three times higher in camel milk than in cow's milk. Levels of vitamins A, E, and B1 were higher in camel colostrum than in mature camel milk. However, the vitamin C content remains higher in adult camel milk. The higher vitamin C content may also be attributed to the higher synthetic activity of the mammary gland tissue during early lactation, suggesting that lactation is (Stahl et al., 2006). Due to the vitamin C content, the low pH value stabilizes the milk and allows it to be stored for almost long periods and also has a very effective antioxidant properties. Therefore, the relatively high content of vitamin C in raw camel milk is of great nutritional importance. Camel milk may be another source of vitamin C under brassy environmental conditions in arid and semi-arid regions (Mal et al., 2007).

4. Camel milk with hepatoprotective effects:

The murine mouse model was used for CCL4-induced liver injury to study the effects of camel milk (Amjad Ali Khan and Mohammad A. Alzohairy., 2011). Their study showed that CCL4 administration caused severe acute liver injury in rats, which was reflected in sensational increases in serum ALT, AST, and ALP levels. Elevated serum levels of AST and ALT are thought to be responsible for damage to the structural integrity of the liver. The changes detected mainly included hepatocyte necrosis or apoptosis, fatty acid accumulation, inflammatory cell infiltration, and other histological manifestations. Pre- and post-treatment with camel milk can ameliorate CCL4-induced hepatotoxicity in rats, which is reflected in decreased serum aminotransferase activity. Pre-treatment with camel milk before CCL4 intoxication showed less increase in serum aminotransferase and alkaline phosphatase levels after CCL4 intoxication. Considering that various cases of acute toxic liver injury are activated by the formation of free radicals and furthermore caused by local inflammatory responses, the potent antioxidant and inflammatory effects of camel milk in these conditions are It appears to be protective. Therefore, the consumption of camel milk has some preventive effects and strengthens the immune system.

Liver fibrosis represents chronic wound healing after liver injury. (Hamed et al., 2017) evaluated the liver response to hepatotoxic drugs in previously liver-injured mice. First, in this study, CCl4 hepatotoxicity in mice was determined by changes in serum parameters. Liver studies were completed by estimating the activities of serum ALT, AST, ALP, LDH and ɤ-GT, enzymes generally present at high concentrations in the cytoplasm. Under these conditions of liver damage, these enzymes enter the bloodstream when the liver is damaged.

Results showed that administration of CCl4 induced a significant increase in enzyme levels in contrast to normal controls. CCl4-fed rats were found to have elevated levels of mainly aspartate aminotransferase and alanine aminotransferase. These most affect cell damage, permeability, and increased hepatocyte necrosis. Prophylactic treatment with camel milk was shown to attenuate the increases in AST and ALT serum activity induced by her CCl4 treatment in rats. This discovery suggested camel milk. Dare to protect liver tissue from CCl4 damage.

Administration of paracetamol to male rats increased the activity of serum enzyme levels GPT, GOT and ALP levels compared with normal rats, whereas rats treated with camel milk and paracetamol showed paracetamol-induced serum- We showed markers indicating an increase in GOT. GPT, ALP mirror prevented. (Fartosi et al., 2011) This study shows that paracetamol can induce significant changes in biochemical parameters and inhibit the function of antioxidant enzymes. Administering camel milk after exposure to paracetamol increases the risks associated with paracetamol. Therefore, camel milk may be beneficial in reducing acetaminophen toxicity.

Oral administration of ethanol at a dose of 0.5 g/100 g to rats biochemically revealed chronic damage induced in rat hepatocytes. Camel milk and camel urine affected significant reductions in serum enzymes that are indicators of liver disease. This demonstrates a specific hepatoprotective effect, surprisingly higher than with the reference drug (silymarin), especially in the case of ethanol-induced chronic toxicity. The hepatoprotective effect of camel milk and urine was It may be due to a possible chelating effect on toxins, especially alcohol, or due to the high levels of vitamins C, B2, E, magnesium and other trace elements found in camel milk and urine. Role It may be an antioxidant and has been shown to help prevent tissue damage from toxic substances. However, clinical studies are primarily needed to validate the potential effects of camel milk mixed with urine as a beneficial drink in preventing chronic hepatotoxicity (Ahmed et al., 2017).

In ethanol-induced hepatotoxicity, rats (Darwish et al., 2012) evaluated the potential prophylactic or therapeutic effect of camel milk. A significant increase in serum levels of liver enzymes (ALT, AST, ALP) was observed after 4 weeks of ethanol feeding. Increased toxic liver damage was accompanied by histopathological changes seen in liver sections from ethanol-treated rats. The aforementioned changes took the form of increased inflammatory cell infiltration, fat changes, collagen fibre growth, and necrotic damage.

Fatty liver (fatty liver) is an early stage of liver damage and is characterized by the accumulation of triglycerides in hepatocytes. Elevated serum triglyceride levels were found after alcohol consumption. This increase can be explained on the basis that alcohol assimilation induces a clear increase in the NADH/NAD+ ratio, boosting triglyceride synthesis and increasing the concentration of glycerol-3-phosphate, which accelerates its accumulation in the liver. increase. Alcohol also damages lipolysis and significantly elevates serum triglycerides. An increase in liver weight or body weight ratio was observed in the ethanol-treated group. This is further confirmation of fatty liver and is also associated with ethanol-induced liver fibrosis as shown by histopathological examination.

Feeding rats with camel's milk normalize liver enzymes and significantly improves liver function, as evidenced by a significant reduction in serum triglyceride levels (TG) and liver weight-to-body weight ratio. Furthermore, the results of the liver histopathological analysis were consistent with the biochemical findings and also showed decreased degeneration of some hepatocytes and decreased collagen accumulation after the camel milk feeding period. The above results demonstrate the hepatoprotective effect of camel milk.

Earlier studies, the effects of oral camel milk administration on poloxamer 407-induced hyper lipidemic Wistar rats (Zuberu et al., 2017). In this study, serum ALT and AST levels, but not ALP, were significantly increased in the hyper lipidemic control group compared with the normal control group. This is probably due to the damage inflicted on the liver as a result of the accumulation of triglycerides and other fats in hepatocytes. However, a significant decrease in ALT and AST levels was seen in the camel milk-fed group. rice field. The reversal may be to prevent the release of intracellular enzymes and enhance membrane-stabilizing activity. This is consistent with the widely held belief that serum transaminases return to normal levels after parenchymal healing and hepatocyte remodelling. Thus, indicating a hepatoprotective role of camel milk against P407-induced liver injury.

The studies of effects of extra virgin olive oil (EVOO) and camel milk (CM) on acetaminophen (APAP)-induced hepatotoxicity in mice. The mice used in this experiment were randomly divided into 6 groups with 6 mice per group. Groupings were controlled, EVOO, CM, APAP, EVOO + APAP, and CM + APAP. Mice in the APAP group were treated with a single dose of acetaminophen (500 mg/kg). EVOO and CM were administered to the prophylaxis group at the same doses as the toxicity group 28 days before his APAP administration.

Liver enzymes, lipid profile, malondialdehyde (MDA) and total antioxidant (TAC) activity were tested. Elevated levels of MDA and TAC were observed in the toxic group compared to the control group with elevated liver enzyme levels ($p<0.05$). EVOO and CM treatment resulted in a hepatoprotective role evidenced by marked reductions in serum liver enzymes and hepatic malondialdehyde, and concomitant increases in TAC compared with mice in the acetaminophen-treated group ($p < 0, 05$).

Histopathological examination revealed massive centrilobular necrosis and oedematous degeneration of hepatocytes in the APAP group. The above changes were reduced in the EVOO and CM pre-treatment groups. This study finally concluded that olive oil and camel milk had hepatoprotective effects against paracetamol-induced hepatotoxicity. Pre-treatment with EVOO and CM specifically reduced paracetamol hepatotoxicity in mice.

(Fahad Al-Hasham., 2009) investigated the protective effects of camel milk against aluminium-induced biochemical changes and oxidative stress in the liver and kidney of albino white rats. The rats used in this study were albino white male rats (230-250 g). The rats used in this experiment are divided into the following three groups. A control group was treated with normal saline, a group treated with AlCl3, and a group treated with camel milk + AlCl3. Each group consists of 10 rats.

The AlCl3-treated group of rats was orally administered 0.5 mg kg-1 AlCl3. Groups of rats treated with camel milk+ AlCl3 were given 1 ml of fresh camel milk 10 minutes before administration of oral AlCl3. All groups of rats were treated daily for 30 days.

Liver and kidney biochemical serum parameters were tested. Lipid peroxidation was determined by tissue concentrations of thiobarbiturate-reactive substances (TBARS) and hydrogen peroxide (HP), and oxidative stress status was determined by glutathione (GSH), superoxide, and glutathione (GSH) in treated rat kidney and liver. Absolute was measured by dismutase (SOD) and catalase (CAT) activity.

Oral administration of AlCl3 reportedly increased urea, creatinine, bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), lactate dehydrogenase (LDH), cholesterol, and triglycerides. Lloyd's serum levels have been shown to increase significantly. significant reductions in total protein and albumin have also been reported. Although rats were fed with camel milk before AlCl3, the above parameters were considered normal. Moreover, oral administration of AlCl3 reduced lipid peroxidation in the liver and kidney, as evidenced by marked increases in lipid peroxidation biomarkers (TBARS and HP) and marked decreases in GSH, SOD, and CAT activities. induced. Lipid peroxidation and oxidative stress parameters were normal in all rats treated with camel milk before the administration of AlCl3. Thus, it is clear that treatment with camel milk prior to AlCl3 exposure reduces AlCl3-associated hazards and protects kidneys and the liver from AlCl3 toxicity.

(Althnanin et al., 2013) focused on investigating the protective effects of camel milk against carbon tetrachloride (CCl4)-induced hepatotoxicity. Therefore, of the 24 rats, he divided them into 4 groups, each containing 6 of her rats and fed a standard diet. Rats in primary and secondary groups were injected intraperitoneally with paraffin oil and fed with water (control 1) or camel milk (control 2), respectively. His third and his fourth groups of rats were injected i/p with CCl4 and fed water or camel milk, respectively.

After 5 weeks (end of the experiment), biochemical and histopathological analyses were performed on blood and liver samples from rats. Results showed that CCl4 increased serum liver enzyme activity and several biochemical parameters, but these effects were prevented by treating rats with camel milk. Histopathological, a superior amount of mononuclear cell infiltrate, necrotic cells and few fibroblasts were observed in the livers of the CCl4-treated group. Finally, (Althnanin et al., 2013) concluded that camel milk treatment may have a protective effect against CCl4-induced liver injury in rats.

The investigation of the hepatoprotective activity of camel milk (CM) in a mouse model of acute (alcoholic liver disease (ALD)) and underlying processes at the gut microbiota and transcriptome levels (Ming et al., 2020). studied. Mice were divided into three groups: normal diet (NC); normal diet and ethanol (ET). A normal diet plus camel milk (CM) and ethanol (ET+CM). Serum biochemical indices and histological analysis revealed a reduction in hepatitis in the ET+CM group. Sequencing of 16S rRNA was also performed, showing that CM balanced the microbial community, increasing the proportion of Lactobacillus and decreasing the gut group of Bacteroidetes, Aristipes, and Rikenellaceae RC9. Comparative liver transcriptome analysis revealed 315 differentially expressed genes (DEGs) in the ET+CM and ET groups (150 upregulated and 165 downregulated). Enrichment analysis revealed that CM downregulated the expression of inflammation-related (ILB and CXCL1) genes in the IL-17 and tumour necrosis factor (TNF-α) signalling pathways. Their study reveals that CM modulates liver inflammation and attenuates intestinal microbial damage caused by acute alcoholic injury, identifying the prospects of dietary CM for protection against alcohol-induced liver injury. Camel Milk (CM) and Nigella Sativa (NS) [common name: black pepper, Tamil names: Karunjeeragam] is traditionally considered therapeutic agent for a wide range of diseases

and has been used as a drug in various parts of the world, especially in Saudi Arabia. We attempted to investigate the anti-hepatotoxic potential of CM and NS-Oil (NSO) on TAA)-induced hepatotoxicity and nephrotoxicity. Thirty female albino Wistar rats participated in this study and were randomized into 6 groups with 5 of her rats in each group.
On the first day, all rats in groups 2-6 received a single subcutaneous injection of TAA (100 mg/kg body weight) to induce hepatorenal injury. For comparison purposes, group 1 served as a normal control and group 2 as a toxicity control. Fresh CM (250 mL/24 h/cage), NSO (2 mL/kg/day p.o.), and NSO + fresh CM were fed to experimental rats in groups 3, 4, and 5, respectively. Group 6 rats received a polyherbal hepatoprotection from Unani called Jigreen (2 ml/kg/day p.o.) for 21 days.
Serum liver and renal function tests were analysed to assess TAA-induced hepatorenal injury and the protective effects of CM and NSO. For biochemical studies, the histopathology of liver and kidney tissue was analysed. The results of this study showed that TAA-poisoned rats had significantly increased levels of alanine transaminase, aspartate transaminase, γ-glutamyl transpeptidase, alkaline phosphatase, lipid profile, urea, creatinine, uric acid, sodium, and potassium. is showing. Rats treated with CM, NSO, and the combination of CM and NSO and digreen significantly reversed the injury, with serum biochemical parameters and lipid profiles reduced to normal values. Histopathological examination also supports the hepatoprotective and renoprotective roles of CM and NSO. This experiment demonstrated the ameliorative effects of CM, NSO, and the combination of CM and NSO on TAA-induced hepatonephrotoxicity in rats.

(Gaber et al., 2018) focused on evaluating the therapeutic effects of camel milk on carbon tetrachloride (CCl4)-induced liver injury in rats. A total of 24 rats were used in this study and the rats were randomly divided into 4 groups, each containing 6 of her rats. Group 1 rats were untreated controls. A rat in group 2 was orally administered camel milk (5 ml/rat/day) by gastric intubation, 3 times a week for several weeks, and he was 5 times a week for 4 weeks. Group 3 rats were poisoned with CCl4 (intraperitoneal injection of CCl44 (1 ml/kg body weight, 3 times a week for 4 weeks)). Groups 2 and 3 rats.

Blood samples were taken at the end of the experiment to estimate serum levels of liver enzymes, albumin, and total protein. Inflammatory cytokines (TNF-α and IL-1β) were measured in liver homogenates and liver mRNA expression of CYT p450 2E1 was detected. Other liver samples were routinely fixed and processed for histopathological evaluation and immunohistochemical assessment of α-SMA expression in liver sections. Results showed that CCl4 caused a marked ($P < 0.01$) increase in liver serum enzymes and a marked ($P < 0.01$) depletion of total protein and albumin with increasing TNF-α levels. And IL-1β induced.

Genetic results show that CCl4 administration caused a significant downregulation of CYP2E1 gene expression in liver tissue compared to controls. In contrast, camel milk treatment markedly ameliorated the hepatic role of serum and inhibited the downregulation of hepatic inflammatory cytokines and the CYP2E1 gene. Some histopathological changes were also noted in the CCl4-poisoned group, which were significantly ameliorated by camel milk administration. As such (Gaber et al., 2018), camels against CCl4-induced liver injury, even though it improved liver function, reduced levels of inflammatory cytokines, and prevented downregulation of the CYP2E1 gene in the liver. proved the therapeutic prospects of milk.

The possible effects of camel milk (CM) and gentamicin (GM) induced biochemical changes in rat serum were evaluated by (Al-Asmari et al., 2014). Randomly he divided the rats into 6 groups, 6 rats in each group. Rats in group 1 were used as controls, rats in group 2 were fed CM, rats in group 3 were injected with GM (intraperitoneal), and rats in group 4 were fed camel milk and GM. injected (intraperitoneally). The results of this study show that GM administration significantly alters serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), and lactate dehydrogenase (LDH) activity levels in rats. I showed that. In Group 4 rats, CM returned these parameters to near-normal ranges.

Furthermore, this study showed that gentamicin-injected rats had increased malondialdehyde (MDA) and myeloperoxidase (MPO) activities, while superoxide dismutase (SOD) and glutathione-S-transferase (GST), etc. showed a significant decrease in the activity of antioxidant enzymes ($P \leq 0.05$). Administration of CM significantly ($P \leq 0.05$) prevented the development of MDA and the activity of MPO and upregulated the activity of antioxidant enzymes (SOD and GST). The comprehensive results of this investigation indicated that pre-treatment with CM protected against GM-induced liver injury, possibly through inhibition of oxidative stress and inflammation, thus camel milk could be recognized as a new therapeutic agent.

They conducted studies in 5 groups of 6 rats each (Abbas MT et al., 2018).

Group 1: Healthy group saline (NS) per tube,

Group 2: 3 g ethanol/kg/day gavage,

Group 3: Oral administration of 1 ml of camel milk per kg per day,

Group 4: Oral administration of 3 g ethanol per kg per day + 1 ml camel milk per kg per day,

Group 5: Fifteen days before conception (prophylactic), camel milk was administered at 1 ml/kg/day, then orally, and then ethanol at 3 g/kg/day.

The duration of treatment is from conception to delivery. According to their study results, treatment of rats with ethanol during pregnancy significantly increased the level of MDA and significantly decreased the activity of the enzymes SOD, CAT, and GSH-PX in the liver tissue of neonatal rats, leading to a significant reduction in the activity of the liver tissue. histological changes. Furthermore, a significant increase in the activity of serum liver enzymes was observed compared to the control group. According to their data, camel milk has a protective and preventive role against ethanol-induced hepatotoxicity in neonatal rats.

5. Conclusion:

In completion, it can be concluded that drinking camel milk is beneficial in reducing the hazards posed by toxic substances. The flavour profile of camel milk is a noteworthy issue. We need to be aware of its health benefits. It is clear from this study that camel milk can have important hepatoprotective effects. useful for blending into formulations that are effective treatments for common liver diseases.

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7. References:

1. Abdulrahman K. Al-Asmari, R. Abbasmanthiri, Abdulrahman M. Al-Elewi, Saud Al-Omani, Saeed Al-Asmary, Sarah A. Al-Asmari., (2014)., "Camel Milk Brneficial Effects on Treating Gentamicin Induced Alterations in Rats"., Journal of Toxicology, Vol.2014 Article ID 917608, 7 pages.

2. Aftab Ahmad, Fahad A Al Abbasi, Saida Sadath, Soad Shaker Ali, Mohammed F Abuzinadah, Hani A Alhadrami, Anwar Ali Mohammad Alghamdi, Ali H Aseeri, Shah Alam Khan, Asif Hussain., (2018)., "Amelioratative Effect of Camel's Milk against *Nigella sataiva* oil against Thioacetamide-induced Hepatorenal Damage in Rats"; Pharmacogn mag. Jan-Mar 2018; 14(53):27-35.

3. Ahmed E. Elhag, F. Bernard, Samia M A El Badwi., (2017)., "Protective Activity of camel's milk and urine mixture (*Camelus dromedaries*) against Ethanol-induced Hepatotoxicity in rats"., Advances in Bioscience and Biotechnology; 8, 378-387.

4. Alric L, M Fort, J Izopet, J P Vinel, C Bureau, K Sandre, J P Charlet, M Beraud, M Abbal, M Duffaut., (2000)., "Study of host and virus related factors associated with spontaneous hepatitis C virus clearance", Tissue antigens; 56(2): 154-58.

5. Al-Hashem, F. (2009)"Camel milk Protects against Aluminium Chloride-Induced Toxicity in the liver and Kidney of White Albino rats". American Journal of Biochemistry and Biotechnology, 5(3), 98-108.

6. Al-Wabel, N. (2008)., "Mineral contents of milk of cattle, camels, goats and sheep in the central region of Saudi Arabia." Asian J Biochem, 3:373–5.

7. Amjad Ali Khan and Mohammad A Alzohairy., (2011)., "Hepatoprotective effects of camel milk against CCL_4 – induced hepatotoxivcity in rats"., Asian Journal of Biochemistry 6(2): 171-180.

8. Ayele Gizachew, Jabir Teha and Tadesse Birhanu., (2014)., "Review on medicinal and nutritional values of camel milk"., Nature and science ; 12(12).

9. Barłowska, J., Litwi, C. Z., Kedzierska-Matysek, M. and Litwi, C. A., (2007)., "Non-Polymorphism of caprine milk αs1-casein in relation to performance of four polish goat breeds"., Pol J Vet Sci, 10:159–64.

10. Coles B, Wilson I, Wardman P, Hinson JA, Nelson SD, and Ketterer B., (1988)., "The spontaneous and enzymatic reaction of N-acetyl-p-benzoquinonimine with glutathione: a stopped-flow kinetic study". Archives Biochemistry and Biophysics; 264**:** 253-260.

11. D'Urso, S., Cutrignelli, M., Calabr,` S., Bovera, F., Tudisco, R., Piccolo, V. And oInfascelli, F. (2008)., "Influence of pasture on fatty acid profile of goat milk"., J Anim Physiol Anim Nutr, 92:405–410.

12. El-Agamy, E., Nawar, M. and Haenlein, G. (2009)., "Are camel milk proteins convenient to the nutrition of cow milk allergic children"., Small Rumin Res, 82:1–6.

13. El-Zeini, H. (2006)., "Microstructure rheological and geometrical properties of fat globules of milk from different animal species"., Pol J Food Nutr Sci, 56:147–54.

14. Farah, Z. (1993). "Composition and characteristics Camel milk"., J Dairy Res, 60:603-26.

15. Fauquant, C., Briard-Bion, V., Leconte, N., Guichardant, M.and Michalski, M. (2007)., "Membrane phospholipids and sterols in microfiltered milk fat Globules"., Eur J Lipid Sci Tech, 12:1167–73.

16. Gabr SA, Zahran F, Mohammed Faten F, Hassanin WF, Sharoud MN, Mesalam NM., (2018). "Therapeutic Effect of Camel Milk Against Hepatotoxicity Induced by CCl_4 in rats"., RJPBCS; 9(1): 601-614.

17. Habatallah A. Darwish, Naglaa R. Abd Raboh, Amina Mahdy., (2012)., "Camel's milk alleviates alcohol-induced liver injury in rats"., Food and chemical Toxicology 50 1377-1388.

18. Haddadin, M., Gammoh, S. and Robinson, R.(2008)., "Seasonal variations in the chemical composition of camel milk in Jordan"., J Dairy Res, 75:8–12.

19. Hamed H, Gargouri M, Bellassowed K, Alkrouti A, El-Feki A Gargowri A., (2017)., "Hepatoprotective activity of camel milk against CCL_4- induced lesions in mice"., Research and Reviews in Biosciences; Vol 12 Iss 2.

20. Hansen, M., Fernandes, G. and Good, R. (1982)., "Nutrition and Immunity: The influence of diet on autoimmunity and the role of zinc in the immune response"., Annual Review of Nutrition, 2: 151-157.

21. Huggette Houghton PJ, Hylands PJ, Mensah AY, Hensel A, Deters AM., (2005)., "In vitro tests and ethnopharmacological investigations: wound healing as an example". Journal of Ethnopharmacology; 100(1-2)**:** 100-107.

22. Jibril Zuberu, Malajiya I. A. Saleh, Abdul Wahab Alhassan, Bello Y. Adamu, Munira Aliyu, Bikisu T. Iliya., (2017)., "Hepatoprotective effects of Camel milk on Poloxamer 407 Induced Hyperlipidaemic Wistar rats"., Macedonian Journal of Medical Sciences; 2017 Dec 15; 5(7): 852 – 858.

23. Khalid G. Al-Fartosi, Orass S. Khuon, Huda issa Al-Tae., (2018)., "Protective rolwe of camel's milk against paracetamol induced Hepatotoxicity in male rats"., IJRPBS; Vol.2(4). ISSN: 2229-3701.

24. Kingshuklahon and Swarnamoni Das., (2011)., " Hepatoprotective activity of *Ocimum sanctum* alcoholic leaf extract against paracetamol-induced liver damage in Albino rats"., Pharmacological Res; 2011 Jan-Mar; 3(1): 13-18.

25. Liang Ming, Xiang Yu Qiao, Li Yi, Dalai Siren, Jing Le Hai, Fucheng Guo, Yuchen Xiao, Rimutu. (2020)., "Camel milk modulates ethanol-induced changes in the gut microbiome and transcriptome in a mouse model of acute alcoholic liver disease"., J. Dairy Sci; 103:3937-3949.

26. Mahrous Abdel Basset Ibrahim, Farooq Ahmed Wani, Shaik Ragiman., (2016)., "Hepatoprotective effect of Olive oil and Camel milk on acetaminophen-induced liver toxicity in mice". International journal of medical science and public health; Vol 6; 186-194.

27. Mal, G., Suchitra-Sena, D. and Sahani, M. (2007)., "Changes in chemical and macro-minerals content of dromedary milk during lactation." Journal of Camel Practice and Research, 14: 195-197.

28. Mansson, H. (2008)., "Fatty acids in Bovine milk fat". Food Nutr Res, 52. DOI: 10.3402/fnr.v52i0.1821.

29. Mitchell JR, Jollow DJ, Potter WZ, Gillette JR and Brodie BB., (1973)., "Acetaminophen-induced hepatic necrosis. IV. Protective role of glutathione". Journal of Pharmacology and Experimental Therapeutics; 187**:** 211–217.

30. Mohammed Talat Abbas, Ali J. Ali, Alaa Abd-Alhasan Hamdan., (2018)., "The prophylactic-protective effect of Camel milk on ethanol induced hepato-toxicity in newborn rate"., EurAsian Journal of Biosciences 12, 503-509.

31. Nakamura W, Hosoda S, and Hayachi K., (1977)., "Purification and properties of rat liver glutathione peroxidise". Biochim Biophys Acta 4; 358**:** 251-261.

32. Richard O. Recknagel., (1967)., "Carbon Tetrachloride Hepatotoxicity" Pharmacological review., 19:145-208.

33. Sai Sruthi Argi, Saim Datri Argie and Lakshmana Rao, A., 2017., "A review on Hepatoprotective ctivity", International journal of current research, 9, (06), 51876 – 51881.

34. Shabo, Y., Barzel, R., Margoulis M. and Yagil R. (2005)., "Camel milk for food allergies in children"., IsrMed Assoc J, 7:796–8.

35. Shabo,Y. and Yagil R. (2005). "Etiology of autism and camel milk as therapy"., International Journal of Disability and Human Development, 4: 67-70.

36. Singh, R., Ghorui, S and Sahani, M. (2006)., "Camel milk: Properties and processing potential. In Sahani, M.S. The Indian camel"., NRCC, Bikaner. Pp: 59-73.

37. Stahl, T., Sallman, H., Duehlmeier, R. and Wernery U. (2006)., "Selected vitamins and fatty acid patterns in dromedary milk and colostru"., Journal of Camel Practice and Research, 13: 53-57.

38. Thnaian Althnaian, Ibrahim Albokhadaim, Sabry M El-Bahr., (2013), "Biochemical and histopathological study in rats intoxicated with Carbontetrachloride and treated with camel milk"., Springer plus; 2:57.

39. Vuyyurvi Bhaargavi, G SL Jyotma and Reshma Tripurana., (2014)., "A review on Hepatoprotective activity"., IJPSR., Vol.5(3): 690-702.

40. Yagil R. (1982). "Camels and camel milk"., FAO Animal production and health paper, Rome, Italy, 69: 68-93

41. Zicarelli, L. (2004)., "Buffalo milk: its properties, dairy yield and mozzarella production"., Vet Res Commun, 28:127–35.