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The Effects of Qinggan-27 Recipe on Nonalcoholic Steatohepatitis Induced by High-fat Diet in Rats

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Running title: Qinggan-27 Recipe on Nonalcoholic Steatohepatitis

Objective: The Qinggan-27 (QG-27) recipe is a traditional Mongolian medicine believed to improve liver function during Non-alcoholic steatohepatitis (NASH). We developed fatty liver models in rats by intragastric administration of a high-fat diet (HFD) to investigate the effects of the QG-27 recipe on the NASH model through the expression of PGC-1 α and UCP-2. **Methods:** Wister rats were fed with specially prepared HFD to create a pathological model for 6 weeks. After successful modeling, the patient was treated with QG-27 twice daily with intragastric administration of QG-27 at 150 mg/kg, 300 mg/kg, and 600 mg/kg, respectively, for 21 days. Biochemical, histopathological, and immunohistochemical analyses were conducted to evaluate the effect of QG-27 treatment. Results: Levels of AST, ALT, LDL-C, TG, and TC were significantly decreased, and HDL-C was increased in the intermediate dose of the QG-27 group compared to the control group ($p<0.05$). Immunohistochemical results demonstrated that an intermediate dose of the QG-27 recipe could significantly improve liver function in rats with fatty liver by increasing PGC-1α and UCP-2 expression. **Conclusion:** The intermediate (300 mg/kg) dose of the QG-27 is a good candidate to be a dietary supplement for reducing fatty liver.
Keywords: disease model, non-alcoholic fatty liver, Mongolian medicine, PGC-1α, UCP-2

Introduction

Nonalcoholic fatty liver disease (NAFLD) is a common liver disease wreaking havoc on people's lives. The prevalence of NAFLD is surging to 24% at present.[1] Progress of NAFLD traces from simple steatosis to nonalcoholic steatohepatitis (NASH), and then pericellular fibrosis in NASH can slowly lead to cirrhosis and hepatocellular carcinoma (HCC).

Furthermore, the most prominent immediate concern is the annual HCC incident rate in NASH patients was 5.29 per 1,000 person-years.[2] NASH is defined as the dynamic condition of hepatocellular injury, usually with ballooning as a hallmark, inflammation, and varying degrees

of fibrosis, which have a high stake in overweight and metabolic syndrome like type 2 diabetes mellitus.[3] In 2018, as a result of a study called "Prevention of Diseases Caused by Wrong Lifestyle Habits" in Mongolia, it was concluded that 54.4% of the population is overweight and obese, which shows that Mongolians are at risk of developing fatty liver diseases.[4]

Inflammation and oxidative stress are key factors in the pathogenesis of NASH, which can be applied as intervention targets for NASH treatment.[5] Elevated reactive oxygen species (ROS) generation causes damage in hepatocytes through systemic oxidative stress, triggering inflammation and fibrosis and ultimately leading to NASH.[6] However, the treatment for NAFLD is still limited. It has been known that mitochondria play a critical role in the development and pathogenesis of NAFLD.[7,8]

Mitochondrial biogenesis is essential to augment mitochondrial capacity, which helps relieve lipid accumulation in the liver. Proliferator-activated receptor γ coactivator-1 alpha (PGC-1α) is a key regulator of energy homeostasis by transcriptional regulation of genes involved in fatty acid oxidation and mitochondrial biology, of which uncoupling protein 1 (UCP1) is of central importance.[8-10] Previous research has shown a 40% decrease in hepatic PGC-1α expression in NAFLD patients, accompanied by mitochondrial dysfunction, lipid accumulation, and insulin resistance.[11,12] Furthermore, the treatment that stimulates mitochondrial function can delay the progression of obesity and diabetes.[8] Therefore, PGC-1α-mediated mitochondrial biogenesis is essential for the improvement of NASH.

Mongolian traditional medicine and complementary medicine have gained more attention for long-term use in treating metabolic diseases like obesity and diabetes due to fewer side effects than synthetic chemical drugs.[13,14] Clinical studies reveal that dietary intake of flavonoids can reduce the risk of NAFLD.[15,16] Qinggan-27 (QG-27) is one of the 1,700 individual medicines, with nearly 2,000 recipes and compositions recorded in the "Mongolian Hospital Treasures (Manag Rinchenjunai)" written in Tibetan by Barga scientist Mindol Nomun Khan Jambal Toin. It has been extensively used as a folk medicine by many cultures. It has many biological activities such as anticarcinogenic, antibacterial, antidiarrheal, antifungal, anti-nephrolithiasis, anti-gastric ulceration, antiatherogenic, and anti-inflammation effects. In addition, it could modify the risk of hypercholesterolemia and have an anti-intoxication impact on the body.[17]

No studies have investigated the effect of QG-27 treatment on key regulators of mitochondrial biosynthesis, such as PGC-1 α and UCP-2, in experimental animals that develop a fatty liver disease model fed HFD. Therefore, this study aims to investigate the effect of the QG-27 recipe on the regulation of PGC-1α and UCP2 in the NASH model induced by HFD in rats.

Materials and Methods

Drug extraction and preparation

Preparation of QG-27 recipe extract. The research work was carried out with the support of the Mongolian International Medical School of the MNUMS and the research center of the Mongolian Institute of Traditional Medicine Technology in the Laboratory of Natural Compounds of the Institute of Chemistry and Chemical Technology, Mongolian Academy of Sciences and the Medical Chemistry Laboratory, and the research laboratory of the National University of Tong Liao, Inner Mongolia. QG-27 ingredients are manufactured as pills at the Mongolian Pharmaceutical Factory of Mongoljin Khoshuu of Inner Mongolia (№M13060377).

We used 10% of QG-27, added 10 g of dry matter to 100 ml of cold distilled water in a glass beaker measuring 250 ml in a ratio of 1:10 (according to the National Pharmacopoeia of Mongolia), and put it in a boiling water bath. After steeping for 15 minutes, remove and cool at room temperature for 45 minutes.[9]

Comparative preparations

The study used Hu Gan Pian obtained from Gui Hua Pharmaceutical Co., Ltd. of Black River Province (China Medical Standard, batch number Z22022091).

Animal experiments

Experimental animals. The study used Wistar rats of 72 males aged 12 weeks (200-220 g). The animals were housed in groups of 12 animals each for 1 week in a temperature—and humidity-controlled room with a 12:12 h light and dark cycle. They were given free access to food and water. After the one-week adaptation period, the animals were used for the study.

Experimental design

The fatty liver model was developed using the high-fat diet (HFD), which was prepared by adding 78.6% soil, 10% pork belly oil, 10% egg yolk, 1% sterol, and 0.2% sulfur hydroxyl pyrimidine to the standard diet. The HFD was prepared every 2 days, kept at 4°C until used, and left at room temperature one hour

before use. Twelve healthy control group rats were fed regular food, and the other 60 rats were fed with specially prepared HFD to create a pathological model, intragastric administration for 24 h for 6 weeks.[12] Animals were weighed, recorded, numbered, and randomly divided into five groups of twelve animals each. All the animals were taken care of under ethical consideration, and the Institutional Ethics Committee duly approved the experimental protocol.

Group 1: The animals received a standard diet and purified water for 24 hours a day for 6 weeks and were considered the normal control group.

Group 2: The animals received HFD for 6 weeks. After 6 weeks of study, 0.5% Na-CMC suspension intragastric administration twice a day for 21 days was considered the HFD control group.

Group 3: Animals received HFD for 6 weeks. After 6 weeks, Hu Gan Pian's drug intragastric administration twice a day for 21 days was considered a comparative group.

Groups 4, 5, and 6: Animals received HFD for 6 weeks. After 6 weeks of study, QG-27 at a dose of 150 mg/kg, 300 mg/kg, and 600 mg/kg intragastric administration twice daily for 21 days, respectively. Biochemical tests. After 21 days, 24 h fasted animals were deeply anesthetized by an intraperitoneal injection of aldehyde chloride 10% (3 ml/kg). Blood samples were immediately 5 ml collected from each rat by cardiac puncture with capillary tubes into clean dried centrifuge tubes. The tubes were then centrifuged at 3000 rpm for 15 min. Clear serum samples were carefully separated using Pasteur pipettes and frozen at −20°C until biochemical analysis.[10] Serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglyceride (TG), total cholesterol (TC) and glucose were measured by semiautomatic biochemical analyzer DIRUI DR7000 (Dirui, Turkey) in each animal groups—histopathological examination. Livers of the experimental animals were removed by careful dissection, washed with cold saline solution, dried between two filter papers, and then photographed for gross observation. For the histological examination, 1/3 of the liver was fixed in 10% formalin, embedded in paraffin blocks, cut into three µm thick sections, and placed on glass slides.[13] The sections were stained with hematoxylin and eosin (H&E) to analyze histological changes under the microscope.[24]

Immunohistochemical staining. The paraffin sections were deparaffinized in xylene, rehydrated in graded alcohols, and pretreated with CC1 (Roche Diagnostics). The sections were washed with reaction buffer followed by incubation with primary antibody PGC-1α and UCP-2 at a 1:500 dilution for 60 min at 42°C. Bound antibody was detected with the UltraView Universal DAB kit (Roche Diagnostics), and sections were counterstained with hematoxylin according to the manufacturer's instructions. Positive and negative control stains were also performed.

Statistical analysis

All the above data were statistically analyzed by PAWS, version 22.0 (SPSS Inc., Chicago, IL, USA). All values are expressed as the means ± SD. A one-way ANOVA test was used to determine the difference among experimental groups. To further explore the data, Tukey's Post Hoc tests are used to analyze the pairwise comparison of the biochemical parameters between all groups; the significant findings are only shown in Tables 2 and 4. $P<0.05$ was statistically significant between the control and experimental groups.

Ethics

This study was approved by the Institutional Ethics Committee of the Mongolian National University of Medical Sciences (MNUMS) and has followed the principles outlined in the Declaration of Helsinki (MNUMS-2019/3-03).

Results

Macroscopic results

During the experiment, no deaths were recorded in animals of all groups. At the end of the experiments, all rats were cervically dislocated rapidly by experts, and their livers were collected for further analysis. The liver of the healthy comparison group of rats is brownish-brown in color, has a smooth surface and sharp edges, and the lobes are not attached and have a soft structure or healthy structure (Figure 1A). The liver of the pathological model group was enlarged, light yellow, with blunt edges, and greasy to the touch, resulting in the pathological model of fatty liver (Figure 1B). The livers of rats treated with an intermediate dose of QG-27 and Hu Gang Pian were red-brown, with sharp edges, and the shape and surface were similar to healthy livers (Figure 1 C and E) compared to low and high doses of QG-27 treated group showed red-yellow liver color, blunt edges (Figure 1D and F). The size and weight of the liver are measured in Table 1.

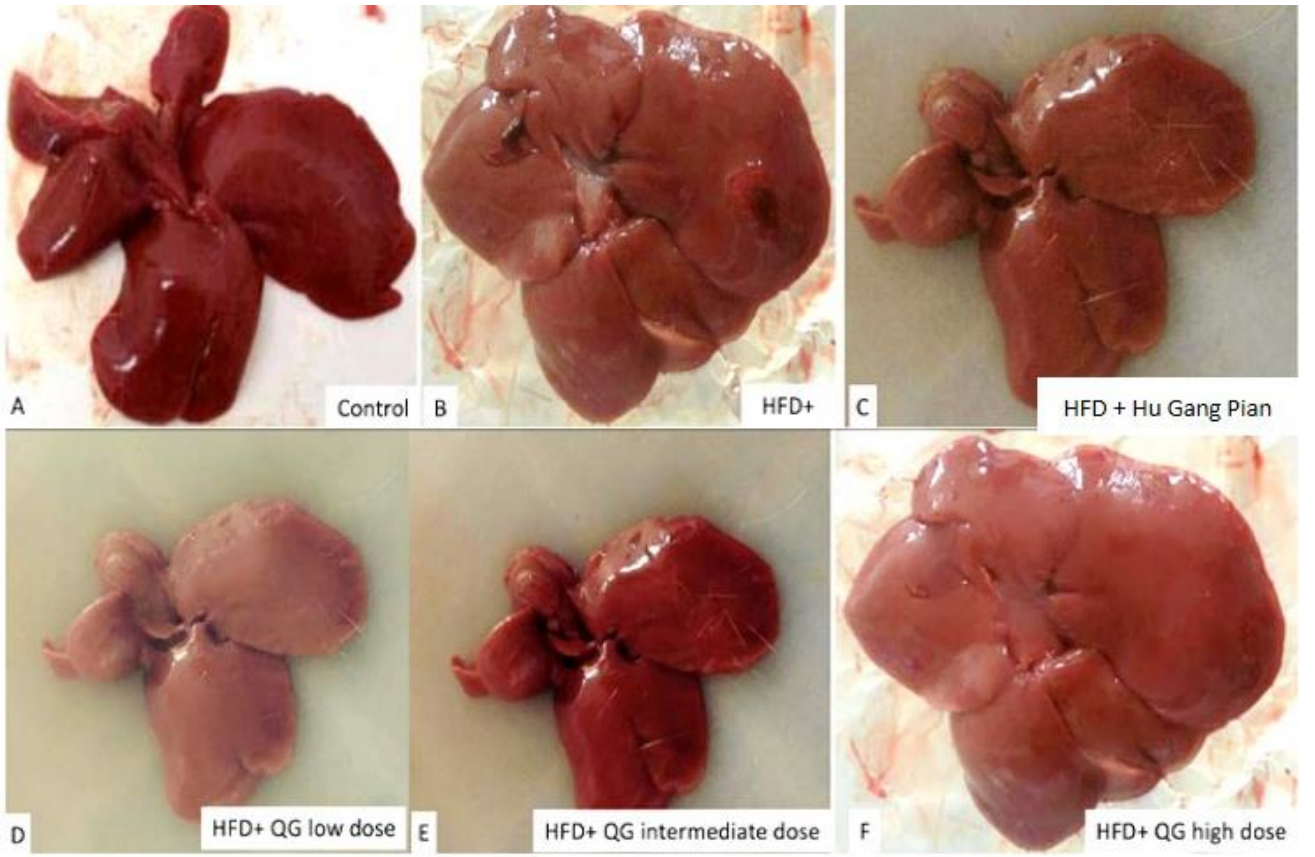

Figure 1. Liver morphology of the control and experimental groups. After completing the experiment, macroscopic examination of the livers from the control group, Hu Gang Pian, and the QG-27 intermediate dose group showed a normal beefy red, smooth, and shiny appearance [A, C, and E]. On the contrary, livers obtained from the high fat (HFG) group, low and high doses of QG-27 groups appeared enlarged, yellowish, and blunt edges [B, D, and F].

Table 1. Liver size and weight between the groups in the HFD model

	Normal	HFD	Hu Gan Pian	QG-27 (600 mg/kg)	QG-27 (300 mg/kg)	QG-27 (150 mg/kg)	F	P-value
Size	5.8±0.33	6.1±0.21	5.8±0.17	5.9±0.25	5.8±0.30	5.8±0.30	3.22	0.011
Weight	11.9±1.55	12.7±1.58	12.09±1.23	10.9±2.94	12.1±1.48	11.45±1.34	0.16	0.136

The liver size of the HFD group was significantly different from that of the Normal group, which indicates that a pathological model of fatty liver has been formed. A significant difference was observed between the QG-27 (300 mg/kg and 150 mg/kg) and the Hu Gan Pian (comparative drug) treated groups compared to the HFD group, indicating that those drugs reduce the amount of fatty liver size (Table 2).

Biochemical results

A comparison between the groups regarding the effects of QG-27 on serum biochemical values in rats-induced fatty livers was listed in Table 3. Plasma AST and ALT activity was significantly decreased ($P<0.05$) in the Hu Gan Pian and QG-27 intermediate dose groups compared to the HFD control group. Serum LDL-C, TG, and TC levels were significantly decreased ($P<0.05$)

Table 2. Multiple compression between the groups by liver size.

Parameters	Groups†	Mean difference	SE	P-value
Liver size	1 vs 2	-.345	0.106	0.026
	2 vs 3	.336*	0.106	0.034
	2 vs 5	.337*	0.106	0.033
	3 vs 2	-.336*	0.106	0.034
	4 vs 2	-.327*	0.106	0.044

*†Group 1; normal group, Group 2; HFD group, Group 3; Hu Gan Pian comparative group, Group 4; QG-27 (150 mg/kg), Group 5: QG-27 (300 mg/kg), Group 6: QG-27 (600 mg/kg) * Tukey Post Hoc tests*

in the Hu Gan Pian and QG-27 intermediate dose groups compared to the HFD control group, and serum HDL-C levels were increased (P<0.05) in the Hu Gan Pian and QG-27 intermediate dose groups compared to the HFD control group. Serum glucose levels were significantly decreased (P<0.05) in the Hu Gan Pian, QG-27 intermediate dose, and QG-27 high dose groups compared to the HFD control group (Table 3). Post hoc revealed that the QG-27 intermediate dose group significantly improved biochemical values more than the HFD control group. Significant interactions were followed up with Tukey's post hoc, as shown in Table 4.

Histopathological examination

Group 1 (Normal control group): The sections obtained from the livers of the control group showed standard histological structure. The liver was formed of classic hepatic lobules, which were roughly hexagonal, with central veins forming their central axis (Figure 2a)—each classic hepatic lobule comprised hepatocytes arranged in anatomizing cords, radiating from the center toward the periphery. The hepatocytes were polygonal in shape, containing rounded vesicular nuclei with dispersed chromatin and prominent nucleoli, and others were binucleated. The cytoplasm of hepatocytes appeared acidophilic with scattered fine basophilic granules. Blood sinusoids were found as a network between the plates of hepatocytes converging toward the central vein.

Table 1. Biochemical parameters between the groups in the HFD model

	Normal	HFD	Hu Gan Pian	QG-27 (600 mg/kg)	QG-27 (300 mg/kg)	QG-27 (150 mg/kg)	F	P-value
AST	106.34±13.80	137.07±16.69	119.13±10.63	121.59±11.45	124.36±18.91	129.53±23.98	17.71	0.001
ALT	39.66±10.60	55.46±11.02	47.71±12.24	52.11±11.97	49.36±6.69	48.4 ±0 4.63	4.40	0.001
TG	0.82±0.22	1.01±0.35	0.80±0.23	0.94±0.34	0.79± 0.22	0.90±0.24	10.4	0.090
TC	1.52±0.15	1.65±0.29	1.47±0.18	1.65±0.27	1.48± 0.29	1.60±0.20	6.01	0.070
LDL-C	0.21 ±0.06	0.33±0.07	0.14 ±0.04	0.30 ±0.05	0.26 ±0.10	0.31 ±0.10	1.92	0.001
HDL-C	1.30±0.26	1.02 ±0.18	1.11 ±0.13	1.00 ±0.17	1.14± 0.15	0.93±0.13	0.57	<0.001
Glu	5.99±0.51	6.80±0.33	5.86±0.67	6.03±0.61	5.55±0.96	5.55±0.73	1.33	0.270

**One-way ANOVA*

Table 4. Multiple comparisons between the groups by biochemical parameters*

Parameters	Groups†	Mean difference	SE	P-value
AST	1 vs 2	-34.67	4.1352	<0.001
	1 vs 6	-15.79	4.1352	0.004
	2 vs 1	34.67	4.1352	<0.001
	2 vs 3	25.49	4.1352	<0.001
	2 vs 4	23.71	4.1352	<0.001
	2 vs 5	31.43	4.1352	<0.001
	2 vs 6	18.88	4.1352	<0.001
	3 vs 1	9.19	4.1352	0.240
	3 vs 2	-25.49	4.1352	<0.001
	4 vs 2	-23.71	4.1352	<0.001
	5 vs 2	-31.43	4.1352	<0.001
	5 vs 6	-12.55	4.1352	0.037
	6 vs 1	15.79	4.1352	0.004
	6 vs 2	-18.88	4.1352	<0.001
	6 vs 5	12.55	4.1352	0.037
ALT	1 vs 2	-16.61	3.7452	<0.001
	1 vs 3	-11.47	3.7452	0.034
	1 vs 5	-12.45	3.7452	0.016
	2 vs 1	16.61	3.7452	<0.001
	5 vs 1	12.45	3.7452	0.016
LDL	1 vs 2	-0.12	0.0335	0.008
	1 vs 6	-0.10	0.0335	0.036
	2 vs 1	0.12	0.0335	0.008
	2 vs 3	0.19	0.0335	<0.001
	4 vs 3	0.16	0.0335	<0.001
	5 vs 1	0.12	0.0335	0.008
	5 vs 3	0.19	0.0335	<0.001
	6 vs 3	0.17	0.0335	<0.001
HDL	1 vs 2	0.28	0.0723	0.004
	1 vs 3	0.27	0.0723	0.006
	2 vs 4	0.30	0.0723	0.002
	2 vs 5	0.25	0.0723	0.014
	2 vs 6	0.37	0.0723	<0.001
	2 vs 1	-0.28	0.0723	0.004
	3 vs 1	-0.27	0.0723	0.006
	4 vs 1	-0.30	0.0723	0.002
	5 vs 1	-0.25	0.0723	0.014
	6 vs 1	-0.37	0.0723	<0.001

*†Group 1; normal group, Group 2; HFD group, Group 3; Hu Gan Pian comparative group, Group 4; QG-27 (150 mg/kg), Group 5: QG-27 (300 mg/kg), Group 6: QG-27 (600 mg/kg) * Tukey Post Hoc tests*

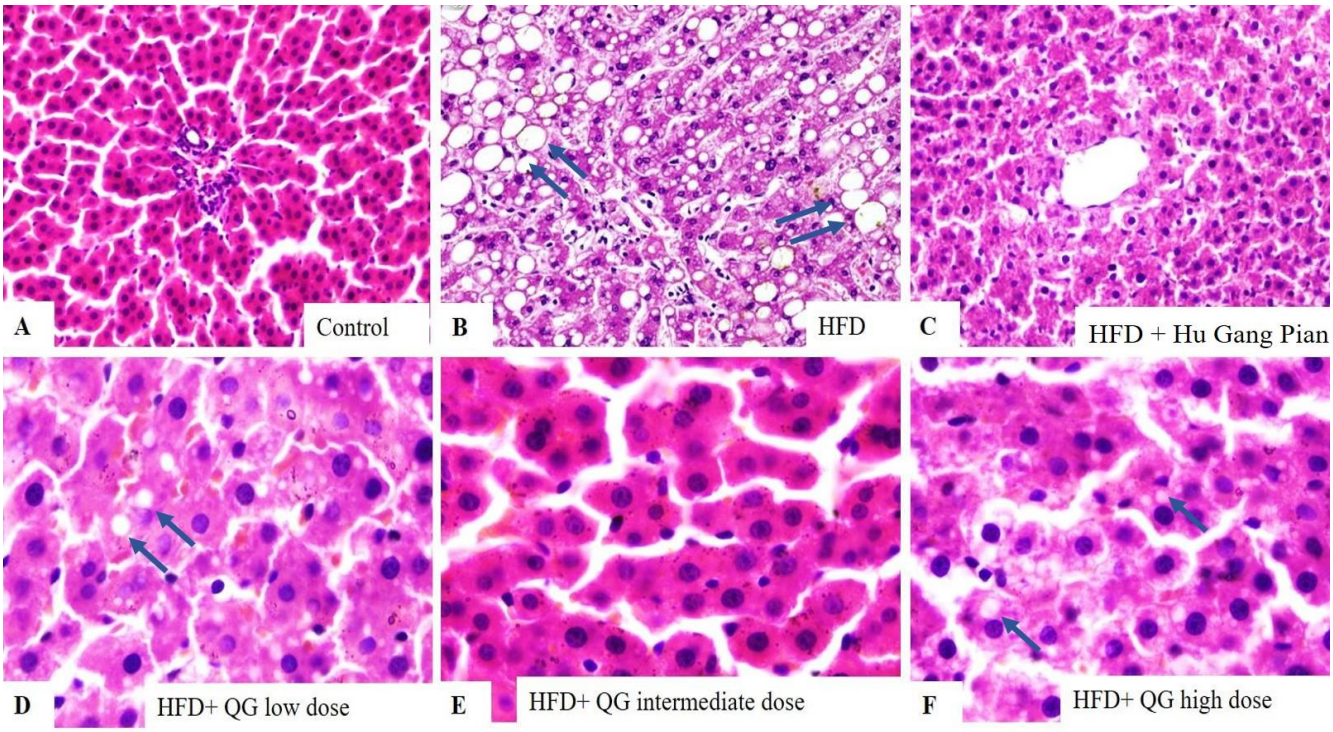

Figure 2. Histopathologic changes of liver cells in experimental groups (H&E).

(A) Hepatocytes from the healthy control group (x10). (B) Hepatocyte nuclei are peripherally located due to fat droplets in the cytoplasm (blue arrow) (x10) (C) The hepatocyte structure is slightly damaged, and partial necrosis surrounds the central vein (x10). (D) Small fat drops in hepatocytes, but inflammatory cells are undetected (x40). Fatty changes and ballooning of large droplets in hepatocytes disappeared (E), and tiny fat droplets were observed in very few cells (F) (x40).

Group 2-6 (fatty liver-induced group): The liver sections obtained from this group revealed several histological changes in the form of disturbed hepatic architecture, marked dilatation of central veins, and blood sinusoids. In addition to cellular infiltrations that can be seen around central veins and between the portal tract's components, most of the hepatocytes showed variable degrees of cytoplasmic fat vacuolations (Figure 2b). The liver sections obtained from animals that received Hu Gang Pian for 21 days were similar to the standard control group. They showed the normal histological structure of the liver (**Figure** 2 a, c, and e). In contrast, the liver sections obtained from both low and high-dose QG-27 groups show mild congestion of the central veins, mild congestion and dilatation of portal veins, and blood sinusoids with few cellular infiltrations around the components of portal tracts. Most of the hepatocytes showed restoration of their cytoplasm, while a few cells still showed some cytoplasmic fat vacuoles (**Figure** 2d and f).

Immunohistochemical results

Immunohistochemical staining from the HFD group revealed an apparent increase in the number of UCP-2 positive cells, whereas there was a decrease in the number of PGC-1α positive cells (**Figures** 3 and 4).

Compared with experimental groups, the PGC-1α and UCP-2 levels in the control group with a regular diet of rats in the HFD group increased, and the difference in expression levels of PGC-1α and UCP-2 in the liver of rats between the control group and HFD group was statistically significant ($P<0.05$). The expression levels in HFD with Hu Gan Pian and HFD with QG-27 intermediate dose group were higher than in HFD with QG-27 high and low dose group, and the differences were statistically significant ($p<0.05$).

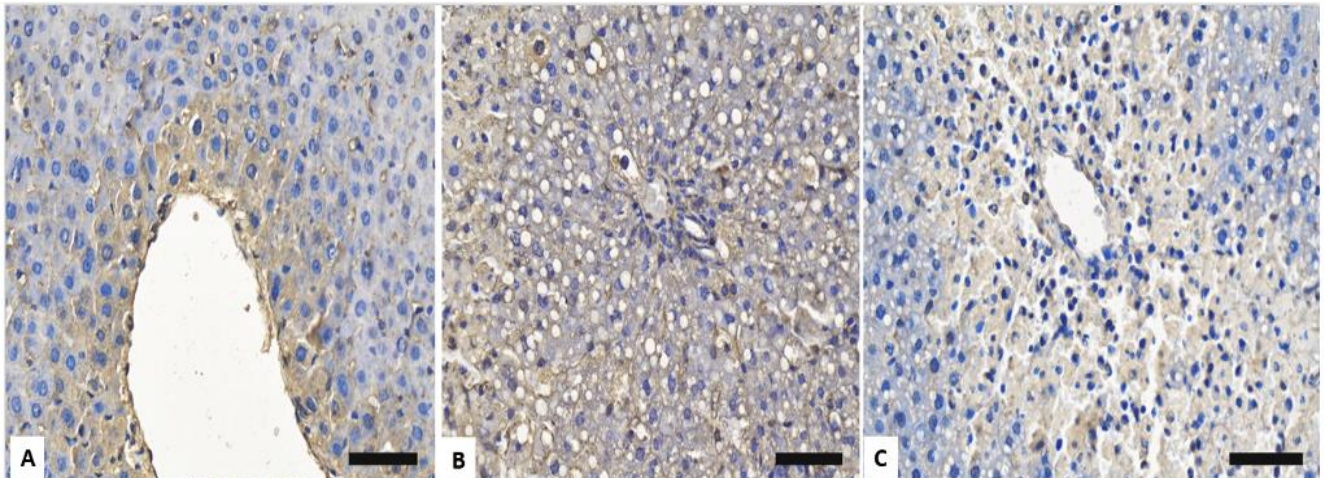

Figure 3. PGC-1α immunohistochemical stain showing (A) PGC-1α positive cells between the hepatocytes in the control group. (B) There was an apparent decrease in the number of PGC-1α positive cells in the fatty liver-induced group. (C) There is an evident increase in the number of PGC-1α positive cells in QG-27 treated groups (IHC stain ×40).

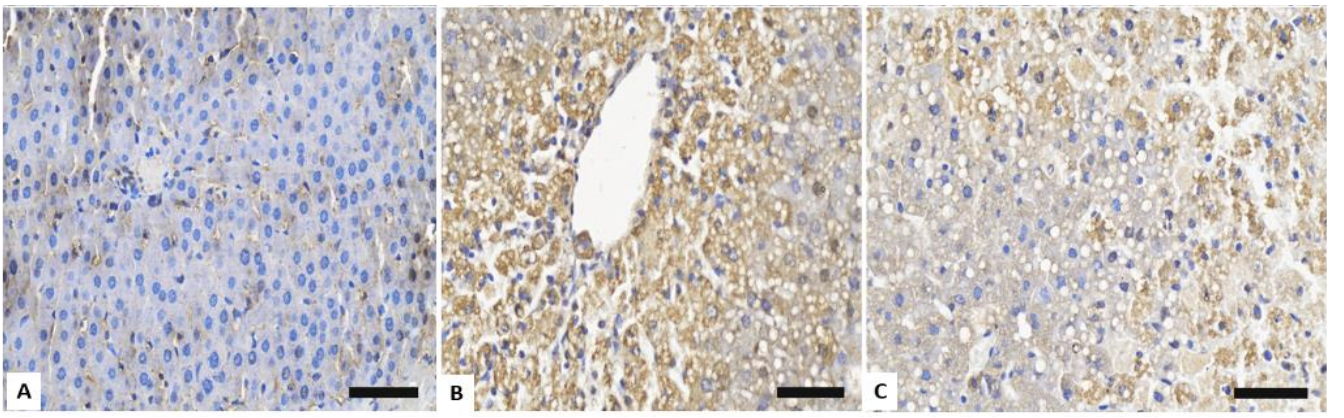

Figure 4. UCP2 immunohistochemical stain showing (A) Few UCP2 positive cells in between the hepatocytes in the control group. (B) There is an apparent increase in UCP2-positive cells in the fatty liver-induced group. (C) There was an evident decrease in the number of UCP2-positive cells in QG-27 treated groups (IHC stain ×40).

Discussion

In traditional Mongolian medical sources, the fatty liver is not mentioned as a single disease. The biochemical reaction or metabolism occurring within the human body is compared to the "maturation of the nutritional essence and waste matter" concept. Non-absorbed maturation of the nutritional essence and waste matter", instead of absorbed, fat deposits in the liver and contribute to the maturation of fat and cause non-alcoholic fatty liver disease.[22]

Phytochemical analysis of the QG-27 recipe revealed the presence of 27 active ingredients that include calcium, Bambusa textiles Mc Clure, gossampinus malabarica, and others, which are rich in flavonoids, could ameliorate fatty liver and insulin resistance in high-fat diet (HFD) fed mice. QG-27 recipes used in traditional Mongolian medicine have several benefits, low toxicity, and a small amount of side effects and harm. Mongolian drugs are widely used in liver diseases, especially non-alcoholic fatty liver disease, but no experimental studies on the action of the drug have been warranted. The prescription QG-27 is usually used for acute and chronic liver diseases, spleen and stomach problems, nausea, loss of appetite, and vomiting.

We identify that QG-27 affects hepatic mitochondrial biogenesis in HFD-induced experimental animals. In our study, we created a fatty liver disease model in experimental animals and studied the effect of the QG-27. On the 21st day of the experiment, AST, ALT, TG, total cholesterol, HDL-C, LDL-C, and blood glucose were measured and the results were calculated. Liver tissue

changes were evaluated histologically in experimental animals.

QG-27 recipe's protective effects on mouse models of alcoholic liver damage may be associated with its antioxidant behavior and inhibition of Wnt/β-catenin signaling pathway activation.[23] PGC-1α-mediated mitochondrial biogenesis enhances mitochondrial capacity and fatty acid oxidation.[18] Increased fatty acid oxidation reduces the lipid overload and lipotoxicity under HFD and produces ROS. Excessive fatty acid oxidation may overwhelm the capacity of the antioxidant defense system and induce oxidative stress and hepatic inflammation.[19] Intriguingly, QG-27 repressed oxidative stress and inflammatory response in the liver induced by HFD, counteracting the adverse effect of enhanced fatty acid oxidation.

In addition to its role in mitochondrial biogenesis, PGC-1α is also reported to regulate antioxidant enzymes in response to oxidative stress.[20,21] PGC-1α reduces mitochondrial ROS production through upregulating antioxidant gene expression, such as Cat, Sod1, Gpx1, and Ucp2.[21] Our results demonstrate that QG-27 alleviates hepatic steatosis in HFD-induced experimental animals. Therefore, QG-27 ameliorates hepatic steatosis and elevated hepatic mitochondrial biogenesis and fatty acid oxidation by increasing PGC-1α and UCP-2 expression.

In our previous acute toxicity study, a high dose of animals showed increased heart rate, respiration, decreased mobility, and increased urine output. There was no statistically significant difference in histopathological findings in the control groups compared to the three experimental groups ($p>0.05$). Prolonged use of QG-27 did not reveal significant changes in the quantity or quality of blood elements compared to control groups. The level of AST decreased in the group administered QG-27 at intermediate and high doses ($P=0.001$). Alkaline phosphatase levels were increased in the group at a high dose ($P=0.5$). Amylase levels increased in the low and intermediate groups and decreased in those receiving QG-27 at a high dose ($P=0.001$). There were no significant changes in histopathological analysis in the vital organs, including the heart, lung, kidney, spleen, and liver.

Sections were assessed in a blinded fashion and observed under a microscope. In our study, the levels of AST, ALT, TG, and LDL-C in the rats treated with the QG-27 recipe decreased while HDL-C increased, significantly different from the pathological model group. Based on these results, it was possible to create a pathological model of fatty liver in experimental animals, and it was confirmed by biochemical and histological analysis that the intermediate dose of the QG-27 recipe reduces fatty liver. The QG-27 recipe used in our research is a good ingredient for removing heat in the liver, suppressing jaundice, protecting the stomach, improving the secretion of clear bile, and helping the fatty liver.

In this study, we only detected PGC-1α and UCP-2 expression in the fatty liver of the included animal model, which is a limitation of our study. In the future, there is a need to investigate the compound of the QG-27 recipe, which further plays a more critical role in regulating PGC-1α and UCP-2, which are the primary regulators of mitochondrial biosynthesis in fatty liver disease models. In addition, a more expanded study is required to explain QC-27 and how to protect against liver cell damage and inhibit liver cell damage biochemical markers.

Conclusion

The pathological model of fatty liver of experimental animals was confirmed by histological analysis. The intermediate (300 mg/kg) dose of the QG-27 was the most effective in protecting liver cell damage and inhibiting the accumulation of fat in the liver with a pathological model is explained by decreasing the levels of AST, ALT, TG, LDL-C, and glucose in the blood plasma and increasing HDL-C.

Conflict of Interest

The authors state no conflict of interest.

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