

The horse liver is an organ that is usually described as part of the digestive system but its functions extend far beyond that. It is estimated that the equine liver performs in the neighborhood of 500 distinct functions. The liver is classified as both an organ and a gland and is the largest internal organ. It is also the only tissue that has any significant ability to regenerate itself.

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What Does the Horse Liver do?

On the digestive end, the liver produces bile which is essential for the processing and absorption of fats and fat soluble vitamins. All blood leaving the intestinal tract and containing digested food must pass first through the liver before being distributed to body tissues. The liver manufactures a protein call metallothionein which binds to trace minerals, both nutritionally important and inherently toxic, to control their absorption into the body by shepherding excess out through the urine or bile. We all know the liver is involved with metabolism of drugs and detoxification reactions but this is only a tiny fraction of the chemicals routinely handled by the liver. With every mouth of food, including grasses, the horse takes into their body they get a huge array of chemicals other than the basic nutrients of vitamins, minerals, protein/amino acids, fatty acids and

carbohydrates. To get some idea of how extensive this is even for a basically benign food go to Dr Duke's Phytochemical and Ethnobotanical Database and enter the food of your choice, e.g. alfalfa or oats. The lists generated are staggering. Every single one of those chemicals has to be processed by the liver, sometimes also the kidneys, and eventually excreted. The liver manufactures the major blood protein, albumin, as well as an extensive array of other proteins that carry hormones or minerals, like ceruloplasmin for copper. The liver also produces the coagulation factors needed for blood clotting. It also plays a pivotal role in metabolism. It stores excess glucose from meals as glycogen, converting that back to glucose when blood levels drop below normal. It can also produce glucose from amino acids and other sugars by a process called gluconeogenesis. The liver can break down fats to acetate for energy metabolism. It synthesizes lipoproteins, converts excess amino acids and carbs into fatty acids and triglycerides, synthesizes cholesterol phospholipids. In protein metabolism, the liver can remove the nitrogen groups from amino acids which are then converted to urea and excreted. The backbone of the amino acid is transformed into glucose or fat. It can synthesize the nonessential amino acids.



Liver Disease in Horses

Liver disease is uncommon in horses, at least liver disease severe enough to cause symptoms. Over 75% of the liver must be destroyed before clear liver related symptoms are seen. Symptoms include weight loss, poor appetite, yellowing of the membranes

(icterus) and in advanced cases encephalopathy, clotting disorders. Colic or diarrhea may be seen but are not consistent signs. Horses on pasture may develop liver disease from ingestion of toxic plants that contain pyrrolizidine alkaloid (PA), including *Crotalaria* spp, *Senecio* spp, *Amsinckia* spp, *Heliotropium europaeum*, *Echium plantagineum*, and *Cynoglossum officinale*. These plants remain toxic when dried so may also contaminate hay. Alsike clover, red clover or panicum pastures may also cause liver damage. Some fungal toxins, such as aflatoxin, also poison the liver. Serum sickness, aka Theiler's Disease, is a sudden onset liver failure with 50 to 60% fatality. It can be seen several weeks after a horse receives a product made with horse serum such as tetanus antitoxin but in other cases the cause is not known. Tyzzer's disease is a usually fatal Clostridial infection of the liver in young foals. Inflammation of the bile ducts and liver can be caused by parasite migration, stones or bacterial infection. This typically causes obvious changes on blood chemistry but is not usually fatal although the horse may be symptomatic. Prolonged treatment with antibiotics is usually effective. Fatty liver occurs in ponies and miniatures with hyperlipemia/hypertriglyceridemia syndrome. In brief, intake of insufficient calories causes an exaggerated release of fat from storage depots and increased fat production in the liver which causes fatty liver among other changes. For more detailed information on this visit this highly informative page.

Fatty Liver Disease in Horses

Insulin resistance or equine metabolic syndrome can also cause fatty liver and this is being increasingly recognized in full size horses as well. Insulin would normally drive both glycogen and fat production in the liver. For reasons that are poorly understood, with IR or EMS the fat drive predominates. To find out more about iron overload as a possible cause of insulin resistance in horses please read this article.

Treatment of Horse Liver Failure or Disease

Treatment of toxin hepatic disease involves removal of further exposure, and bacterial infections are treated with antibiotics. In all cases, supportive care of fluids, minimal fat, maintenance of adequate but not excessive protein and providing adequate calories predominantly from fiber is ideal. Flax is an ideal essential fatty acid source because it is

low sugar/starch and high in soluble fiber. Beet pulp is also a good choice. There is virtually no good equine information available on herbal treatment of fatty liver but click on the picture of the study below for a paper with a brief summary of agents used in humans:

Recent Advances in the Herbal Treatment of Non-Alcoholic Fatty Liver Disease

[Jia Xiao](#),^{1,2} [Kwok Fai So](#),^{2,3,4} [Emily C. Liang](#),² and [George L. Tipoe](#)^{2,3}

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Abstract

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Non-alcoholic fatty liver disease (NAFLD) is one of the leading causes of chronic liver injury across the world. It is also strongly related to other pathological conditions, including obesity, diabetes, cardiovascular diseases, and symptoms of metabolic syndrome. Pathogenesis of NAFLD remains not fully characterized but is generally attributed to the occurrence of insulin resistance, lipid metabolism dysfunction, oxidative stress, inflammation, and necro-apoptosis. Every potential therapeutic strategy should target one or some of these pathological events in the liver. Over the past decades, application of herbal treatment for NAFLD has received increasing attention due to its wide availability, low side effects, and proven therapeutic mechanisms and benefits. In recent years, some monomers and certain functional mixtures of herbs have been extensively examined for their potential uses in NAFLD treatment. In the present review, we selected several herbal derivatives under intense basic and/or clinical investigations by carrying out a PubMed search of English language articles relevant to herbal derivatives and NAFLD, such as polysaccharide portion of wolfberry, garlic-derived monomers, red grape-derived resveratrol, and milk thistle-derived substances. They have been shown to target the pathological events during NAFLD initiation and progression both in pre-clinical studies and clinical trials. Although more detailed mechanistic researches and long-term clinical evaluations are needed for their future applications, they offer unanticipated and great health benefits without obvious adverse effects in NAFLD therapy.

Keywords: Garlic-derived monomers, Herbal treatment, Milk thistle-derived substances, Non-alcoholic fatty liver disease, Pathogenesis, Resveratrol, Wolfberry

Jiaogulan (*Gynostemma pentaphyllum*) has also been demonstrated to be active:

Arch Pharm Res. 2012 Jul;35(7):1241-50. doi: 10.1007/s12272-012-0715-5. Epub 2012 Aug 3.

Protective effects of gypenosides against fatty liver disease induced by high fat and cholesterol diet and alcohol in rats.

Qin R¹, Zhang J, Li C, Zhang X, Xiong A, Huang F, Yin Z, Li K, Qin W, Chen M, Zhang S, Liang L, Zhang H, Nie H, Ye W.

⊕ Author information

Abstract

In the present study, the protective effects of gypenosides from *Gynostemma pentaphyllum* on fatty liver disease (FLD) were examined in rats treated with high fat and cholesterol diet and alcohol. Male SD rats were divided into seven groups: control, model, lovastatin, silymarin, gypenosides high-, medium- and low-treatment groups. The latter 6 groups were fed high-fat and cholesterol diet and administered alcohol intragastrically once a day. Body weight was measured every week for 10 weeks, and the hepatic index was measured after 10 weeks. Compared with model group, levels of serum triglyceride (TG), total cholesterol (TC), free fatty acid (FFA), and low density lipoprotein cholesterol (LDL-C) level, malondialdehyde (MDA), serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activity, and hepatocyte apoptosis were significantly decreased in gypenosides groups; while serum high density lipoprotein cholesterol (HDL-C), superoxide dismutase (SOD) activity in both serum and hepatic tissue and mRNA and protein level of peroxisome proliferator-activated receptor α (PPAR- α) were significantly increased. Moreover, hepatic steatosis and mitochondrial damage were improved. These results suggested that gypenosides could prevent liver fatty degeneration in fatty liver disease through modulating lipid metabolism, ameliorating liver dysfunction and reducing oxidative stress.

This

one shows an added benefit of Jiaogulan when combined with a controlled diet versus diet alone:

Altern Ther Health Med. 2006 May-Jun;12(3):34-9.

The add-on effects of *Gynostemma pentaphyllum* on nonalcoholic fatty liver disease.

Chou SC¹, Chen KW, Hwang JS, Lu WT, Chu YY, Lin JD, Chang HJ, See LC.

⊕ Author information

Abstract

CONTEXT: Other than weight reduction by dieting or physical activity, there are no well-documented medical treatments for fatty liver disease.

OBJECTIVE: To evaluate the efficacy of the add-on *Gynostemma pentaphyllum* (GP) in research subjects with nonalcoholic fatty liver disease.

DESIGN: A randomized, single-blind, controlled clinical trial.

SETTING: Hospital-based clinic.

PATIENTS: Fifty-six research subjects who were diagnosed with nonalcoholic fatty liver by abdominal ultrasound scanning.

INTERVENTIONS: The treatment group and the control group followed a controlled diet for 2 months. After 2 months, the treatment group continued to diet and received 80 mL GP extraction for 4 months; the control group continued to diet and received a placebo capsule for 4 months.

MAIN OUTCOME MEASURES: Body mass index (BMI), biochemistry data, and fatty liver score were measured at baseline, at 2 months, and at 6 months.

RESULTS: After 2 months of dieting, BMI and most biochemistry data decreased in both study groups. There were no significant differences in BMI or biochemistry data at month 2 between the 2 study groups. At month 6, BMI, triglyceride, aspartate aminotransferase (AST), alanine aminotransferase, alkaline phosphatase, insulin (ALP), insulin resistance index (HOMA-IR), and fatty liver score were reduced in both groups. The treatment group saw significant reductions in BMI, AST, ALP, insulin, and HOMA-IR, however. Changes in uric acid levels in the 2 groups from month 2 to month 6 were statistically significant ($P = .028$).

CONCLUSION: GP is an effective adjunct treatment to diet therapy for patients with nonalcoholic fatty liver disease.

There

is also evidence to support the use of vitamin E and omega 3 fatty acids.

Forageplus Talk adds: A highly interesting study and literature search into iron overload in black rhinos suggested that raised liver enzymes were often seen where the liver was compromised through high exposure to dietary iron. It is worth realising that strict mineral balancing is a possible path to correcting

raised liver enzymes and reducing the exposure to high iron in the diet by raising anti-oxidant minerals copper and zinc. A forage analysis is usually very helpful in determining the amount of copper and zinc needed to correct high exposure to the antagonist mineral iron.

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