乐奔拓西洋参人参口服液

——RTB

营养科学部：Jennifer

2025/11/28

目录

1. 肝脏简介


肝脏
内脏巨无霸
重达 1.5 KG
相当于一只
42 码球鞋
性格
肝狠话不多
没有痛觉
没有感情


潜能巨大

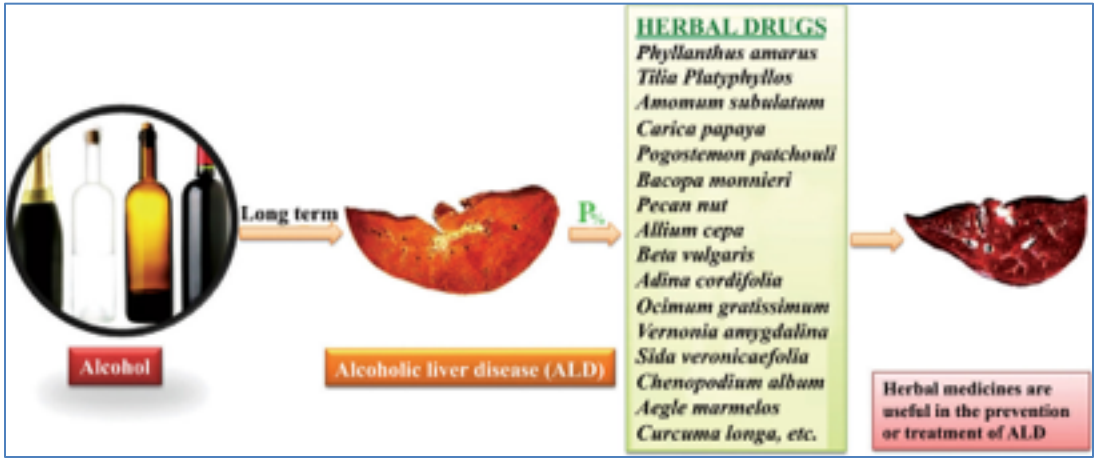

超强再生


解毒高手

1. 肝脏简介

1. 肝脏简介


肝脏不能承受之重
—酒
酗酒伤肝。酒精进入人体后，超90%的解毒工作都留给了肝脏。长期酗酒会让肝脏不堪重负，还可能患上酒精性肝病。
饮酒要适度，不空腹喝酒，如果可以，应尝试戒酒。自身状况不适合喝酒的人应避免喝酒，如胃溃疡患者等。


Long term
Alcohol
Alcoholic liver disease (ALD)
HERBAL DRUGS
Phyllanthus amarus
Tilia Platyphyllos
Amomum subulatum
Carica papaya
Pogostemon patchouli
Bacopa monnieri
Pecan nut
Allium cepa
Beta vulgaris
Adina cordifolia
Ocimum gratissimum
Vernonia amygdalina
Sida veronicaefolia
Chenopodium album
Aegle marmelos
Curcuma longa, etc.
Herbal medicines are useful in the prevention or treatment of ALD

目录

2. 产品信息

产品名称	乐奔拓西洋参人参口服液
产品类型	保健食品
功效宣称	缓解体力疲劳、对化学性肝损伤有辅助保护作用。
核心宣称	每100ml含：总皂苷80mg

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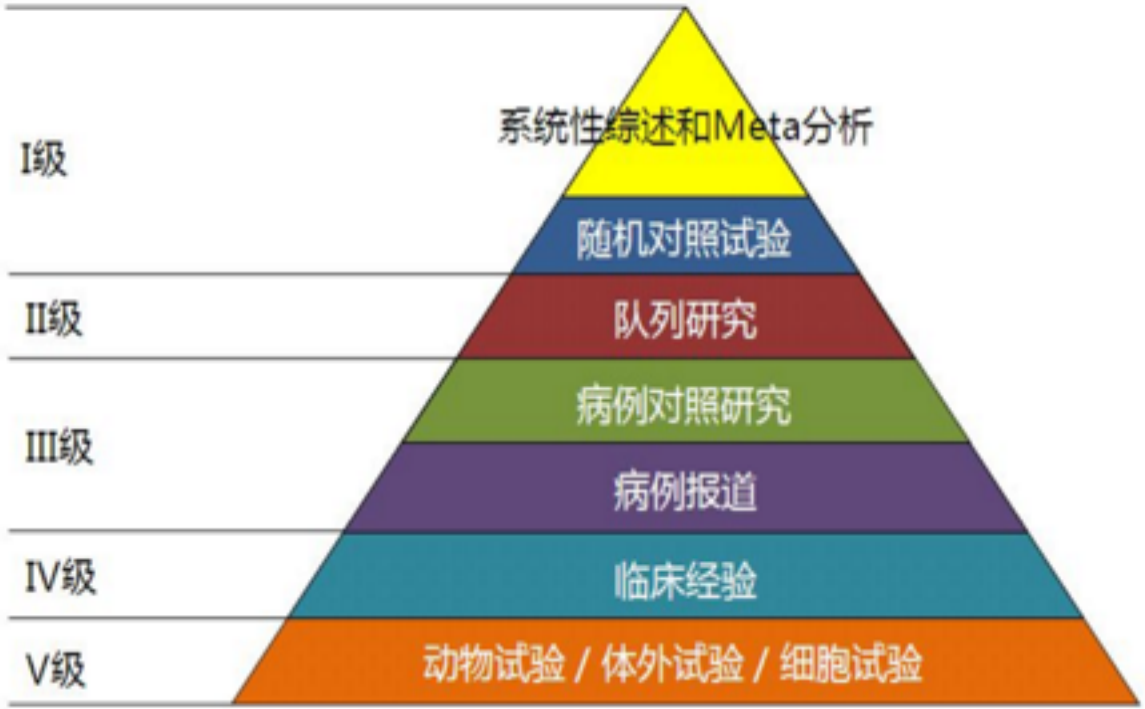
临床医学的循证等级


I级
系统性综述和Meta分析
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3.1 RTB 文献资料支持—护肝

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Research Article

Li-Ginseng powder protects against alcohol-induced liver injury by promoting acetaldehyde clearance and cellular homeostasis

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- 发表期刊：Journal of Ginseng Research
- 发表年份：2025年
- 标题：李人参粉末通过促进乙醛清除和细胞稳态来预防酒精诱导的肝损伤
- 证据等级： V级

3.1 RTB 文献资料支持—护肝

ABSTRACT

Background: Chronic and excessive alcohol consumption is a primary driver of alcohol-associated liver disease (ALD), a global health challenge with limited treatment options. Panax ginseng Meyer exhibits various pharmacological activities, including antioxidant and anti-inflammatory effects. However, its efficacy in preventing alcohol-induced liver injury remains limited, necessitating further optimization and investigation.

Methods: This study evaluated the hepatoprotective effects of Li-Ginseng Powder (LGP), a ginseng preparation enriched in rare ginsenosides, using a murine model of ALD and ethanol-exposed human hepatic L-02 cells. ALD was induced in C57BL/6 mice via daily oral ethanol administration (2400 mg/kg). Serum and liver biochemical markers were measured, and histological changes were assessed using H&E and Oil Red O staining. In vitro assays examined the effects of LGP on ethanol-metabolizing enzyme activity, oxidative stress, mitochondrial integrity, and autophagy.

Results: Ethanol exposure significantly elevated serum levels of aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, total bilirubin, low-density lipoprotein, and cholesterol, as well as hepatic triglycerides and malondialdehyde, while markedly decreasing hepatic levels of reduced glutathione and superoxide dismutase. LGP pre-treatment effectively reversed all these alterations, restored antioxidant capacity, and alleviated histological damage and lipid accumulation to near normal levels. In L-02 cells, LGP significantly enhanced alcohol dehydrogenase and aldehyde dehydrogenase activities, facilitated ethanol and acetaldehyde detoxification, reduced reactive oxygen species levels, preserved mitochondrial membrane potential, and promoted autophagy.

Conclusion: LGP confers comprehensive hepatoprotection against alcohol-induced liver injury by significantly enhancing ethanol catabolism, enhancing antioxidant defenses, and activating autophagy. These findings suggest its therapeutic potential in the management of ALD.

- 乙醇暴露显著升高了血清中的天冬氨酸氨基转移酶、丙氨酸氨基转移酶、碱性磷酸酶、总胆红素、低密度脂蛋白和胆固醇水平，以及肝脏中的甘油三酯和丙二醛水平，同时显著降低了肝脏中的还原型谷胱甘肽和超氧化物歧化酶水平。LGP预处理有效逆转了所有这些改变，恢复了抗氧化能力，并将组织学损伤和脂质积累减轻至接近正常水平。
- 在L-02细胞中，LGP显著增强了乙醇脱氢酶和乙醛脱氢酶的活性，促进了乙醇和乙醛的解毒，降低了活性氧水平，保持了线粒体膜电位，并促进了自噬。

结论: LGP通过显著增强乙醇分解代谢、增强抗氧化防御和激活自噬，对酒精诱导的肝损伤提供了全面的肝保

这些发现表明其在治疗ALD方面具有潜力

3.1 RTB 文献资料支持-护肝

实验类型	用量及时间	产品	测试指标
动物实验 C57BL6小鼠	➢ 对照组：每日灌胃水，持续1个月 ➢ 酒精模型组：从第16天起每日灌胃56%二锅头酒，持续15天； ➢ 低剂量LGP组：每日灌胃12.5mg/kg，持续1个月； ➢ 高剂量LGP组：每日灌胃50mg/kg，持续1个月； 56%二锅头酒在LGP给予4h后进行灌胃，每日一次。LGP处理持续一个月（30天），酒精干预持续15天。	李人参粉末（LGP）	➢ 血清生化指标：天冬氨酸氨基转移酶（AST）、丙氨酸氨基转移酶（ALT）、碱性磷酸酶（ALP）、总胆红素（TBIL）、低密度脂蛋白（LDL）和胆固醇（CHOL）。 ➢ 肝脏氧化应激指标：甘油三酯（TG）、丙二醛（MDA）、超氧化物歧化酶（SOD）活性和还原型谷胱甘肽（GSH）含量。 ➢ 肝脏组织病理学指标：肝组织进行H&E染色和油红O染色。

3.1 RTB 文献资料支持-护肝

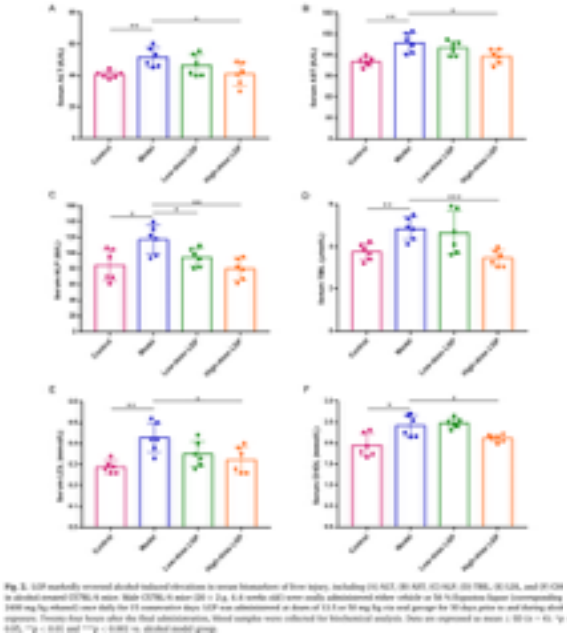

图2. LGP显著逆转了酒精处理的C57BL/6小鼠肝脏损伤血清生物标志物的升高，包括（A）ALT、（B）AST、（C）ALP、（D）TBIL、（E）LDL和（F）CHOL。

3.2. Serum biochemical evidence for the hepatoprotective effect of LGP against alcohol-induced liver injury

To assess the hepatoprotective effects of LGP against alcohol-induced liver injury, a subacute liver injury model was established in C57BL/6 mice via daily oral gavage of 56 % Erguotou liquor (equivalent to 2400 mg/kg ethanol) for 15 consecutive days. Compared to the control group, ethanol-treated mice exhibited significant elevations in serum TBIL, LDL, and CHOL, confirming successful induction of subacute liver injury (Supplementary Table 2).

ALT and AST, enzymes predominantly localized in hepatocytes and involved in amino acid metabolism, are widely recognized as sensitive indicators of hepatocellular injury [17]. Hepatic damage typically results in leakage of these enzymes into the bloodstream [18]. In the model group, serum ALT and AST levels were significantly elevated following ethanol exposure (Fig. 2A and B). Notably, high-dose LGP administration markedly attenuated these elevations, indicating a protective effect. Additionally, alcohol-induced liver damage can impair bile formation and excretion, leading to increased serum TBIL and ALP biomarkers commonly associated with cholestasis and hepatic dysfunction [19,20]. Ethanol exposure significantly elevated both ALP and TBIL levels (Fig. 2C and D). High-dose LGP significantly reduced both ALP and TBIL levels close to those of the normal control group, whereas low-dose LGP decreased ALP but did not significantly affect TBIL.

Liver injury is frequently accompanied by lipid metabolism disorders, resulting in dyslipidemia characterized by increased levels of LDL and CHOL [21]. The model group showed marked increases in CHOL and LDL, which were reduced by 25 % and 14 %, respectively, in the high-dose LGP group (Fig. 2E and F).

- 在模型组中，乙醇暴露后血清ALT和AST水平显著升高（图2A和B）。值得注意的是，高剂量LGP给药显著减弱了这些升高，表明其具有保护作用。
- 此外，酒精性肝损伤会损害胆汁的形成和排泄，导致血清TBIL和ALP升高，这通常与胆汁淤积和肝功能障碍相关。乙醇暴露显著升高了ALP和TBIL水平（图2C和D）。高剂量LGP将ALP和TBIL水平显著降低至接近正常对照组，而低剂量LGP降低了ALP但对TBIL影响不显著。
- 肝损伤常伴有脂质代谢紊乱，导致以LDL和CHOL水平升高为特征的血脂异常。模型组显示CHOL和LDL显著升高，高剂量LGP组分

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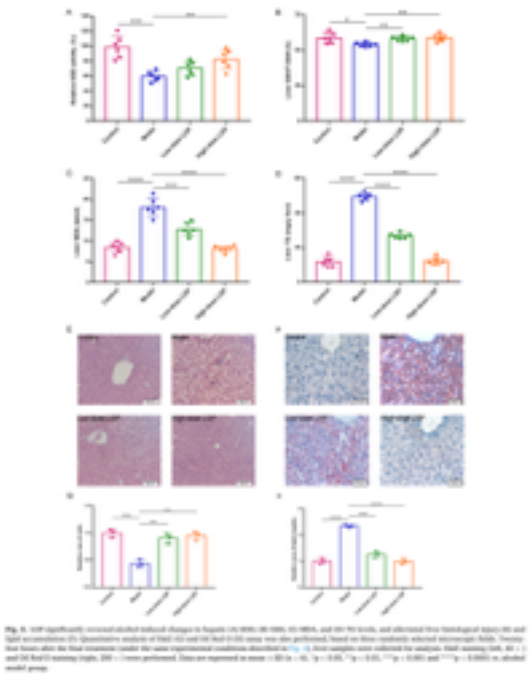


3.3. *LGP alleviated alcohol-induced oxidative stress*

Excessive alcohol consumption promotes the overproduction of ROS

[22], leading to oxidative stress and depletion of critical endogenous antioxidants such as GSH. To assess oxidative damage and antioxidant defense, hepatic SOD activity and GSH content were measured. As anticipated, ethanol exposure significantly reduced SOD activity and GSH/T-GSH ratio in the model group (Fig. 3A and B). Notably, treatment with LGP at 50 mg/kg significantly restored SOD activity and GSH/T-GSH ratio to near normal control levels, suggesting that LGP mitigates alcohol-induced oxidative stress by enhancing the antioxidant defense system.

Disruption of the tricarboxylic acid (TCA) cycle and impaired fatty acid oxidation caused by alcohol further contribute to hepatic lipid metabolic dysfunction and lipid accumulation. MDA, a lipid peroxidation byproduct, serves as a key biomarker of oxidative damage to cell membranes [23]. In this study, MDA levels were markedly elevated in ethanol-treated mice, while LGP administration particularly at the high dose led to a notable reduction in MDA levels, bringing them close to those observed in the control group (Fig. 3C), indicating that LGP effectively attenuates lipid peroxidation and oxidative membrane damage in alcohol-induced liver injury.

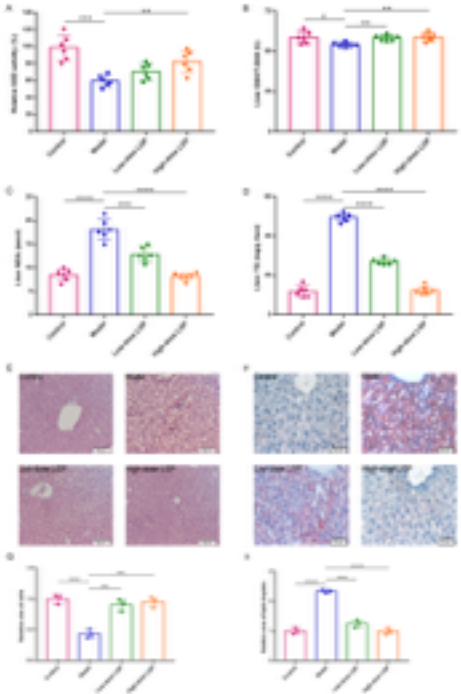
过量饮酒促进ROS的过量产生，导致氧化应激和关键内源性抗氧化剂如GSH的耗竭。

- 乙醇暴露显著降低了模型组的SOD活性和GSH/T-GSH比率（图3A和B）。值得注意的是，用50 mg/kg的LGP处理显著将SOD活性和GSH/T-GSH比率恢复至接近正常对照水平，表明LGP通过增强抗氧化防御系统减轻了酒精诱导的氧化应激。

酒精引起的三羧酸（TCA）循环破坏和脂肪酸氧化受损进一步导致肝脏脂质代谢功能障碍和脂质积累。MDA是脂质过氧化的副产物，是细胞膜氧化损伤的关键生物标志物。

- 在本研究中，乙醇处理的小鼠MDA水平显著升高，而LGP组别，尤其是高剂量，导致MDA水平显著降低，使其接近对照组观察到的水平（图3C），表明LGP有

3.1 RTB 文献资料支持-护肝



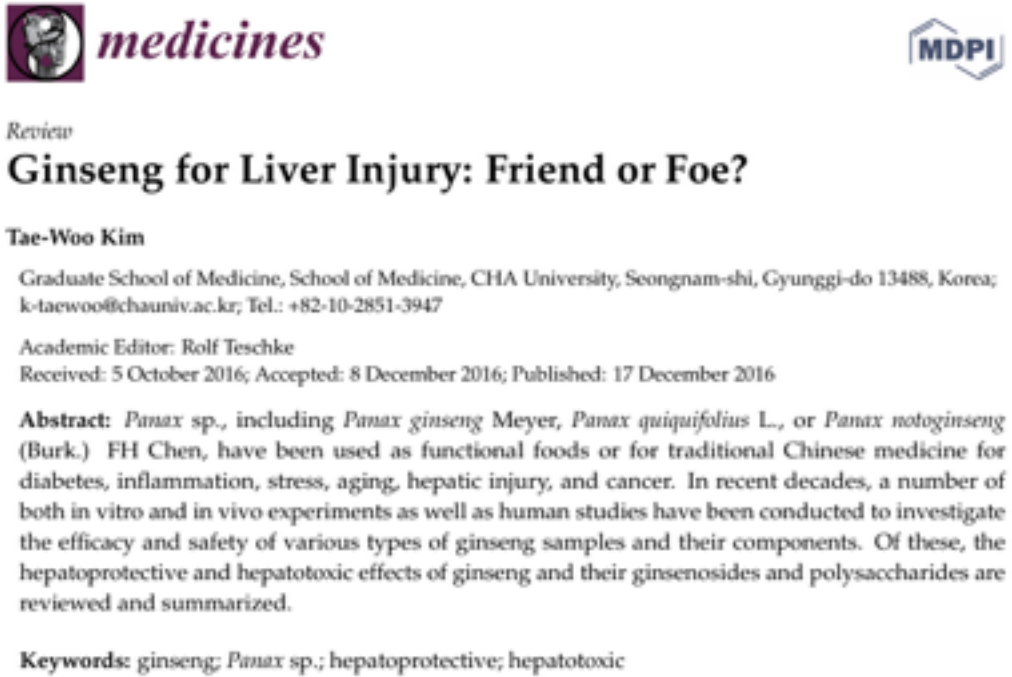
3.4. LGP alleviated alcohol-induced histopathological damage and hepatic lipid accumulation

To assess hepatic tissue integrity and lipid deposition, histological analyses were conducted using hematoxylin and eosin (H&E) staining and Oil Red O staining. Liver sections from the control group displayed normal hepatic architecture, characterized by well-defined cell boundaries, uniform hepatocyte size, and distinct nuclei. In contrast, the alcohol-treated model group exhibited pronounced pathological changes, including hepatocellular edema, inflammatory infiltration, lipid droplet accumulation, and focal necrosis (Fig. 3E and G). Treatment with low-dose LGP markedly reduced necrotic lesions, whereas high-dose LGP (50 mg/kg) substantially restored hepatic architecture, closely resembling normal morphology (Fig. 3E and G). Oil Red O staining revealed severe steatosis in the model group (Fig. 3F and H). LGP treatment significantly reduced lipid droplet accumulation in a dose-dependent manner, demonstrating its effectiveness in alleviating alcohol-induced hepatic steatosis (Fig. 3F and H). Furthermore, hepatic TG content was significantly increased in ethanol-treated mice but was notably decreased by LGP administration; high-dose LGP (50 mg/kg) restored TG levels comparable to those of the control group (Fig. 3D).

为了评估肝组织完整性和脂质沉积，使用苏木精和伊红（H&E）染色和油红O染色进行了组织学分析。

- 对照组的肝脏切片显示正常的肝脏结构，其特征是界限清晰的细胞边界、均匀的肝细胞大小和清晰的细胞核。相比之下，酒精处理的模型组表现出明显的病理变化，包括肝细胞水肿、炎症浸润、脂滴积聚和灶性坏死（图3E和G）。低剂量LGP处理显著减少了坏死病变，而高剂量LGP（50 mg/kg）则基本恢复了肝脏结构，接近于正常形态（图3E和G）。
- 油红O染色显示模型组存在严重的脂肪变性（图3F和H）。LGP处理以剂量依赖的方式显著减少了脂滴积累，证明了其在缓解酒精诱导的肝脂肪变性方面的有效性（图3F和H）。此外，乙醇处理的小鼠肝脏TG含量显著增加，但LGP给药后显著降低；高剂量LGP（50 mg/kg）

3.1 RTB 文献资料支持-护肝



medicines

MDPI

Review

Ginseng for Liver Injury: Friend or Foe?

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Abstract: *Panax* sp., including *Panax ginseng* Meyer, *Panax quiquifolius* L., or *Panax notoginseng* (Burk.) FH Chen, have been used as functional foods or for traditional Chinese medicine for diabetes, inflammation, stress, aging, hepatic injury, and cancer. In recent decades, a number of both in vitro and in vivo experiments as well as human studies have been conducted to investigate the efficacy and safety of various types of ginseng samples and their components. Of these, the hepatoprotective and hepatotoxic effects of ginseng and their ginsenosides and polysaccharides are reviewed and summarized.

Keywords: ginseng; *Panax* sp.; hepatoprotective; hepatotoxic

- 发表期刊：Medicines
- 发表年份：2016年
- 标题：人参对肝损伤：朋友还是敌人?
- 证据等级：V级

3.1 RTB 文献资料支持-护肝

Table 1. Ginseng species and their constituents used in the hepatoprotective experiments.

Cause of Liver Failure	Tested Ginseng		Evaluated Parameter	Reference
	Species	Constituent		
Ethanol	RG	Rg3, Rh2	AST, ALT, MDA	[24–26]
	PN	Rg1	AST, ALT, collagen, TGF-β1, Smad	[27–29]

2.1. Ethanol-Induced Hepatic Injury

The pathogenesis of ethanol-induced hepatic injury involves three stages: ethanol-induced (alcoholic) fatty liver, alcoholic hepatitis, and alcoholic cirrhosis. Alcoholic fatty liver is thus the preceding condition to the more severe pathologies. It is therefore critical to reverse ethanol-induced hepatic injury at the alcoholic fatty liver stage. RG extract improves liver function by attenuating ethanol-induced steatosis and oxidative stress [24]. Chronic ethanol-induced hepatic injury condition leads to elevations in serum aspartate transaminase (AST) and alanine transaminase (ALT). However, pretreatment with RG extract maintained serum ALT activity and malondialdehyde (MDA) concentration within the normal range after short-term ethanol ingestion [25]. Of the constituents responsible for the hepatoprotective effect of RG, Rg3 and Rh2 were found to be the primary ginsenosides of RG that produced medicinal effects against ethanol-induced oxidative injury [26]. The second stage of ethanol-induced hepatic injury is alcoholic hepatitis. Given that the next stage is liver cirrhosis, it is imperative to reverse alcoholic hepatitis to prevent further pathologic progression. PN attenuates the rise in serum AST and ALT due to chronic ethanol-induced hepatotoxicity [27]. More specifically, PN reduces liver ALT and AST levels, collagen fiber deposition, and transforming growth factor (TGF)-β1 expression that are normally observed with the increase in the expression quantity of SMA/MAD homology protein 7 (Smad7) in rats with ethanol-induced liver injury [28]. In particular, ginsenoside Rg1 isolated from PN has been found to suppress ethanol-induced elevations in blood AST and ALT, and TNF in rats with alcoholic hepatitis, which significantly decreases pathological injury as well as improving liver function [29]. These findings suggest that the ameliorating effects of ginsengs, including PG and PN, against ethanol-induced hepatitis may be attributed to their constituents, i.e. such as ginsenosides Rg1, Rg3, and Rh2, which potently inhibit reactive oxygen species (ROS) production and collagen deposition in the blood and liver.

- 红参提取物通过减轻乙醇诱导的脂肪变性和氧化应激来改善肝功能。
- 慢性乙醇诱导的肝损伤状况导致血清天冬氨酸转氨酶（AST）和丙氨酸转氨酶（ALT）升高。然而，预处理红参提取物在短期乙醇摄入后使血清ALT活性和丙二醛（MDA）浓度维持在正常范围内。
- 在红参保肝作用的成分中，Rg3和Rh2被发现是红参中对乙醇诱导的氧化损伤产生药用作用的主要人参皂苷。
- 三七降低了乙醇诱导肝损伤大鼠的肝脏ALT和AST水平、胶原纤维沉积和转化生长因子（TGF）-1表达，同时增加了SMA/MAD同源蛋白7（Smad7）的表达量。特别是，从三七中分离的人参皂苷Rg1已被发现能抑制乙醇诱导的酒精性肝炎大鼠血液AST、ALT和TNF的升高，这显著减少了病理损伤并改善了肝功能。

这些发现表明，人参、三七对乙醇诱导的肝炎的改善作用可能归因于其成分，即人参皂苷Rg1、Rg3和Rh2，它们能有效抑制血液和肝脏中活性氧（ROS）的产生和胶原沉积。

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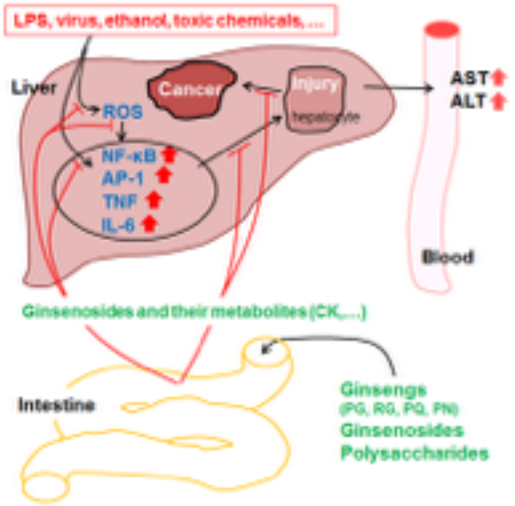

Figure 2. The hepatoprotective mechanism of ginsengs and their constituents proposed in in vitro and in vivo experiments. AP-1, activator protein 1; AST, aspartate transaminase; ALT, alanine transaminase; CK, compound K; LPS, lipopolysaccharide; , IL-6, interleukin 6; NF-κB, nuclear factor kappaB; PG, *Panax ginseng* Meyer; PN, *Panax notoginseng* (Burk.) FH Chen; PQ, *Panax quinquifolius* L.; and ROS, reactive oxygen species; TNF, tumor necrosis factor.

体外和体内实验提出的人参及其成分的肝保护机制。

Table 1. Ginseng species and their constituents used in the hepatoprotective experiments.

Cause of Liver Failure	Tested Ginseng		Evaluated Parameter
	Species	Constituent	
Ethanol	RG	Rg3, Rh2	AST, ALT, MDA
	PN	Rg1	AST, ALT, collagen, TGF-β1, Smad

5. Conclusions

Ginseng extract and its saponins exhibit hepatoprotective effects against various hepatic injuries caused by chemical substances and hepatitis viruses. These causes of hepatitis produce ROS and activate inflammation signaling pathways such as the NF-κB pathway. Ginseng potently inhibits ROS production and the inflammation-signaling pathway. Moreover, as unique constituents of ginseng, ginsenosides have been found to inhibit liver carcinoma proliferation, promote liver regeneration, and prevent liver ischemia through anti-oxidative, anti-inflammatory, and anti-apoptotic mechanisms (Figure 2). Moreover, when used with vitamin or acetaminophen, the ginseng components were observed to exert additive or synergistic effects on hepatic injuries. However, these ginseng components inhibit CYP3A4, leading to serious hepatotoxicity when used with imatinib or raltegravir. In this respect, when a ginseng extract or its component is used clinically along with other drugs, the extract or component needs to be examined carefully to determine if it inhibits a particular CYP450 enzyme responsible for drug metabolism, as it might lead to increased efficacy and ultimately serious hepatotoxicity.

结论：人参提取物及其皂苷对由化学物质和肝炎病毒引起的各种肝损伤表现出保肝作用。这些肝炎原因产生ROS并激活炎症信号通路，如NF-kB通路。人参有效抑制ROS产生和炎症信号通路。此外，作为人参的独特成分，人参皂苷已被发现通过抗氧化、抗炎和抗凋亡机制抑制肝癌增殖，促进肝脏再生和预防肝脏缺血

3.2 RTB 文献资料支持—促进酒精代谢

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Research Article

Li-Ginseng powder protects against alcohol-induced liver injury by promoting acetaldehyde clearance and cellular homeostasis

Ya-Ni Wang [a,1], De-Yu Wang [a,1], Yu-Hui Li [a], Cheng-Yan He [b], Xu-Ming Li [c], Yang Li [a,*], Ying-Hua Jin [a,**]

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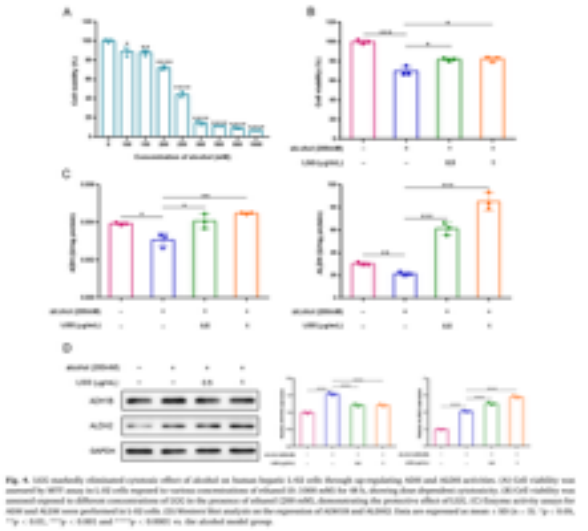
- 证据等级： V级

3.2 RTB 文献资料支持-促进酒精代谢

实验类型	产品	测试指标
细胞实验 使用乙醇处理L-02细胞（人正常肝细胞），建立酒精性肝细胞损伤模型	李人参皂苷组分（LGG，从LGP中提取）	➢ 乙醇代谢：ADH和ALDH的酶活性、乙醛含量、ADH1B和ALDH2的蛋白表达。 ➢ 氧化应激与线粒体功能：ROS水平、线粒体膜电位。 ➢ 自噬：自噬标志蛋白（LC3 II, p62）的表达水平。

3.2 RTB 文献资料支持-促进酒精代谢



3.5. LGG enhanced alcohol metabolism by upregulating ADH and ALDH activities

Building upon the observed hepatoprotective effects of LGP in vivo, further mechanistic investigations were conducted using human hepatic L-02 cells. The MTT result showed ethanol inhibited L-02 cell proliferation in a dose-dependent manner, with the IC_{50} of 238 mM (Fig. 4A). Therefore, a final ethanol concentration of 200 mM was selected for subsequent experiments. Co-treatment with LGG at concentrations of 0.5 and 1 μg/mL significantly improved cell viability compared to ethanol exposure alone, indicating a potential cytoprotective effect of LGG (Fig. 4B).

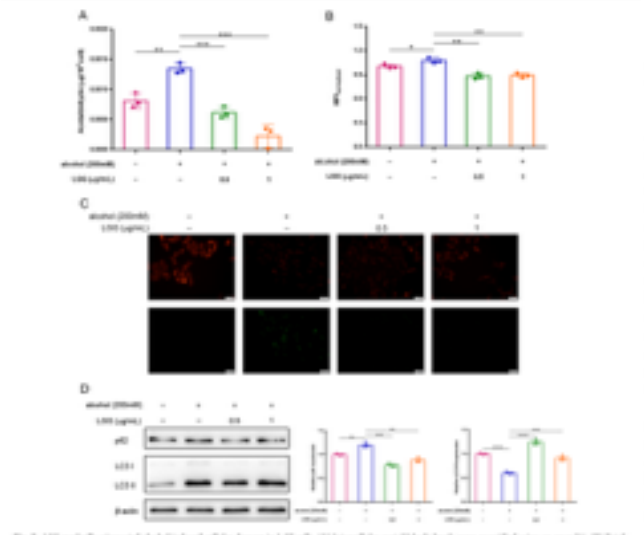
Ethanol metabolism primarily occurs in the liver, where alcohol dehydrogenase (ADH) catalyzes the conversion of ethanol to acetaldehyde in the cytosol, followed by the oxidation of acetaldehyde to acetate by aldehyde dehydrogenase (ALDH) in mitochondria [24]. To elucidate whether LGG modulates this metabolic pathway, we assessed the enzymatic activities of ADH and ALDH. Ethanol exposure significantly reduced the activities of both enzymes compared to untreated controls (Fig. 4C). However, LGG significantly enhanced the enzymatic activities of ADH and ALDH compared to the alcohol group, particularly at 1 μg/mL, suggesting its role in promoting alcohol clearance (Fig. 4C). Western blot analysis further demonstrated that ethanol treatment increased the protein expression levels of ADH1B and ALDH2, potentially as an adaptive response to elevated intracellular acetaldehyde. Interestingly, LGG intervention significantly upregulated ALDH2 protein levels relative to the alcohol group, while simultaneously downregulating ADH1B expression (Fig. 4D), suggesting that the regulation of the ADH family by ethanol may not be limited to ADH1B, but rather a more complex compensatory mechanism exists. These results suggested that LGG promotes alcohol metabolism by increasing enzymatic activities of ADH and ALDH, thereby contributing to its hepatoprotective effects.

总结：结果表明，LGG通过增加ADH和ALDH的酶活性来促进酒精代谢，从而有助于其肝保护作用。

乙醇代谢主要发生在肝脏，其中乙醇脱氢酶（ADH）在胞质中催化乙醇转化为乙醛，随后乙醛在线粒体中被乙醛脱氢酶（ALDH）氧化为乙酸。

- 与未处理的对照组相比，乙醇暴露显著降低了两种酶的活性（图4C）。然而，与酒精组相比，LGG显著增强了ADH和ALDH的酶活性，特别是在1 μg/mL时，表明其在促进酒精清除中的作用（图4C）。
- 蛋白质印迹分析进一步证明，乙醇处理增加了ADH1B和ALDH2的蛋白表达水平，这可能是对细胞内乙醛升高的适应性反应。有趣的是，与酒精组相比，LGG干预显著上调了ALDH2蛋白水平，同时下调了ADH1B的表达（图4D），这表明乙醇对ADH家族的调控可能不限于ADH1B，而是存在更复杂的补偿机制。

3.2 RTB 文献资料支持-促进酒精代谢



LGG显著减轻了L-02细胞中酒精诱导的细胞损伤

3.6. LGG attenuated alcohol-induced cellular damage by reducing acetaldehyde and ROS, increasing mitochondrial membrane potential and promoting autophagy

Ethanol is primarily metabolized in the liver, where it is oxidized by alcohol dehydrogenase (ADH) into acetaldehyde, a highly toxic intermediate that induces hepatotoxicity by disrupting cellular membranes and organelles, forming adducts with proteins and DNA, and impairing normal physiological functions [35]. Measurement of acetaldehyde levels revealed that the LGG treated group exhibited significantly lower concentrations compared to the ethanol only group, and even lower than those in the normal control group (Fig. 5A), indicating that LGG possesses a strong ability to eliminate acetaldehyde derived from both ethanol metabolism and endogenous cellular processes. Ethanol metabolism also generates excessive ROS, contributing to oxidative stress and mitochondrial dysfunction [36]. Our results showed that ethanol exposure led to a significant increase in total ROS and a marked reduction in mitochondrial membrane potential compared to the control group. As expected, LGG treatment effectively reversed these alterations (Fig. 5B and C).

Emerging evidence indicates that autophagy plays a key cytoprotective role in alcohol-induced liver injury, with enhanced autophagic flux helping to alleviate hepatocellular damage [37,38]. We therefore examined the expression of key autophagy-related proteins. Western blot analysis revealed that ethanol treatment increased both LC3 I and LC3 II levels but reduced the LC3 II/LC3 I ratio, suggesting that while autophagy initiation was activated, oxidative stress and mitochondrial damage led to impaired autophagic flux (Fig. 5D). In contrast, LGG treatment reduced LC3 I expression and significantly increased the LC3 II/LC3 I ratio, indicating restoration of autophagic flux and improved autophagy efficiency. Additionally, the autophagy substrate p62, which was upregulated by ethanol, was markedly decreased by LGG, further supporting the activation of autophagy in LGG-treated cells (Fig. 5D).

Collectively, these findings suggest that LGG mitigates alcohol-induced cellular injury by promoting acetaldehyde clearance, reducing oxidative stress, restoring mitochondrial function, and enhancing autophagic activity.

乙醇主要在肝脏代谢，被乙醇脱氢酶（*ADH*）氧化成乙醛，乙醛是一种高毒性中间体，通过破坏细胞膜和细胞器、与蛋白质和*DNA*形成加合物以及损害正常生理功能来诱导肝毒性。

- 乙醛水平的测量显示，LGG处理组表现出显著低于乙醇组的浓度，甚至低于正常对照组（图5A），表明LGP具有强大的清除来自乙醇代谢和内源性细胞过程的乙醛的能力。
- 乙醇代谢还会产生过量的ROS，导致氧化应激和线粒体功能障碍。与对照组相比，乙醇暴露导致总ROS显著增加，线粒体膜电位显著降低。正如预期的那样，LGG处理有效逆转了这些改变（图5B和C）。
- 证据表明，自噬在酒精性肝损伤中起着关键的细胞保护作用，增强的自噬流有助于减轻肝细胞损伤。蛋白质印迹分析显示：乙醇处理增加了LC3 I和LC3 II的水平，但降低了LC3 II/LC3 I的比率，表明虽然自噬启动被激活，但氧化应激和线粒体损伤导致自噬流受损（图5D）。相比之下，LGG处理降低了LC3 I的表达，并显著提高了LC3 II/LC3 I的比率，表明自噬流得到恢复，自噬效率得到改善。此外，被乙醇上调的自噬底物p62被LGG显著降低，进一步支持了

总结：结果表明，LGG通过促进乙醛清除、减少氧化应激、恢复线粒体功能和增强自噬活性来减轻酒精诱导的细胞损伤。

THANK YOU