LivPhcD™

BEST SOLUTION FOR LIVER HEALTH

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AN INGREDIENT WHICH TRULLY PROTECTS YOUR LIVER

BEST SOLUTION FOR YOUR LIVER HEALTH

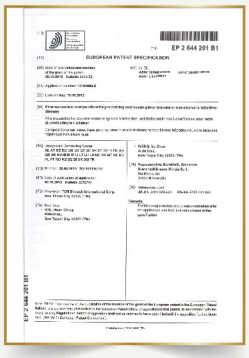
WHAT IS LivPhcD™ ?

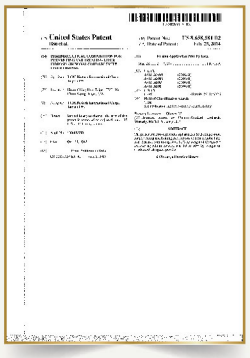
LivPhcD™ is the mycelium of *Paecilomyces hepiali* that rich in adenosine. Adenosine is a nucleoside which plays an important role in various physiological processes,including energy metabolism and neurotransmission.

PATENT FERMENTATION AND PRODUCTION TECHNIQUES

LivPhcD™ is a cutting-edge ingredient which created using patent fermentation and production techniques.
The process involves the analysis of the components of the fermentative broth at different stages in order to determine the optimal cultivation conditions.

PATENTS PORTFOLIO





Pharmaceutical composition for preventing and treating liver fibrosis or nonalcoholic fatty liver disease

Taiwan	TWI501771, TWI554277	Japan	JP6050099
US	US8,858,954, US8,658,181	European	EP2644201B1

Manufacturing patent

US Patent No. US8722056 B2

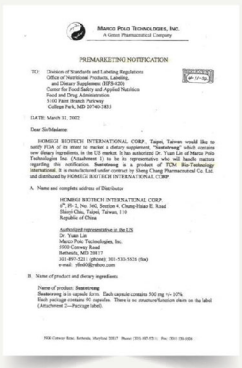
SAFETY OF LivPhcD™

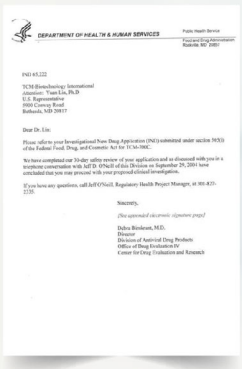
FDA APPROVAL

FDA confirmed that LivPhcD™ was a natural botanical ingredient with decent quality safe enough to be used as dietary supplements' new ingredient in the American market and therefore granted it a pre-market notification.

FDA AGREES LivPhcD™ CAN :

- Promote general health
- Boost energy
- Modulate physical functions


PREMARKETING NOTIFICATION


DEPARTMENT OF HEALTH & HUMAN SERVICES

TOXICOLOGICAL STUDIES

STUDY	SAFETY
Acute toxicity testing	✓
Genotoxicity test-Ames test	✓
Genotoxicity test- Analysis of human lymphocyte chromosomes	✓
Genotoxicity test- Analysis of animal micronucleus	✓
Sub-acute toxicity test	✓

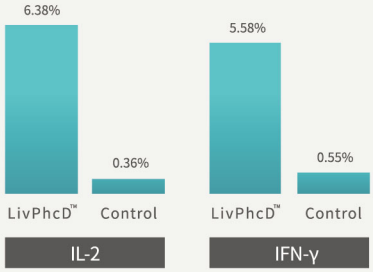
SCIENTIFIC EVIDENCES

EFFICACY INGREDIENT CONTENT

	ADENOSINE	POLYSACCHARIDE
LivPhcD™	>0.35%	>4.0%

IMMUNITY

Activate immunological receptors, CD4 and CD8, then release of IL-2 and IFN-γ.



ANTI-AGING

β-galactosidase inhibition rate is 25%.

ANTIOXIDANT

IN VITRO

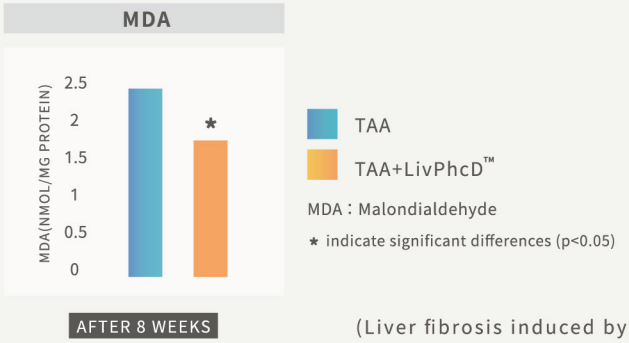
- DPPH scavenging activity is equivalent to $2.5*10^3$μg/ml Vit. E
- Superoxide anions scavenging activity is equivalent to $4.04*10^3$U/g SOD

IN VIVO

- Elevate liver antioxidant enzyme (CAT, SOD, GPX)

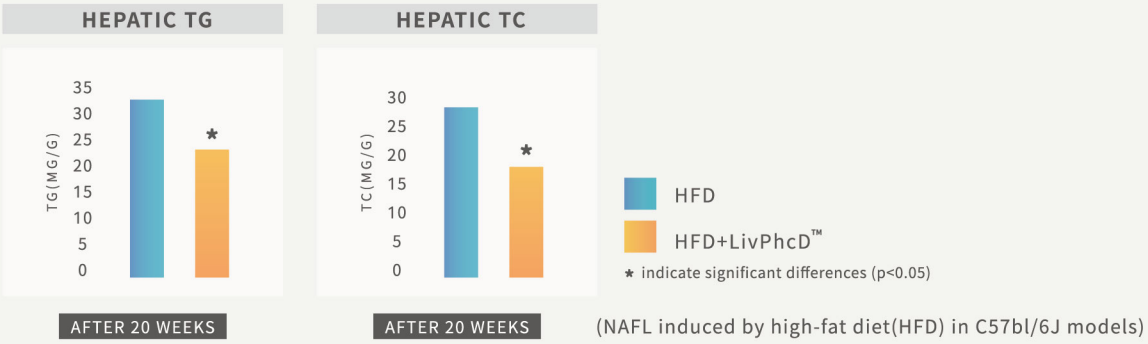
(Liver fibrosis induced by injecting thioacetamide (TAA) in Wistar models)

- Reduce the level of liver lipid peroxidation



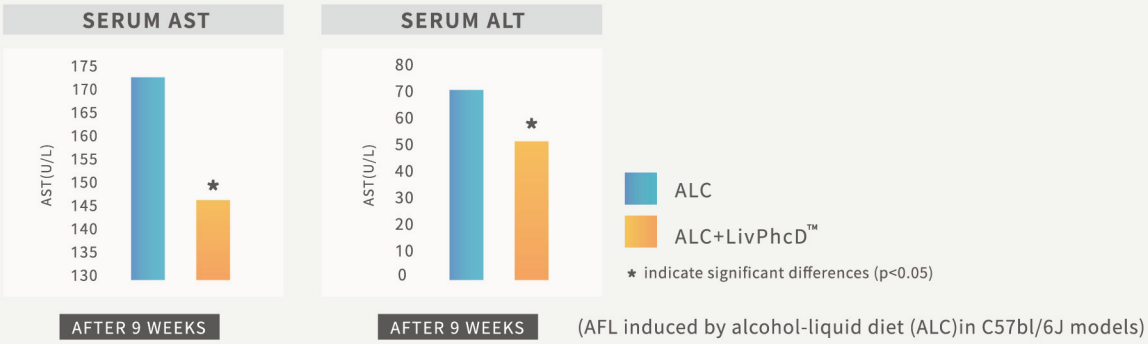
(Liver fibrosis induced by injecting thioacetamide (TAA) in Wistar models)

LOWER HEPATIC TG (TRIGLYCERIDE) & CHOLESTEROL



(NAFL induced by high-fat diet(HFD) in C57bl/6J models)

LOWER AST & ALT



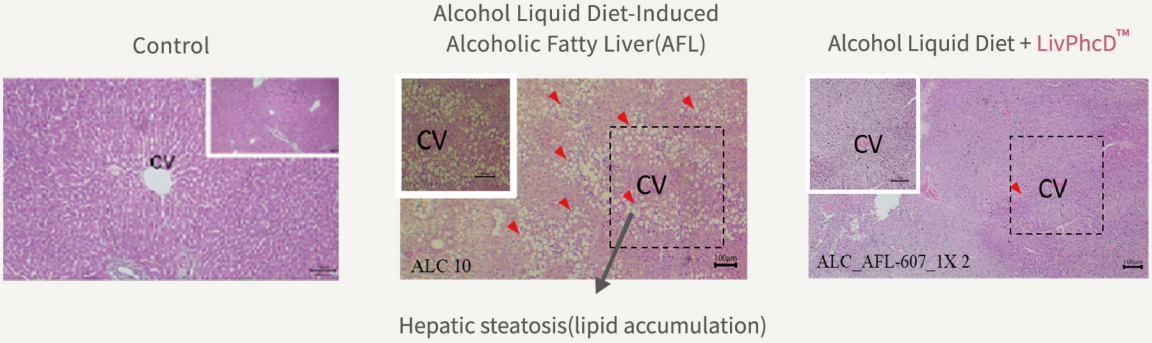
(AFL induced by alcohol-liquid diet (ALC)in C57bl/6J models)

ALCOHOLIC FATTY LIVER (AFL)

AFL is a liver disease caused by long-term excessive consumption of alcohol and characterized by ballooning of hepatocytes, lipid droplets deposition and inflammatory cells infiltration. Lipid accumulation plays a pivotal role in the occurrence of AFL. Therefore, the regulatory mechanism of steatosis remains to be supplemented.

Reduce lipid accumulation in hepatocytes in AFL models

- AFL induced by alcohol-liquid diet in C57bl/6J models
- Feed for 9 weeks

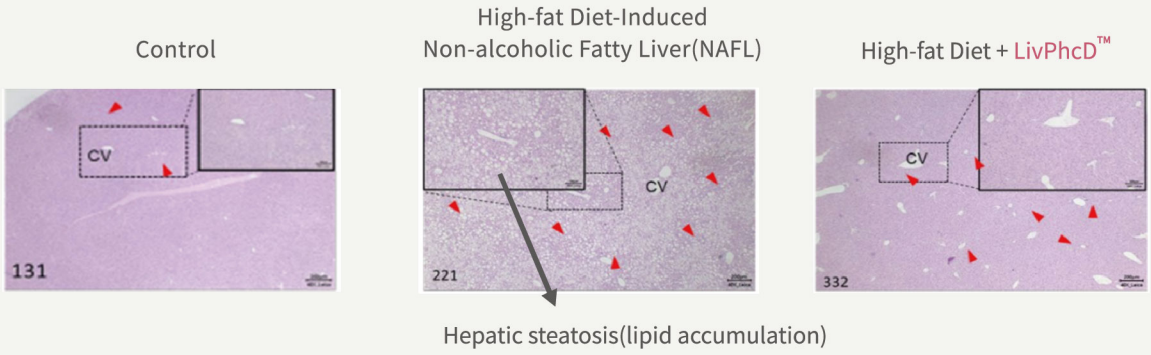


NON-ALCOHOLIC FATTY LIVER (NAFL)

Nearly 25% of the world's population is affected by NAFLD. NAFLD is a disease in which excessive fat accumulates in the liver without alcohol abuse. Fat accumulation can be caused by disordered metabolism of fatty acids by hepatocytes.

Reduce lipid accumulation in hepatocytes in NAFL models

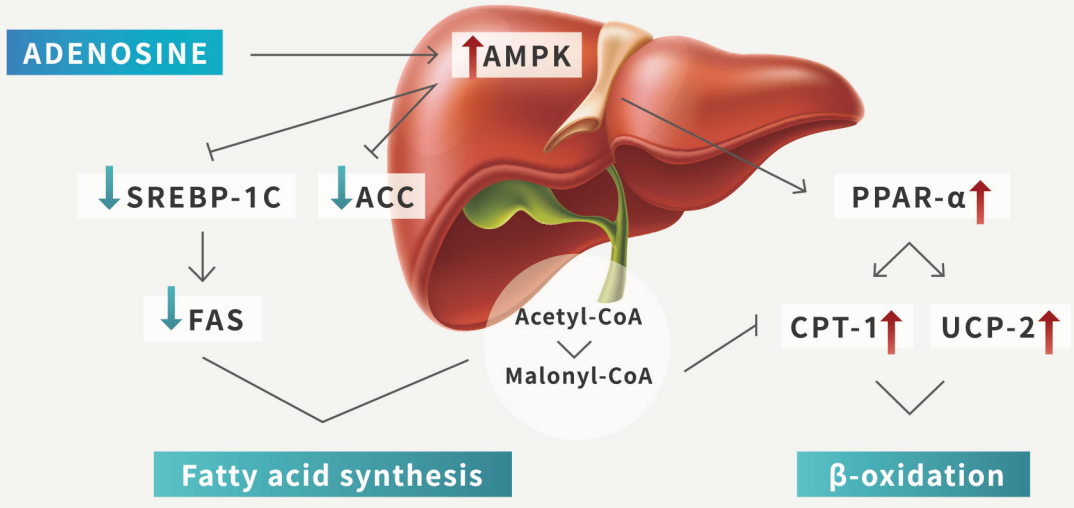
- NAFL induced by high-fat diet in C57bl/6J models
- Feed for 20 weeks



Activate AMPK pathway, leading to the stimulation of fatty acid oxidation and inhibition of lipogenesis

AMPK：AMP Activated Protein Kinase
SREBP-1C：Sterol Regulatory Element-Binding Protein 1C
PPAR-α：Peroxisome Proliferator-Activated Receptor α

ACC: Acetyl-CoA Carboxylase
FAS: Fatty Acid Synthase
CPT-1: Carnitine Palmitoyl Transferase 1
UCP-2：Uncoupling Protein 2



↓↑：Mechanism of LivPhcD™

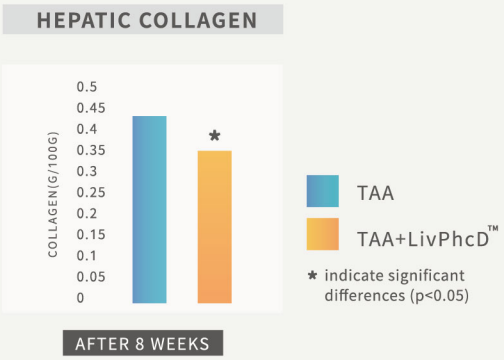
LIVER FIBROSIS

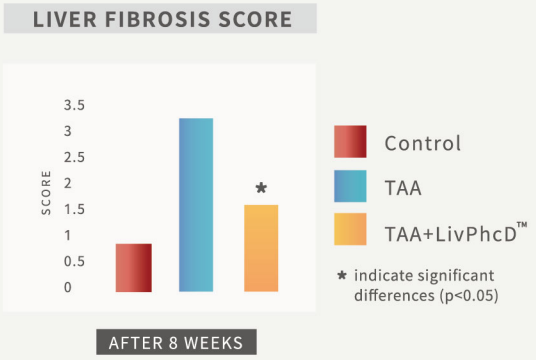
Liver fibrosis develops when the liver is repeatedly or continuously damaged which causes the excessive accumulation of extracellular matrix proteins including collagen occurring in most types of chronic liver diseases. Advanced liver fibrosis results in cirrhosis, liver failure, and portal hypertension and often requires liver transplantation.

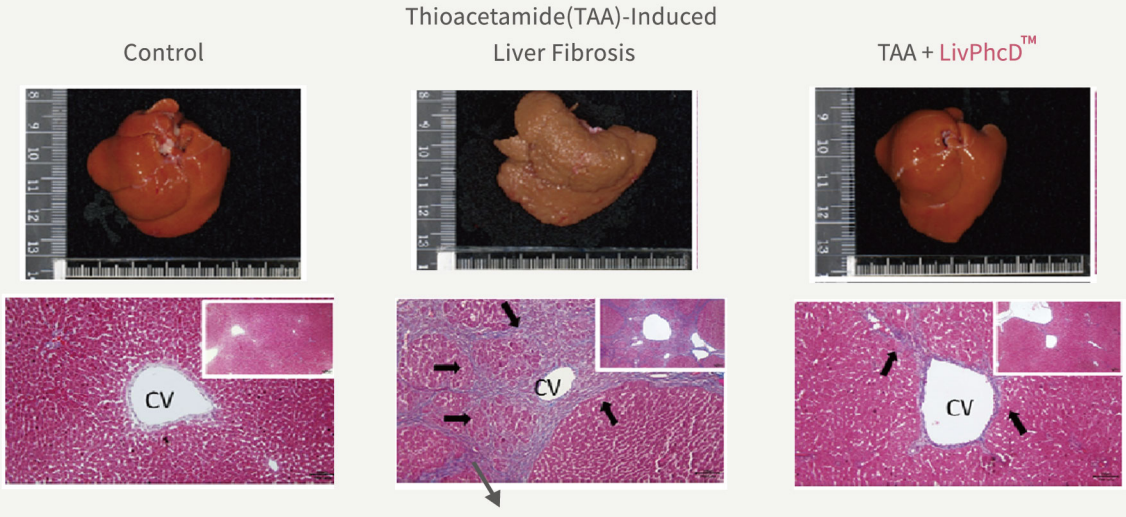
Reduce collagen in hepatocytes in liver fibrosis models

Reduce fibrosis in hepatocytes in TAA models

- Liver fibrosis induced by injecting thioacetamide(TAA) in Wistar models
- Feed for 8 weeks







Blue: collagen(fibrosis)

URL:https://www.tcmbio.com/en/science_5.php

References available upon request.

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LiV PhcD™ TCM BIOTECH INTERNATIONAL CORP.