LV-GB Complex™

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Science first.

By David Brady, ND, DC, CCN, DACBN & Suzanne Copp, MS

LV-GB Complex™

LV-GB Complex provides support for liver and gallbladder function by providing lipotropic substances to aid in the elimination of fatty substances from the liver, as well as promoting proper bile flow (i.e.: L-methionine, L-taurine, inositol and choline, beta-carotene, ox bile). Critical catalysts of hepatic detoxification enzymes are also included (i.e.: pyridoxyl-5-phosphate form of B6 and B12). These are mixed with a combination of hepatic (aid the liver) and cholagogue (aid bile flow) herbs, such as Milk Thistle, Greater Celandine, Dandelion, Fringe Tree, Artichoke, and Beet root for optimal processing and elimination of toxins.

Supplement Facts

Serving Size 3 capsules
Servings Per Container 30

Amount Per Serving		% Daily Value
Vitamin A (as Beta-Carotene)	5000 IU	100%
Vitamin B-6 (as Pyridoxal-5-Phosphate)	5 mg	250%
Vitamin B-12 (as Methylcobalamin)	15 mcg	250%
L-Methionine	150 mg	*
Taurine	100 mg	*
Inositol	100 mg	*
Lecithin (from soy)	100 mg	*
Milk Thistle (*Silybum marianum*)(seed) [standardized to contain 80% silymarin]	100 mg	*
Ox Bile	75 mg	*
Greater Celadine (*Chelidonium majus*) (whole herb)	50 mg	*
Dandelion (*Taraxacum officinale*)(root) [standardized to contain 20% flavonoids]	50 mg	*
Fringe Tree (*Chionanthus virginicus*)(bark)	50 mg	*
Artichoke (*Cynara scolymus*)(leaf) [standardized to contain 5% cynarin]	50 mg	*
Beet Powder (*Beta vulgaris L.*)(tuber)	25 mg	*

*Daily Value not established.

Other Ingredients: Microcrystalline cellulose, vegetable stearate, rice flour.

Dandelion *(Taraxicum officnale)* is a well-established cholagogue (stimulates bile flow), diuretic and appetite stimulant. Milk Thistle *(Silymarin marianum)* is a well-researched protective herb for the hepatocytes (liver cells), and has even demonstrated the ability to promote the regeneration of liver cells in subjects with hepatitis and other liver disorders. It can also aid in the flow of bile to promote optimal gallbladder function. Greater Celandine *(Chelidonium majus)*, Fringe Tree *(Chionanthus virginicus)*, and Artichoke *(Cynara scolymus)* can reduce pain in the bile ducts and the gastrointestinal tract by virtue of their antispasmodic properties, and they are often traditionally used in liver and gallbladder disorders, including non-obstructive gallstones. Beet Leaf *(Beta vulgaris)* is a valuable source of betaine (trimethylglycine), which can act to reduce fatty infiltration and degeneration of the liver. LV-GB Complex is designed to also support optimal digestion and assimilation of essential fats and fat soluble vitamins making this formula appropriate for patients who have had their gall bladder removed surgically and patients suffering from skin disorders.

Who Should Take LV-GB Complex™?

Patients without a gallbladder, patients needing to improve liver or gallbladder function, those with inability to handle fatty foods and those with bloating, gas, GI distress, or skin problems. This synergistic formula will aid fat digestion and improve absorption of fat-soluble vitamins. This product is excellent for detoxification support.

Who Should Not Take LV-GB Complex™?

Patients experiencing acute upper abdominal pain or are known to have a bile duct obstruction should not take LV-GB Complex.

How Should a Patient Take LV-GB Complex™?

Take three capsules per day with meals, or as directed by your health care practitioner.

References

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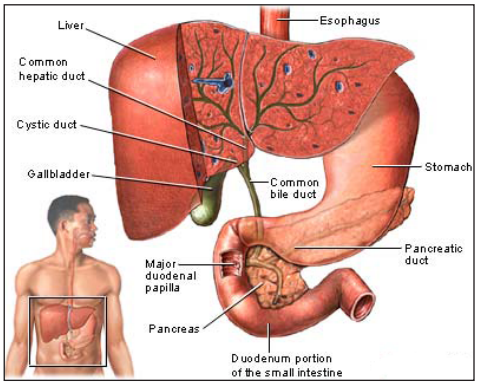
Do you have patients with a history of chronic acetaminophen (Tylenol®) use?

If so, consider the following nutritional support: **SAMe, NAC, LV-GB Complex, Amino-D-Tox and Three A Day Antioxidant**

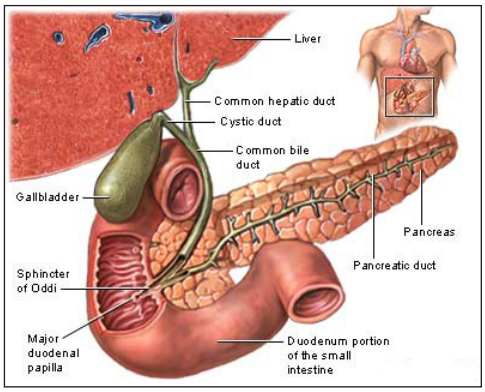
■ *For full protocol on acetaminophen protection (or other protocols) go to* ***www.designsforhealth.com***

S-Adenosylmethionine protects against acetaminophen-induced hepatotoxicity in mice.
Pharmacology. 2004 Aug;71(4):199-208., Song Z, McClain CJ, Chen T.
Department of Medicine, University of Louisville Medical Center, Louisville, KY 40292, USA.



An overdose of acetaminophen (APAP) is the most frequent cause of fulminant liver failure in the United States. Increasing evidence demonstrates that oxidative stress plays an important etiologic role in APAP-induced liver injury. S-Adenosylmethionine (SAMe) is a key intermediate in the hepatic trans-sulfuration pathway and serves as a precursor for glutathione (GSH) as well as the methyl donor in most transmethylation reactions. In the present study, we investigated effects of SAMe on liver injury induced by APAP administration in male C57BL/6 mice. Two related studies were performed. In the first experiment, SAMe (1g/kg BW) was injected intraperitoneally 4 h before APAP (600 mg/kg BW) administration. In the second experiment, SAMe was injected intraperitoneally 1 h after APAP administration. Our results showed that APAP administration induced changes typical of confluent centrilobular necrosis by histological examination and a marked elevation in serum alanine aminotransferase (ALT) activity. APAP administration induced significant decreases in both hepatic and blood SAMe concentrations. In addition, APAP decreased intracellular (both cytosolic and mitochondrial) GSH concentrations along with increased lipid peroxidation in conjunction with mitochondrial dysfunction as documented by Ca2+-induced mitochondrial permeability transition. **SAMe treatment (both before and after APAP) significantly attenuated the liver injury. Exogenous SAMe prevented the decrease in liver and blood SAMe concentrations. Moreover, SAMe treatment attenuated both cytosolic and mitochondrial GSH depletion as well as mitochondrial dysfunction. We conclude that SAMe at least in part protects the liver from APAP-induced injury by preventing intracellular GSH depletion and mitochondrial dysfunction.** Copyright 2004 S. Karger AG, Basel.

An update of N-acetylcysteine treatment for acute acetaminophen toxicity in children.
Curr Opin Pediatr. 2005 Apr;17(2):239-45, Marzullo L.
Division of Pediatric Emergency Medicine, University of Alabama at Birmingham, Birmingham, Alabama 35233, USA.



PURPOSE OF REVIEW: Acetaminophen poisoning accounts for a disproportionate percentage of all toxic ingestions, and can be life-threatening. This article reviews the mechanism and presentation of acetaminophen toxicity, as well as its treatment, including current thinking and treatment recommendations. RECENT FINDINGS: N-acetylcysteine acts to detoxify acetaminophen in several ways, but primarily by increasing the synthesis and availability of glutathione, which binds and inactivates the highly reactive and hepatotoxic acetaminophen metabolite N-acetyl-p-benzoquinoneimine. The US Food and Drug Administration has approved an intravenous formulation of N-acetylcysteine, thus allowing the treatment time to be decreased from the 72 hr most commonly used for the oral regimen, to only 20 hr. This comes after many years of accepted intravenous N-acetylcysteine use in Europe and Canada, and much controversy as to the superiority of both treatments. This review summarizes this controversy, and offers a framework to develop a safe treatment plan that has the optimal outcome for the patient, as well as reflecting knowledge of the potential caveats at work. It describes side effects of N-acetylcysteine treatment, as well as relative indications to choose one route of treatment over the other.

SUMMARY: Acetaminophen can lead to irreversible liver damage and even death in acute overdose. Outcome is related to the swiftness in which the antidote (N-acetylcysteine) is provided. In the United States, there are now available both the oral and intravenous forms of N-acetylcysteine, and pros and cons exist for each. With brisk and adequate treatment using either route, recovery can be complete, and liver function can be restored.