

Alpha Lipoic Acid and Liver Disease
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Alpha Lipoic Acid (ALA, thioctic acid, pyruvate oxidation factor) was first discovered by bacteriologist Irwin C. Gunsalus in 1948 when he observed that aerobic (oxygen-requiring) bacteria could not grow without it. Later, Gunsalus and Lester Reed determined the true structure and named it ALA (1951). **ALA is a natural substance**, produced in every higher-type cell, and it has many functions. Probably most importantly, ALA is the rate-limiting factor for the production of energy from carbohydrates (pyruvate). Without ALA, you could not obtain energy from the food you eat, and you could not stay alive.

ALA is also an excellent antioxidant and recycles other nutrients such as co-enzyme Q-10, vitamin C, and vitamin E. In addition, ALA chelates heavy metals such as mercury, lead, and arsenic, and it stabilizes NF kappa B transcription factor so that it helps to inactivate deleterious genes. It can also help people with diabetes mellitus by increasing the sensitivity of their cells to insulin, and it helps reverse diabetic neuropathies.

The first large human clinical studies using ALA in the United States were carried out by Drs. Fredrick C. Bartter, myself, and associates from the National Institutes of Health (NIH) in the 1970s. We administered ALA to **79 people with severe and acute liver damage** at various hospitals around the United States, and **75 recovered full liver function**.

Dr. Bartter and I were appointed by the FDA as principal ALA investigators, and I went on to use it successfully for the treatment of chronic liver disease. In combination with low-dose naltrexone, I have used ALA to treat various cancers for which no other treatment exists. (For more information, readers might want to go to [PubMed](#) and type in "liver, Berkson.")

My first experience using antioxidant therapy was in 1977, when I was an internal medicine resident. **A man was poisoned and suffering from acute liver failure**. His liver function tests were in the thousands of mg/dL, and he had propulsive diarrhea, projectile vomiting, and dreadfully painful muscle spasms throughout his body. He was the sickest person that I had ever seen. Due to the relentless muscle cramping and pain, he could not find a comfortable resting position. One of the department chiefs told me that nothing could be done to save his life except for an immediate liver transplant, however, a donor liver was not available. I was ordered to administer medical support and to just observe the patient as he went through the phases of

death. I was told to take notes and prepare a report for grand rounds at the hospital.

Death from liver necrosis usually involves four separate stages: (1) ingestion of a poison, such as acetaminophen, a poisonous mushroom, hepatotoxic hydrocarbon solvent, etc.; (2) development of acute and difficult gastroenteritis with dehydration, pain, and electrolyte depletion; (3) a noticeable recovery phase in which the patient is often released from the hospital in a weakened state; and (4) increased weakness followed by coma and death. Because I did not want to see this happen to my patient, I began a search for a way to reverse his condition.

Fortunately, I remembered reading an article about a new drug that had been shown to be helpful in the treatment of severe liver damage. The drug, alpha-lipoic acid (ALA) was stocked at the NIH by Fred Bartter, MD, the chief of endocrinology. Dr. Bartter was interested in this agent because he thought that because it lowered blood sugar levels, ALA might be used as a drug for diabetes mellitus and its complications.

About 30 hours after my patient had ingested the deadly toxins, the **intravenous (IV) ALA** was started. Within a few hours, the patient began to feel better. We were all surprised that he continued to improve, and he was soon discharged from the hospital with nearly normal laboratory values and feeling a little tired, but normal. **He is still well and free of liver disease, 30 years later.**

After I treated three more patients with severe liver damage with ALA and obtained the same remarkable results, most of the hospital chiefs were still skeptical, however, Dr. Bartter and I were delighted. NIH sent a team of doctors to Cleveland to examine my patients, and I was eventually awarded the FDA investigational drug permit for the use of IV ALA. Dr. Bartter and I published three papers describing our successes with IV ALA, and we expected a certain amount of interest in this remarkable organ regenerative protocol. We were disturbed by the lack of attention from the American medical community. Dr. Bartter died in 1985, and I continued to study ALA as a therapeutic agent and as a nutraceutical.

Since my work with Dr. Bartter, I have treated hundreds of patients with IV and oral ALA for acute and chronic liver damage, autoimmune disease, cancer, etc., along with other interesting agents with promising results.

Below are a few case studies of Hepatitis C taken from my office practice.

In my opinion, there are four **laboratory tests** that really tell a doctor what is going on in the liver.

The first is the **platelet count**. It is important because as liver inflammation and scarring progress, the platelet count goes down. So, the platelet count is a very helpful indirect indication of liver health, and a rise in platelet count is an indication of a healing liver.

I believe that the **albumin level** is the most important liver function test. A diseased liver can only produce a small amount of albumin. So a person with severe liver disease has a low albumin level, and as the liver improves, the albumin level rises.

The **ALT** is a liver enzyme that results from damage to the liver. It normally goes up and down from day to day, however, a downward trend may suggest an improvement of liver function. Interestingly enough, in cases of severe liver disease, the ALT is very low because most liver cells have been killed off.

The **prothrombin time** is a very important tool for measuring liver health, because a sick liver cannot produce much of the clotting factors, and thus the prothrombin time (a time it takes the blood to clot) is elongated in severe liver disease. As the liver regenerates, the prothrombin time shortens.

Case 1

Mr. CA, a 68-year-old salesman from Ohio was infected with hepatitis C, following a blood transfusion in the hospital. Soon afterwards, he became ill and was found to have hepatitis C. He was sent to a hepatologist who immediately put him on interferon and ribavirin, which made him feel as if he had influenza for several months, and the drugs ultimately damaged his bone marrow. After the failure of interferon/ribavirin, Mr. CA was told that nothing could be done other than liver transplantation.

Mr. CA presented to my office suffering from fatigue, anxiety, abdominal pain, and anemia, and his abdomen was distended with fluid (ascites). I treated him with my triple antioxidant therapy (ALA, selenium, and silymarin). Within a short time he began to feel normal and was free of the signs and symptoms of liver disease. Some of his results may be seen in **figures 1, 2, 3, and 4**.

Case 2

Mr. EA, a 54-year-old man from California was infected with hepatitis C during a blood transfusion following surgery. He did not feel well for several years following

surgery, and his physician did some laboratory tests that demonstrated hepatitis C. A liver biopsy showed moderate cirrhosis with active inflammation.

Mr. EA presented to my office with fatigue, anxiety, abdominal pain, and some ascites. His ALT was elevated, and his viral load was elevated by the Chiron PCR method. I treated him with my Triple Antioxidant Therapy, and within a few months, he started to feel normal. Some of his results are illustrated in **figures 5, 6, 7, and 8**.

Case 3

Mrs. KVP is a 40-year-old woman in excellent health who developed hepatitis C from a blood transfusion following surgery. Her family doctor sent her to a liver expert who told her that she was seriously ill and must be treated immediately with interferon and ribavirin. KVP had no complaints and had heard that the standard treatment often made people much sicker than doing nothing.

KVP presented to my office, and her blood tests were all normal, except her ALT liver enzyme was elevated at about 300 mg/dL. This indicated that there was viral activity and inflammation in her liver. KVP's original laboratory tests and her progress after being treated with my triple antioxidant therapy over three years are demonstrated in **figures 9, 10, 11, 12, and 13**.

After three years, she once again visited her hepatologist who told her that actually that she was getting sicker because her viral load had increased dramatically (Figure 12). Again, he said that she should be put on interferon and ribavirin and be evaluated for a liver transplant. Incidentally, she had great health insurance.

Mrs. KVP is a health professional and questioned her hepatologist. She asked him if the original viral load was acceptable. He said, yes, however, it had increased from 600,000 to 6,000,000 units, and that showed progression of her disease. She asked him if he knew that the first viral load tests were done by the Chiron method and the second tests were done by the Quantasure method. He did not know that. Then, she told him that viral load is an artificial exaggeration (amplification) of the amount of viruses by millions, and the Quantasure method appears to amplify the amount of viruses by ten times more than the Chiron method. After hearing this reasonable explanation, he answered that viral load was not a very important test anyway.

The three people described in this study continued to stay on the triple antioxidant therapy, and I still see two of them as patients today (Fall 2007). The two continue to improve.

In addition to **ALA**, I added **silymarin** and **selenium** to my **triple antioxidant therapy** because these agents also protect the liver from free radical damage, regenerate the other fundamental antioxidants, and interfere with viral replication. Although my first acute hepatic necrosis patients were treated with ALA alone and did exceedingly well, all the patients presented in this paper followed the triple antioxidant program and recovered quickly from their illness.

The standard-of-care treatments for severe liver damage, especially liver transplant surgery, can be painful, disabling, and extremely costly. From my experience in my practice, interferon and antivirals have less than a 30% improvement rate, and this response is usually not permanent. Liver transplant surgery in a few cases can be lifesaving and necessary, but it is uncertain and tentative, partly due to the residual viremia that ultimately infects the newly transplanted liver. I have found that the highest viral loads are seen following liver transplant surgery, since the residual viruses in the bloodstream and tissues have a new healthy liver on which to feed.

The triple antioxidant therapy offers a more conservative approach to the treatment of hepatitis C that is much less expensive. One year of antioxidant therapies described in this paper costs only a few thousand dollars, whereas liver transplant surgery costs more than \$400,000 a year, and in five years, the person will probably require a new transplant. And, in addition, the transplant patient will require anti-rejection drugs and many doctor and hospital visits. It appears reasonable to me that prior to transplant evaluation or during the transplant evaluation process, this conservative triple antioxidant treatment program should be considered. If there is a significant improvement in the patient's condition, liver transplant surgery may be avoided.

Not too long ago, I was invited by the Internal Medicine Society of Saxony to present my triple antioxidant protocol to the group, in Dresden, Germany. I was asked why viral loads did not always fall to very low levels with my treatment program. I answered that from a microbiologist's point of view I did not believe that one could ever completely eradicate a viral disease without killing the patient. I added that we could only hope to support and "teach" the immune system how to recognize and control a virus. Normally, viruses remain part of our biology for the rest of our lives. And this does not necessarily make a person sick. We are all filled with billions of dormant viruses. As long as we have a healthy lifestyle and avoid unnecessary emotional and physical stress, the viruses should remain

dormant. I believe that one can live to 100 years old with hepatitis C and still be a healthy person.

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