
Chapter 4

Hepatitis C: Toxins Such as Alcohol, Heroin, and Prescription Drugs Suffice as Explanations

*"Where is the hepatitis C virus?
Has anybody seen it?"¹*

Michael Houghton

Alleged co-discoverer of the HC virus

At the 8th International HCV Congress in Paris, 2001

"Toxic shocks like smoking or alcohol consumption can traumatize the liver, causing genetic instabilities. The human cell itself, then, can produce the genetic particles which are fished out by orthodox researchers with their PCR tests and simply interpreted as exogenous viruses. But before jumping on the virus bandwagon, one must have closely analyzed if these really are viruses—which has not happened with hepatitis C."

Richard Strohmman

Professor of Molecular and Cellular Biology

HIV Mania: Detonation for Antiviral Hepatitis C Therapy

Hepatitis C is commonly known as a liver infection caused by a virus (the so-called hepatitis C virus: HCV for short). According to theories, the disease is primarily transmitted through blood and blood products. In the 1970s, American researcher Jay Hoofnagle attempted to strike hepatitis C with medications. In 1978, he joined the US National Institutes of Health (NIH) to continue his research on treating liver diseases.

At this time, leading experts in this area, the hepatologists and even the pharmaceutical companies were still of the opinion that treatment of hepatitis C patients with antiviral medications was too difficult and too dangerous, since substances were so full of side effects, and, directly after ingestion, they landed in the organ that was stricken anyway: the liver. For that reason, advances in medication therapy could hardly be observed.

There were experiments with the antiviral interferon, which was tested on cancer patients. But these trials were anything but a success. Hoofnagle was of the opinion, however, that the antiviral preparations had the potential to fight hepatitis C, even though mainstream researchers didn't share Hoofnagle's optimism. "The idea of treating a liver disease [with medications] went against the grain," Hoofnagle told the medical journal *The Lancet* in 1997. "Liver disease was considered to be a good reason to avoid drug therapies."²

This is no surprise, since substances like interferon ultimately work like chemotherapy and for that reason can severely affect more than just the liver;³ it was also observed that, after interferon administration, herpes developed, or the number of white blood cells (leukocytes) decreased, something that signals a weakening in the immune system. Interferons could also influence the nervous system, causing psychological alterations like depression and confusion.⁴

The side effects of HCV medications are frequently so strong that treatment has to be stopped. "We need medications that are more effective and tolerable than current treatment forms with the active substances interferon-alpha and ribavirin," says Raffaele DeFrancesco, scientific director of the biochemical department at the Instituto Ricerche Biologia Molecolare in Rome. But DeFrancesco only meant that new medications should be developed to defeat the alleged virus.⁵

The virus mania pattern of thought had also infected theories about hepatitis. And so, all at once, the opinion was *en vogue* that liver diseases could, even must, be treated by antiviral medications.⁶

The damage to the human body and particularly to the liver caused by medications is typically less drastic than in the case of—still too often life-long—antiviral AIDS treatments. But, mainly because most patients diagnosed with hepatitis C have just a temporary treatment, with medications such as interferon and ribavirin. And even this frequently leads to severe anemia (iron deficiency) and high fever. Also a risk of cancer cannot be ruled out with ribavirin either, because it has effects similar to chemotherapy.

How To Create a Hepatitis C Virus

Mainstream science says that, based on their studies, hepatitis C is a virus with contagious potential. But the experiments carried out to prove this theory are highly questionable going back to 1978 and a paper published in *The Lancet*. Researchers took blood from four patients; it was assumed that they had obtained their non-A, non-B hepatitis (this is what hepatitis C was called until the late 1980s) through a viral infection via blood transfusion. They also drew blood from a blood donor who

had been mixed up in two hepatitis cases. Then, this blood serum was injected into the bloodstreams of five chimpanzees that had originally been caught in the wilderness of Sierra Leone in Africa.

But none of the animals contracted hepatitis (that is to say, they did not get liver disease). Around the 14th week, liver values were slightly raised for a few days, which can be interpreted as an immune reaction to foreign blood (and not a viral infection). To rule out the possibility that this was an immune reaction, the researchers should have taken a control group of chimpanzees and injected the same amounts of blood from healthy people. But this did not happen. Instead, an animal was simply locked in a separate room and observed, without having been injected with anything at all. These experiments, then, cannot be interpreted as proof that there is a hepatitis virus with infectious potential.⁷

The hepatitis C virus was then created in 1987, by a team of scientists, including Michael Houghton, of the Californian biotechnological company Chiron, and Daniel Bradley of the CDC, whose task was to find a virus that makes hepatitis C.^{8 9} This found virus was then supposed to serve as the basis (antigen) for an antibody test calibrated for hepatitis C virus. Since they couldn't find a complete virus, they decided to forage around for the tiniest traces of a virus, for fragments of genes (nucleic acid particles) presumed to represent a virus. With the help of a special laboratory process, the polymerase chain reaction (PCR), a tiny piece of a gene was taken from a particle that didn't appear to belong to the host's genetic code. From this, the virus hunters concluded that they were dealing with foreign genetic material from a not-yet-discovered virus.

But for the reasons repeatedly mentioned in this book, we must seriously doubt that a hepatitis C virus had actually been found.¹⁰ PCR is much too sensitive. It detects gene-fragments (DNA or RNA particles) which in themselves do not constitute a virus—but which are claimed to be parts of a virus that has not been identified. In any case, certainly nobody has yet managed to detect a corresponding virus structure in the blood serum of so-called hepatitis C patients. As with HIV, the virus purification necessary for a clear identification has not taken place. And there is no paper showing that a so-called high viral load correlates with viruses visible through an electron microscope (viral load is the laboratory parameter measured with PCR—the surrogate marker—upon which basis doctors decide whether to prescribe medications or not).

This even led Michael Houghton, said to be a co-discoverer of the HC virus, to put forward the key question before a large audience at a major hepatitis C congress in Paris in 2001: “Where is the hepatitis C virus? Has anybody seen it?”¹¹

Apart from this, the genetic snippets built up into the hepatitis C virus existed in the apes' liver tissue in such small quantities that they should not have been

considered a cause of a liver disease. But Chiron saw an entirely different picture: there was the evil hepatitis C virus (HCV). And so, on the basis of these gene parts, they began to build their HCV antibody test. The Procleix test alone, with which blood bottles are said to be tested for the presence of HCV antibodies, now brings in more than \$60 million per quarter for Chiron.¹²

Even blatant contradictions are gladly overlooked in this context. This piece of a gene said to come from a HCV can only be found in about half of so-called hepatitis patients.¹³ And a 1997 study printed in the *European Journal of Clinical Chemistry* (today *Clinical Chemistry and Laboratory Medicine*) shows that the gene particles officially classified as the hepatitis C virus had also been found in those who had negative HCV antibody tests. Generally, researchers contend that there is still no convincing evidence that the gene-snippets are indeed a pathogenic hepatitis C virus.^{14 15}

The virus theory does not fulfill any of Koch's three postulates, which must be fulfilled for virus identification. The first postulate requires that a truly pathogenic virus can be found in large quantities in every patient (this is not even close to the case). The second postulate is that the virus can be isolated and made to grow (but a hepatitis C virus has never been found in an intact form). And the third postulate says that this isolated pathogen must be able to trigger the same disease in animal models like chimpanzees. In this case, though, no isolated virus was transmitted, but rather blood; and there was no proper control group either (in which animals would be given blood—but without what was suspected to be the pathogen).¹⁶

Nonetheless, the virus hunters assert that the hepatitis C virus is passed on from junkies through contaminated injections (the CDC even blamed this for most HCV infections in the USA).¹⁷ But a 1999 study published in the *American Journal of Epidemiology* gives us another picture. The paper's goal was namely to find out if needle exchange programs, through which drug addicts are provided with clean needles, help to prevent HCV transmission.

The experiment couldn't confirm this theory. Junkies who used these needle exchange programs tested positive more often than "injecting drug users" (IDU's) who had no access to the programs. The researchers concluded that these programs do not help to prevent a so-called HCV infection.^{18 19} In other words, even when junkies constantly use clean needles so-called HCV antibody tests nonetheless (or with this specific study, especially) still come out positive.

Nevertheless, the hepatitis C antibody tests have been widely used (the blood test was developed in 1994). So, the world now also had a hepatitis C epidemic to contend with. Patients who test positive are stamped as "HCV positive" and it's hammered into their heads that they are carriers of a liver-destroying virus, which allegedly, after a dormant phase of around 30 years, triggers liver cirrhosis (the end-

stage of liver damage). The patients are consequently bombarded over a long period with medications, which ultimately damages the very organ in which chemicals are metabolized: the liver.

Most HCV positive patients have no disease symptoms at all (not even in the liver!),²⁰ and yet they are treated with toxic medications that destroy liver cells and the livers of already sick patients are additionally damaged with medications. The tragic end result of such a treatment was made clear by a study, conducted by Jay Hoofnagle and published in the *NEJM* in 1995. The active substance fialuridine (brand name Fiau) was tried out on hepatitis B patients. Five patients died and two could only be saved by liver transplants.²¹ It is well worth noting that none of the patients had any physical (clinical) complaints before the medicine treatment.

Those who still consider that medications are active in some way should know that in hepatitis C research there are no placebo-controlled randomized double-blind studies with clinical endpoints. This means that, as with AIDS or cancer research, no hepatitis C clinical trials look at two groups of subjects randomly assigned to receive either the active substance or an inactive preparation (placebo). Neither doctor nor test subject (double blind) should know who's taking the active substance and who the placebo. The trials should run for long periods (for hepatitis C around 30 years) and be oriented on clinical endpoints (e.g., survival time). Only then can it be shown whether patients treated with the medications actually do live longer. But without such placebo studies, statements on the effectiveness or a medication's life-prolonging effects are impossible.

Hepatitis C Can Also Be Explained Without a Virus

Just as with HIV/AIDS, there are numerous peculiarities in the theory that a virus triggers hepatitis C. There are patients whose elevated liver values can be observed using traditional blood tests, but they test negative on the antibody test. This prompts some virus-fixated researchers to speculate wildly that these could be "occult" hepatitis C viruses²²—instead of suspecting that perhaps there's no evil virus at work here whatsoever.

There are further inconsistencies. As studies show, it's not uncommon for HCV positive individuals to later, incomprehensibly, test negative, as if by magic, without having gone through any treatment.²³

Most HCV positive patients don't even suffer from any disease symptoms. And, as is the rule, they only have real liver damage if they have consumed alcohol and drugs. Here, there is a very conspicuous overlap: almost 80% of drug addicts are HCV positive.²⁴ To this Rainer Laufs, director of the Institute of Microbiology at the

University of Hamburg and one of the leading advocates of the view that hepatitis C is caused by a virus, says: “It is worth noting that intravenous drug abuse plays such a large role in the spread of HCV infection.”²⁵

Mainstream medicine should ask whether the monocausal virus model for hepatitis C really makes sense. Especially considering that if hepatitis C is indeed a contagious viral disease, the number of cases would show a bell shape: at the beginning a rise in the number of hepatitis infections and—once people have built up immunity against the allegedly evil agent—a following decline. But this is not the case. Rather, the number of those officially declared HCV patients in Germany, for example, has remained at 400,000 to 500,000 for a long time.²⁶

Another worthy investigation would be to look at whether toxins like alcohol, heroin or medications are, at the very least, co-factors for what is called hepatitis C, if not the fundamental cause. It’s fully justifiable to assume that substances like alcohol damage liver cells, cause the production of the genetic snippets on a cellular level, and are then picked up by PCR tests and falsely interpreted as HCV particles by orthodox researchers.

Last but certainly not least, no virus is necessary whatsoever to explain the 30 years that it takes on average until the affected patient’s liver gives up the ghost (liver cirrhosis). Sooner or later, toxic chemical substances like alcohol, heroin or cocaine take care of this on their own (without viral help), by gradually unleashing their destructive effects.

Unfortunately, these simple truths are words in the wind, ignored by the virus hunters. Since the 1980s, hepatitis doctors have been so fixated on antiviral medications that the headlines in the newspapers sound like advertisements for pharmaceutical companies: “Hepatitis C—the underestimated danger”; “Hepatitis C—the unrecognized danger”; “Hepatitis C—the new major epidemic. It’s coming silently but violently.”

A few years ago, in a Northern German city called Itzehoe, the media luridly reported that a HCV positive surgeon had infected many of his patients with HCV. HCV screening took place with antibody tests and a few patients reacted HCV positive. So, the conclusion was drawn that they had been infected by the surgeon, even though there was no evidence that a viral infection had even really taken place—not least because many people are living with what is called the hepatitis C virus; the tests must come out positive in approximately 2% of cases. 2,000 tests could garner 40 positives. So, a doctor could spark a hepatitis C epidemic simply by carrying out the so-called HCV antibody tests on all his patients.

From time to time, media headlines have been a bit more critical, like: “Hepatitis C danger overestimated?” But these articles are the exception to the rule, which is puzzling since anyone who weighs up the various risks of an antiviral hepatitis C

therapy would come to the conclusion that no medications should be prescribed. Mainstream medical research has shown that there is “no lasting success” to be attained with the medications.²⁷ Nevertheless, the virus hunters are tireless and continue to claim that antiviral hepatitis medication produces significant improvements by referring to various studies, such as the one by Hadziyannis et al.²⁸ ²⁹ But all these studies are irrelevant because they prove that the medications do not heal and, even worse, that they cause harm.³⁰

A few years ago, a large American study was published in the *Annals of Internal Medicine*.³¹ The blood serums of the subjects had been frozen between 1948 and 1954, and were now being tested for hepatitis C. The researchers found that there was practically no difference in liver disease between HCV positive and HCV negative patients. Simultaneously, among HCV positive subjects, little liver damage was found and few mortalities could be traced back to liver disease.

The researchers concluded that mainstream research had highly overestimated the risk that a healthy individual who is tested positive for HCV later comes down with liver cirrhosis. At the same time, it is plausible to assume that substances like alcohol and drugs (including several hundred medications known to have damaging effects on the liver)³² could be the main causes. There is no reason, then, to treat HCV positive patients with antiviral active substances.

“My experience as a physician is that a positive hepatitis C test could indicate liver damage, rather than a viral infection,” says Seattle-based naturopath John Ruhland. “The patients I have seen with hepatitis C had liver damage that had primary causes such as alcohol and drug abuse. To truly understand what is causing this hepatitis C ‘epidemic,’ follow the money trail. Millions of dollars are being made by selling drugs and treating people for an often non-existing problem.”³³

Ruhland adds that the human body has a tremendous capacity to heal itself. This principle, known as the healing powers of nature, is the foundation of naturopathic philosophy. Ruhland’s goal as a naturopathic physician is to help restore balance to the body, the mind, and the spirit. An intermediate-range goal may be to focus on preventing specific future illnesses. The long-term goal is to work with the patient to improve his or her health, not just by eliminating illness, but also by promoting wellness.³⁴

Pamela Anderson: The Virus Industry’s Grand Marshall

Unfortunately, an objective examination of the hepatitis C subjects is thwarted time and time again by publications in specialist journals and the mass media, which dwell upon the disease’s alleged infectious and epidemic potential. The best-known

hepatitis C case is probably that of American actress and “Baywatch” nymph Pamela Anderson. Anderson announced in 2003 that she had been diagnosed with hepatitis C, which elicited global consternation. Her doctors had told her she had a maximum of ten years to live.³⁵ Anderson disclosed that she believed she had been infected by her ex-husband, drummer Tommy Lee, when they were tattooing each other.³⁶

Proof of this does not exist. But, the global media had a sensational story to boost circulation and audience ratings—and virus hunters had a global platform to claim that HCV is caused by a life-threatening virus. All of a sudden, after leading a quiet existence for so long, hepatitis C was known all over the world. Just a short time later, Anderson even became “Grand Marshall” of the American Liver Foundation, which promotes antiviral therapy.³⁷ The blonde bombshell made for an effective in-your-face advertisement of medication that had never been proven and certainly its potential damage had never been ruled out.

Chapter 5

BSE: The Epidemic That Never Was

“The assumption that BSE is an epidemic caused by an infectious agent called a prion in meat and bone meal has not been proven. To prove this, at least one controlled feed experiment with cattle herds would be necessary. But this has not been done. A feasible alternative hypothesis is that the BSE epidemic in England was caused by a combination of factors: a genetic defect in the gene-pool of a few cattle herds, which was bred into frequency in pursuit of the best-possible efficiency in milk production, poisoning from insecticides and heavy metals, copper deficiency and/or autoimmune reactions.”

Roland Scholz, Professor of Biochemistry and Cellular Biology
Sievert Lorenzen, Professor of Zoology
(Author of the book *Phantom BSE Danger*, 2005)

BSE: Prophecies of Horror and Wastes of Money

The hysteria caused by the alleged bovine epidemic BSE (Bovine Spongiform Encephalopathy which is a spongelike brain disease) reached its peak in 2001 and caused people to fear that they could contract the so-called deadly new variant Creutzfeldt-Jakob disease (nvCJD or vCJD) by simply tucking into a juicy steak. Scientists and politicians alike initiated the strangest safety procedures, like killing masses of cattle.

“An apocalyptical spirit ruled the country,” cried the German *Frankfurter Allgemeine Sonntagszeitung* in 2002. “Hundreds of thousands of BSE cattle will be discovered in the coming years, predicted serious scientists and self-proclaimed experts. There was talk of thousands, even tens of thousands of expected deaths—human, not bovine—caused by a new form of Creutzfeldt-Jakob disease [induced, according to theories, by ingestion of BSE-infected beef]. Reports of the allegedly impending new plague of humanity were everywhere. Two ministers had to resign.”²

The horror scenarios have not proved true. Not a single German has died from this variant of Creutzfeldt-Jakob disease (nvCJD or just vCJD), although at the end